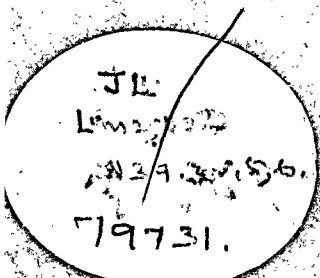
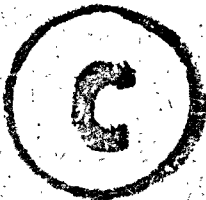


THE ANTISEPTIC

1939

MAY — 36-5

JUNE — 36-6



The Antiseptic

A Monthly Medical Journal

Edited by

Dr. U. Rama Rau, M.L.C. & U. Krishna Rau, M.B., B.S.



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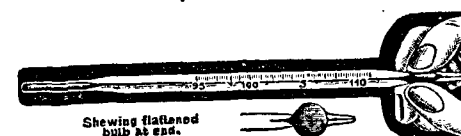
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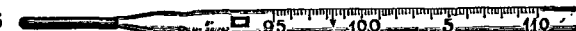
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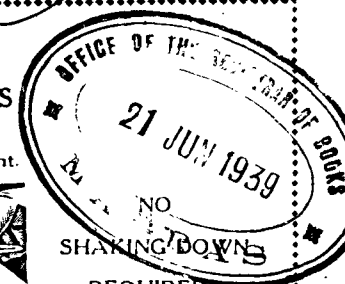
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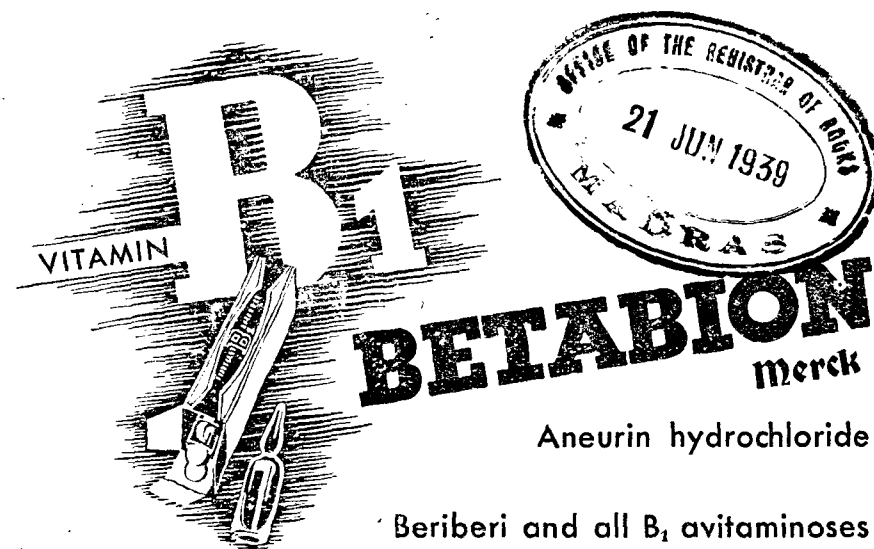
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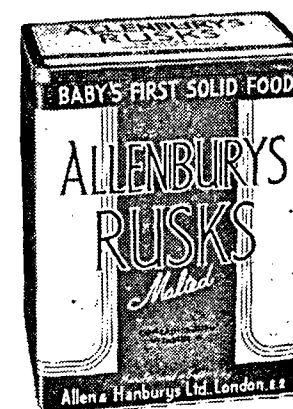
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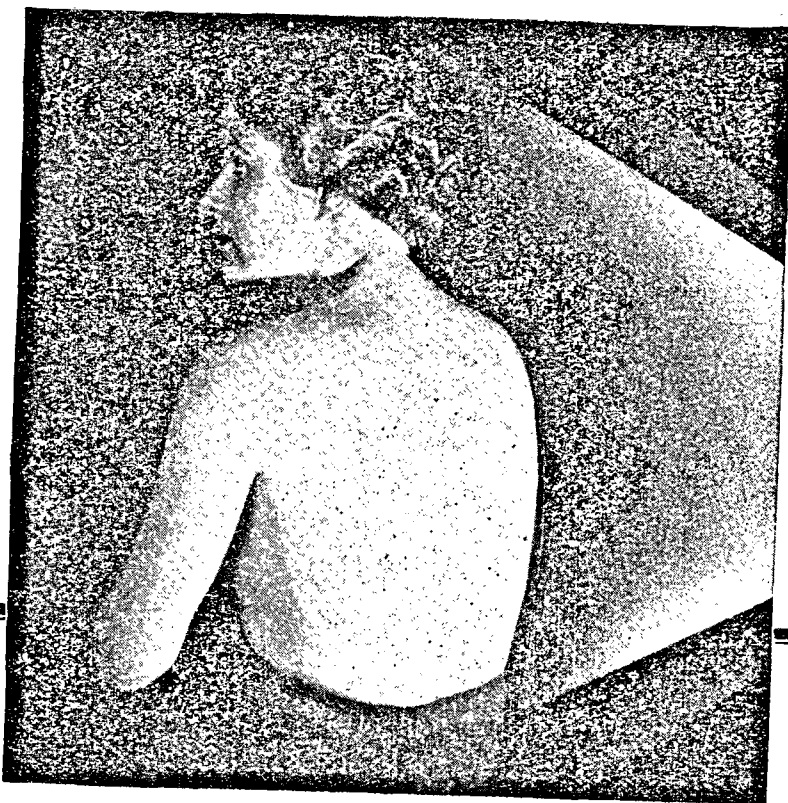
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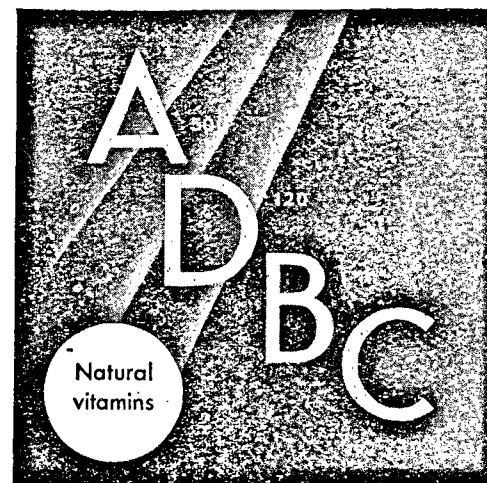
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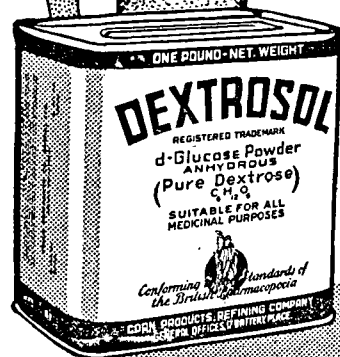
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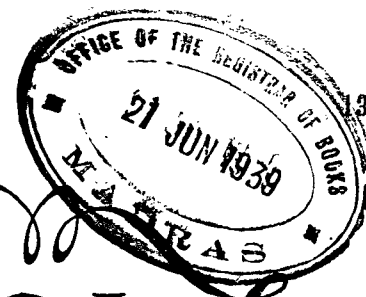
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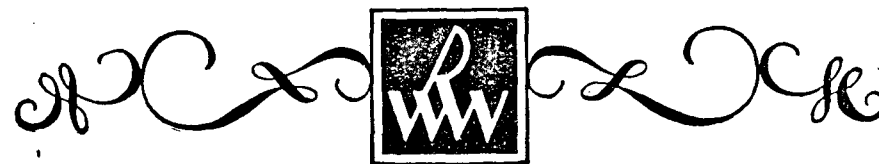
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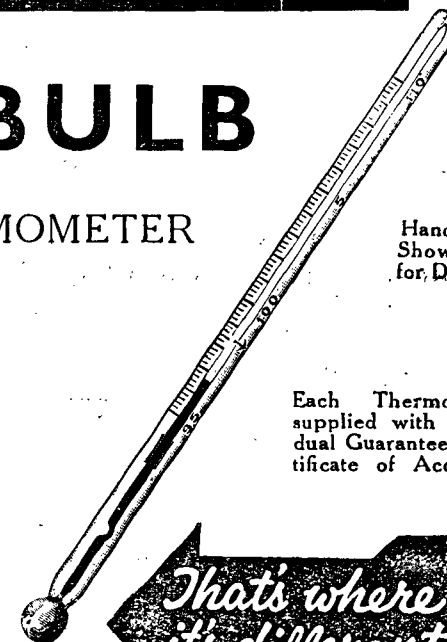
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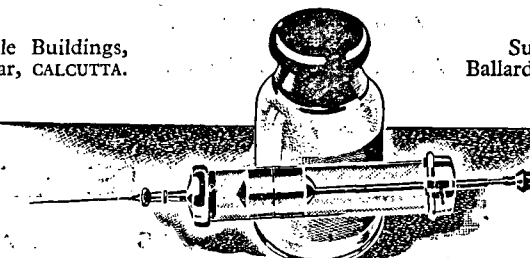
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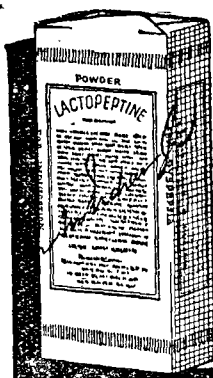


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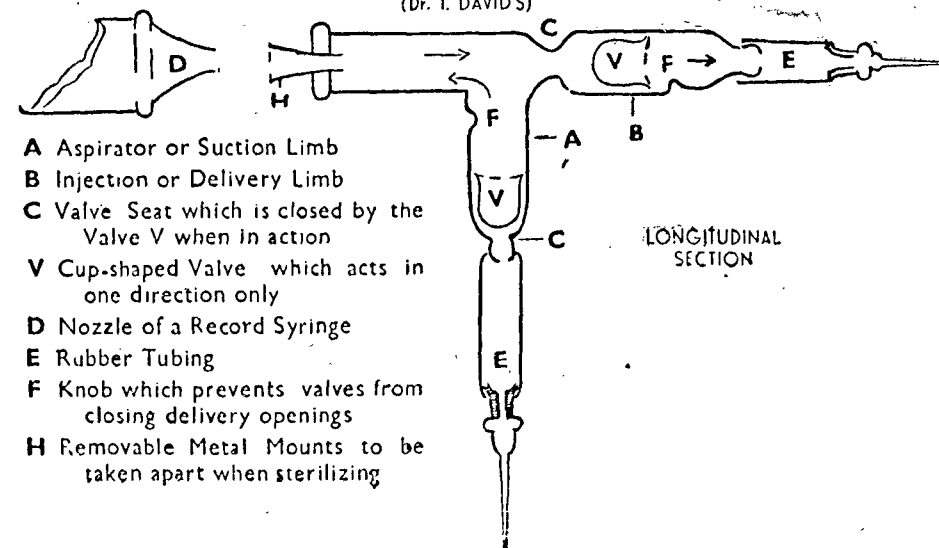
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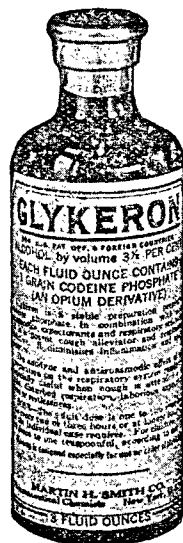
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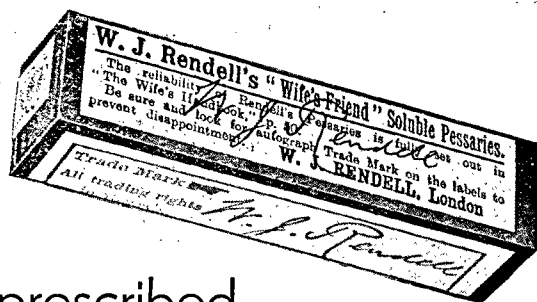
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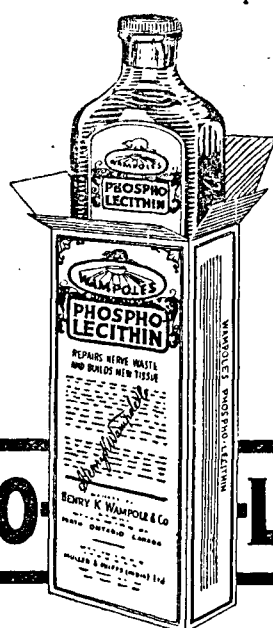
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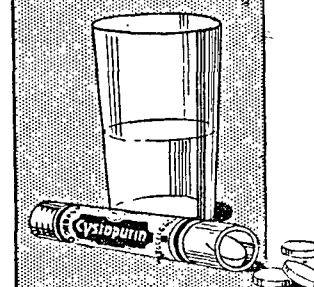
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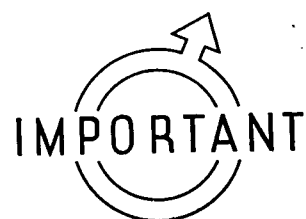
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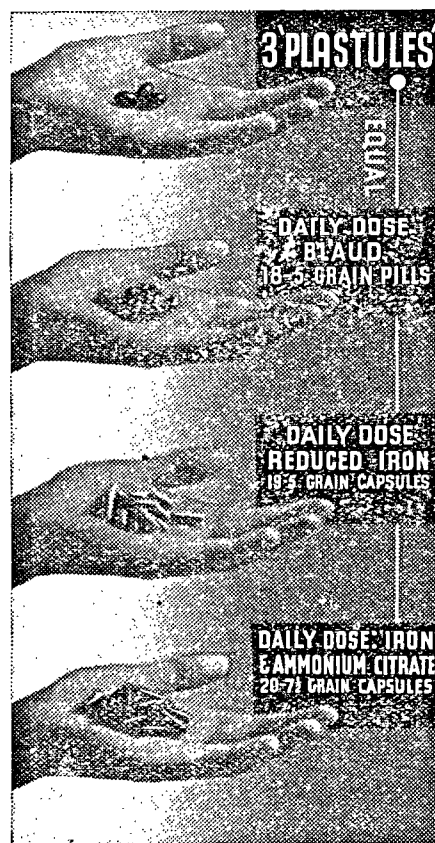


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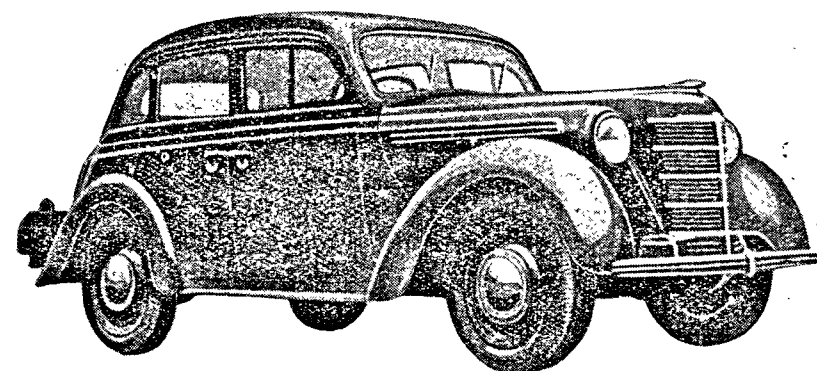
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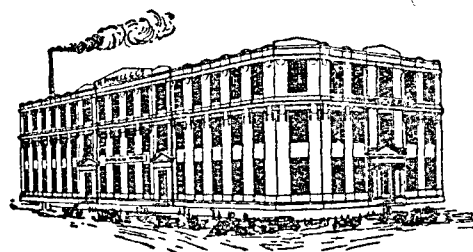
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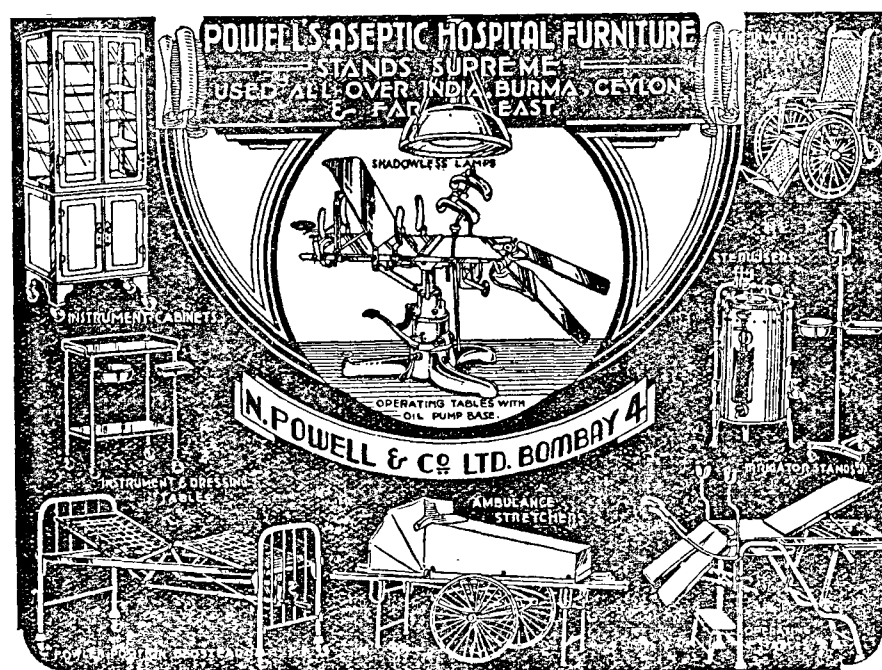
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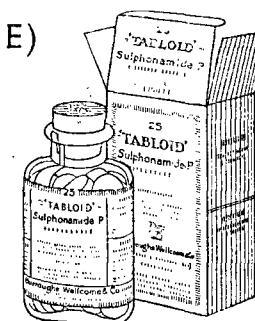


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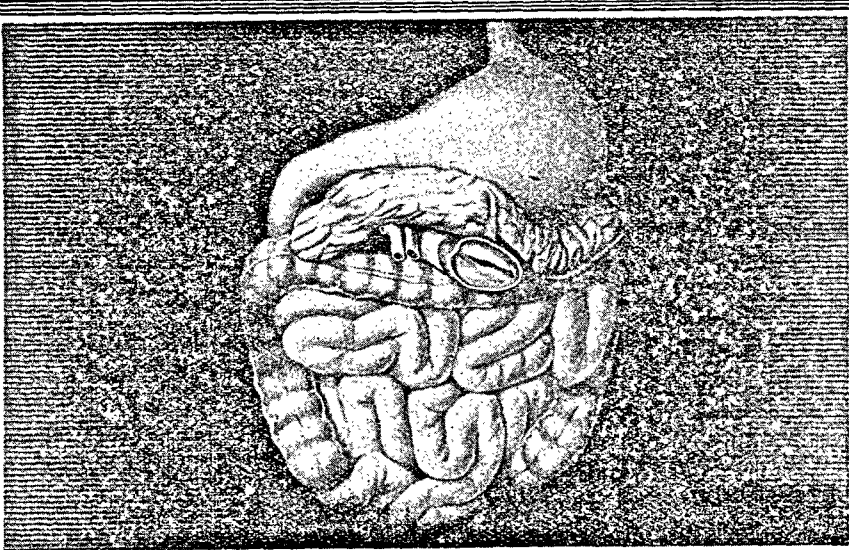
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MAY, 1939.

No. 5.

ORIGINAL ARTICLES

Glimpses of Medicine in the Early Days of Fort St. George*

BY

D. V. S. Reddy, M.B., B.S.,

Medical College, Vizagapatam.

TRAVEL back 300 years in imagination and conjure up a vision of the Coromandal Coast, at the spot now occupied by Madras. At a distance is visible a small steep hill, covered by the ancient church of St. Thomas. Adjoining the shore, is the fortified town of wealth and importance and an early settlement of the Portuguese, San Thome in Mylapore, three miles north of which rose a small hamlet, even then known as, Madraspatam. All the rest of the Coast, was a strip of sand or low sand hills, except for a small lagoon, formed by the mouth of the river, now known as Cooum.

The earliest settlement of the East India Company on the Coromandal Coast was started in 1611 at Masulipatam, then the chief port of the Kingdom of Golconda. The English built there a factory house (ware houses, offices and residential accommodation). Later, due to the exactions of Golconda, the English sought a footing in the southern districts and obtained a grant

* Specially contributed to 'The Antiseptic'.

of a piece of ground at Armegon (in Nellore District) where they built a Fort and factory in 1626. Even here, the English had their difficulties with the local Naik and in July 1639, when Francis Day was chief at Armegon, he was authorised to sail down the Coast and negotiate with the Rajah of Chandragiri for a new settlement. This search led to the selection by Mr. Day of the site, adjoining Madraspatam. He later obtained from the above Naik, Damerla Venkatadri, "Lord General of the Carnatic", a grant of territory and privileges and licence to build a Fort and form a settlement. On 3rd September 1639, Day handed over to the Agent at Masulipatam, a report of the transaction, along with the grant by the Naik, a firman engraved on a gold leaf. There was some delay in getting the approval of the Presidency at Surat for this new venture.

Finally, in February 1640, Cogan and Day, dismantled the fort at Armegon and embarked in the ship 'Eagle' accompanied by another ship 'Unity'. They reached Madraspatam on the 20th February. The party accompanying Cogan and Day comprised Factors, Writers, a Gunner, a Surgeon and 25 soldiers. It is not clear whether the Surgeon mentioned was the one stationed at Armegon or the ship's surgeon. There are entries in records suggesting the presence of a surgeon at Armegon, at least occasionally, as early as 1630. The report or relation of Joe Carter, Captain of the ship 'Eagle' (which remained at Madras road for sometime and was hit by a storm in the 2nd week of March 1640) mentions that his chief men *including the Surgeon* were ashore at Madraspatam at the time of the gale. This would suggest that *the surgeon* that first set foot on the soil of Madras, in 1640, was probably a ship surgeon. It will be highly interesting to know the name of this surgeon and some details about him. The same report mentions a few medical details; that some of the faithful crew were sick and others feigned sickness, during the storm, that a man fell overboard with one of the top masts but miraculously escaped without an injury.

Slowly, people were persuaded to settle down around the Fort, and even some Portuguese from San Thome, migrated to the new settlement. By 1640, besides a number of Portuguese from Armegon, there were three or four hundred families of weavers in the adjoining area. About the year 1641, the Garrison of Fort St. George consisted of 35 Englishmen and as many natives.

It was about this time that the factors on the Coast, wrote

to Surat stating the receipt of the first chest of surgery for Madras and also asked for a surgeon for the coast. "Out of the ship 'Hopewell' we took three of the four chests of surgery sent forth for supply to those that wanted; *one of which was put ashore in Madraspatam, where till then, was not so much as a playster, or wherewithal to make any*; another we took ashore here (Masulipatam) and a third was put aboard the ship, 'Expedition.' But if you shall be pleased hereafter to send us a chirurgeon, he must bring somewhat with him, for the chest, or rather box, was pillag'd of all what was good er (e) sent ashour....."

In 1643, the Agent of the Coast was in a position to give the following report of the progress of the English settlement at Madras.

'And heere wee suppose its not amiss to lett your Worships uderstand that Mr. Francis Day was the first projecture and Contriver of that Forte or Castle in Madraspatam which another, with a greite deals of discontent, laboure and paines hath now brought to some good pass, being a place of securitie on that Coast as the onelie place of secured saiftie with that Title of hours (Castle) that even our nation enjoyed in East India, and therefore in our opinions to bee highlie esteemed. And for its cost, it's certaine that if your Worships continew the Indian Trade, in few yeares it will not onelie quitt its owne Charge, but allsoe produce benefitt and put monies into your Purses by bringeing a Trade thither, raiseing a Custome there, paying of duties by the Inhabitants neere adjoyning, and being replenisht with Merchants (and) weavers, whereby you may have all things necessarie and convenient for you under your owne Command; and happy and glad will manie bee, (wherein you will find the benefitt), to come and live under our nation and be protected by them.'

It is a bit uncertain whether, in 1643, the establishment at Fort St. George, did have on the staff a Surgeon. But a letter dated 20th January 1644, from Fort St. George to Bantam, describes a most unfortunate incident that happened on the 8th of that month and refers to 'our surgeon'. An English sergeant, named Broadford killed one of the natives, not intentionally but by accident. The man was at a most unseasonable time hiding around about the sergeant's house.

'Hee by accident finding of him hid so, and in the darke seizing on him, and the other struggling for

an escape; which the Sargant doubting, drew out his knife, not haveing his Sword about him, and cutt him over the Arme, so far to give him a Marke for to Discover and knowe him the next day. And indeed itt proved soe that *the man was easily to bee found, for what with timerousness and neglect to have his blood stinched, he proved a Corpees.* Wee, having noticed of the Disaster the next morning, and being dubious that our Maters might suffer somewhat, *first sent our Chirurgeon to see what wounds; which was fruitless, for his Parents had intered him early in the morning, being ashamed of the act, for such were his words.....'*

The establishment, in October 1645 consisted of "37 of soldiers and other professions". There is no definite and separate entry regarding a surgeon. But there was much sickness, at about the same time as another letter to the Company adds "What can you expect of 50 well and sick men to defend your Estate and fort against King's power".

In October 1645, was also issued the first medical certificate by the Surgeon at Madras.

"Wee have by yure appointment Bridgman purser of the Advice, and retourned him to Bantam with William Browne, one of master Douwlls accusors; and should likewise have sent Jeremy roote, *had hee not been at deaths dore at the departure of the shipp; which if wee had sent him Aboard, wee must have hazzarded his life, as per attestation here enclosed under the Chirurgions hand.* And if it please god to restore him to health, he shall take his passage for Bantam upon the next shipp....."

An entry dated January 1647 alludes to famine and pestilence, that ravaged the vicinity of Fort St. George.

"The Famine is so great in this Kingdome that wee believe it will bee the Destruction thereof, for there hath not fallen any rayne this yeare for the increase of any graine to relieve the people; and now the season of the Raynes are past, so that, if the Allmighty does not send supply from other parts, the Country will be so Dispopulated that it will be impossible to recover it selve againe in five years time. Likewise we could intreate you to supply us with tenn Englishmen to serve here as soldyers, for Mortality and Moores Campp hath taken all

away to 25 persons, *whereof 4 or 5 are continually sick with the miserie of the time; for we have not, nor is here anything to bee bought to relieve any sick person, unless hee will Eate Carryon beife, which wee procure out of the Moores Campp, which we obtaine by much favour.'*

It is in the consultation of the latter half of 1650 that one come across the name of a Surgeon for the first time, since the foundation of Fort. Edward Whiting, who had been surgeon at Jambee (Bantam) from 1645, arrived at Fort St. George. "It shall bee our care providently to dispose of *both medicines and instruments in the Chirurgery Chest*, rendering our humble thanks both for it and the Surgeon Edward Whiteinge, at whose arrival Nathaniell Lumly lay very sick, and the 7th October last departed this life without makeing any will in this place....."

The year 1654 was a sickly time. "We have had a very *unhealthfull and sickly tyme amongst us ever since Christmas last; hardly a man that hath not beene visited more or lesse, some of us 4 or 5 tymes over.* Wee have buried only 8 of our souldiers, and their Captain James Martin, who after a long sickness left this world the 22th June past." Hence the following request. "When it shall please God to putt your Worships in a way of sending us out a ship for the Coast againe (which we hope will be speedily), wee desire you will be pleased to remember us with a fresh supply of *Chirurgery means, whereof we are in extream want, having neither Purge, Cordiall, or any the least thing elce left us in the Chest we now have by us, what need soever may happen to us.....'*

By this time, the surgeon found a honourable place and mention in the list of men resident at Fort St. George, as there is an entry dated 1654, "Edward Whiteing, Chirurgeon". An interesting passage about this Chirurgeon, refers to a duel he was challenged to fight. Henry Grenhill complained that James Martines, Captain of soldiers, "sent a private challenge to our Chirurgeon Whiteing, even at the time when sickness was most rife amongst us." Whiteing is also mentioned as being an occasional companion of Leigh, during the later's quarrels with President Baker, in May 1654.

"This Leigh hath been known to be drank near upon a month together, and that drawne others of the Companys servants to accompany him in his drunckness, in soe much and it is verily believed that Mr. William Gurney and Mr. James Moore were thereby brought into *such diseases as helped them forward to their last ends...*"

There is also a medicolegal report in that year. Thomas Bland charged his wife "of making away a child which we find by her speeches, was gone with 5 months."

In 1655, the Company was eager to contract their operations. The bigger establishments were closed and the Fort was left in charge of Thompson, the Chaplain. A few months later, after a careful consultation, it was decided to station some more staff in Madras.

Whiting left India in 1657. Henry Greenhill wrote to England in January 1657, bitterly complaining of lack of medicines; "Tis now more than 4 yeares since we received any Chirurgery meanes, nor doe we expect any yet awhile, for these ships will spare us none whatever should happen; and being neglected by your Worshippes from whome we might justly expect reliefe and Comfort, and your people discouraged...; but hope that time will one way or other relieve us of this dropping condition. In the meantime, please to take notice that, upon the request of Edward Whyting, our Chirurgeon, we have granted him lycense to repaire to his Native countrey: for to what end should wee keepe him here upon charge, having not wherewith to imploy him".

The list of men in Fort St. George at the beginning of 1658, contains the name of a surgeon. The entry reads: 'Robert Cooper-Chirurgeon'. It is believed that Cooper was one of the factors recalled from Pegu.

The first surgical procedure recorded in the Annals of Fort St. George, "tapping", was performed in the first week of 1659, on the Agent Greenhill. "2 days past, per hands of the Dutch we had the Sad news of the Death of our Agent Greenhill. which (per the Circumstances) we feare to be too true. They say hee, being very much Sweld, was perswaded per the Surgeons to be tapt, to let out the Beaveridge, after which he lived but 2 dayes, Its an ill wind that blowes noe body good; per this our friend Thomas a Ginger will bee Exalted into a better Capacity of serving his friends..."

There were also others who were sick about the same time. "But, my fine boy is dead; which has been very neare the occasion of my death, for I lay sick hopelese above a moneth and am not yet recovered. And to add to my grieffe, my honorable friend the President is very sick and cannot live ten days, and in his stead is one Mr. Chambres, who claymes kindred with those (of) our country. He hath shewed me divers curtesies in my sickness, and bids me not question but that he will be as

loving to me as his Predecessor..." Thus wrote Roger Myddleton in January 1659.

Beriberi appears in the Fort St. George Records in 1660. "In his stead, we placed Mr. William Hull, who hath since ended his dayes of a languishing sickness commonly called the Barbeers."

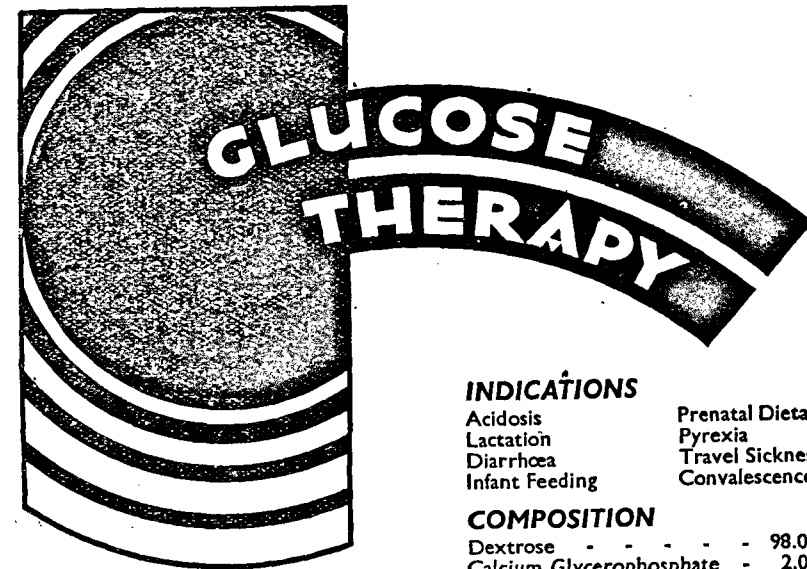
About the same time, appear the interesting passages, attested by Rev. William Issacson. The first complains of the Roman Catholic cemetery. "There is another inconveniencie which to me seemes very preposterous, viz, That the Hon'ble Company shold want a Church for the service of God in their owne Towne, when the Roman Catholiques have one that stands in the heart of the Towne, where they have likewise a churchyard to bury their dead; whilst wee are forc't to carry our dead Corpses out of the Towne. Besides, there are so many of their Pittifull Christians dye of fowle diseases that in time of heates it is enough to Breed Infection. I am certain the smel is very noysome to them that live neere the burying Place. I have spoken sufficiently of the two French Padres Insolencies in another writing; therefore shall not farther enlarge upon that Subject."

The second refers to the activity of Roman Catholic priests in Madraspatam. "In the second place, if any Christians belonging to our Congregation are visited with any sicknesse, they will be soe bold as to intrude into our Ministers Office of Visiting them, and, like the Devill, endeavour to lay their strongest Batteries whilst they are weakest, to seduce them if they can to their Idolatrous custome of setting the Images of Saints before them, that they may pray to them. And this they doe in the night, for our sick men are forced to lye out of the fort for want of Accommodation."

Glimpses of Contemporary Indigenous Medicine.— Belief in witchcraft is clearly indicated by the following passage from the charges made by Greenhill in January 1655. "I shall in the first place rank their Witchcraft, Sorcery, Spells, Medicines in Meate and drinke, Etc., which they ditto Braminees are reported to use; and amongst others to the purpose, had procured a woman Exorcist or Divinatrix, one of Balaams Cast, to curse their Enemies, whoe remain'd long in their howse, untill by the perswasion of Verona, their Cozen, shee was lately sent out of Towne. That such practises are usually in India, too many of the Companys Servants have found true by sad experience, nor have your selfe, Sir, bin exempted forme (r)ly from the like in Bantam. And whoe knowes what mischeife such villaines now

may heere work upon you for their owne ends, haveing all opportunities to Minister, by themselves or Instruments, what they please, by whose Councells you are swayed to our Nations and Masters exceeding dishonour and prejudice..." How the brahmin brethern learned sorcery is mentioned in another charge-sheet. "In this Towne, before the Pagod Permall, the Bramines Caused a little Pagod to be built, and procured a Sorcerer or Wich, whome they kept heere 2 Months and learnd his Art: by whose Charmes and Spells, done upon a Brass plate which they buried under the Stone Idoll in the little Pagod, Like accons are said to bee done to Obtaine the Affections of Governours, abase and destroy or hinder the proceedings of adversaries; and...Wee doe affirme that they determined the Destruction of the Towne; and such actions in these parts are punished with Death..." What they did is thus described: "The Braminee Vincaty procured that Surva Raz, one of their kindred, was made Adigar whoe in the tearm of 2 or 3 Months falling out with him...threatened that hee would accuse Vincaty of Some Accompts or misdemeanors; but tis reported they poysoned him or Murdered him with witchcraft, for soone after, hee dyed suddainly..." The brahmin brothers in their counter-charges against Greenhill mention a barber surgeon of Madraspatam. The article No. 33 reads "In Rudrigas howse two poore beggers about difference in their sects fell out, and beat one the other. The weakest, or one of the two went in and complained to Rudriga; whoe came with a Rattan, and without questioning the matter, strooke the askeman, whoe thereupon cutt his owne throat. For which was no Justice done; and Vincaty gott Dick the Barber to cure him againe."

The First Hospital.— Such was the state of medicine at Fort St. George and its environs, when the Council of the Settlement felt the necessity and established a hospital, in a rented house in the last quarter of 1664, for the sick soldiers of the Garrison. "The Souldiers in the Fort, since your whorships absence, hath bin something strictly held to their duty, and according your order they had noe free guard; soe that the fresh souldiers which came forth this year, takeing up their habitation in the bleake winde in the hall, fell sick. Four of them are dead; aboute tenn remain at this time very sick, and complaine (and it seemes not without reason) that the wages are not sufficient to supply them now in this time of their sickness. Soe, rather then to see Englishmen dropp away like doggs in that Manner for want of Christian Charity towards them, we haue thought it

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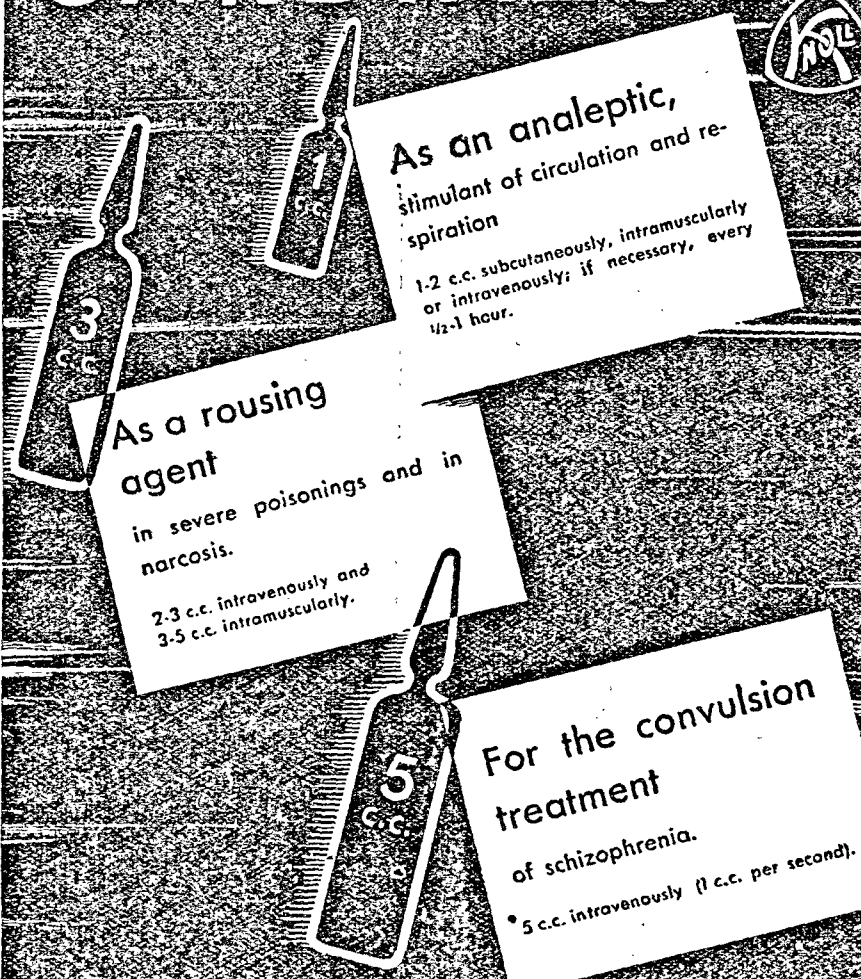
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very Convenient that they might have an house on purpose for them, and people appointed to looke after them and to see that nothing comes into them, neither of meate nor drinke, but what the Doctor alloweth. And have for that purpose rented Mr. Gogans house at two pagotheas per moneth; which we hope you will so well approve of as to continue it for the future." An entry dated January 1665 gives the average number in the hospital. "Perhaps Mr. Buckeridge may give the Company a Large Accompt of my building here; but my last years Letters told them the truth, and necessity of all. For what was done in the Fort is for a place to serve God in (which before was only a shedd and would not keepe us drye), and Lodgings for their Servants, and Godownes for their Goods, and are all soe necessary that it cannot but be allowed and liked off; and yet we have soe little houseing that we are forced to hire rooms for our sicke men eight or ten being downe together."

How this small Hospital, grew in size and importance, with the rising fortunes of the English Settlement at Fort St. George, and formed the nucleus of the now famous Madras General Hospital, is an Epic Story, as fascinating as it is instructive.

Radiology in the Diagnosis and Treatment of Cancer*

BY

Dr. M. J. S. Pillai, F.F.R., (LOND.),
D.R., (UNIVERSITY OF EDIN.)

*Medical Superintendent, Bernard Institute of Radiology,
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OF all the scourges of humanity, Cancer ranks second to Tuberculosis. Its devastating effects are so far-reaching that Cancer research problems are being paid special attention to, all over the world. We, in Madras, have been fully alive to it and the Government have brought into existence special departments in the Institute of Radiology to tackle this great problem. There have also been somewhat similar departments at certain hospitals in the Districts. The personnel of the British Anti-Cancer Campagin have been doing excellent work in this direction and their statistics of the work done is published annually. The public also have been fully alive to this problem, so much so, the Madras Women's Association have been pressing Government for a separate cancer hospital in Madras. Owing to various causes, the institution of a separate Cancer hospital could not be taken

* Being The Madras University Extension Lecture delivered on 11th March 1939, and sent to 'The Antiseptic' for publication.

up now, but the several city hospitals have made arrangements to treat such cases.

The number of Cancer patients is large and the available amount of Radiation energy is comparatively small; so the waiting list of patients is a long one. But the work can be helped by intensive propaganda such as is pursued in the other parts of the British Empire. The chief point is to make people realise that the scourge of Cancer in its early stages is definitely curable. People have to be educated to appreciate the value of early detection of Cancer condition. Women at the menopausal stage suffering from sanious discharge or a rapidly growing lump in the breast, patients suffering from any crack or ulcer in the mouth persisting in spite of treatment, hoarse voice which does not readily yield to treatment with pain and loss of weight should at once warn those sufferers to consult the proper medical attendant to exclude malignancy. Issue of pamphlets describing these conditions, talks on the radio, lectures illustrated by lantern slides, an appeal to people going to hospitals explaining to them such conditions, special instructions to patients, these and similar propaganda should be carried out as an intensive campaign in controlling Cancer. Unfortunately most cancer patients coming to a hospital are in such advanced condition that no treatment will be of any avail and the radiation energy available is to some extent wasted on such incurable cases. In order to dispose of the cases most effectively the following scheme is suggested :—

Centralisation of Cancer Treatment by opening special Wards for treatment of malignant disease in the General Hospital is suggested. Cases admitted in different wards of the city hospitals can all be sent to the clinic which will meet once or twice a week as occasion demands when all those concerned, viz., the Surgeon, the Physician, the Radiologist, the Gynaecologist and the Pathologist can discuss and carry on as a team. Such cases get the benefit of the experience of the specialists in their respective branches. Cases with a good prognosis are taken in the first selection, cases with indifferent prognosis as the second selection and cases with grave prognosis are classified as the third. Cases requiring Radium or Radon applications in the first instance are separated from those that require a pre-operative or post-operative radiation therapy, or cases which require a combined Radium (Radon) and X-Ray therapy, as it has been the experience that even in the early cases with no involvement of glands very often a post-operative therapy is indicated, as one

cannot be too sure about the absence of dissemination. Cases of the first group are entered in their respective case sheets including therein the R.B.C., W.B.C. counts and Hb% these being noted before, during and after treatment with Radiation therapy. In addition the number of reticulocytes per field or per 1000 R.B.C. gives us very valuable information. These patients can be in their own respective wards coming under the observation of the Clinic as often as is necessary during and before finishing the treatment. The line of treatment adopted will be on the lines of the Research Medical Council and the publication of the statistics will also be on similar lines.

One of the Assistants is being deputed to look after the Research work in this direction under the guidance of the Cancer Clinic. Research will be on the same lines as those of the Research Council, England. After distribution of patients presenting a tolerably good prognosis for individual and combined Radiation Therapy, the last class of incurables, destitutes in particular should be kept in a separate institution where they will be looked after during the remaining period of their existence to make their life less miserable and die in comfort. The arrangements will be to alleviate the suffering by morphia injections and keeping the parts affected as hygienic as possible. By thus sorting out the cases, waiting cases can be benefitted by disregarding the claim for priority of admission obtaining at present. By unnecessary prolongation of the waiting period, cases of fairly good prognosis are converted into hopeless classes; this should be avoided at all costs. As the time factor is of great value in the treatment of rapidly progressing malignant lesions, this classification of the patients and early attendance to such cases which can be benefitted is advocated. It is common experience that many of these destitute patients who are afflicted with the fell disease of Cancer feel so miserable that they prefer death. For such class of people, the hospital for incurables, will serve a great purpose. The want of a Poor House for the destitutes is miserable enough, but to leave such miseries afflicted with the disease of Cancer roaming about in roads cannot obtain in any civilized country. Even if the prospect of getting a Poor House established in the Province is remote, the need for a hospital for incurables in order to save the energy and time, for the curables is an urgent necessity. As it stands at present, the strict adherence to priority of claim, acts very prejudicially to the interest of those curables. The time and energy which can be utilised for the curables is wasted on the incurable. The word 'curable' is being used in

the sense that by giving the best treatment available, the average survival period is greater in the 'curables' than in the incurables. If in some of the States of America, voluntary euthanasia is an accomplished fact, there can at least be an attempt to relieve the suffering destitutes in this country to make the few remaining days of their existence as hygienic and as comfortable as possible. I think this should be done as a duty which civilisation owes to the suffering destitutes.

The therapeutic agencies available and at our disposal are the following:—

Deep X-Ray Therapy with 200 and 400 K. V., Chaoul's Low Voltage near distance 3 and 5 C. m. Therapy, Radium, Radon, Ultra Short Wave etc.

It will be instructive here to mention a few facts about the recent advances in Modern X-Ray Therapy and at the end of the lecture, I will show you a few lantern slides depicting the process of radium manufacture and its application on patients and their relative factors.

The Public will have to co-operate in order that we and they may get the best results with the equipment which the Government have placed at our disposal.

A Summary on X-Ray Therapeutics as Practised at Present.—X-Ray therapy has advanced considerably in the last few years. From its inception about 42 years ago, it has passed through various stages of progressive evolution, so much so at present, treatment with the super Million Volt machine is an accomplished fact. The application of X-Rays first as a therapeutic agent to skin lesions, later on to glandular lesions and still later for both malignant and non-malignant lesions has proved the efficacy of Radiation therapy and it has now come to be used for treatment of many conditions.

The apparatus, a single coil set to start with, has now developed to include the step-up transformers of the cascade type, giving rise to a Million volts high power apparatus, with a possibility of increasing it to further high voltage; a short description of the Million-Volt apparatus in use at St. Bartholomew's Hospital, London, will shortly follow. In the experiments conducted for purposes of releasing and splitting the nucleonic protons from the atomic structures, an apparatus of 5 Million volts with a spark gap of several yards in length has come into existence. The feasibility of utilising this super voltage apparatus, for the production of X-Rays (whose wave-length will almost

correspond to the Gamma Rays of Radium) in the treatment of malignant tissues in particular, is in the experimental stage at present.

With increase in voltage, the milliamperage is correspondingly decreased; the intensity factor being proportionately altered. The apparatus now in use, are the 60 K. V., 120 K. V., 200 K. V., 400,000 Volts, 800,000 Volts, one million volts with a potential capacity for one Million and two hundred thousand volts. The 400 K. V., apparatus has been in use for the last four years and has come to stay. The other super-voltage apparatus are being worked on a research basis and the results are awaited.

The results are judged by the year factor expressing them as two year cure, five year cure and then ten year cure etc. The advantages of the super-voltage therapy are, the skin re-action is proportionately less compared with the intensity of the energy absorbed. The biological reaction in the intermediary cellular mass, before the rays reach the centre of the tumour has been lessened, to a greater extent than before and the energy absorbed by the 'tumour' itself, is proportionately large. The skin reaction is not of the uniform erythematous nature as in the 200 K. V. X-Ray therapy. Here the skin gives rise to slight reaction of a 'nodular' type which disappears much sooner and with very little resulting atrophic changes than what obtains with the 200 K. V. apparatus. The fat necrosis is altogether avoided. The X-Ray sickness is considerably reduced in the super-voltage therapy.

A rotating tube energized by 200 K. V. causing equal distribution of energy over a large area in the treatment of Hodgkin's disease has given a greater hope in this most intractable and very often fatal lesion. The Near Distance Low Voltage Therapy for malignant lesions of the skin and the mucous membranes has given very satisfactory results. The glandular and other superficially situated lesions have also given fairly good results with the skin deep X-Ray Therapy.

The dosage interpreted in Roentgen units depending chiefly on the superficial area of absorption and confirmed by measurements at the depth of tissues has resulted in a process which secures a uniform distribution of the radiation energy absorbed by the tissues. Research work on Cancer tissues altering their absorption co-efficient by means of particular preradiation treatment tends to decrease the amount of energy to be delivered to the tissue concerned. For all practical purposes, the 400 K. V. apparatus appears to give a fairly satisfactory result. This has

stood the test of time, whereas the Million volt apparatus is still being worked on empirical formulæ.

The high voltage apparatus has not been tried to any great extent on blood lesions. e.g., Leukæmia, Pernicious Anæmia. Time should decide the utility of this high voltage apparatus in this type of lesions.

Malignant lesions in early cases readily respond to high power apparatus. The study of utilising Ultra Short Wave Therapy prior to X-Radiation has not as yet emerged from the experimental stage though it has conclusively proved that the conjoint treatment means a rapid response to X-Ray Therapy. In the Research Laboratories, the experimental results appear to establish the fact that the high power voltage produces a large amount of responsiveness than the low power voltage therapy. The idea that the tissues develop a sort of resistance or immunity against radiation could not be maintained in the light of present experience.

"The following is a short description of the Million Volt apparatus in use at St. Bartholomew's Hospital, London.

The X-Ray tube of the one million volt apparatus is 30 feet long and weighs ten tons. This consists of a steel tube containing the filament at the target. This is supported by large porcelain insulators at either end and extends from one generator room to the other, across the treatment room. That portion of the tube which lies within the treatment room which is 12 feet in length is surrounded by two protective concentric cylinders, one enclosed within the other, with the interval between these cylinders filled by a six inch closely packed layer of lead shot. The central aperture in this sheath of metal, allows for the passage of the X-ray beam.

This protective sheath is again surrounded by a steel cylinder which supports at the aperture the filters, diaphragms and applicators through which passes the X-ray beam. The protective sheath and the external application cylinder can be rotated independently of each other, so that it is possible to direct the beam of X-Rays as desired either through the applicator towards the patient or into a lead core six inches thick which remains suspended above the tube in position. This arrangement makes the X-Ray tube within the treatment room both shock and X-Ray proof.

In this arrangement since the tube is fixed, it is the platform and the patient on it that have to be adjusted relative to the position of the tube.

The super-voltage installation effectively overcomes the three main limitations of the technique in Radiation therapy, viz., (i) it delivers an adequate dose of Radiation energy to the tumour in its depths without unduly affecting the overlying structures; (ii) this dose is delivered in a comparatively short time and (iii) it ensures and increases the margin of the safety due to the minimised difference, in radio-sensitivity under this particular scheme, between the normal and abnormal malignant tissue.

These are made possible by means of the sufficiently penetrating and highly intensive radiation, the radio-sensitivity of the tissues depending on the wave-length of the radiation employed. Thus by the use of a beam of high penetrative X-Rays, high intensity and shortwave length, we aim at getting results approximating those expected from Gamma Radiations of Radium. This supervoltage apparatus can be operated from 250,000, to 1,000,000 volts and therefore its range and flexibility permits the choice of optimum conditions for treatment in lesions of varying nature.

The physical, chemical and biological effects of X-Rays from this super-voltage apparatus are the subjects of reserach study undertaken in the particular clinics.

The generating plant.—This consists of two generators; one giving a positive potential of 600,000 volts and the other a negative potential of 600,000. The 400 volts A.C. Main feeds the transformers, which raise the potential to 150,000 volts. By means of columns of continuously evacuated thermionic valves and oil immersed condensers, a rectified co-efficient at a continual potential of 600,000 volts at each end can be obtained in this apparatus. Thus when both the generators are brought into use, a constant potential of over one million volts can energize this huge X-Ray tube which gives a varying capacity of current of 5 M.C.

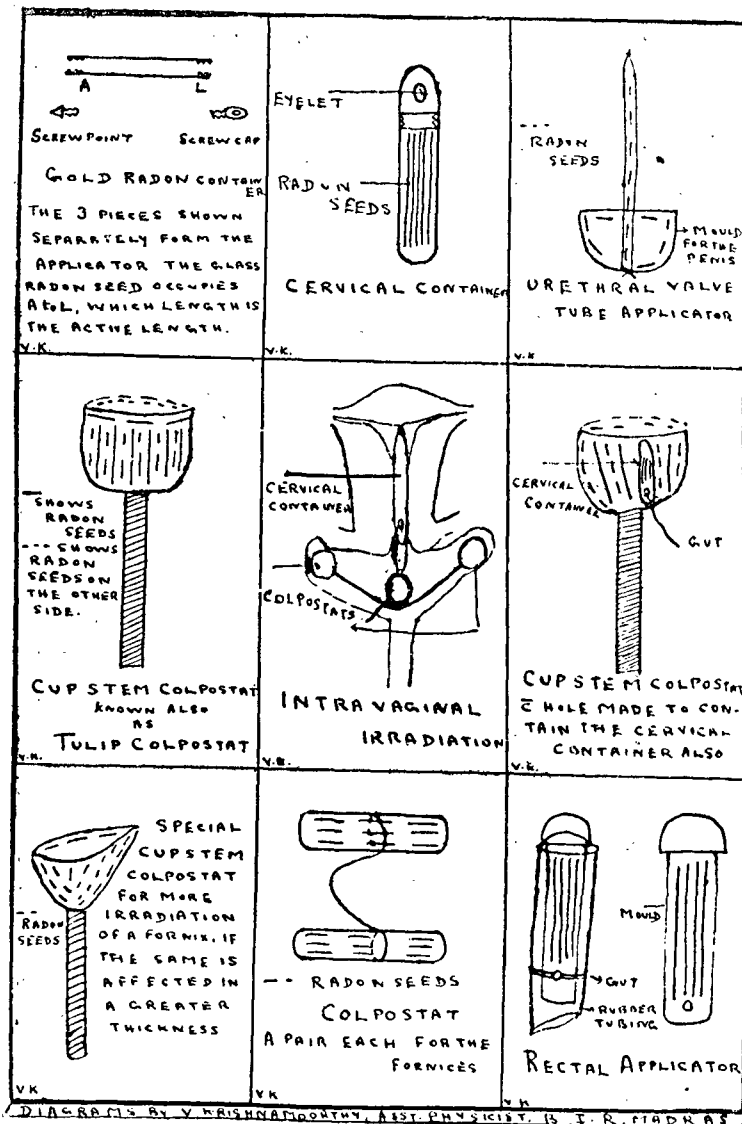
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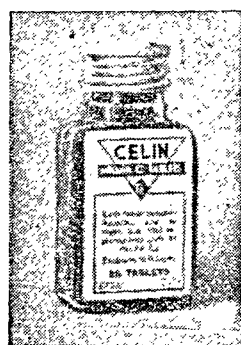
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*Winterton and MacGregor, B.M.J., 1939 I, 5.

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New Aspects in the Therapy of Diphtheria Maligna*

BY

H. J. Hamburger, M.D., (HAMBURG).

Gordon College, Rawalpindi (Americ. Unit. Presbyt. Mission).

I. The student of epidemiology knows that there is a rhythm in the behaviour of certain infectious diseases. There are increases in frequency in seasons, there are diseases with cyclic oscillations of intensity in different years, and there are diseases which show a periodicity only in a survey of long spaces of time.

The diphtheria is the classical example for such a secular rhythm (Doerr)⁽¹⁾. The famous physician Kussmaul said in the second half of the 19th century that the disease described in old French textbooks was never observed by him and he doubted, if the condition existed at all. But only a few years later the great European pandemic started and diphtheria became the most dreaded infectious disease of children.

We are now right in the beginning of a new wave of diphtheria. The advancement in the therapy through the introduction of the antitoxic serum (1894) seems to be paralysed as the disease has chosen a different way of attack against the organism. That is at least the consequence which we have to draw from the reports of many observers about the number of cases fatal in spite of having been treated by serum.

Is there evidence available that the character of the diphtheria has changed? Indeed, de Rudder⁽²⁾ states that there is such a thing as pathomorphosis of the diphtheria, that means a change in the type of the disease. Especially, this change seems to have taken place amongst the more serious cases of diphtheria. In the 19th century, diphtheria maligna—as the most dangerous subvariation is called—was described by its tendency to excessive formation of membranes in the upper respiratory tract. And these membranes migrated swiftly down into the larynx, the trachea and the bronchial tree. They caused croup and bronchopneumonic complications and death occurred mostly from suffocation. Bamberger and Lachtrop⁽³⁾ showed that croup and from the pharynx to the larynx descending diphtheria have become much rarer since 1907. Luebbe⁽⁴⁾ analysed the history sheets of diphtheria cases of a big London hospital and found that there

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too the descending diphtheria had decreased in favour of the pure pharynx diphtheria. Broches⁽⁵⁾ confirms the decrease of larynx diphtheria since 1925 and Schellhorn⁽⁶⁾ finds less bronchopneumonia. With the decrease of the mentioned complications the description of the genuine pathomorphosis is not yet finished. The diphtheria maligna is now characterized by symptoms and complications from the side of the circulation and the general condition of the patient. Stenosis and respiratory troubles are rarer, but heart—and circulatory failure is more frequent. Bamberger and Lachtrop think that this pathomorphosis can be explained thus: the capability of the bacteria to multiply upon mucous membranes has decreased and the production of toxin in the focus of the disease and the absorption into the organism have increased.

This pathomorphosis and the secular rhythm make the diphtheria a most interesting subject for the student of general epidemiology. It remains to future research to explain the reasons for the mysterious ups and downs in behaviour of such diseases.

II. What do we understand to-day by diphtheria maligna? It has been proved that a massive infection does not make a diphtheria a malignant one. The theory has been disproved that a combination of the Klebs-Loeffler bacillus with streptococci means increased fatality. Wright and Rankin⁽⁷⁾ show that there is no or not necessarily any relationship between the clinical type of the infection and the bacteriological variations of *Corynebact. dipht. gravis*, *intermedius* and *mitis*.

We know from experiments that the higher the dose of injected toxin is, the more serious and the more similar to those of malignant diphtheria are the symptoms in the guinea pig. The tragic experience of an accident gives more evidence in this direction. In Baden near Vienna (1924) a number of children were by mistake injected with toxin instead of immune serum.

The early appearance of general symptoms was dependent upon the dose of toxin. Therefore we can say, that the amount of toxin produced and absorbed into the tissues as well as an eventual lack of individual resistance makes the diphtheria maligna.

III. How is the diagnosis of malignant diphtheria to be made? The patient is extremely pale, with light red or pale lips. He is hollow-eyed. Extreme muscular weakness, complete apathy and listlessness are present. His subjective attitude is

that of anxiety and fear, as typical for circulatory failure. He refuses food and whatever little liquid is taken, is vomited. With progress of the disease the adynamia, that is the weakness of the muscles, passes into complete prostration.

The local symptoms are not necessarily as impressive as the severity of the general symptoms. Sometimes, periglandular oedema and swelling of the whole region next to the affected parts indicate the powerful local action of the toxin. But the formation of membranes is frequently limited to the pharynx, and it is characteristic for the malignant diphtheria that these pharynx membranes show little tendency to travel down to the larynx. The membranes show a brownish discoloration and smell offensively due to the putrid disintegration of hæmorrhages in the membranes.

The pulse is hardly palpable, there might be a disturbance of the rhythm, the blood pressure is low, the heart beats can only be heard with difficulty. Hæmorrhages in the skin and mucous membranes occur and are a *signum mali ominis*.

The extremities are cool, but the patient frequently feels so hot that he throws his covers off. The temperature runs a biphasic course: Yieh Pe⁽⁸⁾ noted after an analysis of 1900 cases that the wholesale declaration of diphtheria as a non-febrile condition was not justified. The malignant cases have at least in the first days medium or high temperature. The sub-normal temperatures belong to the second stage of completed toxic action.

Towards the end intestinal troubles, diarrhoea, are observed and a big tender liver is another sign of the circulatory failure which ends within the first two weeks the suffering of the, to the very end, conscious patient.

This early diphtheria death has to be well separated from the late diphtheric lesion, where death occurs perhaps in the 4th or 5th week. Here degeneration of the heart muscle and paralysis of the voluntary muscles prevail.

IV. There are possibilities to verify the diagnosis of diphtheria maligna by means of exact tests. The electrocardiographic examination reveals striking changes under the diphtheria infection of the malignant form. Degkwitz⁽⁹⁾, the head of the German school of pediatricians who have created the here discussed conception of diphtheria and have introduced the later described therapy, says that a malignant diphtheria is present when (a) in two leads at least a negative T is found and S-T is below the

iso-electric line, or (b) when the QRS complex is broadened up to the double of the norm, or (c) when splits in the oscillations of the ventricular complex are found in at least two leads. If several of these abnormalities are combined, and especially if the ventricular complex is only a broad plump elevation going over into the negative ST without a conspicuous knick, the prognosis becomes absolutely bad. The change in the cardiogram precedes the turn in the clinical state from bad to worse, and so does the peripheric capillary pressure. A drop in the pressure of the peripheric capillaries of more than 5 mm water indicates diphtheria maligna and bad prognosis.

It should however be understood that the general practitioner to whom such refined methods of investigation are not available should consider every case of diphtheria with local symptoms of medium or high severity as a very dangerous condition and administer highest doses of antidiphtheric serum.

V. There is a last characterism of diphtheria maligna, which is for the practitioner the most important one: that is the failure of the serum therapy, even under application of highest doses; i.e. 20,000-40,000 Antitoxin units. It was found in the second half of the twenties that the great number of malignant cases observed in the again rising wave of diphtheria did not respond to the serum treatment. And it was one of the great disillusiones at the end of my training as a medical student, when a senior assistant of the clinic took me aside after the round in a diphtheria ward and told me: it really doesn't matter, if you inject the serum with the hypothetic antitoxins in it or if you inject ordinary horse serum; the antitoxin doesn't cure. To-day I know that the belief or disbelief in the 'curative' action of the antitoxin is founded upon a complete misconception of the ideas of E.v. Behring which led him to the introduction of his antitoxic serum. The toxin formed in the membranes of the upper respiratory tract circulates only a few minutes in the body and is rapidly removed from the bloodstream and the body fluids. It becomes attached to the tissue cells. The serum, however, with its antitoxin can't neutralise the toxin once it is fixed to the cells. As diphtheria maligna is characterised by an excessive early toxin production and rapid absorption into the tissue cells, we can understand why the serum does not 'cure', but only prevents lesion. The all important need is therefore early diagnosis and early and repeated serum injection. Schellhorn (14) showed at the clinic of Degkwitz that in spite of improved medical

training, improved means of communication, increased health propaganda and advance in hygiene and preventive medicine, the diphtheria cases of to-day are not admitted to hospital earlier than they were 30 years ago. It seems therefore impossible to improve the results of serum application in requesting earlier injection. The diagnosis and specific treatment of diphtheria before excessive absorption of toxin, remains the ideal, but that ideal will probably remain the exception.

It might be mentioned that the toxins have probably a higher affinity to certain cells than to others. If the disappearance of the lymphocytes from the peripheric blood, stated by Fr. Meyer (11), means that these cells are specially averse to fix the toxin, is not certain. Paulsen (12) however observed in experimental diphtheria leucocytosis and the almost complete disappearance of the lymphocytes. The reappearing of these cells indicates the proper effect of the therapy employed.

VI. As the therapy with serum was so unsatisfactory, efforts were made to find new lines of treatment. It had been thought for a long time that severe disturbance of the circulation in malignant diphtheria was due to an interference of the endocrine glands with the autonomic nerve system. Pathologists had found lesions of the hypophysis. Fr. Meyer verified that clinically and in experiments. There are changes in the thyroid known. The severe damage to the suprarenal glands is observed in such regularity that it is used for a routine test in the laboratory diagnosis of diphtheria. Virchow described already the haemorrhages in the suprarenal, Roux and Yersin showed that the toxin destroys the suprarenals of guineapigs. Bamberger and collaborators (13) made observations at Degkwitz's clinic regarding the collapse of the suprarenal cortex in diphtheria. They collected ample evidence for manifold disturbances in the metabolism, thus leaving the purely immunological line of research for physiological-chemical investigations.

Bamberger made his working hypothesis to be the idea that malignant diphtheria was at least partially due to a collapse of the cortical part of the suprarenals, because the clinical picture of the infection was so similar to Addison's disease. The insufficiency of the suprarenal cortex made the examination of the chloride balance to be the first step in the research. Bamberger and Never (13) found hypochloremia and the excretion of the chlorides through the kidneys was reduced, the nonproteinic N in the serum was found to be increased, the serum calcium dropped.

That together with increase of phosphates and lactic acid and decrease in the total amount of carbon dioxide in the venous blood, was interpreted as reduction of the alkaline reserve. And finally, experiments in animals and clinical observation revealed that the metabolism of carbohydrates was upset. There is reason to believe that this disturbance can be localised in the muscles, which makes it understandable that the diphtheric intoxication leads to (a) paralysis of voluntary muscles and (b) serious affection of the myocard.

VII. Once such ample material being collected about the clinical and metabolic parallel between diphtheria maligna and suprarenal insufficiency, Bamberger and collaborators⁽¹³⁾ tried to substitute by medicaments the missing function of the suprarenals.

We know since the experiments of Biedl in certain fish (selaches, a genus of sharks) more about the different roles of the two parts of the suprarenals. In these fish the cortical part and the medulla are separate. Thus one part can be extirpated without damage to the other. It was proved that the cortical portion was necessary for the sustenance of life. The function of the cortical part can be substituted by extracts containing the so far not yet isolated hormone, while the application of adrenalin, the hormone of the medulla, is useless in cases of failure of the cortex. (Thaddea)⁽¹⁴⁾.

Apart from the cortical hormone, the suprarenals become important through the high content of ascorbic acid or vitamin C. (Szent-Györgyi, Szent-Györgyi and Svirbely)^{(15), (16)}. The preparations used for the additional treatment of diphtheria maligna, in having the advantage to be free of album and adrenalin have the drawback too, to be free of vitamin C, which is destroyed by the Swingle-Pfeffer process of extraction. Bamberger etc.⁽¹³⁾ added therefore some vitamin C preparation and had excellent results. In their first paper they reported f. i. that five children out of eight who would have been lost according to previous experiences, were saved and the fatal end was delayed in the others by 4-5 days.

Why does this new therapy not work in every case? It seems as if through the cortex-vitamin C treatment the weakness of the peripheric capillary circulation was cured. The lesion of the myocard is resistant against the therapy. As long as the

conductive system, mainly the auriculo-ventricular bundle is not destroyed, cure is possible. Auricular fibrillation and auriculo-ventricular block are serious, but the patient may recover. The plump broad oscillation of the ventricular complex, mentioned in (IV) cannot be influenced. But that does not lessen the value of the new therapy as a great number of cases otherwise lost, can be saved.

VIII. The procedure in cases of diphtheria maligna (we regard as such, where an electrocardiograph etc. is not available, all cases with severe local symptoms and those with early appearing general symptoms) would be: 20,000 - 40,000 Antitoxin units. Until the end of the second week 12 c.c. of a cortex preparation are given daily, containing in 1 c.c. 6 cortico-dynamic mice units. In addition vitamin C is given, 400 - 500 mllg dissolved in 10-12 c.c. water. Recently the administration of Sodium Chloride has been added, 2-3 grm, corresponding to about 220-330 c.c. normal saline. The cortex preparation might be given intravenously, intramuscularly or orally, the vitamin C, and the saline best intravenously.

The administration of Sodium Chloride was reported to be successful in diphtheria by Howell and Sears, Harrop, Touw and Noordhoek, Nothroer^{(17), (18), (19), (20)}. It seems obvious to employ it in order to fight the hypochloremia and hypochloruria.

There is another method which is a valuable help in diphtheria. Baar and Benedickt⁽²¹⁾ proposed the blood transfusion for malignant cases. Seckel⁽²²⁾ confirmed their results and obtained in malignant cases with 80% lethality a drop to 40% when treating with specific serum plus blood transfusion. Bamberger could save 63-70% out of malignant cases with 100% lethality according to his stricter standards.

I have not had the opportunity to observe a great number of diphtheria cases in India. But reports from colleagues in different places in the Punjab and Kashmir etc. show that the odd case observed is of extreme malignity. Every few years severe epidemics occur and will continue to occur as long as there is no obligatory prophylactic inoculation. That made it well worth while to draw the attention of the profession in this country in time to the study of new methods in the treatment of this condition.

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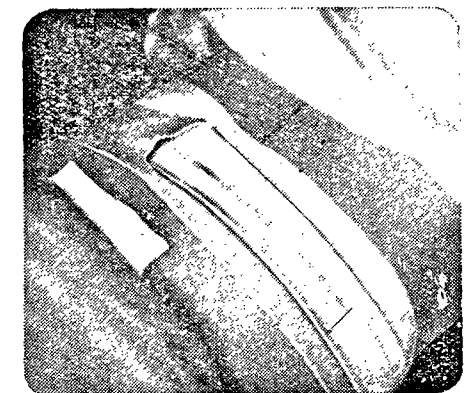


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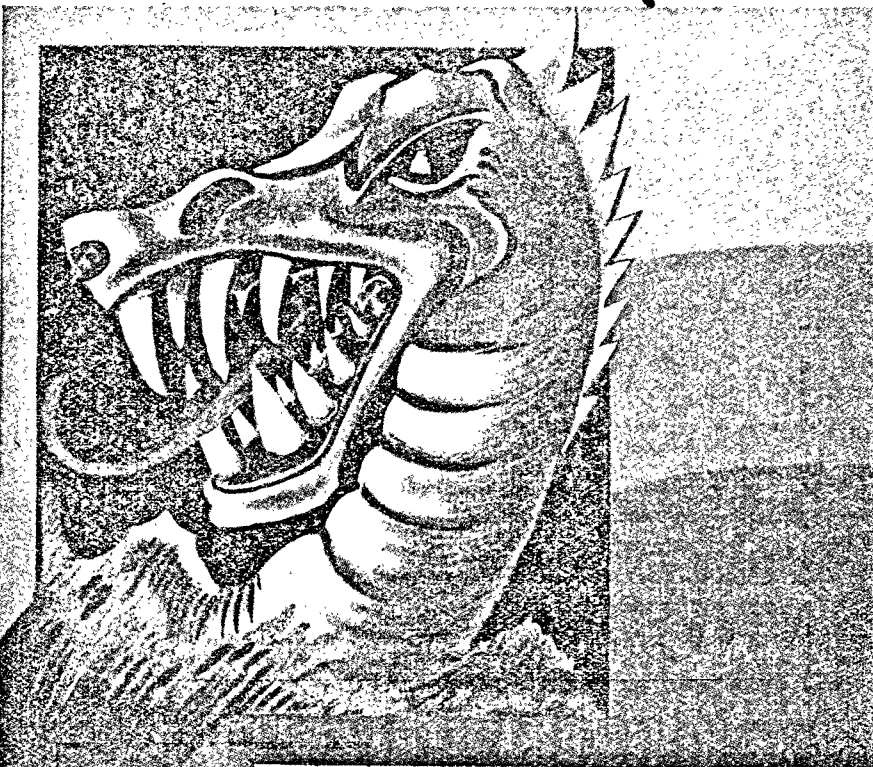
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Medical Control in Sports*

BY

Prof. J. M. Pacheco de Figueiredo,

Medical College, Nova Goa.

WITH the ever-increasing development of Sports, there have been already organized in India, the Sporting Clubs, well-constituted associations which, by their nature, have to fulfil serious responsibilities for the Society. This exists in not allowing any and every one to practise Sports. There is an indispensable necessity of a previous biometrical and medical examination combined with radiography and radioscopy of the cardio-pulmonary apparatus which will, in the near future, form part of the routine of Sporting Clubs.

The organisation of individual report duly elaborated, with a systematic repetition of the examination, will compel the doctor, to have always under his control all those that dedicate themselves to Sport, more or less violent. And what surprises will these examinations spring up?

It was but a few months ago, that we had occasion to examine strictly 120 adults, most of whom dedicated to the Sport Foot-ball in special. We have noted very interesting facts but they are not in scope of this small article. In the meanwhile, in corroboration of what we wrote and especially in our article published in the "Antiseptic", in January 1939 entitled "The hypertrophy of heart among Goan Students" we are giving here below, in rapid synthesis and in some tables, a description of the state of physical health of twenty persons examined by us who have practised Sports.

Number of Persons Examined—20.

TABLE I
Chest Circumferences (Mammary)

720	750	760	770	780	790	800	810	820	830	840	850	890
m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.
1	1	1	1	1	3	4	3	1	1	1	1	1

We estimate from this table that more than half of the examined persons had the perimeters of 800 m.m. or less

* Specially contributed to "The Antiseptic".

TABLE II

Amplitude of Chest Circumference (Axillar)

30	40	45	50	55	60	70	80	85	95	100	130
m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.
2	2	1	1	1	5	3	1	1	1	1	1

The chest amplitude (axillar) of more than half of the examined persons was less than 70 m.m.

TABLE III

Amplitude of Chest Circumferences (Xifoidea)

35	40	45	50	60	70	75	80	90	120
m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.	m.m.
1	1	2	6	3	3	1	1	1	1

More than half of the persons under observation had an amplitude (axillar) of 60 m.m. or less.

TABLE IV

Anthropometric Segment

This element gives us the notions of *relative weight* that is the relation of weight in quilos to the height in decimetres.

$$\text{Formula} = \frac{\text{weight in quilos}}{\text{Height in Decimetres}}$$

The Anthropometric segment among robust individuals must vary between 3.50 to 4.00.

Here is the Result obtained by us:

2.81	to	2.90	—	3
2.91	to	3.00	—	1
3.01	to	3.10	—	3
3.11	to	3.20	—	0
3.21	to	3.30	—	2
3.31	to	3.40	—	3
3.41	to	3.50	—	3
3.51	to	3.60	—	2
3.61	to	3.70	—	1
3.71	to	3.80	—	1
4.13	—	—	—	1

This table shows us that 12 often examined had a segment less than 3.50 among the 20 examined.

TABLE V

Index of Vitality

$$\text{Formûla} = \frac{\text{Chest circumference mamilar} \times 100}{\text{height}}$$

The robust individual must have an index more than 50. Our results:

42	43	44	45	46	47	48	49	50	51	52
1	1	—	—	2	4	3	4	2	1	2

Only three (3) of the examined had the index more than 50.

TABLE VI

Index of Pignet-Mayet

This index sufficiently known has the following formula:

$$\text{I. P. Mayet} = \text{Height} - \frac{(\text{Weight} + \text{Chest Circumferences (Insp. + Exp.)})}{2}$$

Classifications:

Extremely strong	—	Coefficients less than 0
Very Strong	—	„ 0 to 10
Strong	—	„ 11 to 20
Regular	—	„ 21 to 25
Weak	—	„ 26 to 35
Very Weak	—	„ more than 35

Results

Very Strong	Regular	Weak	Very Weak
1	6	6	7

TABLE VII

**Results of Medical and Radioscopic Examinations of
Cardiovascular Apparatus**

MEDICAL EXAMINATION.		RADIOSCOPIC EXAMINATION*	
Ap ⁶ ...	1	Hypertrophy of left ventricular arch. Heart-apex at the level of the 6th intercostal space ...	11
Ap ⁶ and 2nd sound of aorta Vibrating ...	1		
Blood-pressure Mx 16-Mn 8 ...	1		
" " Mx 14-Mn 8 ...	1		
Murmur in mitral region ...	1		
No manifestations ...	6		
Murmur in aortic region and blood-pressure Mx 14-Mn 8 ...	1	Dilatation and hypertrophy of left ventricular arch. Dilatation of right auricular arch. Apex at the level of the anterior arch of 6th rib ...	3
Ap ⁶ & bloodpressure Mx 14-Mn 8½ ...	1		
No manifestations ...	1		
Extra-systoles, 2nd sound in pulmonary region vibrating and blood-pressure Mx15-Mn 7 ...	1	Dilatation of right auricular arch. Pulmonary artery arch prominent. Elongation of left ventricular arch ...	1
No Manifestations ...	1	Elongated aorta. Dilatation of right auricular arch. Dilatation and hypertrophy of left ventricular arch ...	1
Blood-pressure Mx15-Mn-9 ...	1	Globular heart Shadow Elongated aorta ...	1
Cardiac aptitude (Lian) Mediocre. ...	1	Globular Heart ...	1
No Manifestation ...	1	Dilatation of right auricular arch. Narrowing of retro-cardiac space. Hypertrophy of left ventricular arch ...	1
Murmur in mitral region. Heart-beats at the base accentuated &c., blood-pressure Mx14-Mn 8 ...	1	Hypertrophy of tracheo-bronchial glands at right side. Prominent aortic arch. Prominent pulmonary arch. Hypertrophy of left ventricular arch. Dilatation of right auricular arch. Apex at 6th intercostal space filled by hypertrophied glands ...	1
Total ...	20	Total ...	20

Note:—Ap⁶ = Apex in the 6th intercostal space.

* This examination was made by Dr. S. Veiga.

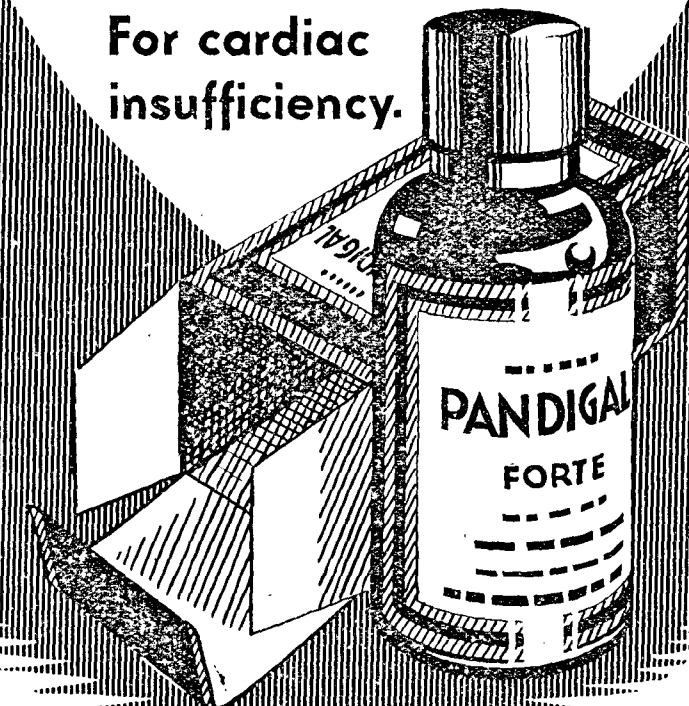
The tables described above need no comments. They speak clearly enough on the necessity of the medical intervention at every step, in the practice of Sports. The following words of George Rosental will never be over-repeated:

*Medical Selection before the Sports, Medical Vigilance
during the Sports and Medical Control after the Sports.*

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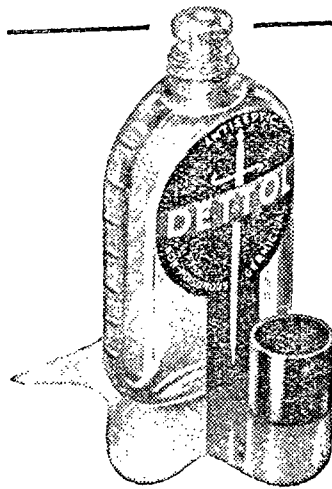
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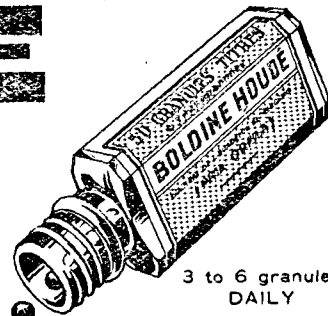
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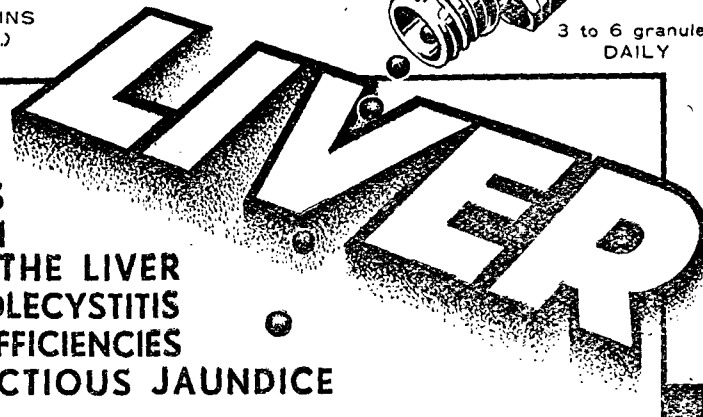
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Diet in Disease*

BY

C. U. Parvani, M.B., B.S.,

Gambat, N. W. Ry., Sindh.

IN our armoury against disease diet has a definite place and like all other remedial measures, it has a limited range of action in the therapeutics. The dietetic treatment is of especial importance in a particular group of disorders and to those disorders primarily attention will be confined.

Diet in Fevers.—Hippocrates, the celebrated Greek physician and teacher, supposed that fever originated from bad humour and intestinal irritation and taught: "Starve a fever". He in order to allay intestinal irritation and to limit the range of temperature, allowed only non-irritating diet. Strange to say this fallacy continued to be adopted by the physicians of middle ages who even invented a pathology to justify this fallacy. Later on, the physicians of nineteenth century basing their teachings on the clinical experience thought that fevers were asthenic diseases and should be treated with liberal feeding and thus reversed the teachings and fed the fevers. Old prejudices die hard and hence this doctrine has never been adopted in toto in practice.

Within recent times the pathological conception of fevers has undergone a radical change. The fever is almost always caused by the action of pyrogenetic substances and which are the products of infective agents—bacteria, protozoa and other parasitic organisms. It is a complex phenomenon comprising of (1) rise of temperature (2) cardiac and respiratory acceleration (3) altered metabolism and (4) functional and structural alterations such as are commonly seen in diseases like enteric and other continued fevers. It has been shown that due to some metabolic changes occurring in tissues, water is drawn towards them especially to muscles. Consequently the blood volume is decreased and there is redistribution of circulation. This decrease in blood volume may be the result of excessive sweating, diuresis, or restriction of fluid intake. Further, it is well known that the excretion of chlorides in fever is decreased, especially in pneumonia and other infectious diseases. This retention of salts is associated with the simultaneous retention of water in muscle,

* Specially contributed to "The Antiseptic".

specially in long continued fever such as typhoid. Thus tissues are relatively richer in water and salts and the blood volume is poorer. In febrile stage, the urine is concentrated having larger quantity of urea, uric acid, creatinin and ammonia salts. As these nitrogenous bodies are largely derived from proteins, the disintegration of protein is increased in fever. This acceleration in metabolism may be due to heat, for warmed tissues metabolise more material than cold tissues and also there may be a toxic element that causes tissue destruction. It is estimated that the increased metabolism rarely exceeds 40% to 50% over normal and the amount of protein loss to the body may be as much as 200 to 500 gm. of muscle tissue per day. Further due to the action of toxin there is marked decrease in protein absorption and there is loss of appetite which in its turn by inducing partial starvation leads to increased protein metabolism. Thus satisfactory planning of diet in fever depends on the consideration of (1) pathological changes (2) daily quantitative requirements (3) personal and racial idiosyncrasies (4) climatic and provincial conditions. We have seen from the preceding paragraphs that (a) there is decrease in the volume of blood, (b) increased protein metabolism and (c) decrease in protein absorption.

In order to increase the volume of blood in fever, plenty of liquid diet and drinks are to be administered. This will further help in diluting toxins and their removal. The daily normal requirements of protein is about 73 grammes. Theoretically, therefore, it follows that sufficient nitrogenous substance should be given to replace the loss. But, in practice, it has been observed that it is impossible to bring about a condition of nitrogenous equilibrium in acute fevers. For the obnoxious action of toxins in tissue cells renders them incapable of assimilating proteins and the broken up proteins are flooded in circulation which being toxic to kidneys and liver do more harm to the system than replacing the loss. Under such circumstances, it is foolish to give proteins in large quantities to the febrile patient. Nature has made a provision for this exigency. Carbohydrates and fats are known as "fever protein spacers". It has been demonstrated that by giving carbohydrate food the nitrogenous equilibrium could be maintained. Experimentally it has also been shown that under carbohydrate diet, the abnormal excretion of creatinin which is probably derived from the break down of muscle tissue, is reduced. Fat, we know by common experiences, is not liked by febrile patients as it induces nausea in them and is not readily digested.

Caloric Requirements.—I have shown in my previous article that an average Indian of sedantary habits requires about 2150 calories per day. By common consent fluid diet is best suited to febrile patient. The milk is then the diet of choice, as it contains, all the approximate principles and digestible animal protein. For necessary caloric value, about 5 pints of milk are to be consumed and few patients will agree to it. The milk protein could be increased by the addition of such preparations as Plasmon and cream to increase the proportion of fat—thus the total quantity of milk could be reduced. Further, milk could be enriched by the addition of glucose, milk sugar, arrow root, barley, oat-flour, sago, honey etc. Fruit juice of orange, grapes, pineapple, pears and pomegranate, may be allowed. Sugar cane if available and if the patient is able to chew it, will be helpful, as it will clean the mouth and the juice is very refreshing. In hot climate iced aerated beverages taken in moderate quantities are very refreshing, but when indiscriminately indulged in, give rise to throat congestion and abdominal distention. Alcohol in moderate quantities is useful. For by dilating peripheral blood vessels, it lowers the temperature and reduces the tissue waste by sparing fat. It helps the failing circulation and induces sleep. In conclusion as the vital processes of the body are reduced, such foods should be chosen as are easy to take, easy to digest and easy to assimilate.

Diet in Typhoid Fever.—A good deal of confusion exists over diet in typhoid fever in India. Though high caloric diet is the routine treatment of typhoid in United States and to a less extent in England, in India, an average physician hardly cares to adopt it. It is strange but nevertheless true that numerous workers repeatedly trumpet the merits and demerits in our Indian magazines, on the new drug in the market or the latest successful development on the Continent, but hardly care to write on the controversial subject of diet in typhoid fever. This absence of authoritative literature coupled with non-existence of nursing profession makes us wary of adoption of more liberal diet. May be the teachings of Unani, indigenous system of medicine, are too strongly implanted in us to be shaken off in spite of ourselves. Besides we have inherited a legacy of some fallacies—namely, that the free administration of food, tends to raise the body temperature of a febrile patient, the febrile patient does not absorb the nourishment from food given, and that the pathological structural alterations in the walls of intestines in typhoid fever make

them liable to perforation and hæmorrhage by the intake of liberal diet. Von Hoesslin's experiments proved that in cases of typhoid, the temperature during the days when food was administered was only 0.11 to 0.3 higher than on starvation diet. Besides the absorption of light diet goes on perfectly in the febrile as in the non-febrile state. The stress of the infection in typhoid fever usually falls on the intestines and the principal sight of lesion is the lower part of the ileum and the ileo-cæcal valve. The wall of intestine gets inflamed and develop characteristic shallow ulcer with undermined edges and tissue paper like base. Sometimes necrosis proceeds farther through the muscular and serous coats and result in dreaded hæmorrhage and perforation. It was considered a miracle that so many ulcers in typhoid patients had stood up against the passage of even fluid and normal intestinal movements. It was concluded that with minimum passage of liquid food, there was maximum chance for the base of ulcer to survive. But the modern view is, that an average typhoid patient living on starvation diet, in order to meet with the requirements of the increased metabolism, has to live on his fat first and then on his muscles, including his intestinal ones. But for this emaciation of the intestinal walls, the walls would have formed a firm base for a typhoid ulcer and good support for a blood vessel exposed by the separation of slough. It has now been conclusively proved that the chance both of perforation and hæmorrhage in the patient living on high caloric diet is considerably less compared to one living on starvation diet. Care should be taken to give only such foods as are easily digested and easily assimilated and which do not reach the lower end of ileum in a solid state.

The orthodox school relying on liquid diet allow only diet of 1000 to 1200 caloric value per day. The milk supplying 600 to 800 calories and rest is supplied by glucose. We have seen that there is increased metabolism by 40 percent in typhoid fever, the average requirement of normal man of sedentary habits being 2150 calories, the total requirement, therefore, is about 3000 calories per day. It is also known that a typhoid sufferer loses about one fifth of his muscle weight in the course of fever. If this loss of weight is to be avoided, sufficient diet of caloric value must be given per day. It is calculated that a diet of 2000 to 2500 calories per day will maintain sufficient weight and strength of the patient. If the milk agrees the bulk of food may be made up of it and the rest of glucose. If milk is not well tolerated, it may be substituted by Ovaltine, Horlicks or some other proprietary

milk preparations. Lactose and dextrin are better than glucose as they are less likely to give rise to fermentation and besides not being so sweet are better liked. Though soups contain little nourishment, are prescribed for a change. Biscuits, bread and butter when thoroughly chewed, though solid when they enter mouth, are as fluid as milk, by the time they leave stomach. Besides they cleanse the mouth. Fruits, vegetables, cooked rice strained through muslin to keep back the coarse fibres, and potatoes mashed with butter, are useful items. Beaten up eggs or lightly cooked ones are allowed. Whatever the diet given, its vitamin contents must be properly assessed and missing ones be supplied. In cases of vomiting and diarrhoea and tympanites, only skilful nursing with attention to daily diet will carry the patient through. In toxic patients who cannot swallow even liquids properly, subcutaneous salines with 50% glucose will often tide over the crisis in many cases, supplying fluid and few calories as well. Water may be given freely and as much as three to four litres of water should be consumed in a day. Barley water, lemonade and soda water are also useful. A good drink is prepared by addition of Soda Bicarbonate and Lactose to water. It should be given as often as patient feels thirsty and is also an efficient diuretic. Patient's likes and dislikes are to be attended to. By the judicious giving of diet the exhaustion is avoided, the heart muscle is supplied with the all important carbohydrates and convalescence is materially shortened.

Ascorbic Acid Content of Some Important Foods

G. Panfil and M. Maxim (Chemicopharmaceut. Laboratory, Univ. of Bucharest) analysed the following foods and obtained the quantities listed in mg. per 100 gm.:

Capiscum shell, raw green...	160.0	Apple-peel	13—15.0
Capiscum shell, raw, red (ripe)	270.0	Melons, green	... 9.8
Capsicum shell, roasted	... 230.0	Melons, yellow	... 3.2
Green cabbage, raw	... 33.0	Pears	... 4.5
Green cabbage, boiled	7.0—20.0	Plums	... 5.5
Cauliflower, raw	... 41.0	Peaches	... 5.7
Cauliflower, boiled	... 9.5	Grapes	... 5.5
Potatoes, raw	22—25.5	Onions	... 3.5
Potatoes, boiled	6—17.5	Carrots	... 5.7
Tomatoes, raw	... 16.5	Beets, boiled	... 2.2
Tomatoes, boiled	... 5.0	White bread	... 2.1—2.4
Cucumbers	... 11.5	Milk, raw	1.2—1.5
Cabbage-lettuce	... 14.0	Milk boiled	... 1.7
Apple with peel	7.5—12.0	Goat's Milk cheese	...

One thus sees that boiling vegetables and peeling fruit cause a great loss of ascorbic acid. Since this vitamin has a great influence on the endocrine glands, particularly on the suprarenals, and thus regulates carbohydrate metabolism, an attempt should be made to eat enough fresh fruits and raw vegetables. (Klin. Wschr., No 44, 1938.)—*Ars Medici*.

CASES AND COMMENTS

An Interesting case of Hydrocephalus

BY

Major R. G. Dani, I.M.S., M.B.B.S., M.R.C.S.,

Nasik.

A CASE was brought to my Nursing Home on the 11th of April 1939 at 10 p.m. in labour. Patient was multipara, the present pregnancy being the second one. The first delivery was conducted at her own home and was normal in every respect, except that the puerperium was complicated by fever for a period of 30 days for which she was treated in a hospital. During the present pregnancy, according to the statement of the patient, she was keeping an indifferent health. I had no opportunity of seeing her professionally during the completion of the pregnancy, till her arrival at the hospital in labour.

Patient is a woman of small stature and build. Her weight is 90 lbs and her height is 4 feet and six inches. The pelvic measurements were as follows:—

Inter-costal: 8 and three quarters inches.

Inter-spinous: 8 inches.

External conjugate: 6 inches and a quarter.

Internal conjugate: 3 and half inches.

Before coming to the hospital she was feeling uncomfortable and indifferent for two or three days. The pains began at 3 p.m. on the 11th and soon after the Liquor Amnii began to flow out, and was flowing out when arrived. On external examination the abdomen looked and was seen to be enlarged out of proportion to the natural build of the patient and in spite of the escape of the liquor amnii in large amount. The fundus reached as high as the ensiform. The lower segment looked much wider than usual. On palpation the uterus felt hard all over except to a certain extent on the right border, where soft parts of the foetus could be moderately made out. All the elasticity of the uterine wall due to the presence of liquor amnii had gone. A hard indefinite mass was found stretching across the lower segment. On vaginal examination, the os was found to be three fingers dilated, and the cervical margin soft. The bag of membrane was ruptured. The caput could be felt softish, in between pains, and tense when pains were on.

Foetal heart sounds were distinct over the umbilicus much more towards the ensiform on the left side. On the right side faint heart sounds were suspected deep below the umbilicus towards the flank. No uterine souffle was made out.

The pains were of a feeble nature, coming at long intervals and lasting for a very short time. Her temperature was 99° and pulse rate at 88 p.m. Under the circumstances present, a provisional diagnosis of twins was made and labour was allowed to proceed naturally. It was presumed that the vertex felt on p.v. was the head of the first on-coming foetus in the third position, and the hard mass felt on the left side in the lower segment, as the head of the second foetus following, in the 1st position, as was strongly evident by the distinct heart sounds heard over the umbilicus on the left side. Patient was given $\frac{1}{2}$ c.c. of infundibulin as the pains were very slow, simulating primary inertia. The injection had only temporary effect lasting for an hour, during which cervical dilatation increased somewhat and the caput descended a bit more. As she was feeling tired out, she was put to rest by administration of Chloral hydras at 1 a.m. She slept on till 10 a.m. of the 12th. The condition of the pains remained about the same. At 12 noon she had a rigor and the temperature went up to 104 degrees. This was probably a malarial rigor as she has, according to her own statement, been suffering from same previously at home. Temperature came down to normal at 6 p.m. The pains kept on being feeble and of intermittent type, as is usual in twin pregnancy, and as the time allowed is longer, she was allowed to go on. At 6 p.m. another p.v. was made and it was found that the os was now four fingers dilated, and could be stretched easily to full dilatation. The caput was bigger and had descended lower. At this stage another injection of infundibulin was given. This had no effect and patient began to show signs of exhaustion. By 10 p.m., it was decided to have recourse to artificial interference. Under chloroform, careful deep P.V. examination showed that the foetal head was occupying the whole of the pelvic inlet and was fixed. The cranial bones were widely separated, and the caput on hard pressure gave a tense feeling very much like water under pressure. The head was fixed in the transversed diameter of the brim and the fixed diameter of the foetal head was the occipitic frontal. The sutures were wide apart. A clear diagnosis of the presence of hydrocephalus was at once made out. It must be stated here that an early diagnosis of hydrocephalus was not possible owing to the peculiarity of the foetus. This to a certain extent was helped on by the fact that

the first delivery was normal, and also the absence of previous examinations previous to the onset of labour. The patient never presented for examination until the labour had progressed well on. The early rupture of the membranes, and the slow, unforcible uterine pains were both in favour of presence of twins, as hydrocephalus in which these may be present is such a rare occurrence. A wait and see policy had to be followed till the patient began to show signs of exhaustion, and the foetus signs of distress.

The cranium was perforated and roughly 20 ounces of straw coloured fluid was let out. The tense and hard condition of the uterus was at once relieved and it became soft, and different parts of the foetus could be distinctly made out. The foetus was removed by traction and the rest of the delivery completed. Slight difficulty was experienced in the delivery of the frontal bones owing to their large expanses. Both the eyeballs were protruding out and the sclerotic showing widely.

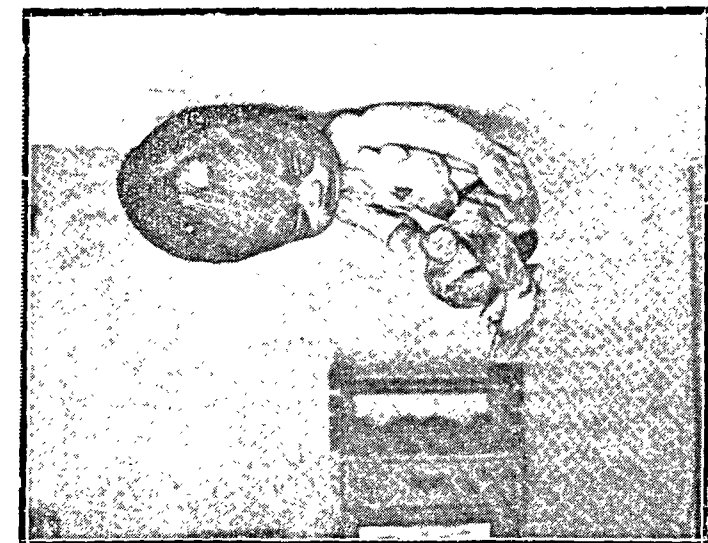
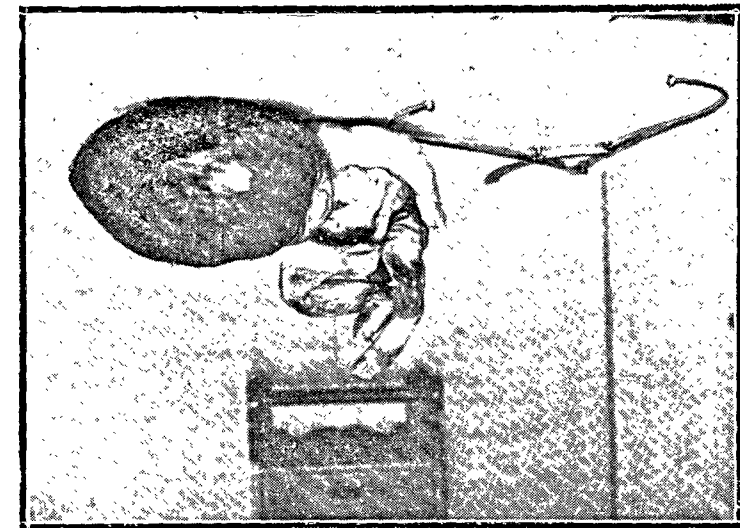
The following are some of the main diameters of the foetal head:—

- (1) The diameter from the occiput to the most dependant part of the vertex was measured at ... $7\frac{1}{2}$ inches.
- (2) Occipito-frontal ... $5\frac{1}{2}$ "
- (3) Suboccipito-bregmatic ... $5\frac{3}{4}$ "
- (4) Occipito-frontal ... 6 "
- (5) Biparietal ... $5\frac{3}{4}$ "
- (6) Bitemporal ... $4\frac{3}{4}$ "
- (7) Greatest circumference ... 20 "
- (8) Smallest circumference ... 17 "
- (9) Weight of the foetus at delivery... 7 lbs.
- (10) Length of the foetus ... 17 inches.

These diameters are taken after the specimen has been hardened and the cranium filled artificially and sutured. In the living state of the foetus these must have been somewhat bigger.

To my mind this case has the following importance. Could an early diagnosis of the true condition be arrived at early? I have given, during my discussion of the case, my reasons for giving benefit of the doubt to the presence of twins. The general lay of the foetus was such as to help diagnosing twins to 75%. I must say that it baffled me, having done and supervised over 4000 cases. In all my experience this is the third real hydrocephalus that I have seen.

An Interesting Case of Hydrocephalus—By Major R. G. Dani, I.M.S.



[To face page 408.]

A Rare Case of "Acute Thyroiditis"

BY

A. S. Dawson, L.M.P.,

Paungbyin, Upper Chindwin District, Burma.

THE thyroid body is a ductless gland which lies beneath the fasciae of the lower part of the front of the neck in close relation with the larynx and trachea. It consists of two lateral lobes joined by an isthmus which lies across the 2nd, 3rd and 4th rings of the trachea. The gland itself has a well defined fascial sheath and a fibrous capsule which dips in between the constituent acini to form the stroma; between these two, the vessels enter and many large veins are to be found.

The blood supply of the gland is very free being derived from the four thyroid arteries which enter at the four corners and possibly from the thyroidea ima artery which runs up to the front of the trachea to the lower border of the isthmus. The glandular acini are lined with cuboid epithelium and secrete a colloidal material which normally contains an albuminous preparation of iodine and thyroxin. There is very close connection between the acini and the surrounding lymphatics and vessels. The secretory activity of the gland undoubtedly plays a very important part in the economy of the body.

Acute thyroiditis is an inflammatory disease of the thyroid gland by pyogenic organisms arising from local lesions, but more commonly it is a blood borne infection and may follow acute infective fevers.

The patient who was admitted to the Civil Hospital, Paungbyin, was a police constable, Chin by race, aged 26 yrs. and his birth place was Chin Hills where Goitre is endemic but when he was enlisted in the force, he had not developed this. Past history showed that he was often on patrol duty suffering from malaria off and on and on his admission to hospital he had an enlarged spleen and this gave him no trouble as he was used to take prophylactic quinine often.

Condition on Admission.—He complained of severe pain in the upper part of the chest and neck, he had 100° fever; examination of the throat revealed tonsillitis. Lungs: Rhonchi over the bases, as he complained of cough also. He had enlarged spleen but the enlargement was only slight. Blood examination showed Leucocytosis only, no parasites were found. The right

lobe of the thyroid was enlarged and tender and hot to touch; the muscles of the neck fixed; the right lobe of the thyroid was also slightly displaced to the central part of the neck, he was unable to move his neck at all and had dysphagia, he could take water only with difficulty, breathing was hurried but not difficult on this day. The fever was ranging from 100 to 100.4 for one week and it was decided to give palliative treatment first.

Treatment :—Hot fomentations with belladonna pigment four hourly and 'entodon' 2 c.c. intravenously and Sodium Iodide mixture grs 20 t. i. d. Later, the mixture was one of Pot. Iodide and Liq. Hydrargyri Perchloride was given for two days. On the 7th day the patient felt better but the signs of suppuration were definite. Under Novocaine 5% with Adrenalin 1 c.c. local infiltration anæsthesia, a dissection of the part was made and the gland tissue was entered and pus evacuated by a drainage tube, the wound was dressed with Dettol antiseptic and he made an uneventful recovery and was cured within a fortnight and was fit for his duty on the 6th of November '38. As this is a rare case and I had never seen one in my 29 years of Service in Goitrous and other hilly tracts in Burma, I am sending this for publication.

Dracontiasis or Guinea-Worm Infestations

BY

Capt. J. M. Ghosh, M.B., (CAL.), D.T.M. & H. (CANTAB),
D.P.H. (LONDON).

Late Chief Medical Officer, Keonjhar State.

IT is an unknown quantity in these parts.

We remember when we were students of the Calcutta Medical College and had just started attending the wards in the hospitals, as juniors, one morning, one of our class friends told us that whilst dressing a case of ulcer thigh, he had found something like a piece of twine in the wound, which snapped when he had attempted to pull it out. In blissful ignorance the matter was allowed to drop at that.

Next morning, the patient, a poor girl from Kathiawar side, was in a terrible state—the temperature was 105, the whole thigh was tender, swollen and angry looking. Multiple

incisions were made and baths ordered. It took about 1½ months before she could leave the hospital—our first case of guinea-worm.

In Central India, where these cases are tolerably known, we met with our second case rather unexpectedly. A knee-joint was being aspirated for effusion when the cannula got blocked by one of these. The joint was opened and a tangled mass of worms removed.

The 3rd Case, the subject matter of these few lines, we came across at Katmandu, Nepal.

One morning, we were asked to see a case with high temperature and severe pain in the right leg, about the knee-joint. We were further told that something like a string had come out through the skin below the right knee.

When we went we saw a match stick dangling just below the right knee and wound round it a few turns of what might have been properly called an extra stout piece of silk worm gut. Temperature was 102°, the knee and the lower part of the thigh inflamed and tender and kept flexed.

The match stick showed that he was a knowing man. On enquiry, it came out that he had learnt the 'trick' when he had been to Rajputana. Unfortunately, his education had been rather defective—he had not been taught what would happen if the worm broke when being twisted round the match stick. Of course, it was a guinea-worm mishandled.

We gave an injection of Neostibosan (.1 gm.) intravenously, in the afternoon. He got a severe rigor and the temperature went up to 105° at night. No injection was given the next day. On the following day, the injection was repeated—.1 gm. being given. No reaction followed. Temperature touched normal. The match stick, we were told, dropped off by itself. The pain was, to all intents and purposes, gone, only a feeling of stiffness remaining. The third and last injection was now given—Same dose as before. The patient came to see us on foot, on the 8th day of our visit.

The drug proved so effective that one could confidently say that any one giving it a trial is very likely to find it worthwhile.

Syphilitic Arthritis of the Left Wrist Joint

BY

Captain, P. S. Gupte, M.B., B.S.,

Nasik.

IT is generally written in the text books that syphilitic arthritis is not associated with pain. The following notes are interesting on this point:—

I had a female patient aged 30 years who was suffering from acute pain in the left wrist for one month before she consulted me for treatment. There was much swelling and redness about the joint. It was so very painful that even a slight jerk would produce an unbearable pain. She had temperature running between 100° and 101°. Her heart was normal. She looked much exhausted on account of sleepless nights.

Soda Salicylas, Quinine and milk injections had no effect. No definite cause could be found out for the trouble. I hit upon giving her one Neosalvarsan 0.45 gm. To my surprise the patient found much relief. After giving her a few more of the injections the trouble rapidly disappeared and the patient made an uneventful recovery.

No blood examination was done. There was no history of abortion or any other evidence of syphilitic taint.

As the Salvarsan gave her complete relief I could not but infer that the syphilis was the cause of the whole trouble.

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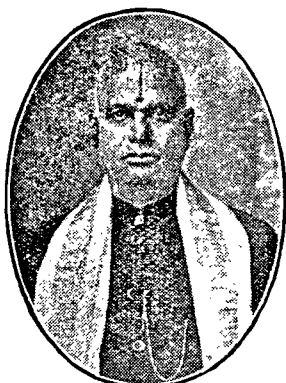
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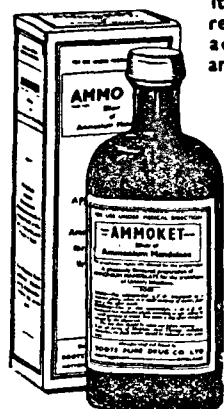
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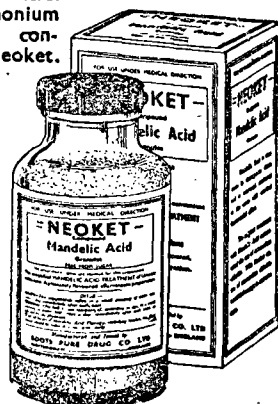
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No. 5.

EDITORIAL

The Madras Public Health Budget for 1939-40

DURING the discussion in the Legislative Assembly in March '39, over the Budget provision of Rs. 28,31,700 in respect of the Public Health Department, the Hon. Dr. T. S. S. Rajan, Health Minister, has clearly and unmistakably outlined the policy and programme of the Government. The criticisms of the opposition may be classified generally under the following heads:—

- (1) Re-organization of the Public Health Department and the appointment of Women Health workers to educate Muslim women in regard to certain harmful social practices prevalent among them.
- (2) Health Propaganda in Villages.
- (3) Vaccination.
- (4) Inadequate Leprosy Relief.
- (5) Rural Medical Relief.

The above criticisms, as the Minister himself had observed, were mostly specific complaints of inadequate treatment. The policy of the Government was laid down by Dr. Rajan in these words:—"The whole phase of the administration is now veering round from cities to villages and those that have observed the working of this Government will realize that villages loom large

in our eyes. It is natural that it should be so. For years, villages have been neglected. For the first time, the villages have come to be recognized as units in the national life which have got to be attended to first."

The scheme for the provision of a protected water supply in every village is a pregnant example of the Government translating their policy into action. Out of the Rs. 15 lakhs earmarked for the purpose, 64 wells had been dug in one year and it is expected that 657 wells would have been dug before this year was out.

Dr. Rajan admitted that more health visitors and propagandists were needed and added that every Government servant had to become a propagandist on the question of public health and sanitation.

In regard to the objection raised against Vaccination by one of the opposition members, the Minister instanced the recent serious outbreak of small-pox in the city and pointed out how it had been brought under control by an intensive campaign of vaccination, nearly two lakhs of people having been vaccinated.

In the matter of Leprosy Relief, the minister said that the special officers of the Government were doing a good deal of work and the Government had now directed their energies to the preventive aspect of the disease. This was a more rational and scientific approach to the problem of relief than having a large number of Leprosy centres.

We entirely agree with this new policy and as children are the most susceptible to infection, efficient and emergent steps must be taken to separate the children from their leprous parents and provide for their healthy living and in the case of children who have already caught the infection, to give them proper and timely treatment. In this connection, we regret very much to note that the only means of detecting cases of leprous children has been abandoned by the Government, we mean, the Medical Inspection of School Children. If prevention rather than cure of diseases is the pivotal policy of the Government, then medical inspection of school children ought to top the list of all Health Reforms. Alongside with it comes compulsory Health Education in schools. On this point, we would like to commend to the Hon. the Health Minister the observation made by Dr. A. W. Stopford Thomson, M.B., D.P.H., Senior Asst. School Medical Officer, Litchamshire County Health Dept., reported in *The Medical Officer* dated 19th March '38, about 'Health Education and School Child': "A glance through the school annual reports for the last ten

years would suggest that education in Health is not a very prominent part of the curriculum at present. More than that—one is left with an uncomfortable feeling that it is rather like a duckling in a brood of chickens—a foreigner not fully understood, tolerated, but, on principle not to be encouraged...The trouble with the re-organisation of education is that while there is general agreement that 'something should be done', each of us has his own particular pet subject, which we hold, should be given prominence before all others. Most people will agree, however, that if education is to be more closely related to life, the promotion of health should be a prior consideration. I have spoken to teachers who readily agreed that hygiene is an interesting and useful subject of study but said that their time tables were so overcrowded already that they were unable to give it the attention it deserves. This is faulty reasoning. The place for health education should be decided first and the other subjects be fitted in afterwards. In this year of grace, I am not likely to be called a fanatic for being dogmatic upon this point."

We hope our Health Minister, will, in consultation with the Education Minister, soon evolve a scheme for the compulsory teaching of Hygiene in Schools, without which no Health Reform will be complete.

Lastly, with regard to Rural Medical Relief, the Hon. The Health Minister prided himself in saying that Rural Uplift was ever before the Govt. and that they had done all they could in this direction and added that the Govt. had provided 75 village hospitals and appointed 150 Midwives. He would admit that there were more villages requiring attention. But when it was realized that they had done so much in one year, it should help them to have faith in Govt.

We should not omit to mention, before closing, 'The Co-Operative Health Scheme' that has now been launched in 8 centres as an experimental measure. Our readers must have been conversant with the scheme and criticisms for and against have already been expressed by Medical Associations and individual medical men. In our own opinion, the subsidized Rural Medical Practitioners' scheme is the best, taking all circumstances into consideration and no less an authority than Major General Sir Cuthbert Sprawson, C.I.E., I.M.S., Director General of Indian Medical Services (Rtd.), and for some time also Surgeon General with the Govt. of Madras, has eulogized the scheme in no uncertain terms. As finance stands in the way of extending this scheme, an alternative has been devised in the Co-operative Health Scheme. This

is on all fours with the Health Insurance scheme of England and is the next best that the Govt. can think of. There are, however, certain lacunæ in the scheme, which should be filled up before it can have the stamp of public approval. They are :

- (1) The flat rate of 2 as per mensem makes no distinction between the rich and the poor. The richest mirasdar in a village is made to pay the same amount as the poorest 1 acre landholder, which is very hard on the latter. A graded scale would be preferable.
- (2) There is no provision for a midwife. We find even the Travancore Co-operative Health Insurance scheme, evidently based on this model, provides for a midwife. This is a serious omission which should be supplied.
- (3) The landless agriculturists have been left in the lurch. They should be treated free and their employers or if they are unattached labourers, Local Boards must meet their medical expenses.
- (4) The amount should be collected by the Revenue authorities, annually along with the kists, otherwise there would be lapses and the Medical Establishment would have to suffer on that account.

We have nothing but admiration for the doings of the Health Ministry in such a short time as 20 months, in the field of Public Health and we hope, it will continue to do its useful work unhampered, and thereby earn the lasting gratitude of the Medical Profession and the Public.

GLEANINGS FROM MEDICAL PRESS

Surgery

Disturbance of the Sexual Functions Following Head Injuries.—Disturbances of the sexual functions, of which the study is habitually neglected, are frequent in association with cranial injuries, after serious fractures as well as when the injury is less severe, and even after violent blows on the head.

Besides the pronounced signs of cerebral disturbance there are others that may be noticed such as vaso-motor manifestations, and in women menstrual disorders as well as disturbed

sleep. One notes also a difficulty in micturition, sometimes slight retention of urine. There are crises of sweating, of disturbed metabolism of carbohydrates, rarer still somnolence, disturbed metabolism of fats and of proteins, etc.

Systematic interrogation regarding the sexual functions furnishes important evidence.

In young subjects of less than 40 years, the disorders are rare and slight, whilst after this age the disappearance of the ejaculations is almost the rule, and that of erections is very frequent.

It has been noted that in certain instances the shock appears to have had contrary effect, acting as a stimulant to the sexual functions up till then deficient.

Sexual disorders may make their appearance several years after the injury, even although the injury was attended by considerable general disturbance.

A grave accident in early life, *e.g.*, at the age of 15, may inhibit the development of the sexual functions.

Attempts at treatment with testicular hormones and with psychotherapy have proved ineffectual.—*Medical World*.

Practical Methods of Examination of the Ear.—An aural examination should always be preceded by a general one (colour of skin, rocking while walking or standing, cranial nerves, reaction of pupils, etc.). In otoscopy the primary rule holds : source of light, ear, and eye must be on the same level; the light somewhat above the head of the patient. The auricle must be drawn backwards and upwards; with sucklings, on the other hand, downwards. A small perforation of Shrapnell's membrane may easily be overlooked if we do not pay particular attention to this small region above the short process of the malleus. Overlooking even a very slight middle ear suppuration (which may, moreover, be the cause of an eczema of the auditory canal that has been existing for years) may endanger the very life of the patient under certain conditions. The most important rule for the practitioner is not to flush the ear, especially after a trauma, when a dry lesion of the tympanic membrane may be present! Sometimes a peculiar sort of crepitation (spasm of the tube), which may disturb the patient very much, is audible; furthermore, pulsating aneurysmal murmurs near the temporal bone may be heard by placing the stethoscope on the mastoid process or a rubber tube into the ear; finally, the

presence of a tympanic perforation may be noticed on making Valsalva's test (patient presses air into the nose, keeping his mouth closed and holding the nose closed).

Some of those affected with middle ear "hard of hearing" (especially otosclerosis) speak very low because the transmission of their cranial bones is increased so much that they hear their normal voices as a roar. Others with inner ear hard of hearing speak very loud, because they lose control of their speech. Defective pronunciation of the "ess" sound also points to the inner ear. The sense of smell may also furnish important clues: foetid or cheesy odour of the secretion, which does not disappear after repeated flushings with H_2O_2 and dabbing the auditory canal with a 2-5 per cent. solution of $AgNO_3$, is indicative of a bone disease, perhaps cholesteatoma; a foetor of the secretion arising in the acute stage points to tuberculosis; garlic, introduced because of a toothache, easily betrays itself in the auditory canal. Palpate at the end of the examination. Sometimes the affected ear feels warmer than the healthy one.

With external otitis the following are painful: pulling the ear, pressing the tragus, or the membranous auditory canal from behind, or the glands under the ear (in childhood they may also hurt with otitis media) and in front of the head of the mandible; with infants, pressure on the auditory meatus may also be painful with otitis media. Pains on moderate pressure on the point and the posterior margin of the mastoid process, as well as on the planum mastoideum are indicative of mastoiditis. Normally pressure on the point of the process is also painful, but especially with otitis externa because of the regional lymph glands. The mastoidal tenderness first becomes a surgical indication if it lasts longer than the first week of an otitis media, or if it reappears in its further course. Its absence does not prove anything because destruction of the bone in the interior of the mastoid may be present, when the cortex is thick, without affecting the bone lying directly under the periosteum. In cases of occipital neuralgia the tenderness is located more to the back. Painful glands at the mastoid almost never indicate mastoiditis, but rather lice. When fever exists after an operation on the ear, a lymphadenitis in the upper region of the sternocleidomastoid points to incipient erysipelas.

When lacking a noise-producing apparatus, the hearing can be tested by plugging the other ear with a moistened finger-tip and rapidly moving the latter to and fro by beating on a sheet of paper lying against the ear. For most occupations it suffices

if each ear is capable of hearing a whisper at a distance of 6 metres. Only words suited to the intelligence of the examined should be used in the test. Distinctly decreased acuity for high tones (*e.g.*, bells), sibilants and high vowels (*e.g.*, sits, beats) is indicative of inner ear hard of hearing; decrease in acuity for low vowels and nasal sounds (*e.g.*, mother, murmur, hundred) indicates middle or external ear hard of hearing. With otitis media or favourable prognosis hearing is improved by politzerization, with otosclerosis mucosal otitis it is not. Practically, an irritating condition of the vestibular apparatus can be excluded if dizziness, spontaneous nystagmus and disturbances in equilibrium are absent.—*Wien. med. Woch.*, No. 41, 1937—*The General Practitioner*.

Medicine and Therapeutics

Magnesium Trisilicate in the Treatment of Peptic Ulcer.

—C. J. Tidmarsh and R. G. Baxter (*Canadian Medical Association Journal*, 39:358, Oct. 1938) report the treatment of 26 cases of peptic ulcer and 10 cases of functional indigestion with magnesium trisilicate, using a preparation that conformed to the standard requirements of Mutch. Of the 26 ulcer cases, 22 were duodenal ulcer, 3 of them complicated by massive hemorrhage, and 4 were gastric ulcer. The ages of the patients varied from twenty-four to sixty-five years with a large majority in the third and fourth decades of life; the duration of symptoms varied from several weeks to fifteen years. In the ulcer cases, treatment consisted in a liberal bland diet with intermediate feedings of milk every two hours from 8 a.m. to 10 p.m., for six to eight weeks; in the cases complicated by hemorrhage the full diet of the Meulengracht regimen was used. A low-residue diet was employed in the cases of functional indigestion. In all cases doses of one dram of the magnesium trisilicate preparation, containing 35 gr. of the trisilicate, were used, and the number of doses to be given daily determined according to the degree of gastric hyperacidity and the clinical progress. Thus at the beginning of treatment patients with ulcer were given 6 drams daily—in 2 cases, 8 drams. Under this treatment the ulcer pain diminished in frequency and severity within forty-eight hours. After the more, severe symptoms disappeared, the dosage was reduced to four drams daily. Treatment was continued until gastric analysis and the x-ray showed no evidence of ulcer. If high acidity continued, patients were

advised to take 2 to 3 drams of the magnesium trisilicate daily. Of the 36 cases 22 were completely relieved of symptoms, including 16 ulcer patients and 6 with functional indigestion; 10 showed marked improvement, including 3 cases of functional indigestion. There were only 4 cases in which the treatment failed—3 cases of duodenal ulcer, and one case of functional indigestion—the latter complicating heart disease. One of the patients with duodenal ulcer that failed to improve subsequently came to operation; a large duodenal ulcer penetrating into the pancreas was found. Six ulcer patients who were nervous and showed marked pylorospasm and spastic colon were given a special mixture containing $\frac{1}{100}$ gr. of atropine sulphate and $\frac{1}{8}$ grain of phenobarbital to each dram of the magnesium trisilicate preparation.—*Medical Times*.

The Anemia of Alcohol Addicts.—A. Bianco and N. Jolliffe (*American Journal of Medical Sciences*, 196:414, Sept., 1938) report a study of 184 cases of alcohol addiction. In 159 cases there was some complicating condition—polyneuritis, pellagra, “alcoholic” stomatitis, “alcoholic” encephalopathy or cirrhosis of the liver; in 25 cases none of these complications and no other “recognized stigmata” of chronic alcoholism were present. Definite anemia was found in 61 per cent. of the complicated group, but not in any of the uncomplicated group. However, macrocytosis was found in about 50 per cent. of both groups. In the complicated group, the macrocytic anemia was found not only in patients with pellagra and cirrhosis of the liver, but also, and with almost equal frequency, in patients with polyneuritis, stomatitis and alcoholic encephalopathy. No correlation was found between the frequency of macrocytosis and the occurrence of achlorhydria, the severity of liver damage, or the presence of an enlarged liver. These findings indicate that the macrocytosis and macrocytic anemia of alcohol addiction are not due to the inability of the liver to store a hematopoietic principle but rather to an extrinsic deficiency of some necessary hematopoietic substance. It is now generally believed that such diseases as pellagra, polyneuritis and cirrhosis of the liver occurring in chronic alcoholism are not due to the direct action of alcohol, but to some associated factors, and especially to the irregular and usually “definitely inadequate” diet that results from frequent and prolonged drinking. Such inadequate diet

most probably also involves a deficiency of the extrinsic hematopoietic principle necessary to maintain normocytosis.—*Medical Times*.

Thiocyanate Therapy in Vascular Hypertension.—Edward Massie, C. B. Ethridge and J.P. O'Hare (*New England Journal of Medicine*, 291:736, Nov. 10, 1938) report the treatment of 14 cases of vascular hypertension with sodium thiocyanate. All of the cases treated were uncomplicated hypertension with good cardiac and renal function; in one case a second study was made after a year's interval. Before the administration of the thiocyanate was begun, there was a control period of three months during which “all direct therapy” was discontinued. Then the sodium thiocyanate was given for three months; and the patients were under observation for a subsequent period of three months. The sodium thiocyanate was given by mouth in such dilution that 4 c.c. of the preparation used contained 0.2 gm. of the drug. In some cases treatment was begun with a daily dosage of 0.8 gm. for the first two days, reduced to 0.6, 0.4, and 0.2 gm. on consecutive days thereafter; in other cases the initial dose was 0.6 gm. for four or five days, then reduced to 0.4 and 0.2 gm. In every case the dosage was determined according to the cyanate content of the blood. The optimum level of the blood cyanate was found to be between 5 and 7 mg. per cent. In all cases there was a definite lowering of the blood pressure during the period when the sodium thiocyanate was given; the average fall in systolic pressure was from 66 to 21 mm., and in diastolic pressure from 33 to 8 mm. This was accompanied in 12 patients by definite relief of symptoms, especially of headache, nervousness and vertigo. Toxic symptoms were usually slight—a transient feeling of weakness and occasional mild epigastric distress; in one patient, however, marked weakness with nausea and vomiting occurred, and another had three attacks of angina pectoris. The authors are of the opinion that thiocyanate therapy is of definite value in hypertension, if dosage is controlled by blood cyanate determinations and patients are carefully selected; but the clinical applications of this therapy “is somewhat limited by these requirements.”—*Medical Times*.

Pineal Therapy in Pre-menstrual Derangements.—Pre-menstrual derangements of various kinds are well known, and for a few days before the menstrual flow begins migraine, skin disorders (acne, urticaria, Quincke's oedema), pains in bones and

joints, "aching all over," neuralgia, mental depression, may make their appearance.

An extract of the pineal gland (epiphysan) has been tried by R. Hofstaetter of Vienna, in order to ameliorate these troublesome complaints.

The treatment consists in three to five injections of epiphysan, made five or six days before the onset of the menstruation.

The first two days of the treatment 1 cm.³ epiphysan is injected, from the third day 2 cm.³, and one may increase up to 3 cm.³

After four or five menstruations, if treatment be still necessary, one may inject doses of 5 cm.³ at one time, on condition that the doses of 2-4 cm.³ are well borne. In general after a few months the dose is diminished.

Good results are claimed in cases where neither anterior pituitary nor ovarian preparations had brought any amelioration, in particular in the cases of migraine, of asthma, of skin affection and "rheumatism," and in the pre-menstrual fever of the tuberculous, observed in young women sexually hyperexcitable.—*Medical World*.

Early Diagnosis of Schizophrenia.—As in all branches of medicine, the characteristic symptoms are not always the most impressive ones. *W. Mayer-Gross* (Maudsley Hosp., Univ. of London) suggests that the diagnostic importance of the symptoms may be arranged in the following order: (1) Loosening of associations (for this reason Bleuler calls it schizophrenia). For example, a young man leaves the following note: "If I should return during my absence, please detain me here until I return." Indeed, at the beginning one first recognizes the disorder after the patient tries to repeat a story just told to him, or explain the meaning of a proverb, etc. (2) Disturbances of will can first be detected on questioning the patient. He maintains that he is no longer master of his will, of his thoughts, his actions; he feels that he is under the influence of others, that he is hypnotized, etc. The disturbance may begin by the patient being unable to reach a decision in things one meets in everyday life, in choosing a profession, in marrying, and similar phenomena. This weakness of will-power may be interrupted by outbreaks of activity, absurd impulses, negativism (unfounded opposition), and ambivalence (going from one extreme to the other.) A striking change in the patient's behaviour is decisive. (3) Affect anomalies; marked coldness of the feelings toward

relatives and friends, anxiety and outbreaks of anger leave an impression that lasts for a long time. Some patients notice the change in feelings and complain about it; many, however, deny the very fact that there has been any change and find nothing strange in their unkind and tactless behaviour. Contrary to a common neurosis, this condition often makes it impossible for the physician to establish normal contact with the patient. The psychosis may also commence with great anomalies in the reactions to the feelings, for example, with unfounded cheerfulness, unaware cruelty (some murderers are incipient schizophrenics!), fits of passion over nothing at all, and so on.

The most important positive signs are (4) autochthonous illusions (delusions), the wrong judgment of things, originating in the patient himself, the sudden arising of abnormal convictions based on insignificant impressions. Thus, for example, the patient says: "I came into the cafe. There were three white tables there; as I noticed them, I suddenly realized that this means the end of the world." Or: "It was eight O'clock; from then on I knew that I must pay more attention to religion or 'something terrible, 'something frightfully important' will happen." The patient is prepared to sink into mystic convictions or into an atmosphere of false interpretations. For the diagnosis it is unimportant whether the affected clings to these delusions or not. (5) Catatonic symptoms (anomalies of movement) are also found in "organic" psychoses, especially with encephalitis lethargica and disturbances in consciousness. Stereotypy, mannerism, fixed attitudes are no longer early symptoms. At the commencement one often finds prestuporous behaviour, a certain poverty of movement. The patient seems to be sleepy, but he is only inhibited in movement and speech. (6) Hallucinations are observed in all forms of mental derangement. At the beginning of the condition they are rare. Particularly hallucinations of hearing, while fully aware, are typical of schizophrenia. The patient lacks understanding although he can distinguish between real and false perceptions. Here, too, the "cleavage" is typical: the hallucinating patient who seems to be lost in a world of delusions simultaneously notices every detail of his real surroundings (double orientation).

The fact that the patient does not find his absurdities remarkable but rather natural, rational and understandable is diagnostically significant. Finally, autism, turning away from reality, should be stressed. The patient rejects everything that could

interfere with these delusions. He feels superior to everything that really concerns him. And in this very fact the key to an early diagnosis may be found. (Brit. Med. J., No. 5, 1938.)—*Ars Medici*.

Insulin in Non-Schizophrenic Mental Disorders.—Prof. J. E. Staehelin (Psychiatric Clin., Basle Univ.): Symptoms of hypoglycæmia (hunger, weakness, relaxation, inability to concentrate, difficulty in recollecting up to vacancy of mind; stupor, sleep, etc.) are found to be agreeable by anxious or excited psychopaths and neurotics; they also make them more accessible to psycho-therapy. The following have most beneficial effects on the mental state: increase in appetite and weight, better sleep (which permits reduction or discontinuation of soporifics), lowering of sexual overexcitability. In the course of an insulin cure there develop, as in schizophrenics, revival of the egocentric attitude or a peaceful, colourless disposition. In most cases these hypoglycæmic states of fatigue or sleep suffice. The patients can be awakened out of these conditions long enough to drink their sweet tea, thus making sugar administration by sound or syringe unnecessary. The peaceful frame of mind and the soothing of excited psychopaths achieved by injecting insulin in the morning, usually lasts throughout the afternoon and helps the patient fall asleep at night. Outbreaks of affects in imbeciles, epileptoids, etc., are prevented much better by insulin than by continuous baths, sedatives, working, and similar therapeutic measures. To this must be added that there is no getting accustomed to insulin; indeed, in most cases an increase in sensitivity sets in, thus making a decrease in the dose essential.

Insulin proved particularly efficacious in demorphinization, in treating alcoholism, addiction to hypnotics, and other toximaniacs. In vegetatively stigmatized persons the principal effect of insulin is tonicization of the parasympathetic, in morphine addicts and drunkards, raising the functions of the liver. Depressive climacteric women, whose conditions have declined much because of having used hypnotics and sedatives for years, are difficult to treat. With the aid of 2 to 3 insulin shocks weekly (up to a stupor and sleep; if necessary, to a coma for 30 to 60 minutes) the patient has progressed far enough at the end of 3 to 6 weeks to allow an extreme reduction of hypnotics, or substitution by pseudo-drugs or complete elimination. In depriving

morphine addicts the symptoms of abstinence are alleviated considerably. 20 int. units, 2 to 4 times daily, for 3 to 5 days mostly suffice; if necessary, real shocks once or twice a day up to coma. Attacks of delirium tremens are shortened and alleviated, even prevented, with the aid of 20 int. units 2 times daily. Alcohol intoxication can be aborted by 20 int. units intramuscularly. In chronic alcoholism tremor, disturbances in sleep, anxiety, etc., are quickly removed. More profound shocks should be avoided with alcoholics because of the frequently bad heart.

Attempts to cure hallucination in postencephalitic psychoses by insulin shocks failed at the clinic (contrary to the hallucinations with acute schizophrenia). Endogenic depressions (contrary to the exogenic ones of asthenic people) could not be abbreviated despite improvement of the general condition, but only assuaged. Two cases of Basedow-psychosis with depressive-catatonic signs, on the other hand, were practically cured after 7, respectively, 14 days of insulin (doses 60 to 120 units); the physical symptoms, too, improved considerably. In one patient severe stupor had to be provoked five times and coma three times. (Schweiz. med. Wschr., Nr. 42, 1938.)—*Ars Medici*.

The Treatment of Pertussis.—In his ward H. Zischinsky (Vienna) was able to test the value of the various methods of treatment in thousands of cases. Specific vaccines were tested in three fully equivalent series of patients. One series was given, according to prescription, vaccine four times at intervals of two days each, the second one physiological saline solution, the third one no injections at all. The disease took the same course in the three series. In marked whooping-cough the vaccine is not even of psychic value; nor is its prophylactic value proven. As little attention should be paid to the attacks of coughing as possible, particularly in the patient's home. If it is very distressing and the child shows signs of exhaustion because of vomiting, small doses of acedicon are most effective: for a little child, a quarter of a tablet at the most; older children get $\frac{1}{2}$ tablet. All other remedies (quinine, bromine, pertussin, etc.) have at best a suggestive value. Coughing is the best expectorant; sudden cessation of coughing due, for example, to a lesion of the brain causes congestion of secretion and cyanosis. Fresh air treatment day and night provides the best help and is most certain to prevent pulmonary complications. Attacks of coughing abate immediately; the children recover more rapidly. In bronchiolitis,

atelectasis, lobular pneumonia, include luke-warm baths (28° C.) and cold showers: in severe cases hot baths (38 to 40° C.) about every 2 hours, alternating with mustard baths, oxygen inhalation, lobelin, sympatol, and (to excite hawks) depressing the tongue or pressing against the trachea; cardiants and vascular agents are given early (digitalis, etc.). Pleuritic pains that interfere with the cough must, if necessary, be combated by morphine. If complications on the part of the central nervous system supervene (meningitis, sinus thrombosis, cerebral oedema), one or two leeches on each temple will sometimes save the life of the patient; for spasms, phenobarbital; if necessary, chloral hydrate for laryngospasmus, calcium bromide, phenobarbital, leeches on the temples. Particular attention must be paid to skin and oral hygiene. (Der Landarzt, No. 39, 1938.)—*Ars Medici*.

Obstetrics and Gynæcology

Neuritis of Pregnancy.—*K. W. Schultze* (Munic. Women's Clin., Wuppertal-Elberfeld) obtained excellent results in neuritis of pregnant women with vitamin B₁. During pregnancy there is increased requirement for almost all vitamins, so that various avitaminoses may develop. Pregnancy hemeralopia as a result of vitamin A insufficiency was frequently observed during the Great War; to-day it is a rarity. The same applies to vitamin D insufficiency, osteomalacia, a rather rare disease in these parts appearing in an abortive form, but still widely spread in Eastern Asia, particularly in times of famine. A deficit in vitamin E may play a role in sterility and habitual abortion, lack of vitamin C may be of importance for anæmia, hæmorrhages and inflammations of the gums. Enough work has not yet been done on these subjects, but we have had vast experience with disturbances due to B₁-insufficiency. It has been proven that this vitamin acts as a fermenting agent in the decomposition of sugars. If a deficit exists, pathologic quantities of lactic acid and pyruvic acid appear in the organism, especially in nerve tissue, as a sign of incomplete combustion of the sugar. This can be overcome within a few hours by the administration of B₁. The requirement for this vitamin is therefore extremely high when much sugar is consumed. In this latitude the classical form of complete B-insufficiency, beri-beri (oedema, cardiac insufficiency, polyneuritis, atrophy and induration of the muscles), is hardly ever observed. Marked polyneuritis is also rare hereabouts,

appearing now and then subsequent to hyperemesis, a condition in which there is almost no resorption at all of the vitamin.

Neuritis gravidarum, however, is not uncommon. In one year the writer observed it 60 times among 750 pregnant women, more frequently in winter, particularly at Christmas time. This is probably related to the diet at this time of the year and to the increased indulgence in sweets. The first symptoms are usually twitchings and indefinite pains in the arms or legs. They mostly set in at night and are accompanied by a feeling of stiffness of the affected extremities. In the arms they are often taken for rheumatism, in the thighs for sciatica or the result of pressure exercised by the pregnant uterus on the nerves of the true pelvis. Spasmodic attacks in the calves are frequent. In other cases there is a feeling of weakness in the legs; the women can hardly get up after sitting, they tire quickly when walking, have difficulties when sewing or writing; milkmaids can hardly lift the cans. The weakness usually affects a specific group of muscles, such as the adductors, the psoas group, the peronei, or the muscles supplied by the radial nerve. The pains are usually described as drawing or pricking, or "like clamps grasping the arm". They may become so intense as to be unbearable; the patients cannot sleep at night and lose weight rapidly. At other times there is a feeling of numbness or numbness, even up to complete insensitivity. Thus one of the writer's patients burned her thumb and index finger while cooking, and first became aware of this after having burned her little finger as well. In this case the median nerve was involved, whereas the ulnar nerve remained unaffected.

Vitamin B₁ has proved very efficacious for treating such a neuritis. The writer administered betabion and betaxin. In accordance with his experiences in the above 60 cases, he recommends the following plan of treatment: as a vitamin depot, one injects 20 to 30 mg.; this is followed by 6 mg. in tablet form daily for 3 to 6 days. This caused an improvement in almost every case. One now injects 10 to 20 mg. and reduces the oral dose to half the tablets. Under these circumstances, a third injection is hardly ever necessary. In severe cases it is better to treat by injections only: 20 mg. three times at intervals of 2 days. Larger doses are at times necessary. Undesired secondary effects were never observed, not even in the case where a patient was injected with 100 mg. of B₁ within 48 hours. Treatment by tablets exclusively, with which one never knows how much is really resorbed, is suitable in very mild cases, for after-treatment

or prophylaxis. Besides vitamin therapy, one must, of course, regulate the diet, limit the use of sugar and prescribe foods rich in vitamins (yeast, nuts, whole-wheat bread, veal). The treatment failed in but 5 cases (8.2 per cent). The future will show whether extremely large doses can be effective in such cases. It is obvious that not every neuritis during pregnancy can be cured in a short time by the administration of B₁; primarily those forms react favourably which developed as a result of B₁-insufficiency. However, the treatment has a favourable effect on other forms as well. Thus the writer observed neuritis of the tibial nerve during child-bed, which was definitely due to a trauma. It hardly reacted to hydrotherapeutic measures and warmth, but healed completely in 14 days with the aid of B₁ injections. If treatment with vitamin B fails, other causes of the neuritic pains must be considered. Pregnancy or child-bed tetany is the principal disease that must be considered in the differential diagnosis. Before the tetanic spasms set in, severe pains in the extremities and paræsthesias may develop. They result from parathyroidal insufficiency and have nothing to do with vitamin B₁. Deep varicose veins may also lead to pains and cramps of the calf. It is self-evident that less common causes of this type of pain (tabes, malignant tumours, etc.) must also be kept in mind. (Zbl. f. Gyn., No. 46, 1938.)—*Ars Medici*.

PROCEEDINGS OF CONFERENCES, SOCIETIES, ETC.

Association of Laryngology and Otology, Madras.

Annual report for the year 1938-39.

The Association was started in the month of April, 1938, with eight members on its roll, subsequently another member was enrolled making a total of nine members. Twelve monthly meetings were held during the year as detailed below :—

- 1938 April —Business meeting to frame the Bye-laws and rules of the Association.
- „ May —Paper on “Tonsillectomy and its Complications” by Dr. V. R. Kamath.
- „ June —Paper on “External Otitis” by Dr. M. Sanjeeva Rao.
- „ July —Talk on “Hoarseness” by Dr. N. Srinivasan.
- „ August —Clinical Meeting at E. N. T. Department of General Hospital, where Dr. P. V. Cherian and Staff demonstrated some interesting cases.

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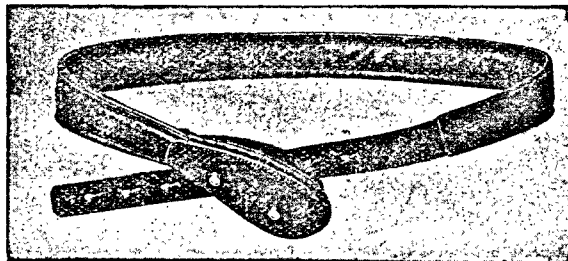
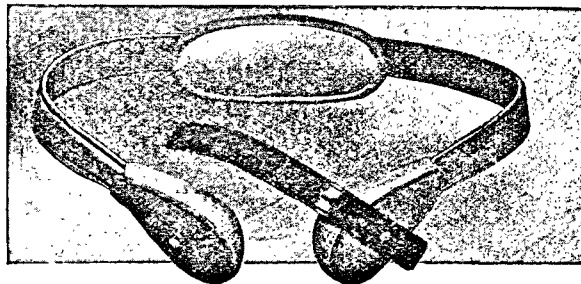
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MAY '39] PROCEEDINGS OF CONFERENCES, SOCIETIES, ETC. 429

- 1938 Sep. —Paper on "Emergencies in Ear, Nose & Throat, work" by Dr. P. V. Cherian.
 „ October—Paper on "Conservatism in Rhino-Laryngology" by Dr. G. P. Rayen.
 „ Nov. —Paper on "Pitfalls in E. N. T. Practice" by Dr. C. V. C. Rao.
 „ Nov. —Lecture on "Radiology in Diseases of Ear Nose & Throat" by Dr. P. Rama Rao.
 „ Dec. —Paper on "Otosclerosis" by Dr. N. Srinivasan, and V. R. Kamath.
 1939 January—Paper on "Clinical Problems in Middle Ear Suppuration" by Dr. P. Govindarajulu.
 „ February—Paper on "Relationship between Eye and Nose and Throat" by Dr. E. V. Srinivasan.
 „ March —Paper on "Rhinosporidium" by Dr. Jacob.

In addition to the above, one committee meeting and one general body meeting were also held during the year.

The accounts of the Association are as follows:—

Expenses:	Stationery including printing note-book, envelopes, etc.	Rs.	A.	P.
	Postage for sending appeals and complimentary copies to mofussil doctors	4	10	0
	Bill-collector for delivering notices	2	1	6
	Typing charges	2	8	0
		1	0	0
Total		10	3	6

Receipts:—Subscription from 9 members at Rs. 6/- each					54	0	0
Deduct Expenses					10	3	6
Balance on hand					43	12	6

The Association conveys its thanks to Dr. P. Rama Rao, Director of the "Radiological Institute" for his excellent lecture on "Radiology in Diseases of Ear, Nose and Throat" and for demonstrating very interesting Radiograph to the members.

The Committee also thanks the President, Dr. P. V. Cherian, for his enthusiastic support and ready guidance, and also all the members who contributed papers for clinical meetings, often at short notices.

The Tinnevely District Medical Association, Palamcottah

(Tinnevely Branch of the Indian Medical Association)

Monthly Meeting.

The monthly meeting of the Tinnevely District Medical Association was held at Tiruchendur, on Saturday the 29th of April 1939.

The meeting began under the Presidentship of Dr. P. Janardhana Rao, L.M. & S., at 6 p. m.

Dr. J. T. Moses, L.M.P., of Tiruchendur, exhibited a Case of Spleen on the right side.

Dr. K. Rama Ayyar, M.B., B.S., read out the following cases which were successfully treated at the Government Headquarters Hospital, Palamcottah :—

- (1) A Case of Tumour in the Right Shoulder (Sarcoma).
- (2) A Case of Myelitis.
- (3) A Case of Stone in the bladder.

Each case was fully discussed by the members. Thereafter, Dr. P. Janardhana Rao, L.M. & S., summed up the salient and important features of all the cases and their differential diagnosis in detail.

BOOK REVIEWS

The Law of Public Health in Madras Province—By C. V. Naidu, Advocate, Tiruvallur (M&S.M. Ry.). First Edition Demy size, 568 pages and 1100 Case Laws. Price : Rs. 5/- net.

This is an exhaustive treatise on the law of Public Health in Madras Province, intended for the edification not only of lawyers and medical men but also of laymen who come under the purview of this Act. This is a consolidated Act embodying the various provisions in the City Municipal Act, the Dist. Municipalities Act, the Local Boards Act, English Public Health Act etc. with certain additions and alterations made to suit modern conditions and to keep pace with the advancement of Medical Science. As failure to obey the provisions of the Act means prosecution and infliction of penalties provided for in the Act, a crop of litigation is sure to arise and this book clarifies the legal position under each section citing case reports and authorities on the subject. Thus, the lawyer is benefited, while the medical man or the layman can avoid unnecessary litigation by analysing his position by referring to the commentaries which are written in clear, non-technical style. The inclusion in this volume of "The Madras Prevention of Adulteration Act, 1918 and The Places of Public Resort

Act" and the Rules framed thereunder, being cognate matters, is specially to be commended, while 'The Table of Cases' cited and the index will go to facilitate reference and make the work of a busy lawyer or a medical man or an uninformed layman light and cheerful. The book is neatly printed and attractively bound and there is no doubt that the author has bestowed much thought and taken enough pains to make an otherwise boring and intricate subject, enjoyable reading. We heartily commend the book to our readers.

The Surgery of Pain—By Reni Leriche, Translated and edited By Archibald Young, B.Sc., M.B., C.M., F.R.F.P.S., F.A.C.S., M.D., Strasbourg, 1939, Illustrated, pp. xii+496. London : Bailliere Tindall and Cox. Price : sh. 21/-

Recent researches and advancement in surgery have made it possible to alleviate effectively pain by surgical means. The Great War has given an impetus for advanced knowledge of great many conditions and the outlook of surgery and surgical methods has entirely changed.

The author has come across good many cases of very painful casualgias following upon gun shot injuries, describes the role of the sympathetic in the causation of these painful conditions, and the great relief that may be obtained by performing operations on the sympathetic nervous system. In the various chapters he details at considerable length, but always in interesting fashion, how his new conception of the role of the sympathetic led him to the adoption of new and primary procedures in the treatment of many painful conditions previously regarded as intractable. We recommend the book to all our readers.

Medical Jurisprudence—By M. A. Kamath, M.B., & C.M., 1938, Fourth edition, pp. 343. Calcutta : Butterworth & Co., (India), Ltd. Price : Rs. 6/-

Since the publication of the previous edition in 1929, there has been an ever increasing accumulation of knowledge of the science, and the present fourth edition has been completely revised, rewritten, much new matter added, and many illustrative cases from author's own experience have been cited to make it up-to-date.

A new feature of the book is the addition of chapter on Workman's Compensation Act. This book has always been a good

guide and a timely friend of the students appearing for their final degree examination, and we hope that with the present additions it would prove very useful. We are sure that the edition would continue to be popular among students and practitioners.

The Students' Hand Book of Surgical Operations—By Sir Frederick Treves, Bart, Revised by Cecil P.G. Wakeley, 1939, Sixth edition, pp. 563 with 246 illustrations. London: Cassel & Co., Ltd. Price: sh. 12/-

There has been a considerable growth of surgical technique since the publication of the fifth edition in 1930.

New operations have come in vogue. Surgery of the sympathetic nervous system, newer operations on the Brain and spinal cord, operations on the skeletal bones for deformity, and joint affections have been greatly improved and perfected. All these have been included in this edition to meet the requirements of the students. There is no need to say anything about the nature of the book as it is too well known for the practical way in which it is written, and the surgical details that it contains. A few new illustrations have been included. The book contains a compact mass of practical information and every student of Surgery should possess a copy.

Jubilee Volume—Prepared in honour of Mr. L.E.C. Dapples, the President of the Board of Directors of the Nestles and Anglo-Swiss Holding Co., Ltd., 1937.

We are in receipt of the Jubilee Volume issued in honour of Mr. L. E. C. Dapples, President of the Board of Directors of the Nestles and Anglo-Swiss Holding Co., Ltd., on his 70th birthday. It has contributions by eminent doctors of the various nations.

The few English articles that are in this volume indicate to a great extent, the nature of the work contained in it. Dr. Eric Pritchard, late Medical Director of the Infants Hospital, London, writes on "Sweetened condensed milk in the scientific feeding of infants". Writing on Mr. Dapples, Dr. Pritchard says that to him more than to any other person must be attributed the credit of building up the great industry of milk preservation to which the world owes so much. He adds that this has enabled him to find the answer to a problem in infant feeding which he should have found extremely difficult to solve without its aid. He is of opinion, that the example with which this great

industrial undertaking has offered to the whole world of the possibility of preserving almost indefinitely so perishable a food as milk, has been instrumental in popularising the preservation of similar fresh foods during the season of their greatest abundance. The writer goes on to speak on the value of condensed milk in infant feeding.

Prof. Kay writes about the "Recent contributions to our knowledge of the Bio-chemistry of milk secretion"—the precursors in blood of milk constituents. Dr. John B. Sparks writes on "Crystallization of Lactose with special reference to the manufacture of condensed milk". Dr. Crocker writes on the "Methods used in milk industry for the Chemical and Bacteriological control." Dr. L. Murdoch McNab writes on "Manufacturing principles" and discusses about the various processes in the preparation of milk foods.

In addition to these which are in English, we find many other articles written in languages other than English by doctors of the various countries of the world. The book is well got-up and very well illustrated and will truly serve as a Homage by the collaborators of Mr. L. E. C. Dapples on his birthday.

A Treatise on Dispensing, Nursing and Hospital Emergencies—By Dr. Mohd. Rahmat Elahi Siddiqui, 1938, pp. 530, with illustrations. Allahabad. Price: Rs. 5.

The author who has spent a number of years as a lecturer to the compounders and practising physicians gives in this handy little book, a quick working knowledge in diagnosing and treating various surgical and medical emergencies.

The first 20 pages are devoted to a concise yet descriptive account of the art of dispensing.

Chapters X to XV deal with surgical practices and handling of surgical emergencies followed by a chapter on general nursing. Two chapters are devoted to treatment and management of medical emergencies. The last chapter deals with acute infectious diseases. The book ends with four useful appendices (i) table of Solubility (ii) Preparations of commonly used prescriptions; (iii) official and non-official preparations of some important drugs and (iv) Posological table (doses of official and non-official drugs).

This book should certainly prove a practical and useful guide for compounders and nurses in the discharge of their daily hospital routine. We recommend it to all the medical libraries.

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MEDICAL NEWS

Cameron Prize for Prof. Domagk.—At a meeting of the Court of Edinburgh University, the Senatus reported that the Cameron Prize for 1939 had been awarded to Dr. Gerhard Domagk, Institute for Experimental Pathology and Bacteriology, I. G. Farbenindustrie, Elberfeld, Germany, in recognition of his discoveries—the well-known Prontosil—which initiated the treatment of diseases of bacterial origin by compounds belonging to the sulphonamide group.

The Cameron Prize is awarded to a person who, in the opinion of the Senatus, has in the five years immediately preceding the award made any highly important and valuable addition to practical therapeutics.

The Dr. B. S. Shroff Memorial Gold Medal of the Bombay Medical Union.

The following subject has been selected by the Bombay Medical Union for competitive thesis for the above Prize for 1939:—

“Alcohol and Alcoholic Drink—Their use and abuse in India, with special reference to their effects on Health and Disease.”

The award will be in the form of a Gold Medal called the Dr. B. S. Shroff Memorial Gold Medal of the Bombay Medical Union.

The thesis, must be sent in six typed copies so as to reach the Honorary Secretaries, Bombay Medical Union, Blavatsky Lodge Building, French Bridge, Chowpatty, Bombay, on or before the 31st October 1939.

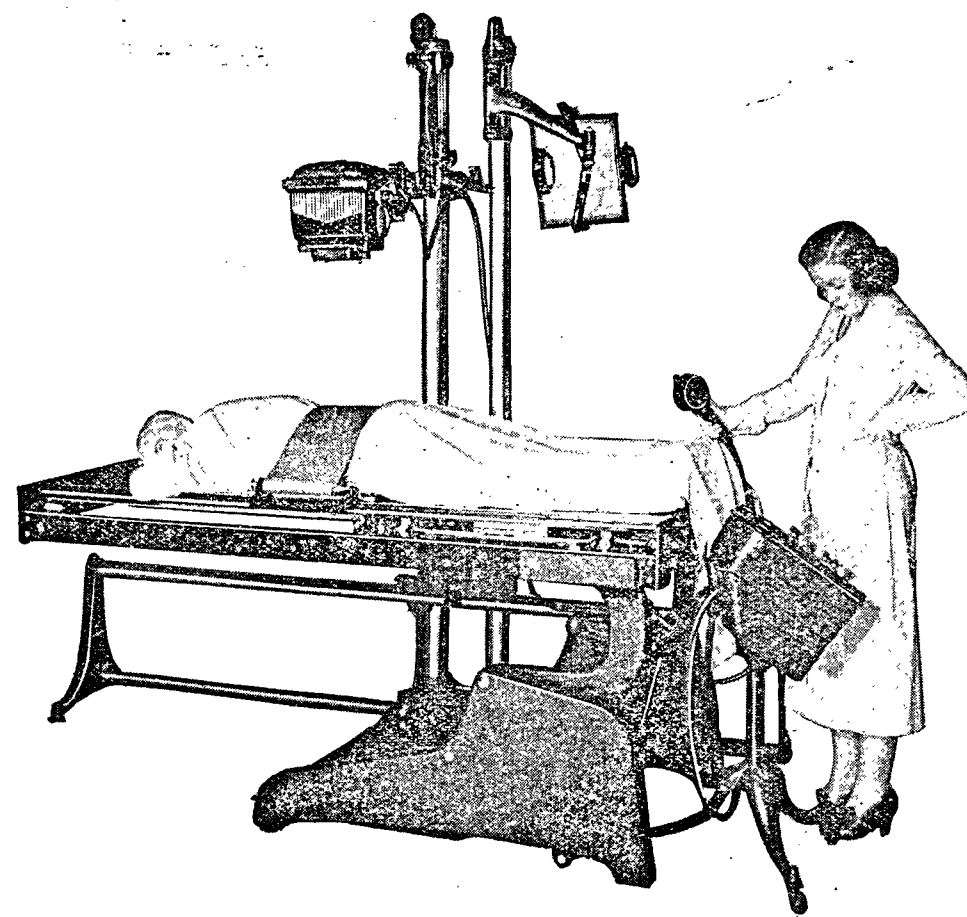
Further particulars can be had from :

Sorab J. Popat, M.B.B.S., K. S. Bharucha, M.B.B.S., Hony. Secretaries, Bombay Medical Union.

CORRIGENDA

“The Antiseptic” Vol. 36, No. 4:—

Page 303, 4th para—last line	for	‘aminograph’	read	‘aminogroup’
„ 309, 3rd para—last line	„	‘in view’	„	‘in vitro’
„ 318, 3rd para—8th line	„	‘if the smear is negative’	„	‘if the smear is positive’



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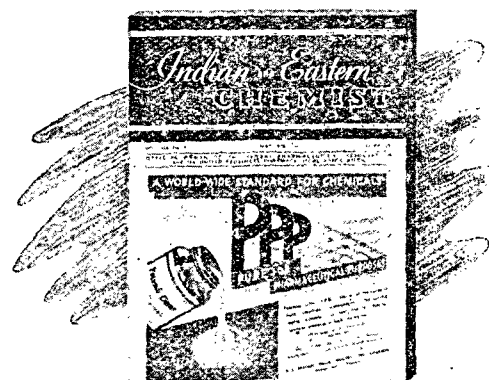
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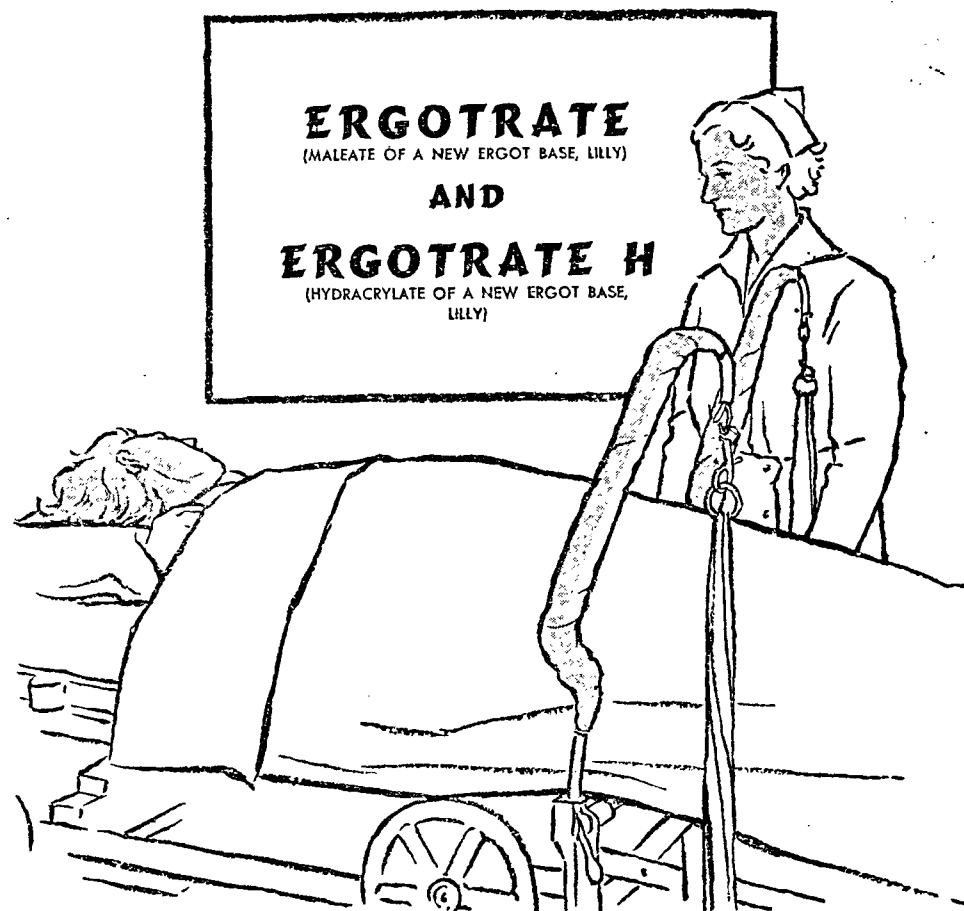
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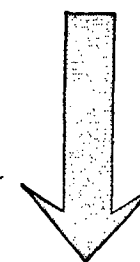
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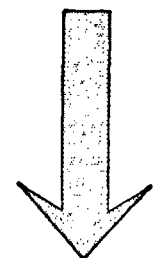


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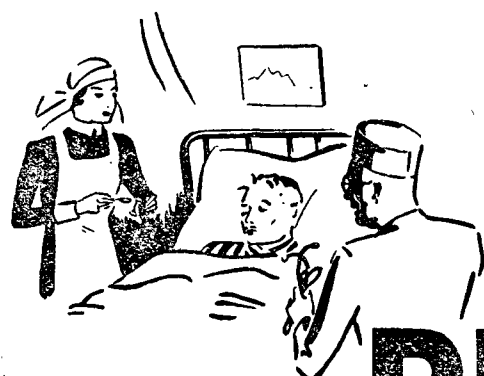
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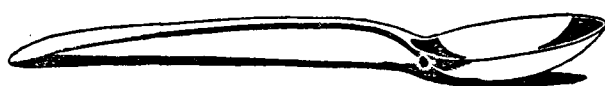
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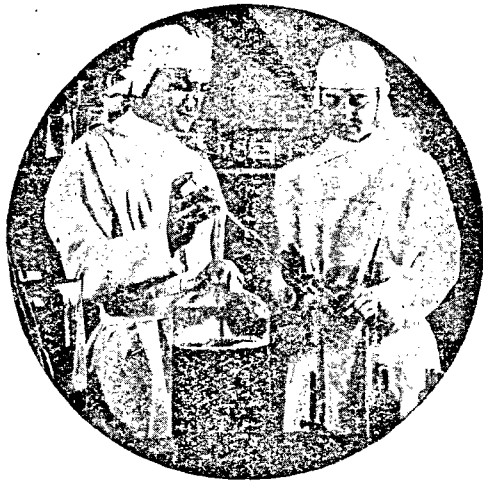
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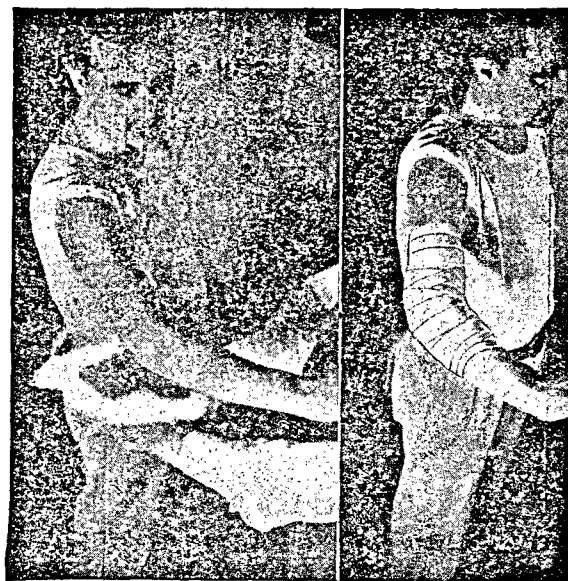
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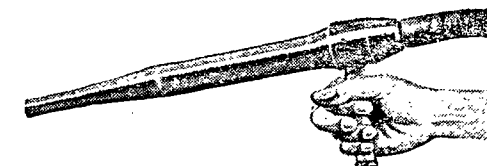
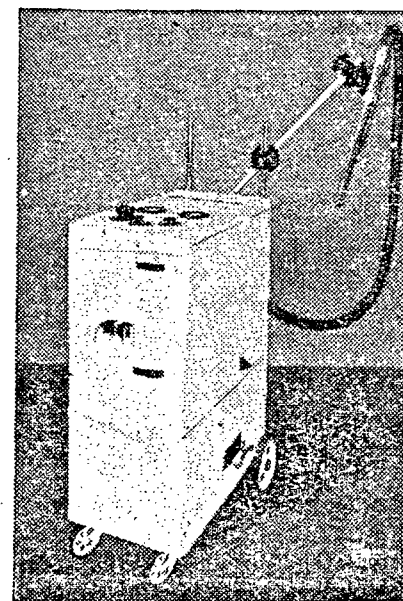
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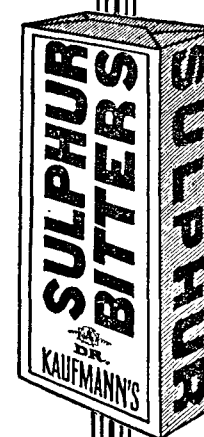
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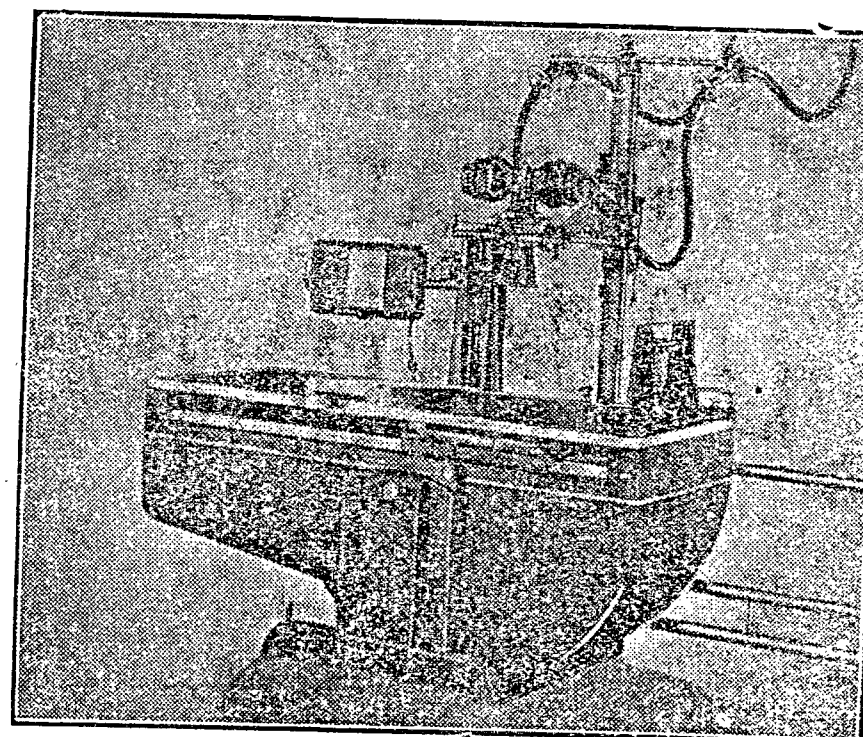


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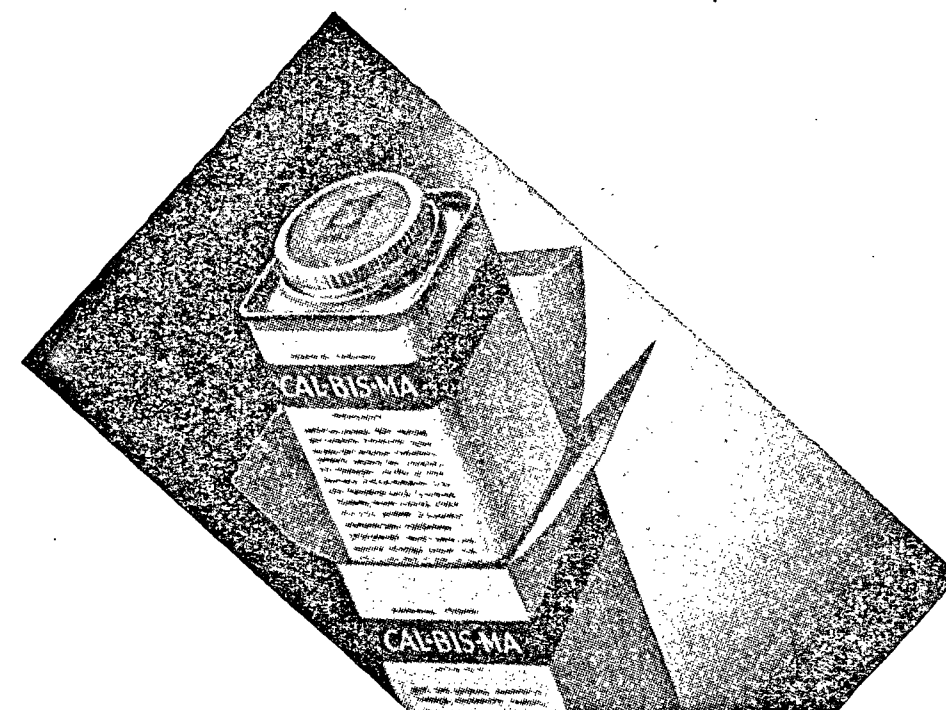
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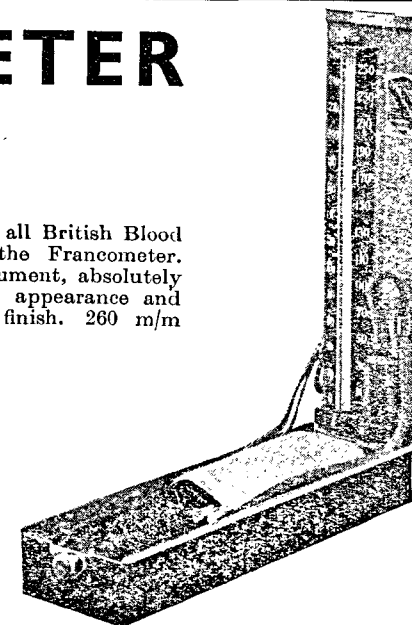
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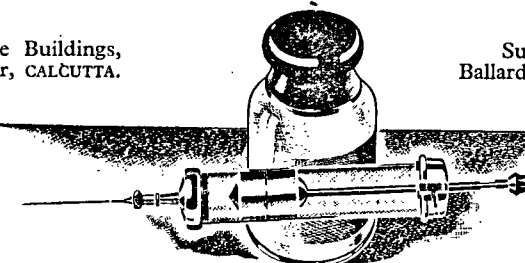
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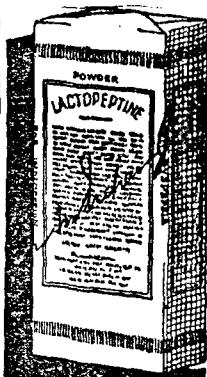
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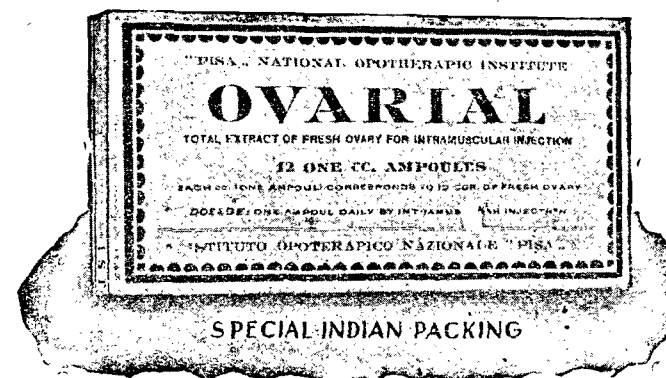
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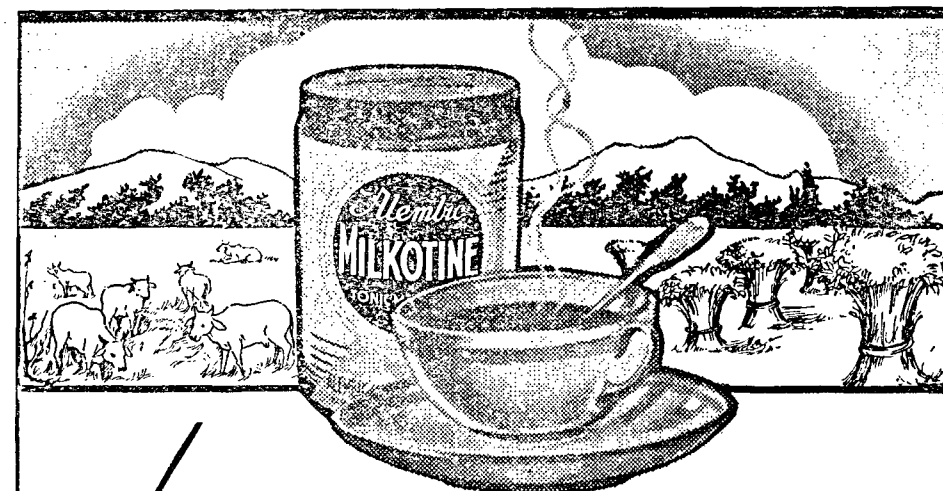
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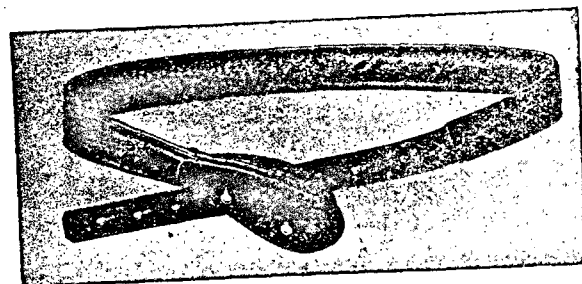
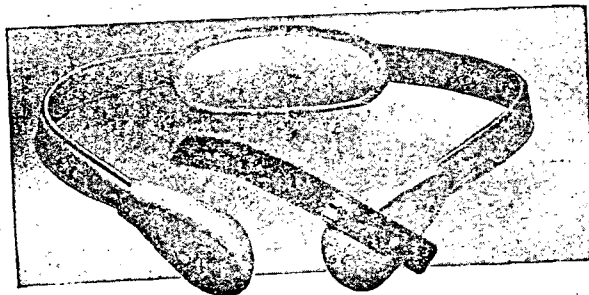
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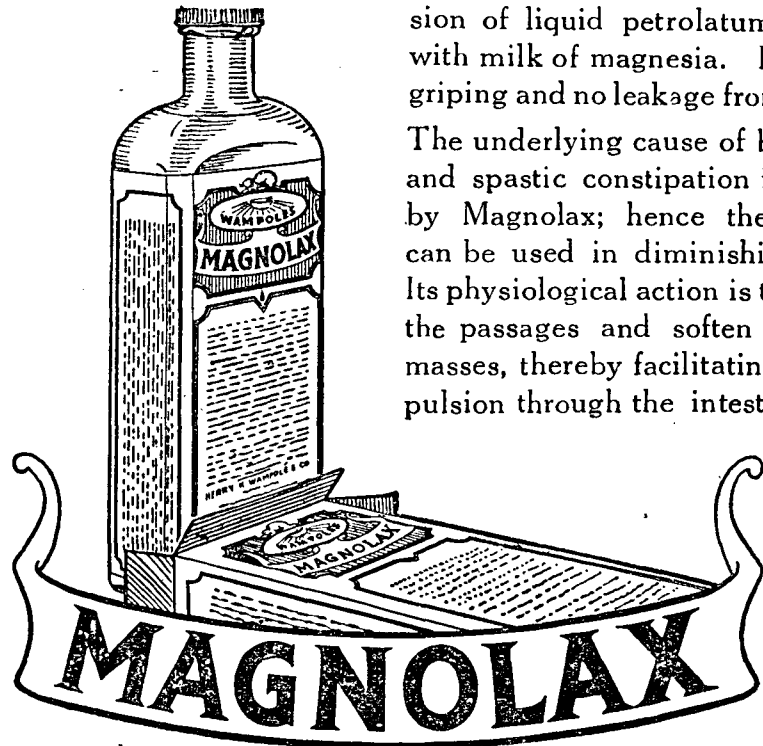
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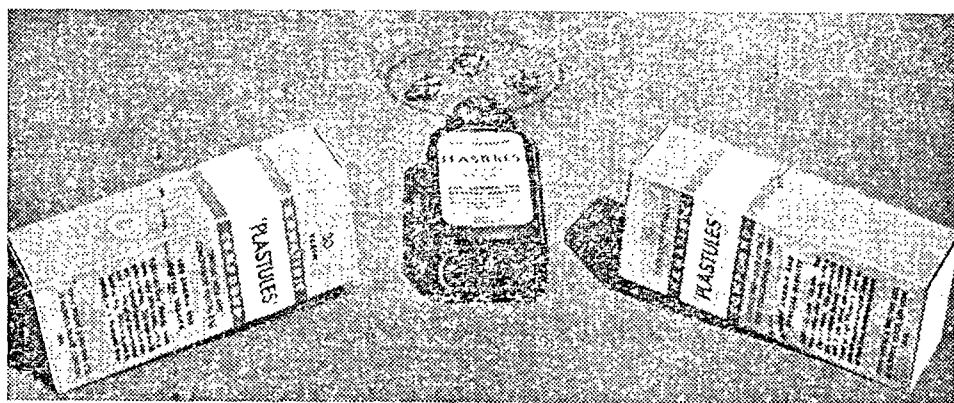
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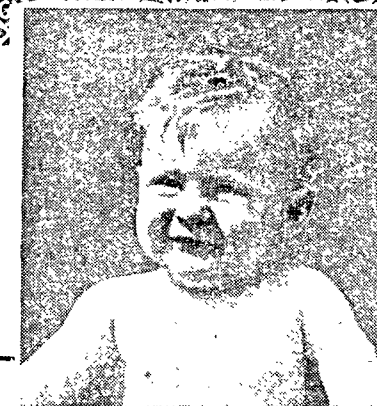
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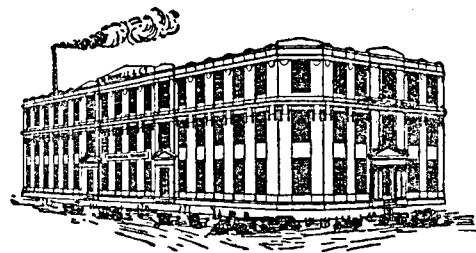
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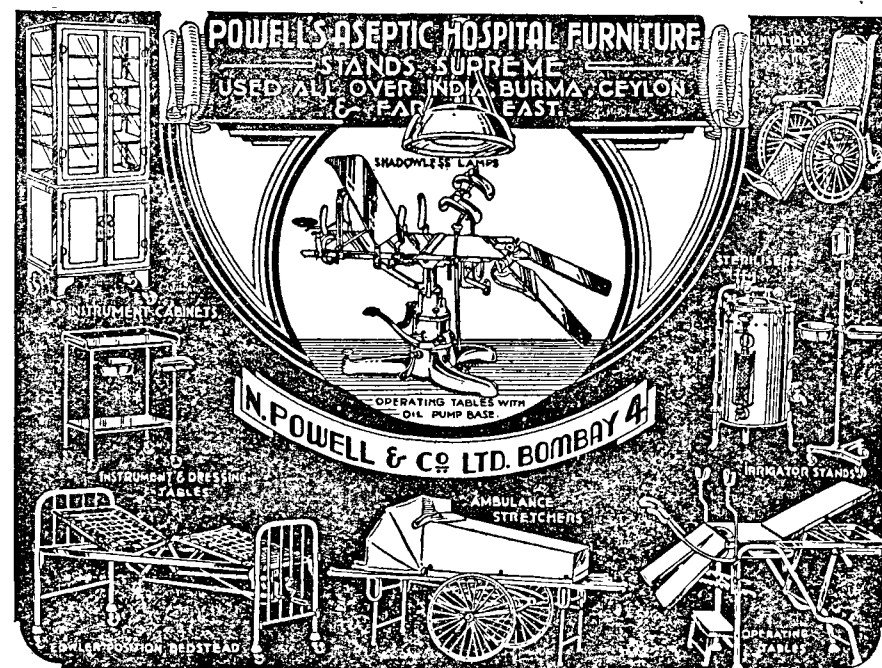
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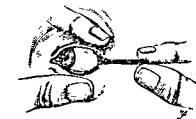
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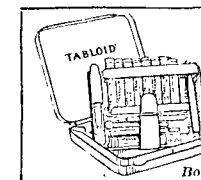
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JUNE, 1939.

No. 6.

Importance of Systematic Examination in Ophthalmic Practice

BY

Major S. Annaswami, M.B., B.S., D.O.M.S., F.R.C.S., I.M.S.,

Superintendent, Central Jail, Midnapore.

THE practitioner who wishes to include Ophthalmology in the scope of his practice should get into the habit of systematic routine examination of cases if he is anxious to avoid pitfalls in diagnosis. He should draw up for his guidance an exhaustive scheme of examination and should master the details of such a scheme by constant application in practice. Once the details are so mastered, he will be able to vary the scheme according to the needs of individual cases. A careful record of the findings should be kept. Rough sketches should if possible be made as they add very much to the value of the record.

Ophthalmic patients may be roughly divided into two groups:—

- (1) Those with manifest objective signs of disease, and
- (2) Those with only subjective symptoms.

In the first group, the manifest external signs may suffice to account for the patient's symptoms. In spite of this, it is necessary to make a thorough functional and internal examination in order to exclude all other evidences of abnormality. This

functional and internal examination may not be possible at the patient's first visit but it must not be omitted on that score. It must be done on a future occasion.

In the second group, where there are no manifest objective signs, after a rapid external examination, one can usually proceed at once to the functional and internal examination. [The best plan in these cases is to test central vision for distance and near, test field of vision by the confrontation method, test pupil reactions, examine tension, test roughly the condition of the extrinsic muscles and then proceed to ophthalmoscopic examination including retinoscopy. More elaborate examination of central and peripheral vision may be done later if it is found necessary as a result of the preliminary examination outlined above.]

The scheme of examination should be elaborate but at the same time elastic. It can be conveniently split up into the following six divisions :—

- I. Taking of History.
- II. General Examination of the patient.
- III. External Examination of the Eye.
- IV. Testing of motility and Muscle Balance.
- V. Functional Examination of Eye.
- VI. Internal Examination of Eye.

Let us now consider the salient points to be investigated under each of these heads.

I. Taking of History.—1. *History of present eye disease* :—Ask the patient what he complains of about his eyes :—*e.g.* Watering of the eyes, discharge from the eyes, pain, grittiness of the lids, photophobia, blurred vision, double or multiple vision, flashes of light before eyes, spots before eyes, etc.

Find out by questioning, how long he has been suffering from the complaint or complaints and whether there is any seasonal variation in the severity of the complaint. Do the lids stick, especially overnight? What is the character of the pain? How severe is it? Where is it felt chiefly? Does he suffer from headaches? If so, where is it felt most? Is the headache present all the time or is it worse in the evenings after use of the eyes during the day? Is there any vertigo? Has he had any recent injury to the eye or does he remember of any foreign body or insect having got into the eye? Has the patient been taking any

medicine containing Belladonna? Has he used any medicine in the eye?

2. *History of past eye disease* :—Has the patient suffered from similar or different eye disease previously? If so let him give brief particulars.

3. *History of any present or recent Systemic Disease* :—Does he suffer from any venereal disease or disease of the kidneys? Does he suffer from diabetes? Has he recently suffered from measles, influenza? etc.

4. *History of any past illness* :—Has he had any head injury? Has he suffered from venereal or any systemic disease? etc.

5. *Family History* :—This will give an indication as to whether the patient has any hereditary tendency to eye disease.

6. *Age and occupation*.

II. General Examination of patient.—1. Note general appearance of patient and position in which he holds his head.

2. Examine face and note if there is any facial asymmetry, facial paralysis, affections of skin like herpes, ophthalmic acne rosacea etc. Examine for stigmata of congenital syphilis.

3. Examine the accessory nasal sinuses.

4. Examine the teeth for evidences of congenital syphilis and rickets.

5. Examine the other systems, particularly the Nervous, Circulatory and Urinary systems. Examine for focal infections. Blood for W. R. if required.

This last under 5. may be deferred till later on in the examination.

III. External Examination of Eye.—Proceed in a systematic way from the superficial to the deeper parts. It is hardly necessary to mention that both eyes should be examined.

1. First examine the *lids*, and note their position, mobility, the width of the inter-palpebral fissure, whether there is ectropion or entropion, whether the skin covering the lids is excoriated and whether there are any chalazia. [Presence of small chalazia can often be detected more easily by gently running the fingers over the closed lids.] Note whether the lids close properly.

Examine the Lid Margins and note their form and outline. See whether the cilia are correctly placed. Note presence or absence of blepharitis and of stytes. Note whether the Puncta Lacrimalia are normally placed and patent. [Sometimes an eyelash may be blocking the lower punctum and may be causing epiphora.]

2. Examine the Lacrimal Sac for evidence of inflammation. Press over the sac and note if contents regurgitate through puncta. [Remember that the major portion of the sac lies below the level of the internal palpebral ligament.]

3. Examine the Preauricular Lymph Gland. This gland is normally not palpable.

4. Examine the Eyeball and note its size, shape, situation in orbit, and direction.

5. Next, examine the *conjunctiva*. Pull the lower lids down and ask patient to look up. In this way the conjunctiva of the lower lid and lower fornix is brought into view. Look for injection, follicles, papillas, scars, concretions, foreign bodies etc. Remember that a favourite site for minute foreign bodies is about 2 m.m. from the lid margin at about the middle of the lid on the palpebral conjunctiva. Look for evidences of subjacent chalazia.

Examine the ocular conjunctiva for injection, altered secretion, phlyctenules, chemosis, hæmorrhages, growths, degenerative changes, wounds, foreign bodies, etc. Embedded foreign bodies may be indicated by patches of soft granulation tissue over their sites.

If injection is present note whether it is conjunctival, whether it is chiefly posterior or anterior conjunctival. Remember that anterior conjunctival injection occurs chiefly in inflammations of the limbus and of the superficial layers of the cornea and that ciliary injection in its pure form occurs in deep inflammations of the cornea, inflammations of iris and ciliary body, and in glaucoma. [Remember that ciliary injection is distinguished from conjunctival injection by the fact that the latter consists of separate vessels which are clearly visible as bright red tortuous lines which branch in an arborescent fashion and which can be moved with the conjunctiva whereas in ciliary injection the individual vessels cannot be clearly distinguished, their colour is a purple red and that the vessels cannot be moved with the conjunctiva.]

Next, evert the upper lid and examine the conjunctiva of the upper lid and fornix. [The simplest way to evert the upper lid is to make the patient look down, place a match stick or thin glass rod on the outside of the lid above the tarsus and keeping a gentle pull on the lashes, turn the lid over.] The points to be noted are the same as in the lower lid.

Prepare a smear of discharge for microscopical examination for organisms, nature of cells etc.

6. The chief points to look for in the *sclerotic* are changes in curvature and colour, ciliary injection and evidences of episcleritis and scleritis.

7. Next, in the order of examination, comes the *Cornea*. Remember that the normal cornea is regularly curved with an even and polished surface, is perfectly transparent, is free from any vessels and is extremely sensitive. Keeping these points in mind, after noting the size of the cornea and its curvature, examine its surface carefully for any abrasions, ulcers, foreign bodies, vessels etc. The best way to examine the corneal surface is by making the patient follow with his eye the examiner's finger in different directions and by noting the character of the window reflex on the cornea. This in healthy cornea is regular and clear cut. As further aids to diagnosis, the use of oblique illumination and instillation of 2 per cent fluorescein may be used. Oblique illumination consists in focussing either day light or artificial light on the cornea by means of a strong convex lens and examining the spot so illuminated with the naked eye or better still through a corneal loupe. Areas on the cornea which are denuded of epithelium are stained green by fluorescein. Remember that a leash of vessels near the limbus may mark the site of a foreign body or a minute abrasion on the cornea.

Next, examine the substance of the cornea for opacities, foreign bodies, vessels etc. Here again oblique illumination and examination through the loupe will be found of great help.

When vessels are seen in the cornea determine whether they are superficial or deep [by keeping in mind the following points of distinction between them. Superficial vessels can be traced over the limbus into the conjunctiva, are bright red and well defined; branch in an arborescent fashion dichotomously and may raise the epithelium over them making the corneal surface uneven. Deep vessels cannot be traced beyond the limbus, are ill defined and dull red, run more or less parallel to each other and branch at very acute angles and do not affect the evenness of the corneal surface.]

Test the sensitivity of the cornea by touching it with a wisp of cotton wool twisted to a fine point.

Examine the posterior surface of the cornea by oblique illumination and loupe for any K. P. (Keratic precipitates.)

8. *Anterior Chamber*:—Note its depth. Remember that it is shallow in old age, glaucoma, dislocation of lens, hypermetropia etc., and deep in buphthalmia, iridocyclitis, posterior dislocation of lens, myopia etc.

Examine its contents. The normal aqueous is perfectly clear and transparent. Abnormalities to look for are hypopyon-hyphæmia, foreign bodies, dislocated lens etc.

9. The *Iris* should be examined next. Examine its colour pattern, and position. Look for adhesions, nodules (gummata and tubercles) and growths. Note whether it is tremulous. Look for holes especially at the periphery (Iridodialysis). The colour may be altered as a result of atrophy or due to the condition known as heterochromia. In this there is a difference in colour between the two irides or between parts of the same iris. It may be congenital or due to iridocyclitis. The pattern is obliterated in iritis. Tremulousness of iris (Iridodonesis) indicates shrinkage, dislocation or absence of lens.

10. *Pupil*:—Note shape and size, whether it is equal on both sides, whether it is centrally placed and whether it is quite black. Look for synechia. Test direct and consensual reactions to light and reaction to convergence. Note whether the reaction to light is sustained.

11. Only the anterior layers of the *Lens* can be satisfactorily examined at this stage by means of oblique illumination and loupe.

12. Examine the *Intra Ocular Tension*.

IV. Motility and Muscle Balance.—Test the range of movements of the eye. If any motor anomaly is present determine its character, extent and cause by an examination conducted according to a systematic plan.

V. Functional Examination of Eye.—This consists in testing the light sense, form sense, and colour sense.

Testing of light sense is difficult and may be omitted.

Form sense is examined by testing the Central and Peripheral vision.

Central Vision: In testing this, proceed according to a systematic plan. Analyse the patient's glasses if he is wearing any. Find out how long he has been using them and how often he has had them changed.

Take distant vision of each eye separately and also that of both eyes together. Use good charts at six metres. [If vision is less than 6/60, find out at what distance the topmost letter can be seen. e.g., V.R.=3/60. i.e., patient can read with right eye the largest type at 3 metres. If largest type cannot be distinguished at all, test with fingers and note at what distance they can be counted. e.g., c.f., at 1 metre. Failing this, see if

hand movements can be made out. Failing this also, note if there is P.L. i.e., perception of light. If no P.L. the eye is completely blind.]

Although 6/6 is taken as normal, most healthy young persons can see better than this, even down to 6/3.

Test manifest hypermetropia, i.e., find out the strongest convex spherical lens which the patient accepts.

Test near vision with Jaeger's or Snellen's types. Note what is the smallest sized print that he can read and also what is the least distance at which it can be read, e.g., J. 2 at 25 c.m.

Determine the range of Accommodation. i.e., difference between refractive power of eye when accommodation is exerted to its utmost and when it is at rest.

If there are no facilities for retinoscopy and if the case is obviously one of refractive error, subjective test of refraction may be done at this stage. But it is important that a systematic plan is adopted. Otherwise subjective refraction test is sure to prove very tedious and difficult.

If a cycloplegic has been used for investigation of refraction and if an error has been revealed, perform a post cycloplegic test with lenses at a future date if necessary.

Peripheral vision is tested in order to determine the state of the perceptive elements of the retina other than the macula which is of course responsible for central vision. The peripheral vision is tested by mapping out the visual field by any of the recognised methods. A rough but fairly useful method is the confrontation test. In this the Surgeon's hand or a square of paper held in a pen holder is used as the test object. In cases where the hand cannot be seen as in mature cataract, a lighted candle or the lamp of the electric ophthalmoscope is used as the test object.

A more accurate determination of the visual field is done if necessary with the perimeter and tangent screen.

A rapid test of colour vision can be made by means of Ishihara's charts, other methods are more elaborate and complicated.

VI. Internal Examination of the Eye.—For satisfactory examination of the deeper parts of the eye, a dark room, a good lamp and a really good ophthalmoscope—preferably an electric one—are necessary. It is also necessary in most cases to dilate the pupils. [A good way of getting rapid and satisfactory dilatation of the pupils is to instil one or two drops of a 2 per cent solution of Homatropine and cocaine, and keep the patient in a darkened room for about half to one hour with the eyes

bandaged.] If it is intended to perform retinoscopy, and if the patient is young it is advisable to paralyse the accommodation thoroughly by the use of cycloplegics. In the very young, the only sure way of attaining this object is by the use of 1 per cent Atropine drops or ointment three times daily for at least 3 days.

Internal examination of the eye should also be conducted in a systematic manner. The following routine should be followed:—

1. Examination with the ophthalmoscope by the *Distant Direct method*. In this the examiner holds the ophthalmoscope at a distance of about 25 c.m. from the eye of the patient and reflects light into the eye. By noting the character of the fundus reflex he can ascertain whether there is any gross error of refraction and whether the media are clear. He can often detect the presence of detachment of the retina. If opacities are present he should note whether they are fixed or floating. He can determine the location of fixed opacities by the parallax method *i.e.* by noting their displacement in relation to the pupillary plane when the eye is moved.

2. Examination with the ophthalmoscope by the *Indirect method*. [This is like examination with the low power of the microscope.]

3. Examination with the ophthalmoscope by the *Direct method*. [This is like examination with the high power of the microscope.] Start with a plus 20 or stronger lens behind the hole of the ophthalmoscope and gradually reduce the strength of the lens and if necessary bring in concave lenses of gradually increasing strength. In this way successive parts of the eye from the cornea down to the fundus can be examined.

4. *Retinoscopy*:—Internal examination with the ophthalmoscope, besides confirming some of the findings already elicited as a result of the external examination, enables one to investigate the following parts of the eye which could not be examined before:—(1) Deeper parts of the Lens. (2) Vitreous. Look for changes in consistency, opacities, foreign bodies, haemorrhages, exudates, persistent hyaloid artery, etc. (3) Fundus. The best order to follow in the examination of the fundus is optic disc, retinal vessels, macula, and finally the rest of the fundus. Examine the whole of the fundus systematically and thoroughly.

N.B.—If a mydriatic has been used to dilate the pupil of an elderly patient for the purposes of thorough investigation do not forget to see that the pupil is contracted by means of Eserine before the patient leaves the consulting room.

Some Common Errors in Diagnosis of Ocular Conditions

BY

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It does not require much imagination to realise how important it is to correctly diagnose ocular conditions and apply prompt treatment. I am sure every ophthalmologist has shared my experience of coming across cases of grossly mistaken diagnosis and delayed treatment, which may have caused the patient the loss of an eye. Even when the blindness is not complete, the functional impairment may be such as to produce severe economic loss. To cite only two recent instances in my practice:

A.J. is a retired Inspector of Schools. One night he got a severe pain and redness in his right eye. The local doctor took it to be a case of "Ophthalmia" and for some reason or other prescribed atropine lotion and on top of it put a bandage on the eye. The pain got worse and the patient noticed he could not see with that eye at all. After wasting a week, which might have made all the difference, he came to Patna and I found it was a book picture of acute glaucoma, with complete loss of perception of light. Iridectomy by dialysis relieved him of the pain, but the vision never returned.

An Engine driver, who had to have 6/6 and 6/9 vision, was rightly failed at the examination by his D.M.O. He came to consult me after having gone round to a couple of opticians who sold him four pairs of glasses but did not bring his vision up to the standard. I found the vision in one eye could not be improved beyond 6/18 due to corneal nebulae. Had that man been given prompt and efficient treatment when he had the superficial corneal ulcer he need not have lost his job.

The last example illustrates how a small scar on the cornea may result in such serious economic consequences. Yet, much bigger scars on other parts of the body may not cause any functional loss and are soon forgotten. Even a badly united fracture may not cause any more than a shortening of the limb and a deformity which may not seriously interfere with the man's vocation in life. How often is one hit with a piece of stone, but the very smallest chip getting in the eye may produce a hypopyon ulcer and result in blindness. I have said all this to emphasise how sensitive is this organ to inflammatory and traumatic lesions. It behoves the general practitioners, then, to pay

a little more attention to the study of ophthalmology in everyday practice.

It is not my object to give a learned discourse on differential diagnosis of rare ocular conditions. I have often told my undergraduate students that I would not mind if they did not diagnose a fundus condition, but I would never forgive them if they made a mistake in the diagnosis of commoner external ocular diseases. It does not matter much if they cannot use the ophthalmoscope efficiently, but they must learn to use their eyes and handle a lens and a loup. Since this paper is primarily meant for the general practitioners I shall confine my remarks to the commoner external ocular conditions and the very essential points in differential diagnosis.

To mistake a case of Glaucoma for Iritis is undoubtedly a very serious blunder which may result in the most tragic consequences. Yet it has happened so often, due undoubtedly to a careless and slipshod examination. The first thing, therefore, is to develop and practise the power of observation and follow a systematic routine of examination, structure by structure. Daylight is often deceptive and you must always be in possession of a good source of artificial spot-light.

There comes a patient to you complaining of acute pain in the eye and on inspection you find the eyeball red with watery or mucopurulent discharge and photophobia. You will immediately ask yourself:—

I. "Is it a case of **simple conjunctivitis**, of **Iritis** or of **Acute Glaucoma**?

You will now proceed, by a process of elimination, to arrive at your diagnosis. You may or may not find the *lids* oedematous or the *conjunctiva* chemosed. You will carefully observe if the conjunctival injection is diffuse and the individual peripheral blood vessels stand out prominently, with comparatively less congestion around the cornea, or that the circum-corneal area is much more congested than the rest of the conjunctiva and the individual blood vessels are not clearly discernible—what is technically called "Ciliary Congestion." The former will denote conjunctivitis and the latter iritis. In acute glaucoma you will usually find the lids oedematous and conjunctiva diffusely injected and chemosed.

The *discharge* in conjunctivitis will be mucopurulent; in iritis and glaucoma watery.

You will then observe the *cornea*. In conjunctivitis you

may not find anything more than aropy sort of mucoid discharge sticking to the cornea; in iritis the cornea is clear or very slightly cloudy; in acute glaucoma it is oedematous and diffusely hazy and practically insensitive.

The *anterior chamber* in conjunctivitis will be normal in depth; in iritis it may be slightly deeper; in acute glaucoma, it is very shallow.

Regarding its contents there may not be any abnormality, though sometimes you may see a plastic exudate or a hypopyon in certain types of Iritis.

Then coming to the *Iris*, you will observe its colour and texture and carefully notice the state of the *pupil*—its size, the regularity of its margin and its reaction to light. In conjunctivitis there will be no abnormality; In glaucoma you will find the iris practically normal in colour and texture, but the pupil though regular, will be widely dilated and oval and not reacting to ordinary light. In iritis, you will naturally find the signs of inflammation. The delicate pattern of the Iris will be blurred and its colour will be muddy, as if a smooth greyish smear has been spread on its surface. The pupil will be contracted and irregular and react very feebly to light or the reaction may not be elicited at all. It is important to ask the patient if any drops have been put in the eye, because sometimes you may find a moderately dilated pupil in a case of iritis when atropine has already been put, but then the irregularity of the pupillary margin will be emphasised. Similarly, with the use of miotics the pupil may have partially contracted in a case of glaucoma, but the margin will remain regular. The greenish haze in the wide pupillary area is often present in glaucoma, but too much importance is not to be attached to it.

Then you will put your fingers on the gently closed eyelids and test the *tension* of the eyeball. You will find it normal in conjunctivitis, normal or slightly raised in acute iritis; but almost stony hard in a case of acute glaucoma.

Pupil and tension, more than anything else, help to differentiate glaucoma from iritis. It is no use talking of nasal contraction of the field or of cupping of the optic disc, as students often do, because in a case of acute glaucoma successful perimetry or ophthalmoscopy is not possible. The rapid and marked diminution of visual acuity, coloured haloes round lights and general foggiess of vision are out of all proportion in cases of acute glaucoma. The nature of pain in the eyeball and headache are minor points of difference, but you hardly ever find the marked

symptoms of severe pain in the eyeball and vomiting except in a case of glaucoma.

Remember it is ever so important not to mistake glaucoma for iritis. You may easily be the cause of the patient getting blind through your negligence, because the line of treatment will be diametrically opposite in the two cases. While you must put atropine in a case of iritis to prevent serious complications, a single drop of atropine in a case of glaucoma may cost the patient his eye. What I have said above is a typical picture of these conditions. But there are cases when even a specialist may at times find it difficult to be sure of his diagnosis, specially when the glaucoma may be secondary to old iritis. Yet with careful observation and cautious line of treatment you will do no damage to the eye and refer such doubtful cases to a specialist.

Perhaps the next patient that comes to you will only complain of gradual failure of vision, with or without slight symptoms of discomfort in the eye. You will now ask yourself:—

II. “Am I dealing with a case of (i) **Incipient Cataract** (ii) **Chronic Glaucoma** (iii) **Optic Atrophy** (iv) Some **Choroido-Retinal Condition**, like retinitis pigmentosa or (v) of some **Error of Refraction**, like progressive myopia?

After a routine external examination, which may or may not reveal anything except perhaps a greyish reflex in the pupillary area, you will naturally proceed to record the vision and if there is an error of refraction you will discover it. The subsequent dark room examination by retinoscopy will exactly tell you if such is the case. Sometimes with advancing age a case of hypermetropia will become more and more “manifest”, or in a young boy the progressive myopia will account for the gradual failure of vision.

You must begin by taking a careful history and asking leading questions. If the patient suffers from night blindness and your subsequent fundus examination reveals signs of retinitis pigmentosa or some other fundus condition, you will eliminate choroido-retinal lesions.

A history of occasional foggiess of vision, discomfort, rapid onset of presbyopia, seeing coloured haloes round lights or of himself or other members of the family, having suffered from epidemic dropsy will give you a clue of chronic glaucoma, which you will follow up by doing tonometry and perimetry and looking at the optic disc.

It is essential to adopt a routine method of examination in

these, as in other, cases. After the external examination, proceed to record the visual acuity for distant and near objects by making him read the usual charts. Try at the same time what maximum improvement can be subjectively brought about through lenses. You will now record the field of vision and take the tension of the eyeball. If the tension is not high you will instil homatropin and cocaine drops three or four times every five or ten minutes and then take the patient to the dark room. You will first examine the eyes by oblique illumination, then do the retinoscopy, then adopt the distant direct method of examination and finally do the indirect and direct ophthalmoscopy. But supposing you are not skilled in these methods you may yet be able to arrive at certain conclusions which will enable you not to give the wrong advice.

I have seen dozens of cases where patients suffering from chronic glaucoma have been advised to wait till they completely lost their visions and then to get their eyes operated on for cataract. When such cases ultimately come to us we find that the glaucoma has become ‘absolute’ and there is no prospect of restoration of vision. Through sheer carelessness or gross ignorance of the ‘doctor’ who gave the advice, the man has to spend the rest of his life in darkness; while, if the doctor had only suspected the possibility of the case being one of glaucoma and not of incipient cataract and referred him to a specialist, a timely operation would have saved his sight. If it is only remembered that every case of gradual failure of vision in an elderly person is not necessarily due to cataract, such tragic mistakes will be less frequent.

In the majority of cases, a carefully elicited history will reveal facts which will make you suspect chronic glaucoma. History of occasional transient attacks of foggiess of vision and of having seen rainbow-like rings round lights, of headaches, of a vague feeling of discomfort in the eyes, of undue eye-strain during near work, and, in this part of the country, of having himself or some other member of his family suffered from epidemic dropsy, will give you sufficient clue. On the other hand, if you find that the eyeball is small and the pupil is perhaps slightly dilated and sluggish in reaction and that the eyeball feels harder than normal, your suspicion will get stronger. You may not have a tonometer but your trained fingers will detect increased intra-ocular pressure. Similarly you may not have a perimeter, but a carefully executed simple “confrontation” test will reveal any gross contraction of the field of

vision, specially on the nasal side. Any text book of ophthalmology will tell you how this test is done. If you have a plane retinoscopy mirror throw the light in the pupillary area, which although in day-light looked grey like a case of immature cataract, will now show a uniform red reflex, unbroken by black spots or radiating spokes as you see in a case of immature cataract. Even if you have slight practice in ophthalmoscopy you should be able to detect the deep cupping of the optic disc which is quite different from the shallow cup of primary optic atrophy, where the disc is grey or dead white, with clear cut margins.

Gradual failure of vision unattended by any of the signs and symptoms mentioned above, showing a greyish reflex in a normal-sized briskly reacting pupil, will most likely be a case of incipient cataract, specially if there is a history of uniocular polyopia and if you find that the vision improves with concave glasses which he did not need before. Periodical examination of such a case will reveal the gradually increasing white opacity of the lens.

Gradual failure of vision with a clear black pupil reacting to light but not maintaining contraction and showing a white disc on ophthalmoscopic examination will make you suspect optic atrophy and you will refer the case to a specialist for detailed examination.

Here again, let me remind you that chronic glaucoma may co-exist with immature cataract. In such cases, be guided by the tension and the pupil and its reaction.

I shall now refer to some of the other commoner mistakes in diagnosis which I have come across in my experience as an examiner for the M.B., B.S., degrees of various Universities.

III. I have often found boys mistaking **Occlusio Pupillæ** for **Cataract**. To them anything whitish in the pupillary area is cataract. A little careful examination of the irregularly contracted pupil, not at all reacting to light, firmly tied down to the anterior capsule of the lens, should tell them that what they think to be cataract is really the old organised exudate from the inflamed iris filling the pupillary area. On two or three occasions they even went so far as to mistake a central corneal opacity to be cataract. This, of course, was inexcusable.

IV. Confusing **Trachoma** with **Spring Catarrh** (Palpebral type) or with **Follicular Conjunctivitis** is by no means uncommon. One has only to remember a few salient points about each of these conditions and then there will be less confusion.

Follicular conjunctivitis is chiefly confined to the lower lid

while trachoma and spring catarrh mostly affect the upper lid. The small round sago-grain-like follicles arranged in definite rows in the lower lid, producing hardly any symptoms and no complications are characteristic of follicular conjunctivitis.

The thick, hard and raised flattened-on-top cobble-stone-like whitish areas marked out on the conjunctival surface of the upper eyelid, the intense itching, the seasonal incidence, and the presence of eosinophil cells in the smear are characteristic of spring catarrh.

Trachoma presents different pictures in different stages of the disease. In the early stage the granules look like those of follicular conjunctivitis but their partiality for the upper lid and earuncle, progressive and obstinate nature of the disease leading to various complications like thickening of the lids producing a mechanical ptosis, scarring of the conjunctiva, entropion, trichiasis, pannus, corneal ulcer etc., and its infectivity should make the diagnosis easy.

V. **Phlyctenular Conjunctivitis** is sometimes mistaken as **Pterygium**. A leash of conjunctival blood vessels running towards the limbus is a common factor in both. But the presence of one or more inflammatory yellowish nodules at the limbus often leading to superficial ulceration and sometimes associated with eczema of the skin of the cheek, pain, lacrimation and photophobia, a tubercular diathesis in the patient and the tendency to completely clear up after treatment but to relapse again, are factors characteristic of phlyctenular conjunctivitis. On the other hand, a pterygium is a degenerative lesion which tends to extend on the surface of the cornea and causes practically no symptoms. Sometimes a fold of the conjunctiva getting adherent to a marginal healed corneal ulcer simulates a pterygium (called 'Pseudo-ptyerygium') but in this case, you can pass a probe underneath it.

Sometimes students call a **Pigecula** a pterygium, which former is a yellow triangular patch on the conjunctiva near the limbus.

A nodule of **Episcleritis** has also been mistaken for a phlycten; but the episcleritis nodule is larger and is 3 or 4 m. m. away from the corneo-scleral junction, is hard and tender, never ulcerates and takes a chronic course and leaves behind a slaty scar to which the conjunctiva gets adherent.

In true **Scleritis** the inflammation is more diffuse and often causes secondary corneal and uveal tract inflammations and, later

on, produces thinning of the sclera which may lead to scleral staphylomas.

Yet another condition which has been mistaken for phlyctenular conjunctivitis is the bulbar form of **Spring Catarrh**. It is usually a dirty white raised gelatinous area round the cornea. Here again the intense itching, the presence of eosinophiles, the seasonal incidence and absence of a definite leash of blood vessels make the diagnosis easy.

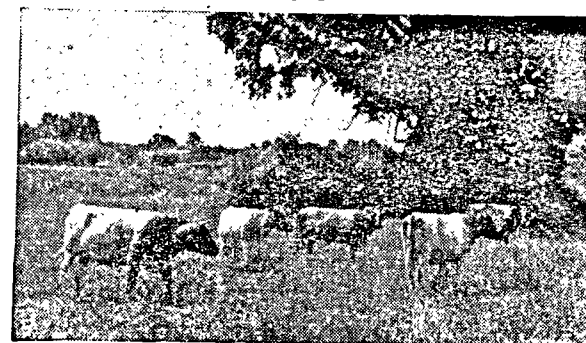
VI. Omitting the rarer condition or conditions which require an ophthalmoscope for diagnosis, I shall conclude this article by mentioning just one more condition often met with in the practice of refraction.

A young patient comes to you complaining of eye-strain and headaches. You see him wearing a new pair of glasses which have been given to him by an optician. He says he sees quite clearly with these glasses, but somehow he does not like them. They seem to have made his headache worse. You find his vision without glasses is 6/12 or 6/9, but with his glasses he reads 6/6. You notice the lenses he is wearing are concave. He has been diagnosed and treated as a case of myopia. Your subjective examination confirms the diagnosis. But if you know your job you will put *atropine* in his eyes for 2 or 3 days and thoroughly paralyse his accommodation. Your retinoscopy will then show that, far from being a myope, he is actually a hypermetrope. Under atropine he will accept plus lenses but without it he won't. This is a case of **SPASM OF ACCOMMODATION** where the young patient endowed with plenty of accommodative power uses an excessive amount of accommodation and converts himself into an artificial myope. The question of treating such a patient involves a great deal of judgment and had best be left to a specialist. What I mean to convey is that if a patient's vision improves with minus lenses do not take it for granted that he is actually a myope. If he still complains of asthenopia send him to a specialist.

In conclusion, let me repeat a final warning: *Whatever mistakes you may make, do not mistake Acute Glaucoma for Iritis and Chronic Glaucoma for Cataract.*

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Scientific evidence is rapidly accumulating to indicate that in the prophylaxis of rickets and other bone diseases the natural vitamin content present in milk, butter and cod-liver oil is more reliable and effective than that artificially produced by irradiation.

Dr. J. C. Drummond, in an address at St. Mary's Hospital, London, 17th May, 1934, stated:

"It may come as a blow to learn that artificial vitamin D is not nearly so active in preventing or curing rickets as is the natural vitamin D which is present in milk, butter and cod-liver oil".

Steenbock, the American pioneer of the irradiation process, has also admitted (Int. Biol. Chem., July 1932) that:

"Ergosterol and yeast when activated anti-rachitically by ultra-violet radiations are inefficient sources of vitamin D for the chicken. Whereas one per cent. of cod-liver oil of average potency resulted in normal bone formation, it required from 40 to 120 per cent. of cod-liver oil equivalence as irradiated ergosterol and from 7.5 to 60 percent. cod-liver oil equivalence as yeast to produce the same result".

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Gastro-intestinal tract: gastritis, gastric and duodenal ulcers, pylorospasm, singultus, constipation, hyperemesis gravidarum, intestinal tuberculosis, gastric tabetic crises.

Bile-ducts: colics, more especially acute biliary colic.

Respiratory tract: asthmatic dyspnoea, allergic dyspnoeic states; whooping-cough.

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Ophthalmic Aid in General Practice

BY

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FROM the socio-economic point of view, diseases of the eye form a class by themselves. Many of the eye diseases, if not attended to in time and in the right manner, will land the patient in blindness, total or partial. To the lay public, and even to the census enumerator, it looks as if only total blindness counts as blindness; they seem to forget that defective vision sufficient to handicap the subject from following his avocation and earning a livelihood is blindness for all intents and purposes and makes him an economic burden on society. Any one with even a passing acquaintance with what may be seen every day in any eye hospital in India will not hesitate to assert that the official census figures on blindness does not cover even a tithe of its real incidence in the country. I am afraid this remark holds good in respect of other infirmities as well.

India is a vast continent of heavy and poor population of diverse habits and culture and of very low literary efficiency. Scientific and satisfactory medical aid is, broadly speaking, available only in the more important centres and townships; even in such places it is not always that one meets with adequate services in special branches as ophthalmology. To the illiterate and poor villager these centres are often beyond their slender means to reach: many may not even be aware where they could get proper help. As a result, the remote and helpless villager falls a ready victim to the ignorant though well-meaning village quack or the unscrupulous itinerant charlatans to stoically suffer and blame his Fate when all is over. The picture may be painful, but true, and demands the earnest attention of social workers.

General practitioners following the Western system are not so rare now, though not evenly distributed as it ought to be. Most villages can, with some difficulty, reach them. These practitioners can be of immense help even in the matter of diseases of special organs as the eye, if they would devote some little attention to the subject. Many eye-conditions, if seen and tackled at an early stage, the general practitioner can successfully treat; in others he can usefully advise and direct the

patients to the nearest place where proper treatment is available. It is unfortunate that the majority of the general practitioners do not devote that attention the subject deserves. No costly equipment or troublesome study is required to meet the ordinary eye-conditions one may encounter. I am aware that the conditions under which the average general practitioner has to work are difficult; his remuneration is often poor, work and responsibility heavy, and he may have often to stand competition against unscrupulous quacks and charlatans. The 'Tradition and Nobility' of our profession will, I am sure, enable even the humblest practitioner to rise above all such difficulties in his sacred work—'Service' to Humanity.

In this article I propose to deal with only such common conditions a general practitioner is likely to meet with, confining myself to a very elementary treatment of the subject. Having been in general practice for some ten years before taking to ophthalmology, I fancy, I am in a position to understand the difficulties and limitations of the general practitioner. I shall therefore delineate only such diagnostic examinations and treatments that are easily practicable to the general practitioner.

Diseases of the lids:—1. Trachoma or granular lids.—Of all diseases of the eye, here in the tropics, this, to my mind, is the one that deserves our attention most. It is very widely spread and is endemic in practically every corner of India; it is infectious in nature, insidious in its onset, such that the sufferer himself may not be aware of anything amiss with him till late; it takes a long course of active and continuous treatment, relapses are common and ends in many and miserable sequelae including total blindness and life-long suffering if not treated and cured before it is too late. Considering its heavy incidence and grave sequelae, it may not be amiss to suggest examination of the lids as a routine measure in all cases, more especially of the young.

In the earlier stages there may not be any complaint; in a good number of cases complaint begins only when pannus has formed. When present, the early symptoms may be an occasional irritation or gritty feel in the eye and some watering. If the upper lids are everted and examined granules can be readily made out. In the very early stage—premonitory stage, as it were—if the everted lids are examined with a magnifying lens and a focussing torch-light, stellate vessel-formations may be noted at the centre of which a granule may be detected. When

the disease is well established plenty of granules can be seen with strands of white fibrous tissue here and there; perhaps blood vessels growing on to the cornea may also be seen at the upper limbus-pannus. In old cases severe pannus covering perhaps the whole cornea, multiple and minute corneal ulcers, thickened, cicatrised and bent tarsus with entropion and trichiasis etc., may as well be seen. Well established and advanced cases present no difficulty in diagnosis. Earlier cases will have to be differentiated from, notably, spring catarrh and follicles. The cobble-pavement appearance of spring catarrh is characteristic and will not be easily missed. If, however, corroboration is required the best one is to examine a smear of the conjunctival discharge for Eosinophiles which will be marked in spring catarrh. Follicles are more pronounced in the lower lids and they appear as strings of pearls. There is no fibrous tissue formation associated with this; in fact no complications will be found. But it should be remembered that, especially in the young, trachoma and follicles may be present together.

In the early stage application of *Ol. Hydnocarpus* is, in my experience, as good as anything else; and it is painless. Pure cold drawn oil should be used and it should not be rancid. If there is discharge the eyes are well washed out with a bland and mild antiseptic as *Lot. Ac. Borici* after fully everting the lid on a Desmarres' Lid Retractor. The tip of the retractor is placed about the middle of the outer surface of the lid; with the thumb and index finger of the left hand the edge of the lid is held and everted on to the blade of the retractor and the retractor used as a lever with the eyebrow as a fulcrum. This exposes the whole of the conjunctival surface including the fornix. The manipulation is painless, not even uncomfortable, and even children submit to it without trouble. It steadies the lid well and gives access to every part. Now oil is applied with a cotton-tipped "kutchi" with a mild rubbing motion till a frothy appearance is produced. The eye should not be washed after the application. In cases with ulcerations on the lids the rubbing may cause a little bleeding just to stain the froth; but it is of no moment. This treatment can be done in the presence of pannus and minute ulcerations of the cornea without danger. This is not to be repeated oftener than every other day: often thrice or even twice a week will suffice. On the intervening days a 1% Yellow oxide ointment may be applied with advantage. If considerable pannus is present injections of "Foudin" may help much. If Foudin is too costly, Tartar-emetic may be given intravenously (3—5 c.c.

of a 1% sterile solution). A 10% black sulphide of Antimony in place of the Y. Oint. will also be found useful in cases of pannus. Trachoma yields to this treatment in 8—12 weeks.

If ulcers are present on the cornea atropine should also be exhibited; if large, measures in accordance with treatment of ulcers should be instituted. (Vide corneal ulcers). The patient should be kept under regular periodical observation for some time after cure as relapses are common. Thin nebula left by superficial ulcers may be treated with 5—10% Dionin oint. and remnants of pannus by sub-conjunctival injections of N. saline.

Cases of entropion can also be treated easily by the general practitioner. After injecting the whole length of the lid on its outside with a local anaesthetic (1 c.c. of 2% eucaine with some 5 m of adrenalin is found satisfactory) the lid is everted as before on a Desmarres' retractor. With a sharp small scalpel the tarsus is cut through the sulcus along the whole length and through its whole thickness. Now with a snip of a blunt pointed scissors the attached ends of the now separated tarsal rim are severed. This need not include the skin; it is sufficient if the mucous membrane and the tarsal plates are severed. At the inner side this snip should be outside the punctum.

After-treatment consists of application of some simple non-irritating antiseptic ointment and a bandage for three or four days. The effect is good and permanent in the large majority of cases; and there is no disfigurement.

Cases complicated with large corneal ulcers, heavy pannus, thick disfigured and ulcerating tarsii etc. are better sent to the nearest ophthalmic clinic. Follicles demand no treatment beyond some 1% Y. Oint. Spring catarrh is very difficult to treat successfully and is best treated by a specialist. Fortunately it has few complications and is not fraught with any grave dangers.

2. *Cysts*:—Two common forms are styas and chalazia. It is useful to remember that frequent styas and chalazia are often associated with errors of refraction and a correction of the error is indicated in them. In rare cases even auto-vaccine may be required to control the cases. Styas are easily opened, all that is required being just to lance at the lid edge where it is always situated. Chalazia are cyst formations with a wall and are embedded in the tarsus. The lid is everted and the cyst is lanced. Then the cyst wall is removed by a sharp, small spoon. If a chalazion forceps is available, it is better to use it to steady the lid and encircle the chalazion in its fenestra. For the first day, a

mild antiseptic ointment and a bandage are required. After-treatment consists of use of Y. oint. to hasten absorption.

3. *Entropion and ectropion of lower lids*:—Marked ectropion of the lower lids, both primary and those due to cicatricial changes demand complicated surgical procedures and are therefore best directed to an ophthalmic clinic. Simple eversion of slight degrees can be corrected by a "V-Y" operation. After local anaesthesia a "V" shaped incision is put below the lid, the upper ends of the V commencing on a level with the bony orbital wall below the outer and inner ends of the lid, and the lower tip falling about $\frac{3}{4}$ " below the lid margin. This "V" is allowed to retract fully; it may even be desected if the eversion to be corrected is great. Now the skin wound is sutured into the form of a "Y". It is better to overcorrect a little than undercorrect. Sutures may be removed on the sixth or seventh day.

In inversions of the lower lids removal of an elliptical piece of skin below the lid and with its long axis parallel to the length of lid, and resuture may be done. These cases should not be overcorrected.

4. *Punctum conditions*:—The commoner ones are (1) a foreign body or inspissated secretion blocking the puncta and (2) eversion of puncta. The complaint is always epiphora. Sometimes a fallen eye-lash may get stuck in the mouth of the puncta and give rise to severe and distressing watering and irritation. Removal of the offending hair with a forceps is all that is required. In the case of inspissated secretions blocking the punctum or canal, dilatation and syringing may be required demanding special blunt needles etc. To deal with eversions of puncta also special directors etc., are required. Hence, though both the procedures are simple enough, are not considered here.

5. *Dacrocystitis and Lachrymal cyst abscess*:—Chronic cases can easily be diagnosed as regurgitation can be demonstrated by pressing on the sac area. In a good number of cases, out here, rhinosporidium of the nose or arising in the sac itself is the root cause. All cases of dacrocystitis are a source of potential danger and the patient should be advised accordingly. As there is no urgency it is best to advise him to seek an eye-clinic. The same is true in respect of discharging lachrymal sinuses. If, however, the case presents itself as an abscess involving the sac, the abscess must be opened and drained in the usual way and the patient advised to go to an ophthalmic clinic for radical treatment.

6. *Blepharitis*:—A simple chronic dry blepharitis may be a

concomitant effect of some error of refraction. Hence, for a permanent cure such cases should get their errors of refraction corrected. In children of dirty habits blepharitis may be the result of pediculosis blepharæ. In these cases nits as well as pedicula may be seen on careful examination. Simple forms including the pediculosis type are quickly cured by the use of a 2-3% Yellow oxide ointment or a $\frac{1}{2}$ —1% Hydrarg. Ammon. ointment applied two or three times a day to the lid edges. Blepharitis are often associated with angular conjunctivitis and if found so, that condition should as well be treated. ZnSO_4 , 2-3 gr. to the ounce with a little ac. boric and adrenalin drops in normal saline will be found satisfactory.

In the ulcerative types, the scabs should be removed at least once a day with the help of a 2% Sod. Bicarb. solution and cotton wool and an ointment containing 3-5% Ichthyol. 2-3% Y. oxide or 1% Hydr. Ammon. should be liberally applied three or four times a day. When the inflamed and angry look has subsided, Zinc oxide oint may be substituted. These cases are often found associated with chronic trachomatous conditions which should also get adequate treatment for a permanent cure.

7. *Injuries to lids*:—Simple wounds are to be treated on their own merits. If the lids are torn, after proper cleaning, the torn edges should be carefully sutured ensuring correct apposition. Horse hair is the best material for suture in these situations. In suturing from outside the conjunctiva need not be included. If conjunctiva requires suturing it should be first done and with fine silk. If a lid is torn off and lost it is essential to take adequate measures to protect the cornea from irritation from outside or from the dressings to be applied to the wound. Such cases are best sent to an eye clinic. As a first aid protection, after cleaning the wound, the whole of the exposed area may be filled with plain sterile vaseline and covered over with a sterilised piece of any old rubber glove and dressed with plenty of cotton wool. If only one lid is destroyed, the other lid may be stretched over the eye and fixed in that position with sticking plaster or a couple of temporary sutures and dressings applied over as a first aid expedient.

Diseases of the Conjunctiva.—*Simple conjunctivitis, catarrhal conjunctivitis, "pink eye"*:—This though a simple ailment can become troublesome. Iritis may be mistaken for pink eye and lead to serious trouble. The differential diagnosis from iritis will be considered under iritis. The symptoms in simple

conjunctivitis are redness, gritty feel and some watering. If left untreated the discharge may assume a mucopurulent or even a purulent nature. In many cases a single application of a 2% AgNO_3 solution followed by milder treatment is all that may be required. In severer cases, AgNO_3 may have to be applied twice or thrice with at least a days' interval between each. In applying silver nitrate, care must be taken not to allow the solution to come into direct contact with the cornea; and after the application the eye should be well irrigated. As in these cases considerable mucous discharge is to be expected the eye should be washed before the application as well. This is to be followed by Boric irrigation and instillation of, say, a 12% Argyrol Sol. 3-6 times a day according to the severity of the case. In severe cases hot fomentation twice or thrice a day will also be beneficial. To avoid sticking of the lids a thin smearing of boric ointment or even plain vaseline on retiring to sleep will be very useful.

Purulent conjunctivitis:—In many cases Gonococcus is the offending organism. Streptococcus can produce a condition which cannot be clinically differentiated from the gonococcal form. The local treatment is the same irrespective of the causative agent. In gonorrhœal cases the associated urethral condition must also get its proper treatment. Those cases having a serosaneous discharge and those with marked oedema of the lids and conjunctiva are the worst. In these, corneal infection and its sequelae form grave complications. Therefore, the urgent aim must be to protect and preserve the cornea from danger.

If only one eye is affected, the other eye should be protected, as a prophylactic measure, with a Buller's shield. A circular piece of mackintosh large enough to cover the orbital area, with sticking plaster spread at its edges for a width of about $\frac{1}{2}$ " and with a window cut out in the centre for vision, over which a watch glass or a round spectacle glass is stuck with sticking plaster, would answer the purpose. The idea is to prevent discharge from contaminating the unaffected eye. The good eye must be viewed daily and if any suspicion of infection arises the shield should be removed and treatment begun early.

If there is no brawny oedema of the lids, AgNO_3 may be applied as before; if there is oedema AgNO_3 may do more harm than good. If there is much oedema and consequent difficulty of opening the eyes well, a free canthotomy will help much by

avoiding accumulation of secretions in the eye. In all cases the following treatment will be found effective. Every two hours or so while the patient is awake, the eye is well irrigated with warm sterile saturated solution of Mag. Sulph. from a can taking care to keep the lids well opened with a wire retractor; this irrigation must be complete and should remove all traces of secretions from the fornices. If there is much oedema this irrigation should be followed by hot fermentation. This is now followed by an instillation of a 2% Mercurochrome solution. The lid edges may be, as before, smeared with a trace of HgCl_2 Oint. 1:1000. On retiring at night the same ointment may be applied in plenty to the eyes. In severe cases Prontosil 3-5 c.c. daily for two or three days may cut short the disease wonderfully.

If ulcers are present on the cornea, besides these measures, atropine will have to be applied and pupil kept in a dilated condition. Under no circumstances should such eyes be occluded by bandaging. The ulcers may be touched with Tr. Iodine or even pure carbolic taking good care that the caustics do not come into contact with the healthy cornea. If electric cautery is available it can also be used to cauterise the base of the ulcer. If the ulcer is fairly large and there is much hypopion, a paracentesis is indicated. It is advantageous to do this in such a manner that the cut in the cornea passes through the base of the ulcer, bisecting it. If the pupillary area is free and there is a fear of the ulcer encroaching on to it, the section may be planned to fall beyond the progressing edge of the ulcer, on healthy cornea, between it and the pupillary area. This often limits the encroachment effectively.

If there is bad chemosis producing a raised conjunctival wall around the cornea, where, in the moat formed thereby, secretion could remain in contact with the cornea and destroy its vitality, this wall must be removed with scissors to obliterate the moat. This procedure becomes imperative if the cornea begins to show a dull appearance at its periphery or if tiny peripheral ulcers begin to form.

General measures such as cleanliness, good ventilation, proper nourishment etc., will go a great way and must not be neglected. Good sleep must be ensured for which sedatives may be required. Old Bromide and Hyoscyamus is as good as any other thing. If gonorrhoea is present adequate treatment should also be given, and the patient warned against transferring the infection from the urethra to the eyes. It is one of the most important duties of the profession to give a true and correct idea about

the infectious nature of the trouble and to give such advice to the patient and his friends to enable them to avoid the infection.

Ophthalmia neonatorum is practically a disease of the same type occurring in the now-born. Though in many cases the causative agent may be gonococcus, it should be remembered that it could be a non-gonococcal trouble. The line of treatment is more or less the same as in purulent ophthalmia. The patient being an infant only a few days old, very careful and tender handling and milder measures are required. It is worth while to remember that proper Crede's prophylactic treatment of the eyes soon after birth is efficient to prevent neonatorum developing.

It is no good to direct these cases to carry out the frequent irrigations etc., by themselves or untrained friends. A well trained nurse's services are essential in these.

Affections of the Cornea.—Besides ulcers due to injuries and secondary to diseases like purulent ophthalmias, there are many inflammatory conditions some superficial and some deep affecting the cornea. Of the superficial types, the "Madras Eye" is perhaps the commonest. It is perhaps infective in nature: it is wiser to recognise it as such, until the contrary is established. Some watering, irritation and dimness may be the complaint. They appear as whitish, superficial, circular spots scattered over the cornea. As a rule they do not stain and complications are practically nil. A mild associated conjunctivitis is always present. Iris involvement is rare. The disease sometimes occurs in an epidemic form.

In the earlier stage, a mild antiseptic as chloretone oint. or mercurochrome oint. is all that is needed. If there is any circumcorneal injection, atropine may also be exhibited once in a way. Usually, this is not necessary. The eye becomes quiet in a few days leaving multiple nebulae and reducing the vision often considerably. The seriousness of the disease lies in this. The treatment of the leukomata takes a long time and a perfect clearing is almost impossible. For the clearing, Dionin 5-10%, Y. oint. 1-2%, subconjunctival saline etc., are required in rotation.

Of the deep forms, Interstitial keratitis is the most important. The appearance is characteristic; the deeply imbedded broom-shaped vessels forming the "salmon patches", hazy ground-glass cornea, K. P. in the anterior chamber, and the presence of congenital syphilitic stigmata in the large majority, make the diagnosis easy and clear. A blood examination may show positive reaction. The disease is as much a disease of the uveal tract

as that of the cornea: hence the use of atropine is essential. Atropine and yellow oxide or atropine and dionin in the form of ointment form the most important local applications. The main treatment is antisyphilitic. Arsenical preparations of the Salvarsan type may be used and will hasten the cure. Time-honoured Grey powder in $\frac{1}{2}$ —1 gr. doses T.D. is also satisfactory. A judicious combination of both is the best. The treatment including the clearing of the cornea is a fairly long one, but the result is very satisfactory. Though this is a disease of young adults, it may occasionally be met with in grown-ups as well as the very young.

Of the ulcerative forms, the commonest are septic ulcers resulting from injuries, ulcerations arising during the course of trachoma, purulent ophthalmia etc; deficiency types as keratomalacia and Mooren's ulcer etc.

Management of corneal ulcers in general:—The aim must be to limit the ulcer from spreading, to heal it quickly, and to see that no posterior or anterior synechia form. In all inflammations of the cornea the iris will be more or less affected, and would require treatment. In all cases where there is no septic discharge from the conjunctiva, it is advantageous to bandage up the eye to give rest to the injured part by arresting the frequent rubbing by the lids in winking.

The surface of the ulcer, if unhealthy, may be scraped, or cauterised with Tr. Iodine, Pure carbolic, or the actual cautery. A 2% Mercurochrome oint. is one of the best antiseptics which can be used without any fear of injury to the healthy cornea. Atropine should be used to dilate and to maintain the dilatation till the ulcer is healed. If there is not much discharge the eye must be kept tied up till the ulcer heals. If the ulcer tends to progress in spite of these procedures a paracentesis may be done as described before.

Keratomalacia and Mooren's ulcers are deficiency diseases and do not stand strong measures. Xerosis of the conjunctiva with or without night blindness must be recognised as a fore-runner of keratomalacia and suitable treatment instituted. The prime essential in the treatment of these conditions is to make up the A and D vitamin deficiency with cod liver oil, milk, butter, sheep's liver etc. Keratomalacia patients are as a rule children in a very low general condition; a bad gastro-enteritis with bad diarrhoea is common. There may be general oedema and albumin in the urine. Such cases cannot accommodate cod liver oil by mouth; they can be very successfully treated as follows:

Hydrarg. Cum creta in $\frac{1}{3}$ gr. doses with a gr. or two of soda are given 3 or 4 times a day, the patient being fed on whey or diluted milk with glucose added in place of sugar. This controls the diarrhoea quickly. At the same time Haliverol in three to five drops are given by mouth three or four times a day, while cod liver oil is used in the form of an inunction to thighs and the lower abdomen.

The corneal ulceration is treated on general lines but cauterisations and similar strong measures are not to be used. Boric irrigation, a 2% mercurochrome oint., atropine oint., and bandage twice a day will meet the requirements. A drop of Haliverol to the eye with the ointments is found very useful in controlling the progress of the ulcer. By these procedures it has been found possible to save eyes where the cornea on admission was more than half involved.

Mooren's ulcer also is one in which the vitality of the subject is low. Here also the same type of treatment as in keratomalacia is found useful. The proceeding edge of the ulcer may be scraped and freshened to start with. If this treatment fails and the ulcer tends to progress, covering the ulcer with conjunctiva may save the eye for which he may be advised to seek the services of an ophthalmic clinic.

Injuries to Cornea and Conjunctiva including Foreign Bodies.—*Foreign bodies, conjunctiva*:—If there is a history of foreign bodies the first place to be examined should be the sulcus on the under surface of the upper lid which is the commonest place for a foreign body to lodge. When found, it can be removed with the tip of a piece of stiff paper, eye washed with a little boric lotion and patient dismissed. If the foreign body is imbedded in the conjunctiva, it may be removed with a fine forceps, or better still, the bit of conjunctiva including the foreign body may be picked up with a fine forceps and sniped off. No suturing is required, but the eye must be dressed with some mild antiseptic for a day or two.

Foreign bodies on cornea:—This if superficial, may be removed with the tip of a sharp needle—flat Hagedorn type will do—or of a fine scalpel if regular foreign body instruments are not available. After removal of the foreign body, all corneal injuries should be well cleaned, and dressed as a corneal ulcer.

All penetrating injuries of the cornea or sclera, particularly if there is prolapse of intra-globular structures, if foreign body is suspected inside, should be sent over to an eye clinic after a simple first aid dressing.

Diseases of the Iris.—Iritis is the only condition worth considering here. This is a common disease and one entailing serious consequences. In the early stages it is likely to be confounded with simple conjunctivitis. Hence it is imperative to look for circumcorneal injection in all cases of pink eye, particularly so if there is only watering and no discharge. Headache and pain in the eye are early complaints. One of the earliest signs is that the iris fails to react to darkness; *i.e.* if the suspected eye is shaded the pupil will not dilate but will remain contracted. If the whole conjunctiva is red and a specific circumcorneal injection cannot be made out, instillation of a few drops of adrenalin will in a few minutes blanch the superficial vessels leaving the deep circumcorneal vessels unaffected and rendering it prominent by comparison.

It is essential to differentiate it from glaucoma as the treatment for the two conditions are opposed to each other. It should be remembered that in congestive glaucoma marked circumcorneal injection can be present, and in iritis with much posterior synechiae, some rise in tension. Absence of tension is in favour of iritis. Tension may be roughly noted with the fingers. It may be useful to remember that the normal tension of the eye is about that of a new tennis ball. Any harder feel should be reckoned as an increase in tension.

Treatment.—In both iritis and congestive glaucoma certain measures of treatment are common. A smart saline purgative to start with is good in both. So are leeches to temple and hot fomentations. In iritis atropine application is urgent and essential. This should never be done in glaucoma, even if only suspected. In iritis atropine helps in many ways; it dilates the pupil and thus avoids the chances for the formation of posterior synechia, the serious sequel of iritis; by vaso dilatation it promotes better circulation in the iris and ciliary body and thus tends to reduce the inflammation; and by paralysing the sphincter iris, it gives rest to the inflamed structure. If atropine alone does not produce sufficient dilatation, a sub-conjunctival injection of 2 m of a 1% atropine solution with some 5 m of adrenalin will be found very useful. Daily use of atropine may be in the form of a 1% oint. instead of solution as the oint. rarely produces atropine-irritation even when long continued. If there is any suspicion of tension—and dilatation is still wanted, adrenalin in the form of subconjunctival injection may be given with advantage, as it both dilates the pupil and lowers the tension at the same time.

Common underlying causes of iritis are Rheumatic factor, gonorrhoea and syphilis. Of these syphilitic types, as a rule, are not prone to recur. In these, if the iris is carefully examined with a magnifying glass and a strong focussing light, nodule-like formations may be seen. History may not be always reliable; the result of blood examination, where possible, is better. Besides the focal treatment antisiphilitic measures should be adopted.

In the other two types, a differentiation is not always possible. Hexamine by mouth in good doses is often very useful; Sodii salycilas is also serviceable. Pot. Iod. for absorption of exudates is useful. The aim must be to discharge the patient without synechia, as they predispose to recurrence. If, on discharge, there are strong or multiple synechia an iridectomy is often indicated and the patient may be advised to go to an eye clinic.

Glaucoma.—Congestive glaucoma is an emergency and must be attended to. The differential diagnosis has been already referred to. Wide pupil which does not contract to strong light or does so only tardily, a rise in tension, marked injection, and severe headache and pain in the eyes are the characteristic symptoms. An acute rise in tension is capable of producing permanent destruction of the optic nerve, by throttling it as it were, in 24-48 hrs. Hence, quick and efficient treatment is essential. Smart saline purge, multiple leeches to the temple, and hot fomentations to the eyes are all very useful. Eserine in 1% sol. may be instilled into the eye every two hours till pupil assumes pin-point contraction, and thereafter, continued in $\frac{1}{2}$ % strength. Sub-conjunctival injection of adrenalin is very effective to lower the tension by constricting the vessels. When the tension has subsided a little, and if the practitioner is quite conversant with the technique and is well equipped, a broad iridectomy may be done. It is always better to send the patient to a proper eye clinic after the first aid treatment. If by the aforementioned medical measures the tension does not tend to lower itself within 12-14 hrs. it is better to send the patient to an eye clinic without loss of time. When directing the patient to an eye clinic it is essential to supply the patient with some $\frac{1}{2}$ % eserine sol. for frequent application till he reaches the clinic.

Chronic or Non-congestive Glaucoma.—This demands only appraisal and advice at the hands of the general practitioner. The complaint may be frequent headaches with pain in the eyes

and dimness of vision. On questioning, classical symptoms like haloes around light etc. may be forth-coming in the case of intelligent patients. It is important to remember that tension may not be high at the time of examination if the patient is then in a quiescent period. A rough estimate of the field is perhaps more trustworthy. This can be done by placing the patient facing the doctor in such a way that their eyes are on a level and the right of one looking into the left of the other and vice versa. Now the eye of the patient not under examination is closed; the doctor also closes his eye which is not opposing the one under examination. In this position the doctor moves his fingers in a vertical plane midway between himself and the patient in at least eight quarters, up, down, right, left and the intermediary angles. The finger is moved from the centre outwards, and the relative places where the doctor and patient misses the finger are noted. If the patient misses earlier than the surgeon the field in that quarter is reduced. A very fair idea of the patient's field of vision can be estimated by this procedure if carefully done and if the patient is one of average intelligence. If the restriction of the field on the nasal side is positive, it is a very presumptive evidence of glaucoma. When chronic glaucoma is suspected it is the duty of the general practitioner to warn the patient and direct him to a proper place for treatment.

It often happens that a case of chronic glaucoma is mistaken for an early cataract and the patient advised to wait for it to mature, with disastrous results. If the condition of the field and tension are examined, this danger can be mostly avoided. If the practitioner is accustomed to ophthalmoscopic examination it is better that he does it in all cases of suspected glaucoma and early cataracts.

Cataract.—The one responsibility of the practitioner is to warn the patient from falling into the hands of couchers. The practitioner can save much time and trouble of the patient if he could keep cataract cases under observation and direct them for operation when fully mature. Inability to count fingers at a foot may be taken as a fair criterion. The best test, however, is to see that no shadow of the iris is seen on the lens when strong light is thrown slantingly on to the iris. If complications are suspected as evidenced by a rise in tension, loss of vision disproportionate to the lens opacity etc. the case ought to be sent to a specialist without loss of time.

Cataracts in children and those below 25 need not wait till full maturity as these can be successfully treated even in the earlier stages; if vision is below 6/18 they may be advised to seek surgical help.

Glioma.—This is a deadly disease affecting little children. It is important to recognise it early, as invasion of the optic nerve will nullify all surgical assistance. In the earlier stages all that is seen is a "cat's eye" appearance unless carefully examined. A final diagnosis in the early stages demands transilluminations and ophthalmoscopic examinations which are beyond the average general practitioner. The commonest danger is to mistake it for cataract and consequent advice to wait. It is to avoid this danger, among other things, that in the section on cataract it was pointed out that cataract in children need not wait for maturity to go to an eye clinic. If there is the least suspicion the benefit of the doubt should be given to the patient and he should be advised to seek proper help without loss of time. The pupil may be dilated with atropine and examined with a strong torch light and a magnifying lens, when, behind the clear lens a whitish mass with fine blood vessels may be made out. It is not necessary for the general practitioner to differentiate between gliomas and pseudogliomas; not even from an infantile cataract; it is sufficient to advise the patient to be taken to a specialist without loss of time, but without alarming him.

In this connection it may be noted that the incidence of glioma is comparatively high in Travancore.

Panophthalmitis.—This may be described as an "abscess" of the eye ball. Cases of panophthalmitis are mostly the result of injuries and ulcerations, the sepsis having gained entry from outside. The whole eye will be swollen and lids oedematous and not covering the proptosed eye ball. There will be much pain and headache; some fever may as well be present. If the cornea is clear, as may happen in endogenous infection, yellow pupillary reflex can be made out. It is not worth while to send these cases to an eye clinic. Treatment consists in evacuating the contents of the eye ball. Regular evisceration can be done without special instruments. A narrow bladed bistoury, a small scissors curved on the flat, and a fairly large blunt spoon will suffice. After cocaineising, the cornea is transfixed and bisected and the two halves cut out with scissors at their junction with the sclera. Now the contents of the globe are removed with the spoon taking care to remove every remnant of the uveal tissue. The inside of

the globe is now well washed out with 1:3000 HgCl₂ lotion, wiped clean with sterile gauze, painted with Tr. Iodine and again washed and the eye is dressed. The inside of the globe need not be packed. After-treatment consists in daily cleaning and dressing and fomentation if there is much swelling.

If there is any difficulty in doing this, the cornea may simply be slit open (fully bisected) to let out the pus and the patient treated as before or sent to the nearest clinic.

Retina, Choroid etc.—Unfortunately most general practitioners do not seem to interest themselves in the use of the ophthalmoscope. The training will be very useful and handy even in general practice. Papilloedema, papillitis, optic atrophy, albuminuric, diabetic and leukemic retinitidis, are only a few of the conditions that will interest the general practitioner. Ophthalmoscopy is easy to master; and as cheap and efficient luminous instruments are readily available, there is no reason why every practitioner should not equip himself with one and train himself in its use. A general practitioner could double his services, and incidentally double his income too, if he equips himself to meet ordinary ophthalmic demands. A cheap and simple luminous ophthalmoscope, a cheap binocular loupe, a condensing lens, a strong ordinary focussing torch light, a couple of Desmarres' retractors (small and medium sizes), a simple eye speculum, a foreign body needle and spud, a couple of plain fixation forceps, one iris forceps, one or two cataract knives, a chalazion forceps, one chalazion spoon, one evisceration spoon, one fine conjunctival scissors, which can be used as an iris scissors as well, one small blunt pointed scissors curved on the flat, a few fine fully curved suture needles and some fine silk, are almost all that may be required. These can be procured within a couple of hundred rupees even if one goes for the best make. A small cheap trial set, a retinoscope, and vision charts will complete the equipment which can turn out much useful work.

I should advise every new graduate who contemplates going into general practice to spend a few months in any good eye hospital as well as a maternity clinic to get some useful practical training in these two special branches of every day demand, so that he could be of better and wider service to those around him.

Finally, I conjure the profession in general and general practitioners in particular to honour and revere the tradition of Bharatavarsha by avoiding every trace of commercial sense from our work and considering the service we are lucky to render as True and Sacred Service to HIM, The Universal SELF.



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GROUP	TYPE	CASES	RESULT
I Adherent Normal Testis Normal Child	Unilateral - -	5	Failed
II Subnormal Testis Subnormal Child	{ Bilateral - - Bilateral with hypospadias - - Unilateral - -	{ 6 2 3	Cured
III Subnormal Testis Normal Child	{ Unilateral - - Bilateral - -	{ 3 3	Cured Improved

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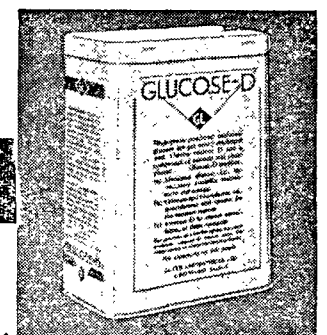
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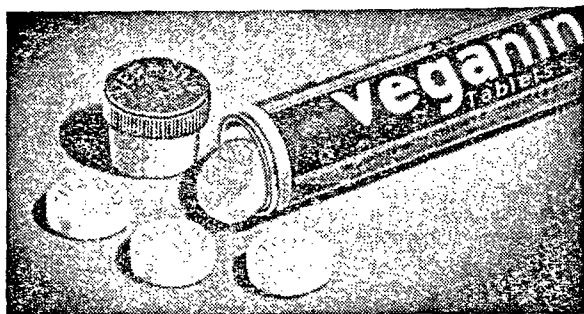
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The Relation of Eye to Ear, Nose and Throat

BY

Dr. E. V. Srinivasan, M.B., & C.M.,

Hony. Ophthalmic Surgeon, Govt Royapettah Hospital, Madras.

The Sinuses in Relation to the Eye.—The relation between the orbit and its contents and the complex system of air cells in the bones of the face almost completely surrounding the orbit is of first importance to the ophthalmologist.

More than two-thirds of the orbit is made up of thin bone which at the same time is the wall of the sinuses. It has to be borne in mind that the extent of variation of the formation of sinuses is extraordinarily great and, therefore, the topographical relations between the orbit and the optic canals are not constant.

Post-operative defective vision and blindness of nasal origin have been reported time after time. Rare cases of functional interference with optic nerve have developed immediately after or even during intra-nasal operations. Though it is impossible to explain these calamities in the light of text-book relationships, still abnormal situations of the various sinuses are quite common to explain the blindnesses recorded after operations on frontal sinuses, ethmoidal sinuses, after resection of nasal septum, removal of polypii and conchotomy. The optic nerve may be free in the sphenoidal cell and be injured during curettage. The optic nerve canal is sometimes in a large posterior ethmoidal sinus. Besides the anatomical explanations of these accidents, there is the possibility of embolus passing from nose to the retinal vessel, without going through the pulmonary circulation.

Recently an European nun whose turbinates were cauterised consulted me regarding her disturbances in vision. She had a central scotoma in the eye following the cautery.

Paraffin injections for deformity of the nose are said to cause embolic blindness.

Many of the earlier symptoms of the disease of sinuses bring the patient to the ophthalmologist. Anomalies of the field of vision, presence of scotoma, central and paracentral, enlargement of the blind spot play a conspicuous part in the detection of the sinus disease. Unilateral epiphora, oedema of the lids, episcleritis, fugax (fleeting) and proptosis point to sinus disease.

Relation of Nose to Eye.—The headache of the nasal disease very closely resembles the headache of refractive error and so the oculist sees these cases. The location of headache is not characteristic of any particular sinus disease, although the pain of frontal sinusitis may be located over the area of the sinus. These sinus headaches occur at nights, unusual time for ocular headache. They are frequently paroxysmal occurring at regular hours, usually in the mornings and ending at a corresponding time each day.

Ethmoidal pain is most frequent at the root of the nose or behind the eye and may be exaggerated by ocular excursions.

The pain of Antrum is often referred to the teeth of the corresponding upper jaw and to the ear in the back or side of the head, markedly increased by stooping.

Sphenoidal produces occipital and temporal headaches and pain in the back of the eye and base of the brain. Cases are frequently free from pain finding a mention only in books, though percussion of walls may elicit tenderness.

Easy exhaustion of eye muscles or asthenopia is very often due to nasal disease probably reflex—as the nerve supply is derived from a common source, the fifth nerve furnishing the sensory roots for the motor-cranial nerves. Photophobia and lacrimation may be due to a conjunctival congestion occurring during a sinusitis or to involvement of ocular muscles including the ciliary muscles. Another symptom of sinusitis is pain in the corresponding eye noted upon looking up while reading. Application of cocaine and Adrenalin to the swollen midturbinals gives relief to the above symptoms, showing the necessity for cauterising the turbinals. The midturbinals may cause pressure on the septum which is very sensitive at this spot and synechia between the septal spur and the anterior end of the midturbinal is a frequent source of supra-orbital neuralgia, lacrimation, conjunctival hyperaemia with burning, smarting and blepharitis etc. When midturbinals impede respiration and prevent air from reaching the superior meatus in the so-called attic of the nose these various reflex symptoms arise. The Sunday morning headache of some patients is due to prolonged pressure of midturbinal on the septum from sleeping unusually late. It never comes on when patient lies down or sits quietly.

Many cases of iritis which are considered to be rheumatic, idiopathic or of auto-intoxication are undoubtedly due to nasal disease. A bulging sac is oftentimes cured by packing the inferior meatus with adrenalin and weak silver solution.

Narrowing of the outlet of the frontal sinus into the middle meatus by means of a bullæ in front of processus uncinatus behind or merely by deflection of septum causes the so-called vacuum frontal headache which is a low grade constant pain, worse in the morning and aggravated by near work and unrelieved by glasses.

Unilateral Trachoma and nasal disease of the same side have been reported as concurrent. I know that this cannot be true trachoma in the light of the recent advances in the diagnosis of trachoma or trachoma must be bi-lateral with so-called unaffected side, really affected, seen by an examination with the slit-lamp.

Orbital inflammation is always due to the extension of the inflammation from the accessory sinuses of the nose. Swollen lids, widening of lid aperture and displaced eye-ball result, the direction of dislocation pointing to the offending sinus.

These orbital abscesses unless drained will lead to optic nerve trouble, if not to ulceration of cornea and pan-ophthalmitis. These are mostly posterior ethmoidal sinus occurring in children as well as in adults.

Acute Frontal Sinusitis.—Secondary to acute rhinitis beginning with sensation and pressure above the eye accompanied by fever and malaise. The pain may be severe and of a bursting character. Percussion of the sinus area is painful. Ewing's sign—pressure of the upper and inner angle or orbit elicits tenderness. Stooping increases pain and causes vertigo. Blurred vision and restriction of field of vision with accommodative asthenopia amounting to pain on reading with photophobia and lacrimation are symptoms. Superior oblique muscle paralysis causing diplopia is common.

A fistula of the frontal sinus may produce an abscess at the inner canthus simulating dacro-cystitis. Chronic cases have only headache, the diagnosis chiefly depending on presence of secretion in the upper part of middle meatus. Ewing's sign will be present. Trans-illumination has a corroborative value. Rontgen Ray photographs are of greater value especially with negative nasal findings.

Sphenoidal Disease.—Symptoms are purely anatomic; headache is occipital, temporal or behind the eye. Post nasal discharge draining into the nasopharynx. When the secretion is profuse the dropping is felt at the pharynx and also crusts on the pharyngeal vault. Sixth nerve passes into the orbit in a groove in the sphenoidal sinus wall; hence affected.

Mental symptoms frequent in sinus disease are most pronounced in sphenoidal disease; such as lack of concentration, loss of memory, depression, hypo-chondriasis and neurasthenia. Mild attacks of sphenoiditis may occur with each attack of acute rhinitis until finally the process becomes chronic with almost constant secretion draining post nasally. The optic nerve changes may vary from slight oedema to an active papillitis. Dimness of vision or absolute amaurosis, uni-lateral headache increased by stooping, a heavy meal or alcoholic drink. The menstrual period is accompanied by increased headache. This symptom was present in a female aged 40 who came to me with a vision of finger counting at 3 meters in the right eye and the left eye normal. She had only occipital headache and papillitis. The sphenoidal sinus was opened by Wright with subsidence of papillitis, though the vision continued to be the same.

The most frequent change in the field of vision is uni-lateral central scotoma for colours and for white in advanced cases.

A case of retrobulbar neuritis with blurring of central vision gradually extending to the rest of the field and one of optic neuritis with narrowed visual field have improved by attending to the nose by simple douche and nasal plasma.

If the obstruction caused by the hypertrophied mid-turbinal and polypoid masses is overcome by their removal, the field improves and visual acuity returns. A very favourable symptom in optic nerve cases is the improvement in vision which frequently follows the thorough contraction of the swollen nasal mucosa by adrenalin and cocaine. In such cases, we reasonably expect a satisfactory result from the re-establishment of free communication between the diseased cells and the nasal cavity.

A case of bi-temporal hemi-anopsia due to acute suppuration of posterior nasal sinuses. There was deviation of septum and enlargement of the lower turbinal, started with absolute central scotoma in the left eye ending in hand-movements. Pus was seen in left middle meatus. Originally sinus was free to radiography. Nasal septum was submucosally resected. Posterior ethmoidal and sphenoidal cells were exenterated. Improvement followed.

Long ago, I had a boy patient who had an incurable oedema of the right upper lids of six months' duration, unaccompanied by oedema of conjunctiva or proptosis. This boy was suffering from a musical lung as well, which gave the clue, the cause being a myxomatous mid-turbinal with polypii, on removal

of which by the late Dr. T. A. Sankaranarayana Pillay, oedema of lid disappeared in a couple of days. The drainage in the anterior ethmoidal and frontal sinuses has evidently caused oedema to disappear, closely followed by the disappearance of the patient which left us in the dark about the improvement or otherwise of the asthmatic condition.

Chronic empyema of the antrum has a toxic effect on the first division of the fifth nerve causing paresis and anaesthesia of the cornea and dendritic keratitis. I had to do tarsorrhaphy in a case.

Ocular headaches are always bi-lateral, rarely severe, accompanied by nausea and vomiting and it is practically a daylight headache. When the lights are turned out no use is made of the eyes—no eye-strain and no reflex pain. It is often associated with hyper-metropic astigmatism and latent hyperopia, following only nearly always near work. Prolonged and continuous eye-strain brings about also redness of conjunctiva, blepharitis and other symptoms of ocular involvement.

Vertigo is also a sign of sinus disease. The ocular vertigo is associated with diplopia disappearing by shutting one eye. The history of other eye symptoms like blurring of print while reading will help.

Exophthalmos due to sinus disease is mostly unilateral. Cause is suppurative sinusitis due to frontal in adults and ethmoidal sinus in children. These are mostly acute and inflammatory. Symptoms are characteristic. Pus will be seen rhinoscopically. We treat cases externally, symptoms are local and constitutional.

Non-inflammatory exophthalmos is due to mucocoele or pyocoele of the frontal or ethmoidal sinus gradual in onset without any signs of phlegmon, rather difficult to diagnose from orbital neoplasms. Direction of dislocation of the globe gives a clue to the sinus affected.

Frontal—forwards outwards and downwards.

Anterior Ethmoid—forwards and outwards

Posterior Ethmoid—straight-forwards.

Maxillary—forwards and upwards.

Recently, a Mohamadan boy of nine years turned up with a bluish elevation of the inner end of the upper lid said to be of a month's duration. It was quite soft, not suggestive of any neoplasm. The eye was proptosed forwards and downwards. Pressure on eye balls made the bluish swelling tense. There were no signs of inflammation. But the vision of the eye with proptosis was 6/36, the other being normal. Straightaway, I put

a needle into the elevation making it tense by pressing the globe backwards. The syringe filled itself to about 3 c.c. of dark blood and the eye went back. I bandaged the eye, gave him Ascorbic acid, tomatoes, oranges, etc. The next day, the eye was maintaining the normal position, except for a very very slight exophthalmo. This is a case of sub-periosteal haemorrhage due to scurvy. The boy recovered quite well.

A case of an intermittent exophthalmos of nasal origin due to varicocele at the apex of orbit; (the venous constriction being due to the spreading of osteoperiostitis, from the affected posterior ethmoidal cells,) has been reported.

Retro-bulbar neuritis and sinus disease.—Sinus disease causes papillitis, papilloedema, atrophy, or most frequently retrobulbar neuritis. The last presents the greatest difficulty in diagnosis. Examination of the field of vision shows characteristic central scotoma, enlargement of blind spot ending in a large oval scotoma, including and surrounding both fixation point and blind spot. These symptoms can be caused by the retro-bulbar neuritis due to other causes, such as multiple sclerosis, tubercle, syphilis, dental inflammation, rheumatism, tumours or cysts of the basal portion of the frontal lobe, lactation, acute infectious disease, metabolic disturbances, and Leber's Disease. But the ophthalmologist has in the eye no sign to distinguish the origin of retro-bulbar neuritis and the sinus affection cannot be definitely settled by X-ray unless exploratory operation is done. The affection of the nerve is due to (1) direct spread of the inflammation, (2) pressure by the walls of the dilated sinuses, (3) damage by toxins, oedema and congestion.

Shall we operate on the nasal sinuses in optic nerve disease of unknown origin?

The *pros* and *cons* of the association of retro-bulbar neuritis with sinus disease have to be considered.

Pros:—(1) Operation on posterior sinus greatly benefits the nerve condition

(2) The researches of Onodi and others have shown the close association which exists between optic nerve and sinuses and in particular the manner in which the optic canal can be encroached upon and narrowed by the sinus cells.

Cons:—(1) Sinus infections though common in children, retro-bulbar neuritis is not.

(2) No gross pathological changes in Sinuses opened though the retro-bulbar neuritis gets cured.

(3) Lillies in 225 cases of retrobulbar neuritis and optic neuritis at the Mayo Clinic included only one case in which the condition was actually due to sinusitis.

In Edinburgh, Traquair says true and genuine retro-bulbar neuritis is a very rare condition.

Treatment.—*T. A. B. Vaccine*:—Packing the nose with cocaine and epinephrine causes secondary hyperæmia of the mucosa, and the continued congestion of the mucosa of the sinuses after operation and the rise of temperature is in effect an auto-vaccination. So, similar results by packing two or three times daily with mild Silver protein or painting with 2% iodine the nasal mucosa, can be obtained. Such being the case, operate only if pus is proved to be in the sinus.

Tonsils and Adenoids.—If you remember the fifth cranial nerve is responsible for the sensation of the cornea, conjunctiva and posterior nares, the presence of adenoids is certainly reflexly responsible for the intense photophobia and lacrimation added to the locally existing phlyctenulosis. And so one cannot too well impress the necessity for careful examination of the throat in all cases of phlyctenular disease and the early removal of the tonsils and adenoids if present.

The Tonsils one of the triads along with teeth and toxæmia, are accused to cause various structural diseases of the eye such as keratitis, iritis, cyclitis, choroiditis, retinal hæmorrhages optic neuritis and retro-bulbar neuritis besides the functional disorders of asthenopia, insufficiency of accommodation and convergence.

Long ago a student of the Presidency College, aged 20, had ptosis and intense neuritis of the right eye only. Vision finger at 4 feet. Duration one month. No cause found except that he had chronic follicular tonsillitis, of the same size. Treatment with Sodium Salicylate and touching the tonsils with 10% Silver Nitrate, cleared the condition in 10 days.

Striking results in cases of Spring Catarrh by enucleating tonsils—reported by Brinton of Johannesburg.

A case of optic neuritis reported cured in 36 hours after ablation of tonsils.

Follicular conjunctivitis in children is very often associated with tonsils and adenoids.

Eye and Ear.—The only eye disease known to cause deafness is sympathetic ophthalmia and it is suggested that the pigment in the labyrinth is sensitised by the affected eye in the same way as the pigment of the sympathising eye. The ear disease causing affections of the eye such as sixth nerve paralysis and thrombosis of the cavernous sinus is Otitis Media. Sixth nerve is closely related to the bone and as it traverses the narrow cleft known as Dorello's space, it is readily liable to suffer from the effects of pressure. Dorello's space is a triangular Osseo fibro-canal formed by (1) apex of petrous temporal; (2) posterior clinoid process; (3) petro sphenoidal ligament. In this region sixth nerve is extra dural and is isolated from other nerves and it is easy to see how it may be pressed upon and involved in suppuration affecting the apical pyramidal cell resulting in Gradenigo's Syndrome (acute middle ear suppuration + intense headache + sixth nerve paralysis).

Purulent Otitic disease may be complicated by choked disc or optic neuritis and a distinction between the two should be made as it influences diagnosis, prognosis and treatment. In choked disc the cause is probably chemotaxis and if the ear is cured, the disc becomes normal.

In a case of double mastoiditis and symptoms of thrombosis of lateral sinus on one or other side, it was decided to operate on the side on which the choked disc was most marked and this proved to be the side of thrombosis. In optic neuritis of ear disease, the cause is purulent meningitis and cure is not possible.

If the third or the sixth nerve paralysis with an ipso-lateral otorrhoea accompanied by headache and vomiting is present in a case, auditory apparatus must be explored to determine the extent and limits of the disease.

I have a case of bilateral uveitis with deafness at present. It is said that degenerated uveal pigment acts on the pigment of the labyrinth by exerting its antigenic qualities, but in my case the deafness was quite early when the uveal pigment cannot have exerted its antigenic qualities. Besides Koyanagi says there is no pigment in the labyrinth and its physiological action is unknown.

It is said that sympathetic ophthalmia and uveitis are associated with Alopecia poliosis, (premature grayness), vitiligo and deafness. I have not met with vitiligo but in the present case I have on hand when I blistered the temples, the blisters that generally heal without loss of colour have turned out

vitiliginous (white patches). Deafness may be due to treatment with sodium salicylate.

There is another Syndrome blue sclerotic, brittle bone and deafness. The deafness is oto-sclerotic in type and labyrinthine in advanced cases. The cause of the syndrome is congenital deficiency or weakness of the mesodermal structures throughout the body and the deafness oto-sclerotic or labyrinthine must be of mesodermal origin.

Deafness is also seen where choroid is mainly affected both in inflammatory cases and in degeneration of high myopia. The cause is not definite, though in interstitial keratitis, along which deafness appears Spirochaeta is the exciting cause.

Pigmentary disturbances also go with deafness. Degeneration of retinal pigment has been demonstrated in waltzing mice which have defective labyrinths.

Deaf-mutism is associated with optic atrophy, retinitis pigmentosa and general disturbances of retinal pigment. The field of vision and light and colour sense show no abnormality worthy of note. This is astonishing having regard to retinitis pigmentosa which always goes with restricted field of vision.

Diphtheria:—A young boy of seven years had just recovered from an attack of mild angina with paralysis of soft palate. He walked into my office with an ataxic gait and his speech was affected too. He was referred to me because of sudden bad sight detected during convalescence after four weeks from the commencement of attack. The paralysis of accommodation was so complete as if atropine was used for four days. His distance required five dioptries and close vision an additional two dioptries.

Quinine:—Thin arteries, concentric diminution of field of vision, pale discs characterise quinine blindness. Some are amblyopic, others are absolutely amaurotic. The ischemia of the retina by the direct action of drug on the vessel wall causing starvation of the retina or the selective toxic action on the cells of the retina is the cause of the blindness. The aural symptoms of quinine poisoning go by the name cinchonism. They are temporary and pass off with stoppage of the drug.

Vertigo:—This is a very widespread symptom for which patients often go to the ophthalmologists with the idea that vertigo starts from the eye. It is a subjective symptom like headache with no objective changes.

This is finally an E.N.T. man's affair as it is due to irritation of the vestibular branch of the VIII nerve which is concerned

with our senses of equilibrium, oculo-motor control, orientation of body and extremities. Vertigo is a syndrome of sudden attack of dizziness, objective or subjective, feeling of nausea, loss of equilibrium and in-co-ordination with nystagmus. I can only enumerate the kinds and causes of vertigo attributable to the eyes.

Vertigo due to false localisation:—When one wears glasses for the first time, things appear at a higher or lower level than the usual especially with cylindrical corrections. Again, looking not through the centre of the thick glasses prescribed after cataract operation, causes a prismatic deflection, the sighted object appearing in a wrong place. Paralysis or paresis of the external ocular muscles cause very troublesome vertigo, of course disappearing on closure of the paralysed eye. Frontal sinusitis affecting the superior oblique giving rise to vertigo is seen and reported also.

False Movements of the Objects caused by False Localisation.—This is a greater cause of vertigo in the paralysis of eye muscles and this vertigo is very great—of course disappearing on closure of paralysed eye.

In the ordinary nystagmus originating in childhood no false movement occurs because the patient is accustomed to the nystagmoid movements and no vertigo results, but in miner's nystagmus, nystagmus due to ear and nystagmus due to disseminated sclerosis, false movements of objects and consequent vertigo do occur.

Vertigo forms a triad with headache and vomiting in intracranial increase of tension.

A sensation in the head combined with a feeling that everything is black before the eyes is due to the sudden changes of circulation in the brain occurring in anæmia when sitting posture is assumed suddenly and as a precursor of syncope and in arteriosclerosis of the arteries of the brain. Disorders of stomach cause vertigo which can be induced sometimes by pressure on the epigastrium.

Ear and Eye in Sea-sickness.—Is the eye or ear responsible? Is it labyrinthine stimulation or optic nystagmus that causes the sea-sickness? Personally I think optic or railway nystagmus is easily tolerated and not accompanied by depressive gastric phenomenon. I have travelled mostly in railways and have yet to see a railway sickness. On the other hand, during my sea-voyage, I met a number of cases of sea-sickness more in a

heavily tossed about light boat, than in a steady heavy boat. This observation may not be accurate. Is this due to the rocking and tossing movement of the vessels which is absent in the railway train which travels in a perfect plane, causing greater disturbance of the semi-circular canals? Another interesting fact in support of the ear alone being responsible in sea-sickness is that sea-sickness is caused by excessive motion during sleep or in the dark indicating that retinal stimulation is not necessary. Both eye and ear may be responsible as when a man is about to get sea-sick, it may be inhibited by looking steadily at the horizon-line. The matter is certainly more complex as it is well-known that over-eating or indigestible food increases the liability to sea-sickness. The eye is said to set up stimulation of the semicircular canals on the basis of an irritation of a definite band of sympathetic fibres said to exist between the eye and the ear though the ear may not be primarily responsible for the sickness.

Nystagmus is another symptom where the ear—and the eye—man may be consulted. Visual nystagmus is vibratory pendulum-like—the swing in the one direction being just as swift as that in the opposite. Aural-labyrinthine is a resilient nystagmus in which the eye makes a slow swing in one direction followed by a quick return.

Visual nystagmus goes with conditions causing bilateral amblyopia in infancy which I need not enumerate here. A person born totally blind never gets nystagmus. It is occupational as in miner's nystagmus due to insufficient light and consequently fatigue of muscles. Aural nystagmus is due to irritation of the labyrinth due to rapid rotation of the body, syringing the ear with hot or cold water or galvanisation of the ear.

We are not concerned with nystagmus of nervous diseases or poisons. Apparent movement of objects is a prominent and distressing symptom in miner's and vestibular nystagmus, also present, though not always, in nystagmus of nervous disease. Hence, vertigo is marked in miner's and vestibular nystagmus.

In this connection of experimental nystagmus being produced by instillation of hot or cold water into the ear, one is reminded of the preservation of reflex movements referable to semi-circular canals during paralysis of voluntary lateral conjugate movements.

Fix the object in the mid-line. Head is passively rotated in the direction opposite to that of the motor defect. It will be found that the fixation is maintained *i.e.* movements which

seemed impossible have actually been performed. Here, the path from the muscle nucleus to the cortical centre necessary for the voluntary act is interrupted, the track from semi-circular canals to the nucleus is intact and forms the afferent side of the reflex arc. Movement of the head sets up current in the fluid contained in the semi-circular canal, such as normally connected with certain directions of movements and these, in turn, excite reflex movements of the eyes. Nystagmus on instillation of hot or cold water into the ear depends on the soundness or existence of this reflex arc.

Trichinosis—The predominant severity of special symptoms viz. pain in the frontal region and oedema of lids turn these cases either to the aural or ophthalmic surgeon.

Eosinophilia, muscular pain and swelling, chemosis of conjunctiva and oedema of both lids in a pork-eating patient is characteristic of trichinosis.

Muscle shows encysted trichinae, within the cyst is the coiled worm.

Nasopharyngeal Tumours.—(Carcinoma, Sarcoma and Endothelioma) Generally located in the fossa of Rosenmüller—chief characteristic is to spread widely beneath the mucous membrane, so that involvement of the mucous membrane of the nasopharynx with ulceration and haemorrhage is late in the disease. The growth burrows in deeper planes spreading in all directions involving Eustachian tube, vault of the pharynx, sinuses, sphenoidal maxillary and pterygo-palatine fossa, the sphenoidal fissure, the orbit and the structures at the base of the skull. Lymph chains in the upper pharyngeal and cervical regions are involved. Symptoms are nasal and extra-nasal.

Nasal :—Bulging of the soft palate, nasal obstruction, dysphagia, haemorrhage, foul odour and taste, alterations of nasal voice, obstruction of the Eustachian tube, tinnitus and obstructive deafness.

The extra-nasal symptoms are early and the ophthalmologist is often the first to be consulted. Pain in the eye, forehead, side of the face and temporal region, tooth-ache, ear-ache, tinnitus, a sense of fulness in the ear, diplopia, blindness, proptosis, paraesthesia of the face, enlarged cervical glands, dysphagia, aphonia, hoarseness and metastasis in distant parts.

Nasopharynx should be examined in all these cases where there are inequality of pupils, involvement of V nerve, Horner's Syndrome (mycosis, ptosis and sweating of face and exophthalmos,

caused by paralysis of sympathetic) involvement of VI, VII nerve, Ophthalmoplegia externa, interna, exophthalmos, ptosis, papilloedema and papillitis, optic atrophy, and mass in the orbit-syndrome of the sphenoidal fissure (Paralysis of III, IV, VI nerve and the ophthalmic divisions of V nerve).

It is part of our work to recognise tumour of naso-pharynx. Many obscure cases in which there are pain in the head and involvement of cranial nerves naso-pharyngeal tumour must be considered. One can evolve an ocular syndrome of naso-pharyngeal tumour involvement of the V sympathetic, VI either alone or together with symptoms referable to the ear.

A word about intra-nasal dacryocystotomy:—

The situation of the lacrimal sac is outside the eye and outside the nose and it will be perfectly ethical for either the rhinologist or the oculist to tackle it. So far as Madras goes, I see it is monopolised by the ophthalmologists, though they cannot be proud of their results in dealing with dacrycystitis. The epiphora is a constant menace to the eyes from which the sacs have been ablated.

West's dacryorhinocystotomy in which an oblong piece of bone consisting of a flimsy piece of lacrimal and a tough piece of the nasal process of the superior-maxilla is removed, thereby laying open the floor of the lacrimal sac, subsequently excising a piece of the sac wall and stitching the edges to the nasal mucous membrane is an ideal operation indeed, though the noses of children and noses with deflected septum or any disease and the narrowness of adults may not admit of successful tackling.

My own method of avoiding this bug-bear or at least minimising it, is by leaving a thin strip of gauze leading from the wound to the nose. This can be done by first dilating the passage after removal of sac with a pretty stout dilator and then driving the gauze into the meatus from the wound with the help of a probe. The nasal end of the gauze will be hanging loose in the nose which is of course plugged with cotton. This of course is kept for nearly 10 days and finally removed. It can be replaced in 2 or 3 days by fresh strip by a trickish manœuvre. Make a hole in the gauze end at the wound by insinuating a probe or kuchi. Introduce just the end of the fresh strip. Pull the nasal end of the original one and this will bring down the long fresh strip into the nose.

A Few of the Nervous Disorders Seen by an Ophthalmologist

BY

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THE following is a short but sketchy and by no means an original attempt at the enumeration of a few nervous disorders of interest alike to the general practitioner and the Ophthalmologist. The diseases are treated from the point of signs and symptoms, beginning from those obvious on inspection.

First, is the patient with pain and headache limited to one half of the head, coupled with perhaps a few visible eruptions on the forehead. The eye may be a little congested. A closer examination shows a few more vesicles in the scalp, the hyperaesthesia of the region affected, and the anaesthesia to light touch of the cornea.

An early diagnosis of the case, as of Herpes Zoster, will save the eye from damage. A confusion in severe cases with Erysipelas may be avoided by remembering the strict hemi-distribution of the former.

Next in the obvious of disorders is the patient with droop or ptosis.

In the Myasthenic, ptosis is often the first symptom and monocular at that. The abnormal muscular fatigue, the increase of the distress of the symptoms with the duration of the active and waking periods and the absence of any abnormal reflex or sensory signs, are diagnostic.

It is a pleasure to watch the relief of the trouble, albeit temporary, after an injection of prostigmine.

Ptosis of some degree is pathognomonic of Tabes and occurs as an isolated symptom or with paresis of other muscles. The drooping lids with the compensatory wrinkling of the forehead constitute the 'Tabetic Facies'.

Ptosis is common in Encephalitis Lethargica, the elevator muscle of the eye lid being the favourite seat of selection of the virus in nearly 50% of the cases.

Ptosis is usually the first to appear in 'chronic progressive ophthalmoplegia Externa of Willbrand and Saenger'—a slowly

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progressive bilateral lesion beginning early in life and gradually progressing to total or nearly total ophthalmoplegia externa.

Ptosis is one of the symptoms of Horner's syndrome to be referred to later.

Next in the order of apparency is lagophthalmos. It may be due to a peripheral or nuclear or local lesion.

Of the peripheral one, Bell's palsy is so well known.

Of the nuclear conditions, two syndromes merit mention.

FOVILLE'S SYNDROME:—A lesion of the VI and VII nerve nuclei and the mesial fillet and the spinophthalmic tract, resulting in paralysis of the homolateral facial and external rectal muscles and contra-lateral hemianaesthesia.

MILLARD-GUBLER'S SYNDROME:—Total paralysis of VI and VII nerves of one side and crossed hemiplegia, the lesion being lower pontine.

Local lesion.—Lagophthalmos is one of the complications of anaesthetic leprosy with the distressing sequelae of corneal damage partly due to exposure and partly to anaesthesia.

The next symptom that springs to notice is Nystagmus. Disseminate sclerosis, Friedreich's ataxia and Syringomyelia and cerebellar lesions be they neoplastic or inflammatory are the disorders commonly exhibiting Nystagmus as a prominent symptom.

Next, mention must be made of squint due to paralysis of any one or group of extra-ocular muscles.

Commonest amongst these is paralysis of the External Rectus.

In children it is the result of a birth injury. Involvement of the VI nerve has been already referred to in the two previous syndromes. Yet another is Gradenigo's where acute otitis media is followed by abducent nerve palsy and intense unilateral headache. The VI nerve is involved in the apical abscess of the petrous bone.

Abducent palsy is not infrequently due to cold or rheumatism and followed by complete recovery. These are associated with tenderness on pressure of the globe and slight proptosis.

Besides parasymphilis, syphilis accounts for nearly a third of the cases involved of the III, IV and VI nerves. However, congenital syphilis is markedly free from any ocular paresis. In addition to syphilis any one of the following may be responsible for a lesion of the III, IV and VI nerves.

Intracranial :—Tumours, haemorrhages, aneurysms, arterio-sclerosis, polioencephalitis, disseminate sclerosis.

Orbital :—Grave's disease, growths, infections etc.

Trauma :—Fracture of skull.

Toxic :—Diphtheria, diabetes, botulism etc.

There may be no discoverable cause, the lesion then being ascribed to cold or rheumatism.

In a few, it occurs as a congenital or hereditary affection.

Weber's Syndrome due to a lesion of the junction of the pons and the crus cerebri is marked by paralysis of the III Nerve and contralateral hemiplegia and supranuclear facial paralysis.

BENEDIKT'S SYNDROME :—III nerve paralysis with tremor of the opposite limbs due to a lesion of the red nucleus.

The troublesome symptoms of these extraocular palsies which the patient seeks relief for, are diplopia and vertigo, unless there is complete paralysis of the levator palpebral muscle and the globe is hidden from view. In addition there is a characteristic compensatory tilting of the head.

Next come the disorders of the pupil, either myosis or mydriasis.

Myosis met with in Horner's Syndrome is an important symptom of paralysis of the cervical sympathetic and accompanied by ptosis, narrowing of the palpebral fissure, vasodilation of the vessels of the side of the face and head and absence of perspiration. This may be caused by tumours of the medulla, pressure by growths, enlarged glands or aneurysms in the thorax, cervical rib, goitre, glands and aneurysms in the neck, syringomyelia etc.

The Myosis of Argyll Robertson Pupil :—"The pupil is small, constant in size, unaltered by light or shade, reacts promptly and normally in convergence" with very few exceptions is due to syphilis.

"Argyll Robertson phenomenon is an occasional occurrence in diffuse toxi-infective states other than neuro-syphilis"

Next is the dilated pupil of which 2 illustrations may be given :—

- (1) The tonic pupil with or without absent knee jerks. The latter—Adie's syndrome—is met with almost exclusively in women between the 2nd and 3rd decades of life. They quite suddenly develop an enlarged pupil with attendant visual disturbance due to deficient focussing or accommodation superficially

examined the pupil is found fixed to light and accommodation, but if patient persists in staring for 20 or 30 minutes, the pupil contracts more than its fellow and takes a longer time to dilate. There is no reaction to light. The cause is not known.

- (2) The fixed pupil :—A dilated pupil which reacts promptly to miotics and is inactive to any other stimulus—the lesion being in the efferent pupillary pathway.

Paralysis of accommodation of nervous origin is met with mostly in children after an attack of diphtheria, commencing about a fortnight after the attack and disappearing within a couple of months. Children are hardly ever likely to complain of that disability and as such is probably missed more often than not.

Next mention may be made of the affections of the optic disc.

Sudden loss of sight of one eye in a young adult is due to acute retrobulbar neuritis which again is a manifestation of disseminate sclerosis. Probably that may be the one and only symptom of the disease and if the case is followed on into later life perhaps even upto 15 or 20 years, is bound to develop into the well established disease.

Any history of fleeting diplopia with transient loss of sight, should remind one of disseminate sclerosis between the age periods of its incidence *i.e.*, 15 to 40 years of age.

Optic Atrophy is met with amongst the young in Amaurotic Family, idiocy and allied disorders like cerebromacular degeneration and in the hereditary Ataxias and Juvenile Tabes.

Atrophy of the primary type is seen in Tabes often 15 or 20 years before the locomotor symptoms *i.e.*, may be the first sign of Tabes.

Any grade of primary atrophy may be met with in Disseminate Sclerosis.

About 8% of the general pareitias show optic atrophy.

"Besides Tabes and G.P.I. Syphilis is responsible for primary atrophy as an independent para syphilitic manifestation."

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Some Common Nutritional Disorders of the Eye

BY

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As the economic condition of the villages is worsening and due to a false notion that rice is a diet of superior class of people, whiter the rice, higher the scale of refinement, one now-a-days comes across a large number of diseases traceable to malnutrition. If one examines very carefully all the patients attending an out-patient clinic, he can find that up to 50% of their condition is due mostly to poverty and in the case of the middle and upper classes, to rich but ill-balanced diet.

Eye being the organ through which malnutrition is first revealed, eye clinics have been the first to draw attention to the importance of Vitamins. In our eye clinic, every second child admitted suffers from one or other of the deficiency diseases. Xerosis and night blindness leading on to keratomalacia, angular conjunctivitis, blepharitis and ulcers of the cornea going on to hypopyon ulcers are among the commonest. With the present-day clearer understanding of different types of vitamins, we are able to tackle them more successfully. Some of the deeper affections as choroiditis, some forms of optic and retinal atrophies associated with night-blindness and some types of cataracts are also associated with a-vitaminosis. With these are associated general affections as diarrhoea, dysentery, angular stomatitis, running of the nose, otorrhoea, dry skin, lichen spinulosus, glossitis, enlarged tonsils, pharyngitis etc.

Xerosis of the Conjunctiva.—A disease common among children; first attention of the parent is drawn to it when the child at night finds it difficult to move about and feels for the objects.

On examination, the bulbar conjunctiva is found to have lost its glistening appearance, a dirt-like triangular patch is observed on either side of the cornea, more marked on the temporal side, the colour getting deeper with the intensity of the affection. This area shows a wrinkling and the whole has the appearance of the outer covering of an onion, which could be better observed if the conjunctiva is moved. This is followed by a pigmentation of

the conjunctiva with white foamy patches at the angles of the palpebral fissures, more marked on the temporal side.

In more advanced cases, the cornea loses its lustre, turns yellowish and goes on to keratomalacia.

Night blindness is one of its early symptoms. On a systematic examination of children from several schools, it was found that about 5% of them have objective signs as xerosis and night-blindness. In vitamin A deficiency, the visual purple fails to reform at the normal rate. This failure is discovered by a dark adaptation test, in the dark room. On a systematic examination of the above children it was found that about 20% of the smaller school-going children showed this symptom. I found the village schools showed a higher percentage. The vision in the daylight is normal as well as the fundus. But in more advanced cases general health is low and vision defective which improves rapidly with proper dieting. There is no definite change in the fundus, except a little discoloration often called slate fundus. Sometimes this follows prolonged illnesses like typhoid, measles etc., and improves with the general health.

A study of these cases by Dr. H. K. Indra of Calcutta showed that the incidence of xerophthalmia in children is about 16 times as high as adults and that whereas in adults only $\frac{1}{5}$ th of xerophthalmia develop keratomalacia, in children if neglected, nearly half of these pass on to this stage. Children respond quickly to treatment, but in adults it persists inspite of prolonged treatment.

From an experimental study on rats by various experts and from clinical observation it has been proved that deficiency in vitamin A is the cause and the role of vitamin D is still a doubtful factor. It is prevalent among the rice eating races; perhaps the real cause is not rice as such but several secondary causes as over-milling and the poor keeping quality of undermilled and hand pounded rice etc. Calcium deficiency also is an indirect contributory cause and it has yet to be investigated whether calcium is necessary for the proper assimilation of vitamin A.

Among other causes mention should be made of:—poor quality or absence of mother's milk, bad substitutes for mother's milk, want of sufficient fat in food, living in congested, ill-ventilated, damp surroundings, lack of sufficient exposure to ultra-violet rays of the sun, which is an important link in vitamin absorption more associated with D.

Prognosis in these cases is quite hopeful provided the patient is able to change his diet to a more balanced one and also his surroundings.

Keratomalacia.—This is the last but one stage of vitamin A deficiency. To be more accurate it is a complex where not only vitamin A deficiency but imperfect assimilation of vitamin, due to toxemias and pathological changes brought about in the mucous membranes of the system also play a roll. This condition is very rare in the western countries and among North Indians who take whole wheat products. This is on the increase in Southern India; this is evident from the fact that about ten years ago I used to see one case a month, on the average in an out-patient clinic of 150 a day; but of late, the number of out-patients being 200, I meet on an average one a week. In India more people close their eyes from this disease than from Gonorrhœal Ophthalmia.

Its onset may be in the form of xerosis described above and going on to necrosis of the cornea, the cornea melting like wax within 24 hours; sometimes the cornea melts within a few hours. It is very common among children below five years, generally, the history being that the mother is dead or the mother became pregnant before the child weaned. It is not uncommon in adults as a sequel of chronic debilitating diseases and in the case of women in latter months of pregnancy of early purpereum when sufficient iron and calcium are not supplied.

It is not uncommon that keratomalacia sets in without xerosis. A faint gray haze makes its appearance usually on the temporal side of the cornea. It enlarges and the surface of the cornea becomes very hazy, an yellowish infiltrate makes its appearance and a hypopyon develops. There is no congestion or lachrymation though at times lachrymation is excessive. The cornea melts and sloughs away. Though the general condition is low and susceptibility to infection is very great, very few of them go on to panophthalmitis. Generally, it begins in one eye and the second follows within a day. The skin is very dry, patient is very ill, diarrhœa supervenes followed by pneumonia and death. After the patient has come under treatment and diarrhœa is arrested, if diarrhœa recurs, it invariably ends in death.

Though prognosis is grave, if brought under immediate treatment and progress kept up, the cornea clears up and useful vision is obtained after an iridectomy.

Angular and sub-acute conjunctivitis, associated with stomatitis, whitish fissures at the nasal orifice, along the borders of the lips, dentures along the borders of the tongue, bad blepharitis are all intimately related to a-vitaminosis. These are found in

groups of children in orphanages and poor houses, in famine-stricken areas and poor villages. In my experience these improve with the addition of more vegetables and vegetable oils and fat to their usual food. Yeast also has a specific effect on these conditions.

Treatment.—*Preventive* :—"Prevention is better than cure" is an old and valuable dictum but more difficult to practise. One is very slow to adopt preventive measures but when once the disease sets in, serious attention is paid and money is spent freely to get out of the danger often incurring debts out of proportion to the capacity of the individual. This preventive work is not only the province of the Health Department but also belongs to the Medical Profession as well as the general public including politicians. The whole system of dietary has to be reformed and this is very intimately connected with economics. So, the politician, general public, health staff and medicos have all to join hands to fight the growing nutritional disorders of the nation before they assume dangerous proportions. I undertook to write this article, more to emphasise this aspect of the question. I shall leave out of discussion the economic aspect with only this remark, *viz.*, that duty is laid upon every Indian Doctor that he, instead of copying the western system of dieting from western text books, evolves a system of dieting for Indian patients suitable for different diseases and as far as possible within the means of average bread winner. In these days of unemployment, Allopathic doctors could improve their positions by working along these lines which will mean more patients drawn to the Allopathic doctors.

The second important question is making the living quarters and the surroundings of poorer people cleaner and more fit for healthy living. Here doctors could advise and educate patients and at the same time help the health staff. This is part of our civic duty.

The third is a study of different Indian Diets and their nutritive value and to suggest to the patients their relations and friends a balanced diet for their adoption. Plenty of propaganda work has to be done along these lines. What is the use of giving a prescription with costly proprietary medicines—very valuable, but the patient is not able to purchase it. The following are some of the factors to be considered in proposing a balanced diet. The Nutrition Research Institute, Coonoor, has prepared a Health Bulletin No. 23, published at Delhi by Government. It contains valuable data which can be supplemented by those of Bangalore

and Calcutta science institutes and on animal nutrition by the Agricultural College, Coimbatore. A mixed diet is very essential—Kambu (Tamil) Bazra (Hindi) is the richest cereal grain—even superior to wheat—in Protein, Fat, Iron and Vitamins A and B. It is cheap, keeps well after cooking, taken with curd or buttermilk is very delicious and not hard to digest. Fermented water which has a good effect on Xerosis, stomatitis and glossitis because of its yeast content is easily obtained from it—Ragi is the next best, it is poorer than Kambu but its great value is its calcium content—which is three times as much as that of other grains—hence a very valuable adjunct to diet for growing children and Tuberculous patients. The above two, supplemented with a few vegetables, fruits and greens, makes a cheap balanced diet perhaps within the means of average villager. Rice when de-husked with a wooden chaki has all its pericarp preserved and is the most nutritious. Next best is hand pounded rice—the economy of it when done at home is that the coarse bran is given to the cow—better milk; finer one to the fowls—more eggs; and the finest mixed with jaggery to the children who will surely be healthier than at present.

Among the vegetable oils, Gingelly oil is richest in calcium and cocoanut oil the poorest—Among leafy vegetables—Agathy, Drumstick leaves and Amaranthas should form part of the daily food—by boiling too much they lose their taste. It is enough if they are steamed, hence vitamins are not spoiled and they also move the bowels easily. Among the fruits, Dates are the best. If more attention is paid along these lines, it must be possible to prescribe easily assimilable cheap Indian diet for diseases like typhoid, pneumonia etc.

Meat in different forms, egg and fish are very valuable in a balanced diet. Yet if there be any sentimental objection to a meat diet it is possible to have a pure vegetarian diet still well balanced.

Treatment.—As the line of treatment follows an attempt at supplying the deficiency, I just describe a common line of treatment for the above conditions, followed in our clinic. The quantity and intensity of the treatment has to be varied according to the needs of the individual cases.

1. Cod liver oil still holds the field. It is richer in vitamins A and D, the cruder the better. It is enough if it is given in teaspoon doses. Red palm oil whose cerotone content is equal to vitamin content of Cod liver oil is a good substitute in such cases where cod liver oil produces diarrhoea or is not tolerated. This is better tolerated and is given in the form of an emulsion with Trag co. and flavoured with Oleum menth pip. The dose is 30 to 60 minims repeated twice or thrice a day. This costs only

half that of cod liver oil and is at present available from Tata Oil Company, Ernakulam in one gallon tins.

In delicate conditions these can be replaced by Ostelin, halibut liver oil, Preparation A., adexolin, Vigintol and similar preparations which all claim to be richer in vitamin A. These have to be supplemented by calcium in the form of lactate or gluconate orally or by way of injection of one of the calcium preparations in the market. Iron is an important addition. This is given in the form of syrupus ferri iodide, fersolate or col-iron. These are well tolerated and have a constipating effect on the diarrhoea. Diarrhoea and lung complaints and other complications are treated according to their cause or symptomatically. Where liver seems to be affected one of the liver preparations campolan or hepatex etc. should be given.

Dieting is very important. In milder cases patients easily take to the diets mentioned under preventive treatment. In serious conditions orange juice and gulcose is the only diet and if there is no diarrhoea milk is added. These are given at frequent intervals. As the condition improves curd or buttermilk and marmites are added with one meal of rice a day. Skimmed milk is cheaper and effective.

Treatment with sheep's liver is the line followed in India from time immemorial for night blindness. This holds good even now. Meat, eggs, fish and milk are valuable adjuncts to treatment.

In cases of angular stomatitis etc. under Dr. Aykroid's suggestion, I had been giving 10 to 15 grains of yeast repeated thrice a day and this has wonderful effect. The same result is also obtainable by giving sour rice, kambu or ragi water and sweet toddy. As mentioned, more oil and vegetable added to the usual diet brings about a cure also.

In all the above conditions, I give as a routine, if the lung is not involved, injections of the Sodium Morrhuate solution twice a week beginning with a dose of 0.10 for children and 0.20 for adults. This dose is increased by 0.10 c. c. at every subsequent injection if there has been no reaction till the maximum of 1.00 c. c. is reached. This is kept on until the whole condition improves.

Local Treatment.—Keratomalacias are treated as ulcers of the cornea. Xerosis cases clear with yellow oxide of mercury ointment 1 to 2 %. Instillations with cod liver oil or halibut liver oil or ostelin are also effective especially in advanced cases. In angular and blepharo-conjunctivitis, some preparation of Zinc salt is effective.

Eye Symptoms in General Practice

BY

T. S. Duraiswami, L. M. & S.

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Introduction.—The General Physician is accustomed to regard the eye as a more or less isolated organ, subject to its own peculiar diseases with which he has no professional concern, pertaining exclusively to the ophthalmologist. This is a mistake. Quite aside from its own local pathology, the eye, from its intimate relationship with the general and special functions of the body, frequently furnishes useful indications of certain important diseased conditions. Various visual defects, ophthalmoscopic appearances, different types of hemianopsia, abnormal changes in the visual field, and sudden occurrences of squint or blindness, are all of inestimable value in the detection of systemic disorders, their differentiation and interpretation. They not only serve in many instances to establish diagnosis, but furnish indications for treatment, and offer valuable data for prognosis. It is a proverbial saying that "the eye is the window of the soul", and it may equally be said, that it is the window of the body. In no other part of the body can the physician observe, with his own eyes, the living pathology, functional and organic, of circulatory and nervous diseases; and through no other organ do systemic disorders manifest themselves with such sensitive subjective symptoms.

It is with the hope of encouraging the general practitioner to avail himself more generally of this invaluable means of diagnosis and prognosis, and of helping him a little in searching for, and interpreting the ocular signs of disease, that this little paper is offered. It does not pretend, of course, to cover the whole ground, but only to serve as a guide.

I. Subjective Eye Symptoms.—I am giving here some interesting diagnostic facts gleaned from my own experience (from my practice) of over fifteen years as an ophthalmic physician and surgeon concerning the value of subjective eye symptoms in systemic conditions.

1. *Corneal sensitivity*:—This is best tested by gently touching the cornea with a piece of cotton. If sensitivity is diminished or lost, and is unilateral, we may suspect tumour of the cerebellum on the same side; if so, we should next look for nystagmus and staggering gait.

2. *Scintillating Scotoma*:—This is a rather common condition, and, while not a true neurological manifestation, so far as brain disease is concerned, yet is of such frequent occurrence, that it is worth considering. It is characterized by vertigo, sparkling lights before the eyes, and contraction of the field of vision. It lasts from fifteen minutes to half an hour, after which it abates and vision returns to normal. The etiology is an *angioneurotic* disturbance of the circulation, and its most frequent cause is some constitutional disturbance, such as constipation, gastro-intestinal toxæmia, or some focal infection such as sinusitis, or bad teeth.

3. *Nystagmus*:—This is usually divided into two special types, namely, (a) *ocular* nystagmus and (b) *vestibular* nystagmus. (a) Ocular nystagmus has a special character, in that both movements of the eyes occur with equal rapidity and amplitude of excursion, and are never rotary. (b) Vestibular nystagmus is distinguished by rhythmical movements of unequal velocity, or has two components, quick in one direction and slow in the other, and is the one, with which we are here concerned. Nystagmus may be produced by various conditions, as alcohol, tobacco, intestinal intoxication, seasickness, brain abscess, brain tumour, meningitis, fracture of the skull, mastoiditis, and labyrinthine disease.

[In spontaneous nystagmus, which is always pathological, the rotary element is usually present with nystagmus in another plane. The quick component is toward the diseased side, unless the labyrinth is diseased or destroyed, when the quick component is toward the sound side for a few days, after which it subsides. But if it returns to the diseased side, meningitis is present on that side.]

The presence of nystagmus may be easily overlooked unless you have the patient look to the extreme right or left. Another way to determine a pathological vestibular nystagmus is by turning the patient in one direction for ten seconds, and if the nystagmus lasts only half as long as that produced by turning him in the opposite direction for ten seconds, we can reasonably assume some intracranial complication.

4. *Diplopia*:—Double vision is practically always, with a very few rare exceptions, due to some degree of squint, which, however, may not be sufficiently marked to show itself as an obvious squint, and may not be permanent, but may come and go. In children, diplopia is generally the manifestation of some dysfunction of the ocular co-ordinating mechanism due to

refractive or fusing errors. But in adults, and especially in persons of middle or past-middle age, who have never before had any ocular troubles, the sudden occurrence of diplopia is almost always an indication of some degree of ocular paralysis, either as the result of some grave brain lesion, (*e. g.* tumour, abscess, etc), or a disease of the nervous system, or the advance of *arteriosclerosis*.

II. Ocular Diseases.—Certain eye diseases have long been known to be local or focal manifestations of systemic disorders of which the commonest are :

1. *Iritis*:—This eye disease is never primary. It is always a sign of some deep-seated constitutional disease. The diagnosis usually lies between rheumatism, syphilis and tuberculosis. Rheumatic infection can usually be determined by the history of the case. Syphilis is confirmed, but not necessarily ruled out, by a Wassermann test or Khan's test or both, positive or negative. Tuberculosis, as it affects the eye, is generally more intractable than syphilis, the nodules lasting longer and the Van Pirquet's test will help to solve the problem. It must not be overlooked that iritis may also be a symptom of gonorrhoeal infection.

2. *Glaucoma*:—The cause of glaucoma is still unknown. The reference and the title of this means the discussion will be very wide, and hence will have to be dealt with in a separate paper. As predisposing causes many conditions are recognised, such as hypermetropia, shallowness of the anterior chamber and narrowness of the filtering angle, and small size of the cornea. Some of these conditions, for example, shallowness of the anterior chamber, are often the result rather than a cause of glaucoma, and others are manifestations of imperfect development of the eye. None of these conditions causes glaucoma, though they render its occurrence more probable.

The chief features present in glaucoma cases are, first, that the patients are elderly, and secondly, that they are of an anxious, nervous temperament. When glaucoma occurs in the young, there is usually a family history, indicating the predominance of hereditary factors. It is a disease which is common in all classes of society, and is therefore largely independent of nutritive or hygienic conditions. It is also important to note that both eyes are always affected, though the intervening time interval may be considerable. We must therefore regard glaucoma as a disease of the patient and not a disease of the eye, and the

recognition of the glaucoma patient, before he exhibits a glaucomatous eye may, in the future, become an important, though, no doubt, a difficult and uncertain part of the early diagnosis of the conditions. At the present time the diagnosis can hardly be made in this stage unless accidentally, during routine examination.

3. *Phlyctenular Conjunctivitis or Keratitis*:—When ulcers and syphilis can be ruled out, phlyctenular eruptions of the eye can almost always be attributed to deficiencies of diet, particularly shortages of protein and vitamins of all kinds. I find in my experience, that correction of the diet by the addition of proper amounts of protein, and plentiful supplies of the various vitamins, not only clear up the phlyctenulosis of the eyes but greatly improve the patient's general health.

III. Ophthalmoscopy.—No attempt will be made here, of course, to give any instruction in the use of the ophthalmoscope, which can be obtained from any book on the subject, but merely to mention a few of the outstanding signs of systemic diseases seen in the fundus of the eye.

1. *Optic Neuritis*:—This consists of an inflamed condition of the nerve-head or optic disc, the vessels which emerge with the nerve, being swollen and tortuous, and spreading out over the eye-ground, so as, almost to obliterate the circular boundary line of the disc. Often, it indicates a brain disease, such as meningitis, or a general inflamed state of the entire nervous system; but quite often, too, indicates some infected and congested condition of adjacent structures, *e. g.*, of the sinuses.

2. *Choked Disc*:—In this condition the nerve head appears itself swollen and balloon-like, surrounded by swollen dark-coloured veins, the arteries being small and almost empty, and the surrounding eye-ground rather pale than otherwise. Choked disc is invariably the sign of grave intra-cranial disease, *e. g.*, brain tumour, abscess, etc.

3. *Optic Atrophy*:—In any form of atrophy the appearance of the disc is extremely white. Its exact hue varies, depending on circumstances,—chalky, snowy, pearl-like, bluish, greenish, etc. But always it is very white, and has lost its soft healthy lustre. The atrophy may be the end-result of a choked disc (as stated above), that is, atrophied by strangulation, in which case it is usually a dirty greyish-white, with indistinct edges and narrow, tortuous vessels. But quite often, it is a sign of some general degenerative nervous disease, *e. g.*, *tabes dorsalis*, or brain

disease, in which case it is of a brilliant pearly white, very prominent, with the vessels almost normal.

4. *Sclerosis*.—This condition pertains to the arteries alone. They become opaque, and often white in appearance, due to light reflected from their opaque walls. In places they are practically empty. The hardened arteries, pressing upon the veins where they cross, obliterate the latter, so that, they appear interrupted where the arteries cross them. There are areas of pallor over the fundus where the nutriment is cut off by the obliteration of the vessels. It is, of course, an indication of a general arteriosclerosis.

5. *Hæmorrhages*.—In certain conditions the ocular arteries give way and produce hæmorrhages into the retina. Sometimes there is one large hæmorrhage, but more often numerous small ones. The ophthalmoscope shows a cloudy retina, with variously shaped red blotches scattered over the ground,—round, flame-shaped, linear, stellar, and even irregular. These are the appearances seen in profound systemic diseases which raise the blood pressure and degenerate the vessel walls, such as nephritis, diabetes, malaria, etc. They are often accompanied by albuminuria, glycosuria etc.

6. *Anæmia*.—This is not always easy to recognize, since eye-grounds differ so much in different individuals. The arteries and veins are equally affected, hence, their relative sizes remain the same, but both are much smaller than normal, often exactly alike and pale. There is a general pallor of the entire fundus and disc, but the lustre of the latter is not lost. The appearance indicates a general condition of anæmia, and is particularly marked in pernicious anæmia, where the colour of the fundus is a peculiar saffron yellow.

Strabismus (Squint).—Squint is often a manifestation of ocular paralysis, due to some systemic disease. But it is also, of course, quite frequently the result of some dysfunction in the co-ordinating function of the eyes, due to refractive error, etc.

Paralytic Squint is usually the result of an infectious disease, such as diphtheria, which poisons the peripheral nerve supplying the ocular muscle, or to a central nerve lesion in the brain, due to syphilis, tuberculosis, meningitis, brain tumour, etc. Paralytic squint, appearing suddenly in adults is almost always of brain origin.

Pupil.—Because of the intimate connection of the iris with the sympathetic and the central nervous systems, the size,

behaviour, and shape of the pupil furnish an extremely valuable data in health and disease as to the state of the body, manifested through the two nervous systems, of which only a brief summary can be given here.

In health, the shape of the pupil is almost perfectly circular. Distortions are practically always due to local mechanical causes, the dragging of the iris in one or more directions, either by reason of being caught and stuck down to the lens or the cornea by exudates, or being trapped in the wound of an ulcer or operation.

The size of the pupil in health is a difficult thing to specify, because it is continually varying under physiological stimuli. What may be called its absolute or constant is a personal idiosyncrasy. Since light, which is the principal agent of stimulus for contraction, is a more or less constant factor, it is to the action of the sympathetics, or dilators, that we must look for an explanation of individual variations in the size of the pupils under similar conditions. In general, it may be said, that sensitive neurotic persons, whose sympathetic system is easily excited, and reacts readily, have larger pupils than phlegmatic individuals.

Much more important, are the variations in the size of the pupils due to abnormal or pathological causes, concerning which the following rules may be laid down:

Mydriasis (dilation of the pupils) is caused by any condition which (1) paralyzes either the central or peripheral endings of the third nerve, or the short ciliaries, (2) inhibits the function of third nerve, through the brain, or (3) stimulates the sympathetics. Example of the first kind are tumours of the brain or along the third nerve, toxins of acute infections such as diphtheria, etc., and the action of certain drugs, notably atropine. In the second group are fevers, comatose conditions, excited states of the brain. Of the third variety are pain, sensory irritation, and abdominal diseases.

Myosis (contraction of the pupil), on the other hand results from influences which (1) irritate the third nerve, or (2) depress the sympathetics. Photophobia, from whatever cause, meningitis, and certain drugs such as eserine, are instances of the former; spinal diseases, aneurysms (by pressure on the nerves) and opium, of the latter.

Inequality.—Normally the two pupils are equal in size. Inequality is practically always due to interruption of the motor-oculi nerve path, either central or peripheral. Central causes are

brain tumours, brain syphilis, tubercle of the brain, and the like. Peripheral causes are injuries to the eye and toxins from acute infections. Occasionally, one pupil is dilated because of pressure on the sympathetics by an aneurysm. It must not be forgotten that diseases which interfere with light stimulus of the retina, (cataract, optic atrophy etc) will sometimes produce unilateral dilation.

Pupillary Reactions.—In health the pupil reacts with great quickness and sensitiveness to stimulus on both sides of the nervous circuit, i. e. to light and to sensory stimulus. A sluggish or immobile pupil may, of course, be due to adhesion of the iris to the lens or cornea, but usually, it is a sign of paralysis of the third nerve, central or peripheral. Most of these cases are those of cerebral syphilis.

The Argyll-Robertson pupil, in which the pupil contracts to accommodation, but fails to respond to light, occurs in locomotor ataxia, myelitis etc., and is supposed to be due to involvement of Meinert's fibers.

Wernicke pupillary reaction is a test for determining location of a suspected lesion in the optic tract, employed in cases of hemianopsia. If the light reflex is present in the blind half of the eye the lesion is back of the optic thalamus; if not, it is in front of this ganglion; because at that point the reflex and optic paths part company.

Hippus, a periodic contraction and expansion of the pupils, is seen in psychic diseases, such as mania, hysteria etc.

Visual Field Charts.—It is not to be expected, of course, that the general practitioner should be an expert in exploring and interpreting the visual field, or even, that he should make exact and exhaustive chartings. Like all other diagnostic procedures, it has almost unlimited possibilities, requiring long experience and painstaking patience, to which physicians, under present conditions, cannot devote the necessary time. Perhaps he cannot and will not make field chartings himself at all, but will have them made for him, just as, he has his laboratory work done for him. But it does not follow that he should be ignorant of or utterly neglect this most valuable diagnostic measure.

Therefore this must be treated at some special length in a separate paper.

Ocular Signs in Pregnancy.—Here I may point out one or two valuable diagnostic indications to be gained from the eye in

the pathology of pregnancy. The hæmorrhages into the retina which occur in albuminuria gravidarum are, of course, well-known to all, and differ in no respect from those which occur in any case of nephritis, and which are mentioned under the heading of ophthalmoscopy.

Pernicious Vomiting of Pregnancy.—The Ophthalmoscope often affords great assistance, in differentiating simple reflex vomiting of pregnancy from the more severe types of pernicious vomiting which occur in pregnant women. In the former case the fundus of the eye manifests no definite variation from normal but in the severe toxic type, even in its incipency, the ophthalmoscope will usually show a halo of cloudiness around the nerve head, due to oedema, often extending to the entire retina.

Eclampsia.—This is such a distressing condition, that any definite warning of its threatened incidence is of great practical value. The Ocular indications are at times strongly prophetic and serve to put the physician on his guard. The earliest danger signals are: defective vision, spots before the eyes, coloured lights, and even sudden blindness. The ophthalmoscope shows nothing special, but the presence of the pupillary light reflex, proves, that for the present the temporary pathology is confined to both visual areas of the cerebral cortex. Under such conditions, and with possible evidences of renal insufficiency, I should recommend that pregnancy be terminated without delay. If matters are allowed to take their course, other portions of the cerebral cortex will become similarly involved, with coma and eclampsia resulting.

Ocular Manifestations of Thyroid Dysfunction.—The relation of the eye to the thyroid and its disorders has long been recognized, and principally exemplified in the protruding eyeball of exophthalmic goitre. In this connection, I would point out a type of eye-strain which is peculiar to thyroid dysfunction, differing in its manifestations from ordinary eye-strain.

"There is," of course, "the red lids, headache, attacks of fatigue, bad temper, irresponsibility, and inability to work. And in some cases eye fatigue is not present at all. In the instances of ocular vagotonic types, a special case of spasmophilia is indicated, from one end of the body to the other, from the pylorus down through the intestines, gas, belching, hyperacidity, hæmorrhoids rectal fissures. Other manifestations appear in other parts of the body, such as asthma. We have these cases which show improvement under atropine. What we have done heretofore is to give them glasses. Yet, after the atropine wears off they will come back, with all their vagotonic manifestations. The correcting glasses were of benefit only, while the atropine was working. That is almost a diagnostic point. The reaction of the atropine, on the patient, is a valuable indication of vagotonic make-up."

I do not know whether the glandular secretions affect the structure of the eye or not, yet indications are that the hyperopic eye is of the pituitary type and the myopic eye, of the thyroid.

Eye Signs in General Anæsthesia.—This subject is of great interest to surgeons and anæsthetists.

The ocular signs consist of (1) lid and corneal reflexes, (2) globular signs, and (3) pupillary signs.

1. The first group consists of the conjunctivo-palpebral reflex, Parson's sign, and the corneal reflex. The first two are only present in the primary stage of anæsthesia, while the corneal reflex is usually present in the primary and intermediate stages, disappearing or being feebly present when the advanced stage is reached. These sensory reflexes, may disappear much earlier than normal, especially in those patients that have had morphine premedication; also in the debilitated and very sick; and they are seldom present in small children.

2. The globular signs are dependent upon the activity or degree of tension of the recti muscles. The eyes are usually shifting actively from side to side in the early primary stage, and gradually cease active movements as the intermediate stage is reached. During the intermediate stage, divergent strabismus usually occurs. If, however, narcosis becomes lighter after having been in the advanced stage, the recti muscles will again resume their tension. In babies and children the eyes are usually parallel in all stages of narcosis. But in adults the globular signs are so constant as to constitute an invaluable working sign in anæsthesia.

3. The pupillary signs exhibit a light reflex that is active all through the three stages of surgical anæsthesia. It is abolished only when the toxic degree is reached, at the point where the pupils begin to dilate from overdose or toxicity. Therefore, an active pupillary light reflex is a positive assurance that the toxic degree of narcosis is not present. The sizes of the pupils vary from many causes. However, I believe, one should have the utmost respect for dilated pupils. Dilation may occur at either end of the anæsthesia, *i.e.*, in too light anæsthesia, due to the irritation of the sympathetics, or in too deep anæsthesia, due to profound sympathetic toxicity. Unless there are unmistakable evidences of light narcosis, I would suggest always giving the patient the benefit of the doubt, and ceasing to administer the anæsthetic until definite signs of light narcosis appear, especially in Ether anæsthesia. Ignoring this has often led to grave errors. If the dilated pupils are due to toxicity, a few breaths of fresh air will reduce their size. On the other hand, if due to light narcosis, they will be unchanged or even augmented in size.

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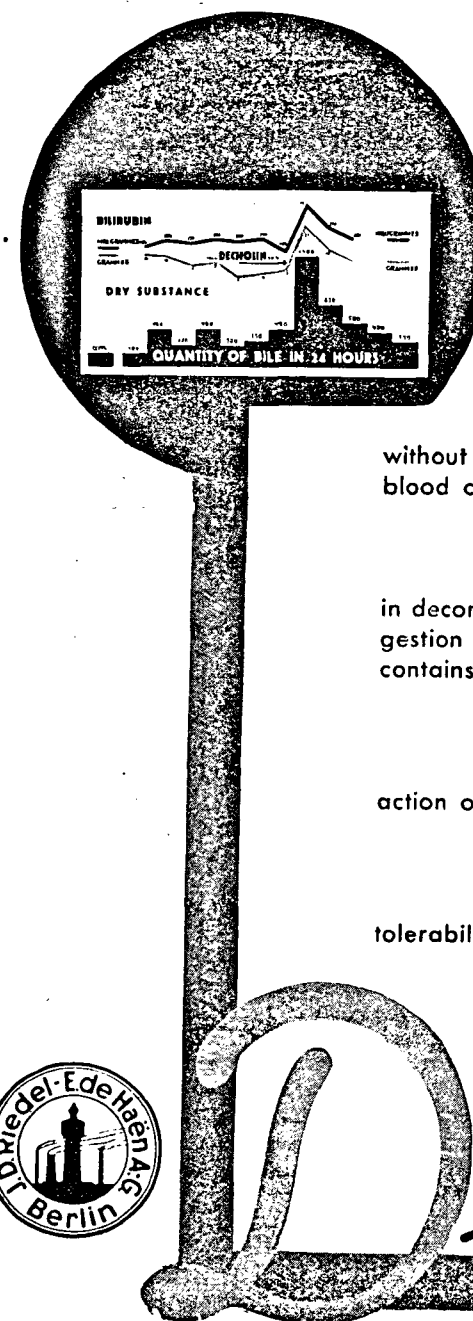
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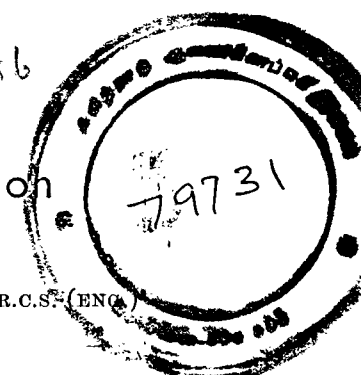
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Lachrymal Obstruction

BY

Dr. G. Zachariah, B.A., L.R.C.P. (LOND.) M.R.C.S. (ENG.)
D.O.M.S., (LOND.),

Eye Specialist, Madras.



LACHRYMAL obstruction is a common enough condition especially among the lower classes. One frequently comes across patients who complain of watering from one or both eyes. A certain number of these patients on examination are found to be cases of lachrymal obstruction. Lachrymation is often the only symptom of such a condition in its early stages. The tears accumulate in the sac. Such an accumulation in which numerous micro-organisms are also present produces a chronic inflammation of the mucous membrane of the sac. The sac thus becomes filled with a mucous discharge which later becomes purulent. This mucopurulent discharge remaining in the sac produces swelling in the region of the sac. If pressed upon it regurgitates back into the conjunctival sac producing a mucopurulent discharge in the eye. Later still, the persistence of the discharge in the sac produces gradual atrophy of the mucous lining of the sac.

The contents of the sac are highly virulent as it contains numerous pus cocci, especially the pneumococcus. It is a constant source of infection to the eye especially as any corneal abrasion is likely to be easily infected and produce a septic ulcer. For the same reason operations on the eyeball are also dangerous under such a condition as infection is sure to occur.

Occasionally, an acute inflammation may be set up by the passage of this virulent material into the surrounding tissue through any epithelial defect in the lining of the sac. The skin over the area becomes tense, hard and swollen. The swelling extends to the lids and even to the conjunctiva. Upwards the swelling rarely extends above the margin of the internal palpebral ligament. There may be severe pain and fever. After a few days the skin perforates and a quantity of pus is discharged. The pain is relieved now and the swelling subsides. The discharge may gradually clear up giving place to a thin watery fluid which keeps on escaping through lachrymal fistula.

Lachrymal Obstruction may be seen at any age—but it is more common in elderly people. It is also more common in

women probably 'on account of the more frequent use which women make of the lachrymal apparatus'.

Occasionally new born babies exhibit lachrymation from one or sometimes both eyes with a slight discharge from the eye. This condition is often mistaken for a mild conjunctivitis and treated as such with no benefit in most cases. The condition is due to an obstruction in the lachrymal passages, there being an unperforated diaphragm of mucous membrane at the lower end of the nasal duct.

The lachrymal passages are developed from a rod of cells which canalise throughout its length except at the puncta and the lower orifice. These open up at the 7th and 8th month of foetal life. Occasionally, the lower orifice remains closed even at birth. Hence the obstruction seen in the new-born.

Occasionally, in infants a persistent dacryocystitis may be tubercular or syphilitic in origin.

Lachrymal obstruction of adults results from a stricture of the nasal duct from any cause, the commonest cause being an extension of inflammation of the nasal cavity or an extension of inflammation from the sac itself. The usual position of obstruction is at the junction of the lachrymal sac and the nasal duct which is the narrowest part of the channel.

Treatment — Lachrymal Obstruction as seen in the new-born is as stated above generally due to the presence of an unperforate membrane at the nasal end of the channel. Treatment of such a condition is therefore not difficult. In many cases pressure in the sac region with the ball of the thumb rotated downwards is enough to burst the membrane. In those cases which are not relieved by this means probing of the passages under an anæsthetic. *Once* is all that is needed. The probing should be done very carefully as the delicate structures of an infant are very easily injured. If the condition is seen later when the child is 5 or 6 months old the appearance is that of a chronic dacryocystitis with a good deal of muco-purulent discharge. At this stage one probing may not occasionally be sufficient. A second probing may also have to be done. If it does not clear up even after that there is probably a more serious underlying cause of a tubercular or syphilitic nature. The sac should never be excised in an infant.

Before discussing the treatment of Lachrymal Obstruction in adults it would be well to remind ourselves of certain anatomical points.

The lachrymal sac lies in the lachrymal fossa behind the internal palpebral ligament with its fundus peeping just above the ligament. The sac is separated from the ligament by muscle fibres and the lacrimal fascia. The lacrimal fascia is a thick layer of the orbital periosteum which bridges across over the sac from the posterior lachrymal crest to the anterior lachrymal crest. In front of this lies fibres of the orbicular muscle. The lachrymal sac is about 12 m.m. long and about 4-8 m.m. broad. At its lower end it passes into the nasal duct. The duct opens into the roof of the inferior meatus of the nose behind the anterior end of the inferior turbinate body. The direction of the lachrymal channel is slightly backwards and outwards. Hence if 2 probes are inserted into the two channels on the two sides of the nose, the probes will meet in the middle line on the forehead.

In early cases of obstruction the condition may sometimes be relieved by syringing the sacs. The sac is syringed with a mixture of adrenalin and cocaine. A few drops of argyrol are then instilled into the conjunctival sac. When there is obstruction in the nose itself, adrenalin should first be applied to the inferior turbinate before syringing. The syringing should be carried out once or twice a week. In the intervals 1 or 2 drops of adrenalin followed by argyrol are instilled into the eye twice daily.

Cases of long standing are not relieved in this manner. Probing has been advocated for such a condition but the relief is only temporary. In such cases operative measures alone afford relief.

One type of operation advocates conservation of the sac and making a new passage into the nasal cavity through the lacrimal bone. This may be done either through the nose or externally. Operation through the nose, an example of which is West's operation, attempts to make a passage in the mucous membrane of the nose, a hole in the lacrimal bone and an opening in the sac directly communicating with the bone. The external route—for example Toti's operation—attempts to create the same kind of passage into the nose from the sac side. Sometimes the opening in the sac is stitched on to the opening in the nasal mucous membrane through the hole punched out in the lacrimal bone. Both these operations claim 90-95 % successes. But they are both difficult of performance for the average surgeon.

The other type of operation aims at the extirpation of the

sac. This is easier of performance and affords relief in most cases especially when the obstruction has gone on to a chronic dacryocystitis.

In performing this operation, the incision is made in the skin from just above the internal palpebral ligament along the orbital ridge for a distance of about 20-25 m.m. The skin should not be pulled upon in making this incision. The angular vein lies about 8 mm. medial to the inner canthus as a rule and hence is easily avoided. When the superficial fascia is cut through fibres of the orbicularis are exposed and the inferior margin of the ligament is brought into view. The fibres are cut below the lower border of the ligament. We then come across the dense fibrous membrane, the lacrimal fascia. When the fascia is carefully incised the pale bluish wall of the sac is exposed. The sac is freed all round and severed from its connection to the nasal duct and dissected upwards and finally removed by severing its connections with the lacrimal canals. This will be found easier in most cases especially when one does not want to cut the palpebral ligament.

Conjunctivitis

BY

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THE conjunctiva is the mucous membrane of the eye and it will be proper very briefly to explain its histological formation. The epithelium of the conjunctiva at the lid margins is epidermoid in character and consists of stratified epithelium. In the tarsal region the epithelium is of the columnar type and then proceeding towards the limbus it resumes its stratified formation. Phlyctenes attack the stratified layers and therefore are seen at the limbus and at the lid borders. Also in this connection may be mentioned the fact that in all acute conjunctivitis the palpebral conjunctiva is more affected than the ocular which is covered by the bacterial resisting stratified epithelium.

The adenoid layer is absent at birth and begins to develop at the second month. This layer is infiltrated with numerous lymphocytes and stretches from the sulcus subtarsalis to the limbus. In trachoma and allied diseases this layer assumes tremendous importance by formation of follicles.

The conjunctiva is liable to be inflamed:—

- (1) By germs carried to it from outside, *e.g.* by dust, fingers, towels, soorma stick, insects etc.
- (2) By toxins carried through blood *e.g.* tubercle, gonococci and local septic infections.
- (3) Direct extension of a surrounding inflammation from the orbit, nasal passages and the skin lids.
- (4) Allergic processes through change of resisting capacity of the individual cells to various allergens. It may be added in this connection that the conjunctivæ like the mucous membrane of the bronchial tubes in asthmatic individuals is liable to become sensitive to animal and vegetable proteins, such as feather, wool, pollen etc. Spring Catarrh and other allergic conjunctivitis will be dealt with hereafter. Suffice it to say that Atropine Conjunctivitis also comes under this group and Atropine being a salt it is assumed that atropine in combination with the protein of the mucous membrane of the conjunctiva forms a compound which gives rise to a specific allergen.

Classification of Conjunctivitis.—As it is not possible to classify conjunctivitis by the causative bacterial lesion since so many different species of bacteria produce the same clinical result and the same brand of germs at times may cause to appear different naked eye manifestations, we shall proceed to group conjunctivitis by the symptoms:—

Acute Catarrhal Conjunctivitis:—It is caused by such micro-organisms as Influenza bacillus, Pneumococcus, and Koch Weeks bacillus. Conjunctivitis occurring during measles, mumps, influenza also come under the same group. Simply finding pneumococcus or influenza bacillus in the conjunctival smear will not justify us in labelling the germ as the causative factor. The micro-organism should be present with the cells of the discharge showing its parasitic nature. Mere presence may indicate its saprophytic tendency as pneumococci and streptococci are present also in the normal conjunctiva. Conjunctivitis produced by endogenous toxins or metastatic origin have been seen in cases of gonococcal infection, bacillary dysentery and meningitis. It is difficult sometimes to differentiate this latter type of conjunctivitis from episcleritis.

Subacute Catarrhal Conjunctivitis due to the Diplobacillus of Morax and Axenfeld:—This variety is easily diagnosed by the clinical signs. The skin of the angle of the lids and the eyelashes are reddened and excoriated and present an eczema-like appearance. The diplobacillus secretes a protein-dissolving substance, which has very slight action on the mucous membrane as it is covered by the tears which by their antitryptic action protect the mucous membrane from the ravages of the protein-dissolving substance while the dry skin is attacked and inflamed. In old standing cases the mucous membrane also becomes involved. There is very little doubt that this saprophytic organism by exuding a toxin, primarily affects the skin. Zinc sulphate acts as a specific in this infection and the best way of administering it is by ionization, using 1% solution of 1 m.m. strength for one minute. The Zinc salt does not kill the organism as shown by test tube experiments but inhibits its power of secreting the ferment which has a tryptic action on the tissues.

Diphtherial Catarrhal Subacute Conjunctivitis:—It is seen mostly in new-born babes and smear preparations and culture would settle the diagnosis. It is mostly associated with discharge from the nostrils and swelling of the pre-auricular glands.

Symptoms of Acute Catarrhal Conjunctivitis.—Subjective sensation of gritty feeling in the eye and slight blurring of

vision due to mucous on the cornea are complained of by the patient.

Injection:—The congestion is most marked in the fornices and the tarsal conjunctiva at the beginning. In severe cases it soon spreads to the ocular conjunctiva and by anastomoses the ciliary vessels also soon become engorged. Capillary hæmorrhages also occur. Congestion of the conjunctiva is absent in anæmic cases.

Discharge:—At the beginning it is watery but soon becomes mixed with mucus and serum oozing out of the swollen conjunctiva. The mucopurulent discharge does not collect in the fornices as in purulent conjunctivitis but is seen dried up at the corners of the lids. During sleep the discharge clogs the eyelashes and causes the lid borders to stick to one another.

Infiltration:—The tissues of the conjunctiva are infiltrated with blood, leucocytes and serum. Infiltration may be diffuse or get collected into pustules and phlyctenular nodules. In some susceptible individuals lymphocytic collection gives rise to follicle formation.

Treatment of Acute Catarrhal Conjunctivitis.—It consists in:—1. Application of 1% silver nitrate on the lids once a day by the physician. To minimise the smarting it is better to put a few drops of 2% Novocaine solution before the application. One may wash away the excess of silver with plain water.

2. Patient may instil into his eye a few drops of 10% argyrol 3 times a day.

3. Fifteen minutes after the application of the argyrol drops the patient is instructed to wash his eyes with 1 in 10,000 Hydrarg. Perchl. lotion from an undine. If this is not done the argyrol produces by itself a foreign-body-like sensation.

As regards washing, it should be noted that the tears contain a potent antiseptic lysozyme whose beneficial action may be retarded by too frequent bathing which dilutes the tears. Specially during the recovery period the lysozyme content of the tears is not to be impaired by too frequent washings which unnecessarily dilute the lacrimal secretion.

As soon as the acute inflammatory stage of the inflammation is passed the washing should be done by a bland solution which is isotonic with the tears and is nearly of the same alkaline reaction. Boric acid solution though harmless would better be

substituted by saline solution such as can be prepared from compound saline tabloids of Messrs. B. W. & Co., Ltd.

It is not good to put the drops immediately before going to bed as it tends to stickiness of the lids.

4. A mild ointment such as borofax or Noviform should be smeared on the lid before going to bed. The ointment keeps the lid borders free from sticking and has a mild antiseptic action.

The silver nitrate solution is to be continued daily for about a week whilst the instillation of argyrol should continue for a few days more after the cessation of symptoms.

Purulent Conjunctivitis.—Gonococcal infection of the eye is a very serious disorder. After an incubation period which varies from a few hours to two days the conjunctiva gets congested, swollen, and secretes serosanguineous fluid. In the course of 48 hours, an yellow purulent secretion is poured out which is so copious that it runs on the cheek. Meanwhile the limbal conjunctivæ gets enormously swollen and the cornea at the periphery may be deprived of its nutrition with serious consequences developing. The clinical diagnosis must be confirmed by the bacteriologist and a very energetic treatment immediately instituted. Without treatment and sometimes even with treatment the prognosis is grave. The chief cause of anxiety is the lesion of the cornea, the toxins having a solvent action on the corneal tissue.

Treatment.—1. Services of a trained nurse is essential in this connection. The fellow eye must be protected from infection by means of a watch glass fastened on the orbital margins by means of sticking plaster.

2. The eyes should be frequently and copiously washed with 1 in 10,000 Hydrarg Perchloride solution. The pus, as soon as it is formed, must be washed out, sometimes it is difficult to separate the lids and canthotomy should be resorted to.

3. Instillation of 1 per cent solution of silver nitrate on the everted eyelids (and if eversion is not possible drops should be put in) from the first day. It does no harm though some people wait till the pus appears, before instilling silver. As soon as the pus begins to ooze out, daily application of 2 per cent silver nitrate solution should be begun.

4. Intradermal injection of Gonoderm (Parke Davis & Co.) is highly beneficial and I believe it has taken the place of intramuscular injection of milk 5 to 10 c.c. twice a week which undoubtedly exerts a favourable influence on the course of the disease.

5. General condition of the patient and urethral discharge if any should be carefully attended to.

Ophthalmia Neonatorum.—During labour the eyes are tightly shut and the various forms of bacteria in the vagina which are almost always present do not get a chance of entering the eyes. Most of the infections occur immediately after birth from soiled dressings or infected fingers and in 50 per cent of the cases the gonococcus alone or in combination with other bacteria is responsible for the formation of the gonorrhoeal or purulent conjunctivitis of the new-born. The conjunctival smear sometimes shows the presence of pneumococcus, streptococcus, bacillus coli etc. Also it is not uncommon to fail to find any infective virus in the purulent secretions. Inclusion bodies in the epithelial cells are also seen both in the conjunctival discharge and discharge from the maternal passages, the causative factor of which is not known.

Prophylaxis:—Instillation of 1 per cent solution of silver nitrate in the infant's eye immediately after birth is almost certain to abort gonorrhoeal conjunctivitis in the newborn. If legislation is passed compelling notification of every case much good can be done to minimise the ravages of ophthalmia neonatorum.

Prognosis is usually good though much protracted treatment is necessary.

Treatment.—1. Washing the eye copiously with 1 in 5,000 pot. permanganate lotion according to the severity of the discharge. As soon as the secretion is collected it should be cleaned out.

2. Daily instillation of 1 per cent silver nitrate solution on the everted lids by the Ophthalmic Surgeon.

3. A couple of drops of Proflavin in castor oil (1 in 1,500) three times a day.

4. If gonococcus is found intradermal injection of Gonoderm of Parke Davis & Co. once a week.

The treatment is to be continued till the discharge is completely stopped lest a relapse should occur.

Pseudo-Membranous and Membranous Conjunctivitis.—The false membrane is a coagulated fibrinous exudation lying on the surface of the mucous membrane. The true membrane is formed when the virulence of the causative organism produces exudation in the substance of the conjunctiva itself and necessarily it is a very serious lesion.

The false membrane is produced by a host of bacteria *i.e.* Klebs-Löffler's bacilli, pneumococci, streptococci, meningococci and bacillus coli. Micro-organisms of low virulence may produce a false membrane in patients whose general vitality is low.

Chemical irritants such as lime, silver nitrate, carbolic acid may also produce a false membrane, in the conjunctiva.

Course of Pseudo-membranous Conjunctivitis.—The disease begins like acute catarrhal conjunctivitis, there being the formation of a false membrane on the everted lids and fornices. The area involved by the false membrane varies, which, when it is peeled off shows a denuded area underneath. The membrane gradually disappears leaving behind clinical manifestation of acute catarrhal conjunctivitis. In rare cases specially in very debilitated subjects the cornea is severely affected, causing permanent diminution of sight.

Treatment.—The bacteriological findings determine the treatment. Antidiphtheritic serum or anti-streptococcal serum should be administered if smear examination warrants it.

Locally, at the initial stage, Fairchild's Enzymol dropped two or three times a day coupled with washing with 1 in 10,000 Hydrarg perchlor lotion is effective. 1 per cent silver nitrate should be instilled as soon as the membrane is loosened and boric acid ointment smeared on the lid at night.

True Membranous conjunctivitis.—This is a very serious disease and the cornea is frequently involved. The lids are enormously swollen and the membrane cannot be peeled off the surface of the conjunctivæ. It is not possible in some cases to divert the lids owing to swelling and hardness. Within a week pus pours out and the membrane is gradually extruded. If the ocular conjunctiva is involved the cornea is sure to be affected and the eye sight may be lost.

Treatment.—Isolate the patient and antidiphtheritic serum should be administered. Enzymol drops and washing with weak perchloride lotion is recommended. Noveform ointment should be introduced in the palpebral cavity.

Follicular Conjunctivitis:—This can be divided into:—(1) Simple Follicular conjunctivitis; and (2) Trachoma.

1. *Simple Follicular Conjunctivitis:*—In some individuals irritants applied to the conjunctiva give rise to a formation of

follicles which consist of collections of lymphocytes. School-going age is specially liable to get adenoid hypeplasia and this disease is most common in school children. It sometimes occurs in epidemics and hence it is believed that some micro-organisms not yet isolated may be the exciting cause. It is to be noted that all lesions of the conjunctiva associated with folliculosis take a long time to heal and chronic follicular conjunctivitis has a course running into two years. However, the cornea is not affected and there is no scarring left at the end. Compared to Trachoma it is a benign disease and an examination with a loupe or slit lamp of the upper cornea easily differentiates it from Trachoma, where the signs of beginning pannus are almost always seen.

Treatment:—Consists in washing the eyes with saline or very weak mercuric perchloride lotion and boric acid ointment at bed time at the lid margins.

If there is discharge, silver nitrate solution 1 per cent should be instilled on and off.

2. *Trachoma:*—It is impossible within a short space to depict trachoma but the most salient points of the disease would be touched in the briefest manner.

DEFINITION:—Trachoma is a persistent, intractable conjunctivitis associated with the formation of lymph follicles.

Sometimes acute trachoma is seen but usually the symptoms are those of chronic conjunctivitis.

DIAGNOSTIC POINTS:—1. Negative results of conjunctival smears.

2. Unlike simple follicular conjunctivitis, the conjunctiva is rendered thick and opaque.

3. Formation of papillæ which are shown as red points and which are seen in all conjunctivitis of a chronic nature.

4. Presence of follicles.

It should be noted that follicles are present in many eye diseases.

5. The most important single symptom on whose presence the clinical diagnosis can safely be based is the behaviour of the capillary loops at the upper limbus. The capillary loops seen under magnification show a tendency to migrate on the surface of the clear cornea. This is the earliest most pathognomonic sign of trachoma and if it is absent, caution should be exercised in pronouncing judgment. After a time, the cornea near the

upper limbus shows minute pits (Herbert's pits) and soon the appearance of diffused superficial vascular keratitis (pannus) places the diagnosis beyond the pale of doubt.

6. The tarsus is softened and deformed and the upper lid border assumes an 'S' shaped curve.

7. Scarring:—At a late stage, scarring is noticed as pale blue lines or as shallowing of the fornices when they are stretched.

Treatment.—Daily application of copper for 1 or 2 years is yet regarded as the best remedy.

If corneal ulcers are present, silver nitrate in 1 per cent solution is indicated.

If there is too much of follicle formation, expression by means of roller forceps is indicated.

A thick tarsus may be removed by Tracectomy.

Allergic Conjunctivitis.—The cells of the conjunctiva, both of the surface and the substance, become sensitive to animal and vegetable proteins which can come from the outside or are brought to the membrane from the lymphatic and blood stream.

1. *Simple Allergic Conjunctivitis*:—Simple allergic conjunctivitis is caused by various allergens i.e., dust from skin, feather, mattresses. The prominent symptoms are suddenly appearing oedema of the lids, conjunctiva, itebiness and slight discharge. Often the conjunctival smear shows the presence of eosinophiles.

Treatment.—Treatment consists in the removal of the allergen which is sometimes a very difficult matter. It is particularly to be observed that toxins from chronic septic foci are liable to induce this particular variety of conjunctivitis and the removal of the cause will produce a permanent cure.

Adrenalin drops are beneficial in allergic conjunctivitis and calcium should be given by mouth.

2. *Interstitial Allergic Conjunctivitis*:—In this category might be placed two important diseases:—(1) Phlyctene; and (2) Spring Catarrhal.

(1) *Phlyctenular Conjunctivitis*:—The nodular phlyctene is produced by the hypersensitiveness of the cells due to bacteria or their products brought through the blood. The modern theory that phlyctenules are anaphylactic phenomena due to tubercle bacilli is generally accepted. Whilst in most of the cases definite allergen of tubercle is present, it is sometimes

seen that in gonococcal and staphylococcal infected persons conjunctival phlyctenular nodules may be produced by reinfection of the same virus.

In majority of cases of ordinary phlyctene, tuberculin injections are indicated.

(2) *Spring Catarrh*:—It is a chronic bilateral interstitial conjunctivitis in which the upper tarsal plates and limbus are specially affected.

Unlike trachoma, it usually affects children of the well-to-do. Col. Herbert was the first to show that the conjunctival smear as well as the blood count shows an increase of eosinophiles.

Spring Catarrh is at present classified as an allergic disease though nothing is known of the definite allergen.

For the last five years, I have treated successfully many intractable cases of this disease by preparing a sort of vaccine from the foreign proteose found in the urine during the height of the attack when the subjective symptom of itchiness is most annoying to the patient.

I am indebted to Dr. Khanolkar for the technical procedure and so far the vaccine has been introduced intradermally.

In one patient, very recently, Dr. Khanolkar prepared drops which are instilled in the eye with very encouraging results. In this particular patient intradermal injections had not acted very successfully.

Some of the Common Forms of Conjunctivitis met with in the Tropics and their Treatment

BY

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CONJUNCTIVA being a mucous membrane, when it is inflamed or irritated it acts just like other mucous membranes in the way of increased formation of mucus. The goblet cells of the conjunctiva are shed along with their contents, varying with the amount of discharge in a case of conjunctivitis. As distinct from this mucous discharge which is gummy there is a lacrimal discharge—which is watery—as well, but this is not a prominent feature of conjunctivitis as opposed to its occurrence in other forms of ocular irritation. Clinically a gummy discharge or a sticky secretion means a conjunctivitis.

Conjunctivitis may be a manifestation of general disease or it may be due to exogenous causes. This article will deal primarily with conjunctivitis due to exogenous causes. Under this head conjunctivitis again may be due to exposure to irritants such as hot metal particles, alkalies, acids, caustics, smoke, dust, heat, sun's rays, etc. However, the commonest and most important cause is infection with pathogenic micro-organisms.

Irritative Conjunctivitis.—Caustic of any kind introduced into the eye should be washed away as quickly as possible. Plain water is always at hand to wash freely. Burns by caustics are liable to produce a condition of symblepharon and require special care for its prevention by way of introduction of simple ointment or vaseline into the eye twice or thrice daily to prevent formation of adhesions between the opposing granulating surfaces. The eyes should be protected by well fitting sun goggles from heat and dust etc., Crooke's synthetic glasses are of much use in the tropics.

Bacterial Conjunctivitis.—This group may be subdivided into acute and chronic forms. The acute forms are :—(1) Simple catarrhal; (2) Mucopurulent; (3) Purulent; (4) Membranous.

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Phlyctenular Conjunctivitis though an allergic condition may as well be considered with these as it is an acute form of conjunctivitis.

Catarrhal Conjunctivitis :—This is the mildest group, but liable to become chronic, when it is very difficult to treat. The symptoms are a sense of grittiness, some burning and discomfort on using the eyes at near work. The discharge is not much and it is mucoid in nature. On examination the eyes are not red, the palpebral conjunctiva however is congested and somewhat swollen especially that of the upper lid. In this type of conjunctivitis, the most commonly found micro-organism is staphylococcus aureus.

Muco-Purulent Conjunctivitis :—This is a much more acute and distressing condition than the catarrhal form. The bulbar conjunctiva is congested, the eye is blood shot, the redness being less pronounced in the region near the cornea. The palpebral conjunctiva is red and swollen and in severe cases the lids are oedematous and preauricular glands may become enlarged and tender. The discharge is yellowish and sticky, the amount varying with the severity of the case. If the discharge is allowed to dry on the lashes a condition simulating blepharitis superficially may occur; the lid margins, however, are healthy. This type of the disease is very contagious. Complications however are rare, but corneal ulcers may form if abrasions are caused by using strong lotions or powders. This form of conjunctivitis frequently complicates phlyctenular conjunctivitis as well. The micro-organism most commonly met with are Koch-Weeks bacilli, pneumococci and staphylococcus aureus. This form is frequent in measles.

Purulent Conjunctivitis :—Most acute type of conjunctivitis but fortunately not so common in tropics. It is mostly due to Gonococcal infection, and is especially a disease of the new-born (Ophthalmia Neonatorum). Gonococci can attack and destroy the healthy cornea thus causing blindness. The discharge is very copious and pure pus wells out on opening the lids which are very much swollen. The conjunctiva is chemotic and there are general constitutional disturbances. Corneal ulcers are the rule in this form of conjunctivitis, and there may be a diffuse general haze of the cornea or distinct infiltrates. Often there is a ring of ulcers at the margin on account of chemosis of bulbar conjunctiva. Perforation is very common. Besides the gonococcus, Streptococci and Diphtheria bacillus may produce this type of the disease.

Ophthalmia Neonatorum. — Much less common now than formerly. It is mostly caused by Gonococci, less so by others as *B. coli*, pneumococci and streptococci. It occurs after birth usually noticeable on the 3rd day. Both eyes are involved. Conjunctiva is red, intensely swollen and chemotic discharging thick yellow pus. The lids also become swollen and tense, later they become softer and the conjunctiva puckered and velvety and then there is free discharge of pus and serum. Ulceration of the cornea is the rule rather than exception. The ulcers are central and perforate causing prolapse of the iris and sometimes the lens itself is extruded. Perforation is followed by adherent leucoma, anterior staphyloma, anterior capsular cataract, panophthalmitis etc.

Membranous Conjunctivitis. — It is very seldom seen in tropics and is due to infection with *Corynebacterium diphtheriæ*. A false membrane is formed on palpebral conjunctiva, otherwise the case is like muco-purulent conjunctivitis. Raw surfaces of the opposing conjunctiva may become adherent to form symblephron. Cornea may become involved. It is rare to obtain evidence of primary diphtheria of throat.

Phlyctenular Conjunctivitis. — In this form one or more small round yellowish nodules somewhat raised above the surface are seen on the bulbar conjunctiva mostly near the limbus. As muco-purulent conjunctivitis practically complicates every case of Phlyctenular Conjunctivitis, besides phlyctens there are all the features of muco-purulent conjunctivitis as well. The disease is commonly met with in children of school-going age, who are generally strumous. It may follow an attack of measles. Adenoids also frequently co-exist. Very frequently the skin of the lid and cheeks show an eczematous condition. In simple phlyctenular conjunctivitis there is discomfort, irritation and lachrymation; generally however there is muco-purulent conjunctivitis superadded. Complication in the way of fascicular ulcer is the rule, and then there is increased lachrymation, muco-purulent discharge and photophobia is intense. There is a great tendency for recurrence, when there is any intercurrent malady or defective nourishment and unhealthy surroundings.

Treatment. — A stimulating line of treatment locally in the way of bathing eyes frequently with weak sublimate lotion, yellow oxide of mercury ointment. If corneal complications are present atropine must be incorporated in the ointment. In simple cases however I have found dusting of finely powdered

calomel as the best treatment. The eyes are not bandaged unless ulceration is very severe, a shade being better. General treatment however must be carried out assiduously. Cod liver oil, good food *e.g.* fish, milk, and raw vegetables should be incorporated in the dietary of every child.

Treatment of Acute Infective Conjunctivitis. — The conjunctiva being a very delicate mucous membrane, only very weak solutions of antiseptics should be used. Theoretically this means use of such lotions as possess no germicidal properties. Clinically however there is a curative effect of appropriate antiseptic solutions, which is not possessed by such bland solutions as normal saline.

In the treatment of two common groups, catarrhal and mucopurulent conjunctivitis, a mercurial solution such as Hydrarg perchlor 1 : 10,000 or Hydrarg oxycyanide 1 : 8,000 can be used. I generally use the latter solution which is less irritating and equally effective. The conjunctival sac is washed 3 to 4 times a day, varying according to the severity of the case. To prevent sticking together of the lids, I generally put a drop or two of liquid paraffin into the sac. Use of 2 % silver nitrate solution on the palpebral conjunctiva daily for two or three days in the early stages of the mucopurulent conjunctivitis cuts short the duration of treatment. When the lids however are very much œdematous, I resort to frequent washing of the conjunctival sac and use of liquid paraffin and paint the *external* surfaces of the lids with 2 % silver nitrate daily till the swelling subsides. Optochin 1 % is useful in cases of mucopurulent conjunctivitis really due to pneumococcus, but is rather painful. Conjunctivitis in infants apart from *Ophthalmia Neonatorum* is commonly of the mucopurulent variety. The infant's conjunctiva is even more sensitive than adults, and can only bear Hydrarg oxycyanide solution in 1 : 10,000 strength and this may be prescribed as drops to be used after washing with boric lotion or normal saline three times a day. After each application boric ointment or simple vaseline must be introduced into the sac. Argyrol 10 % solution is also useful in cases which do not tolerate the mercurial drops, or acriflavine 1 : 1000 solution may be used.

Gonococcal Conjunctivitis is so serious that it should receive expert treatment. Essential factor in treatment is frequent, nay, continuous washing out of the sac to prevent prolonged contact between the infected discharge and cornea. Use

of 2 % silver nitrate as a paint on everted lids is resorted to as soon as the brawny swelling of the lids lessens and the discharge becomes free. Atropine should be used in all cases where cornea is involved. In babies both eyes are infected, but in adults primary infection is monocular. The normal eye should be protected by strapping a Bullar's Shield. Crede's method of prevention of ophthalmia neonatorum is now sufficiently common even in tropics.

Membranous conjunctivitis should be treated on the same lines as other Diphtheritic infections by antitoxin infections. The local condition being treated by frequent washing of the sac with boric lotion. Silver nitrate paint is however *not* to be resorted to.

Chronic Conjunctivitis.—The main types under this head are :—

1. Simple chronic conjunctivitis.
2. Angular conjunctivitis.
3. Trachoma.
4. Follicular conjunctivitis.
5. Spring Catarrh.

1. *Simple chronic conjunctivitis* :—Occurs as a continuation of simple acute conjunctivitis which has not been properly treated, or when irritation is a continuous one, such as smoke, dust, heat, errors of refraction, dacryocystitis etc. There is a sense of burning and grittiness. The lids may stick together on waking. The lids feel hot and dry. On pulling down the lower lids the posterior conjunctival vessels are seen to be congested. The palpebral conjunctiva of both the lids may be congested as well. In very chronic cases the Meibomian glands become infected and so the infection persists.

Treatment.—Besides the treatment for eliminating the cause as ordering spectacles, operating for the sac etc., the local treatment consists in ordering a stimulating treatment. In mild cases zinc sulphate gr. 1—2 to $\frac{3}{4}$ 1, alum lotion gr. 4— $\frac{3}{4}$ 1 two or three times a day using an Eyebath. In severe cases silver nitrate 2 % or protargol 5/10 % is painted twice a week. In very severe cases, the secretion of Meibomian gland is squeezed out by pressure with a glass rod. Some such ointment as Ichthyol 1 % is rubbed into the lid margins. In chronic pneumococcal cases, optochin 1 % is helpful.

2. *Angular conjunctivitis* :—This is a form of conjunctivitis which is produced by a special micro-organism (Morax and

Axenfeld Diplobacillus) and presents a typical clinical picture. In it, the reddening of the conjunctiva is limited to the intermarginal strip and the bulbar conjunctiva of the angles of the eye. Besides there is excoriation of the skin. There is a slight mucopurulent discharge and some nasal discharge. Cases become chronic generally through want of proper treatment and there may be blepharitis as well.

Treatment.—This type of conjunctivitis respond readily to boric lotion with the addition of grs. 2 to $\frac{3}{4}$ 1 of zinc sulphate. Since there is sometimes an added mucopurulent conjunctivitis it is best to use oxycyanide of mercury lotion (1 : 8,000) with the addition of zinc sulphate.

3. *Follicular Conjunctivitis* :—This type occurs in children who are seldom robust, and suffer from adenoids. It is a collection of lymphoid follicles in the lower fornices. It is liable to be mistaken for trachoma, but conjunctiva is neither reddened nor swollen and they persist and have no after-effects in the way of scarring etc. They cause only slight irritation of the eyes.

Treatment.—A weak astringent lotion is useful. If follicles are much enlarged silver nitrate solution may be painted. General health should of course be looked after.

4. *Trachoma* :—The commonest chronic form of conjunctivitis met with in the tropics and in fact the commonest single eye disease causing vast amount of defective vision and blindness. Both eyes are affected. Every one is familiar with the appearance of palpebral conjunctiva in a case of trachoma, hence, I may only briefly recapitulate. The changes met with in the conjunctiva are either papillary enlargement and congestion which passes into a uniform jelly like thickening, but usually, however, there are follicles, and their distribution is characteristic. They begin in the fornices and spread into the tarsal conjunctiva, being common about the caruncle. The disease itself is very chronic, but sometimes it takes on a very acute form. There is irritation, photophobia and lachrymation and if mucopurulent conjunctivitis is superadded, then, much mucopurulent discharge as well. As it progresses the large follicles become gelatinous looking masses and some of them burst and extrude their contents into the conjunctival sac. More often, however, fibrous tissue is laid down and scars form, which are very characteristic. The disease is very contagious and flourishes among people crowded together and living in unhealthy rooms

and using common articles of toilet. The disease is conveyed by transference of conjunctival discharge on fingers, towels, etc.

Research is still going on as to the true causation of this disease. It is now thought to be virus closely related to an infra-organism of the Rickettsia family. Experiments with cultures grown in the anal canal of louse on apes and man seem to prove that trachoma is a rickettsiosis and that louse forms a reservoir of infection and is perhaps one of the vectors of trachoma virus. Under the circumstances delousing and improved sanitation should put an end to this scourge of the East.

COMPLICATIONS:—Trachoma rarely attacks the bulbar conjunctiva but the cornea is invaded invariably.

- (1) **Trachomatous Pannus:**—It is a lymphoid infiltration, with vascularisation of the margins of cornea usually the upper half but if untreated may spread over the whole cornea. The vessels at first are superficial but later on the substantia propria is involved. In progressive pannus at the end of the vessels there is an area of infiltration and haze, while in pannus that is healing or retrogressing the vessels extend beyond the area of the haze. In very severe cases the whole of the cornea becomes vascularised from all sides and becomes opaque. Pannus is due probably to direct infection of the cornea, from the close and constant contact of the infected upper lid. In early stages it may resolve completely and before the Bowman's membrane is destroyed the cornea may become quite clear but generally permanent opacity results. In neglected cases the corneal substance becomes weak and a condition of ectasia develops.

- (2) **Corneal ulcers:**—They are commonest at the advancing edge of the pannus and are superficial, very indolent and irritable, causing much lachrymation.

SEQUELÆ:—It is the sequelæ that make the trachoma such a dreadful disease. The chief ones are:—(1) Results of pannus and corneal ulceration; (2) the distortion of the lids. This is the result of scarring. When it is extensive the shape and position of the eyelids especially of the upper ones are altered. At first due to much infiltration of the lids, there may be eversion of the lid margins giving rise to ectropion. Generally however due to softening and absorption of the dense fibrous tissue of the tarsus there is inversion of the lid margins (Entropion) and consequent trichiasis, the eyelashes rubbing on the cornea.

Treatment.—There is no cure for trachoma. Sulphanilamide group of drugs has been claimed to effect a cure if used in conjunction with the local treatment, but as yet sufficient time has not elapsed to gauge the benefits of the treatment.

Local Treatment.—When there is much discharge and congestion, the conjunctiva of the lids is painted once daily with 2% silver nitrate solution and the sac washed with very weak sublimate lotion three or four times a day and boric ointment smeared to the lid margins. When there is less of the conjunctivitis then I use copper sulphate crystal, rubbing it all over the palpebral conjunctiva especially to the fornices. Only when there are corneal ulcers or the case is too far advanced and of gelatinous variety that I refrain from using the copper sulphate crystal. In these cases most reliance is placed on the silver nitrate paints in the former case and simple boric ointment in the latter. For home use I have found copper citrate ointment (10%) very useful or the proprietary medicine—cuproargentique ointment (5 or 10 %). I seldom use any of the stronger applications such as concentrated solution of sublimate of mercury in glycerine or the use of carbon dioxide snow etc.

Of the mechanical treatments, I generally use expression of follicle and have found that the length of treatment is cut short by this procedure. I prefer the Knapp's roller forceps and then scrape the caruncle with an ordinary scraper. Sometimes, I resort to excision of the fornix and have found it to be useful if there are large follicles especially in the fornices. For treatment of pannus the use of copper sulphate crystal is the best but if it is progressing rapidly then I perform invariably the operation of periotomy and thus give a chance to the usual treatment to act advantageously.

5. **Spring Catarrh:**—It is a recurrent conjunctivitis, but the lesions persist always, only taking on more acute form during spring and autumn months. Young people of both sexes are affected. There is not much mucoid discharge but there is lachrymation, burning, itching and photophobia. There are two types:—(1) Palpebral form; and (2) Bulbar form, and though the text books state that they seldom occur together, I have found them mostly associated together though to varying extent. In the palpebral form the conjunctiva is hypertrophied and mapped out into fleshy polygonal areas, not unlike hypertrophied unhealthy granulations with rough surfaces. The presence of Eosinophle leucocytes in them and the scrapings is very characteristic of this form of conjunctivitis. In the bulbar form the conjunctiva of the superficial layers of the cornea are covered with thick fleshy masses not unlike pterygium masses but totally adherent at the limbus and over the cornea and much thicker than pannus of trachoma.

Treatment.—Smoked goggles should be fitted, weak acetic acid drops, yellow oxide of mercury ointment should be used. Excision of follicles is useless. General health should be built up. Calcium, and arsenic internally are reported to be good.

Gonorrhoeal Conjunctivitis of Adults

BY

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Resident Medical Officer, The Minto Ophthalmic Hospital, Bangalore.

BLENNORRHOEA and Purulent Conjunctivitis are two terms used to designate Gonococcal Ophthalmia of adults. In the following pages a few observations are made on the subject from the experience gained during my eleven years work in Minto Ophthalmic Hospital, Bangalore. Cases of Ophthalmia Neonatorum are not considered here.

Mode of infection.—In the vast majority of cases, the germs of the disease are conveyed to the eyes of the patient from discharges from the patient's own genital passages. Infections from soiled water-closets, used towels, bath tubs and swimming baths mentioned by some writers did not come to the notice of the author. Two cases of children of 5 and 6 years of age, who got the disease from the hands of ayahs, who were suffering from gonorrhoea were seen.

Age Incidence.—The disease may occur at any age, the youngest patient we had being 5 years and the oldest 57 years old. There was one case of a woman, who had twins aged about 6 months, and all the three had purulent conjunctivitis, derived very probably from the mother.

Sex.—More males are affected than females, perhaps owing to more free drainage of discharges in the latter. During five years, 1934 to 1938, the total number of cases of gonorrhoeal ophthalmia of adults admitted in the Minto Ophthalmic Hospital was 32, of which 19 were males and 13 were females.

Incubation Period.—Although the incubation period of gonorrhoeal infection in cases of the genital passages is said to be 2 to 5 days, the period is very much shorter in the case of the adult eye. The conjunctiva and cornea of new born babies seem to be much more resistant to the infection than that of adults, and numerous cases of babies as old as 4 to 6 weeks and suffering from ophthalmia neonatorum almost from 2 to 3 days after birth have been seen to have clear corneae. In the adult, however, as short a period as 36 hours after infection has been found to be enough to produce extensive ulceration of cornea. Frequently, cases are seen with gonococcal conjunctivitis of both eyes and corneal ulceration on one

side. Even hospitalisation and careful and efficient treatment cannot guarantee the safety of the other eye, which may commence ulceration at any time. It is impossible, thus, to foretell the course that the disease is likely to take, or prophesy the ultimate result. Gonococcal corneal ulcers have a great tendency to extend rapidly by coagulative necrosis of corneal tissue, and to perforate, often producing large irregular unsightly iris prolapses, which are slow in healing. Some such eyes pass on to panophthalmitis, while others heal up into painful phthisis bulbi, which are a source of recurring inflammation and suffering. In the past eleven years, the author has seen only nine adults who suffered from purulent ophthalmia, who came to the hospital with clear cornea and possessed clear unaffected cornea on discharge. At least superficial scarring, if not denser leucomata and adherent leucomata is the rule.

A curious fact regarding the change in bacterial flora of the eye has been noticed in many cases. In the earliest stages of catarrh in this disease, the conjunctival discharge is somewhat thin and mucopurulent and the gonococci are mostly extracellular. Within 12 hours after this, the organisms become intracellular and usually it is after this stage corneal ulcerations occur. As the disease gets controlled by treatment, the number of gonococci becomes less, the few organisms appearing degenerate and staining faintly. When the gonococci disappear, it is noticed that the conjunctival discharge contains large numbers of pneumococci. These last a few days, to be replaced by xerosis bacilli. The explanation of this perhaps is that the virulent toxins of gonococci destroy all other organisms in the eye and while these hold sway no other organisms can exist. Once they disappear, the pneumococci from the nasal passages become predominant and ultimately, in their turn, yielding place to xerosis bacilli, which are the normal inhabitants of the conjunctival sac.

Treatment.—No attempt can be made in this paper to enumerate the various local and general treatments adopted by the various hospitals and clinics, except to mention, that frequent irrigations of the eyes with concentrated solution of Magnesium Sulphate or a 1 in 6,000 solution of Potassium Permanganate are employed to clean away the discharges, improve the circulation of blood and lymph in the eyes and advantage taken of the oxidising properties of potassium permanganate against the gonococci, which are anaerobic organisms. Some use

ice-cold solutions for the purpose, while others use luke-warm ones. Eye drops in the form of Argyrol 40%, Choleval 10%, Mercurochrome 2%, Rivinal 1%, Sulphanimide (soluble) 2%, Trypaflavine 1%, are generally employed after the irrigations. Atropine sulphate, either as aqueous solution or in oily suspension is invariably instilled to counteract the iritis and a bland ointment like Collargol ointment $\frac{1}{2}\%$, or Boric ointment 2%, finally introduced into the eye. After the swelling of the lids has subsided a little and chemosis is a little less, Silver Nitrate solution 1 to 2% is used as eye drops, before the irrigation. Hot or cold compresses are soothing to the eyes. Every attempt is made to remove accumulations of conjunctival discharges at frequent intervals with cotton wool moistened in dilute Condy's Solution.

Instillation of gonococcal vaccines into the eyes as eye drops has been advocated by some writers, but the author has found them useless. Gonococcal serum has not been tried. Gono-yatren may be of some benefit.

Injections of vaccines are of limited value in purulent ophthalmia. Injections of milk and its products are widely employed in the treatment of the disease, sterilised milk being injected in doses rising from 2 to 7 or 8 cc. on alternate days. In a few cases, however, it has been noticed, that a little over-enthusiasm with milk therapy accelerates and augments the already existant coagulation necrosis of the cornea, to such an extent that the ulcerated cornea literally melts away before one's eyes leaving a large complete prolapse of the iris. Such enthusiasm is generally not in the interest of the patient.

Owing to some technical difficulties, the statistics of cases of the Minto Ophthalmic Hospital could not be obtained and this article is necessarily in a narrative form for which the author tenders his apologies.



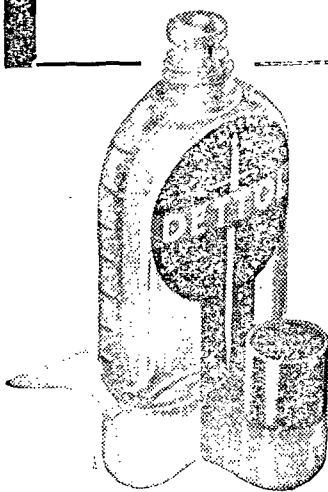
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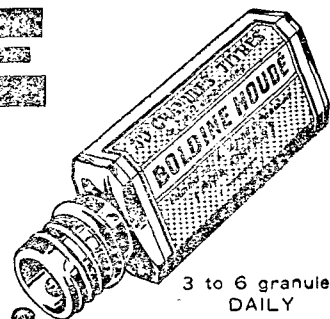
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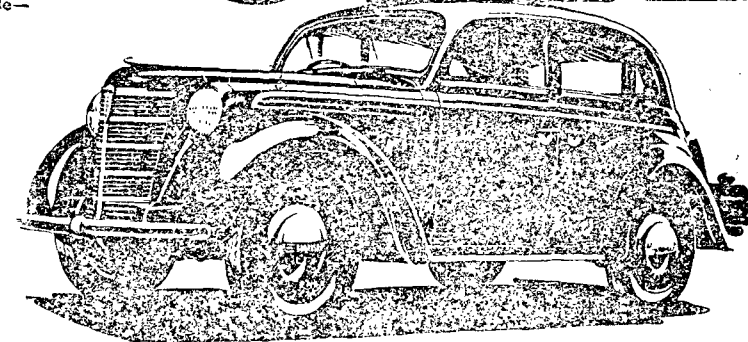
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Silver Acetate for Crede's Method*

A Safer Drug for Preventing Ophthalmia Neonatorum

BY

Dr. Victor, C. Rambo, M.D., F.A.C.S.

Mission Hospital, Mungeli, Bilaspur Dt., (C. P.)

IN a country like India it is important that every effort be made to extend the use of the sight saving protection of Crede's method.

A more fool proof drug than silver nitrate is needed. Fortunately one is at hand which cannot be made into a stronger solution than that needed for the treatment and yet one which has proved equally effective in protecting the infant against ophthalmia neonatorum.

I reported the possibilities of using silver acetate in the Journal of the Christian Medical Association of India in 1931 and later in America¹. I am using these articles by quotation and abstract to present the facts about silver acetate.

"When the writer visited Professor Emile de Grosz, at the Royal Hungarian University Eye Hospital in Budapest, he found that Hungary was using 1 per cent silver-acetate solution as a prophylactic for ophthalmia neonatorum. The advantage of this salt over silver nitrate is in its safety. A 1 per cent solution of either salt is equally effective in preventing gonorrheal ophthalmia. The solubility of silver nitrate is very high, 1 gram being soluble in 0.4 grams of water. The solubility of silver acetate solution is 1 gram to 100 c.c. of water. Because of the low solubility of silver acetate, a stronger solution than is needed for infants' eye cannot be made up with water at room temperature."

In an American town three infants had a 10 percent silver nitrate solution dropped into their eyes by mistake. One infant had squeezed out the drops, but the other two had severe reactions. Such occurrences are more common than is generally known and emphasize the danger of using silver nitrate.

"In Hungary, the midwives are employed in peasant homes at the time of delivery. They are given a bottle containing a watery solution of silver acetate, with a considerable excess of silver acetate at the bottom. When the

* From the Jackman Memorial Hospital Bilaspur, and the Mungeli Area Christian Hospital, Mungeli, C. P., presented before the Chhattisgarh Medical Association, Champa, 15th April 1939.

solution is used up, more water may be added. The midwife, therefore, has a ready, potent solution on hand, for as long as the silver acetate lasts. It cannot become too strong a solution. Such a procedure is not recommended particularly at a time when the use of silver nitrate is so common. But it shows the safety of the solution of silver acetate, and gives more assurance of having a potent solution for use by persons distantly situated from sources of the necessary drug."

"According to Merck's report for 1906, 'Zweifel pointed out years ago the advantages of silver acetate as compared with silver nitrate, for the prophylactic treatment of gonorrhœa neonatorum; this view was shared by Scepiades and Bischoff, while Leopold and Dauber held silver nitrate to be the better remedy. J. Thies held that, the two being of equal efficacy, the only possible advantage of one over the other would be due to a greater tendency of one to produce irritation. To settle this matter the author made trials with 2,000 children. After mechanically cleansing the eyes, he placed in the right eye a few drops of a 1 percent solution of silver acetate; in the left eye, a few drops of the usual 2 percent silver nitrate solution. The eyes were then bathed with a normal saline solution. He found silver acetate to work better than silver nitrate, although the difference was not great."

"Silver acetate has other properties, however, which would appear to make it more suitable to the needs of practice than the nitrate. While the latter is readily soluble in water, its solutions may gradually become concentrated by evaporation. Silver acetate crystallizes out as soon as a concentration of 1.2:100 is reached. Thus the danger of using too concentrated a solution is entirely precluded in the case of silver acetate. A further advantage of the acetate is said to exist in the fact that, in case silver becomes set free from the solution, the acetic acid thus liberated is less irritant than the nitric acid set free, under similar circumstances, from the nitrate. For prophylactic treatment in midwifery, the acetate would thus appear to be preferable to the nitrate of silver."

For several years silver acetate was unavailable in India. When I was in America in 1937, I obtained this drug and sent samples of it to both Bilaspur and Mungeli Mission Hospitals

for trial. This drug has been used constantly in both these hospitals for over a year and a half, in both eyes of over 200 infants on birth. In only four of these there was slight congestion following its use. In no case has there been a suggestion of ophthalmia neonatorum. The fact that there are surprisingly few cases of ophthalmia neonatorum seen from the villages where no prophylactic whatever is used makes this number inadequate to show the effectiveness of the drug. Yet these statistics do add to the large number in other favourable reports, and it is a practical demonstration that it is a drug that can be safely used in India. Before leaving America in the spring of 1938 the large Pennsylvania Lying-in Hospital was using silver acetate for the application of the Crede's method.

An incident will illustrate the safety of this drug. I went into the drug room in the Bilaspur Mission Hospital and found the compounder stirring a mixture vigorously with mortar and pestle. She looked up at me when I came in and said there was something wrong with the salt. I asked what it was and what she was trying to make. She answered that she was making a 2% solution of silver acetate. I then told her that this could not be done.

Conclusion.—Silver acetate is a recognised drug for use in application of Crede's method for prophylactic against ophthalmia neonatorum. Being only 1% soluble it cannot be made into a stronger solution as can silver nitrate which from time to time injures infants' eyes because of the strength of the solution. A small group of statistics is presented to show that the use of the drug in India is practicable. The author is probably right in stating that the use of silver acetate in Mungeli and Bilaspur is the first use that has been made of silver acetate in India. It is slightly more expensive than silver nitrate, but should become cheaper as its use is extended. A persistent demand for the drug will make it readily available in all markets.

Reference :

1. Rambo, V. C.—*Silver Acetate as a Prophylactic for Ophthalmia Neonatorum*, 'American Journal of Ophthalmology', Vol. 21, No. 4, (April) 1938.

Treatment of Ulcerations of the Cornea

BY

Dr. P. R. Chhibber,

Eye Surgeon, Nowshera Cantt, N. W. F. P.

IT consists of:—I. General; II. Local treatment.

I. General treatment.—It consists in: 1. Looking after the hygienic conditions of the patient.

2. Regulating the condition of the bowels for which a purgative of calomel at night followed by Mist Saline next morning is given.

3. To relieve the pain of the eye-ball salicylates and opiates are administered in the form of Aspirin, Phenacetin and Dover's powder and even injection of Morphia Atropine is indicated in severer cases. In milder cases Ortal (P. D. & Co.) or Veramon (Schering) given in milk proves beneficial.

4. Iodine in the form of either iodine (B. C. P.W.) or Entodon (Bayer) 2 c.c. should be injected intravenously or intramuscularly, respectively as often as is necessary. In ordinary cases one injection suffices. Iodine helps to lessen the pain and inflammation. The scar left is also less dense than otherwise.

II. Local treatment.—Bathe the eye with lukewarm 6% mag. sulph lotion (filtered sea water is also good). When it irritates, then prior to the wash, instil a drop or two of Cocaine Lotion, otherwise use normal saline or 2% Boric Lotion instead. Then a drop of 1% Mercurochrome or 6% protargol is used. Following which a drop each of 2% Dionine and 1% Homatropine is instilled and finally introduce a small lump of $\frac{1}{2}$ % yellow ointment between the lids. The last thing is to apply on the eye a warm pad of antiphlogistine, its under surface being covered by a piece of sterile gauze tissue or a double folded piece of sterile gauze, and place a pad of cotton wool over it and keep it in position by a bandage which should be firm but not unduly tight.

This treatment repeated three times a day is quite sufficient for mild cases.

Severer Septic Cases.—Homatropine drops should be substituted by atropine sulph 1% ointment and yellow ointment by 50% Iodoform ointment or compound colour substances with organo-bismuth ointment (blachetube). If there is no improvement in 24 hours then cauterize the Ulcer with any one of the

following things:—(1) Tr. Iodine; (2) Carbolic acid pure liquid; (3) Electric cautery. In the absence of an electric cautery, a white hot needle cautery may be used. Usually one sitting suffices; otherwise it may be repeated as many times as is necessary.

Trachomatous Ulcers.—In this condition Mercurochrome should be replaced by 2% silver nitrate touchings washed away by normal saline. It is performed under a local anaesthesia and repeated every 24 hourly and two dressings in between should get instillations of acriflavine $\frac{1}{4}$ % prepared in normal saline.

Phlyctenular Ulcers.—Here the yellow ointment should be replaced by a 50% Calomel ointment. In recurring cases, local drops of Cod liver oil are beneficial.

Hygienic conditions with exercise in open air, good and nutritive food and change to a hilly station help and supplement the treatment.

Injection of Tuberculine or A. K. B. is useful.

Chronic Ulcers.—They are usually cured by Insulin ointment prepared by Organon, England.

Keratomalacia

BY

Dr. P. V. Paul, M.B., CH.B.,

Medical and Surgical Hall, Ernakulam.

KERATOMALACIA (Keras-Horn, Malakos-Soft), softening of the Cornea, is the ultimate result of a nutritional deficiency disease occurring mostly in children, and less commonly in adults. The title is used in its widest sense to include all the manifestations of deficiency of the Fat-Soluble Vitamin A. It is one of the most devastating diseases in children in Malabar. Keratomalacia destroys many infantile eyes and lives and a few adult eyes every year in Malabar. Fortunately, ophthalmia neonatorum is very rare in these parts. Ophthalmia Neonatorum is as frequent in Western Europe as Keratomalacia is in certain parts of India. Keratomalacia is almost as prevalent as Trachoma in these parts.

Signs and Symptoms.—1. One of the earliest signs to appear is *Xerophthalmus Epithelialis*, a dry, white, foam-like patch, on the bulbar conjunctiva, in the palpebral fissure. These occur as Bitot's spot, a triangular patch of epithelial Xerosis with its base towards the limbus on the bulbar conjunctiva in the palpebral fissure, on one or either side of the Cornea; or as smaller or larger irregular white patches scattered on the conjunctiva. One or more of these patches are frequently seen often in both eyes, less frequently a unilateral lesion is also found. Epithelial Xerosis affects the cornea rather very late in the disease. The earliest symptom is the loss of lustre, and the loss of sensibility of the cornea. The irregular white plaques of various sizes appear scattered in the centre, or periphery of the Cornea. The whole cornea may soften by degeneration, or disintegration beginning in the superficial layer and later affecting the whole thickness. This condition is known as Keratomalacia. Xerosis Bacilli are found in the epithelial debris of the cornea and conjunctiva. Pigmentary changes from brown, to dark discoloration of the conjunctiva, especially in the caruncle, as also scattered in other parts of the conjunctiva, are seen early in the disease. Wringling of the conjunctiva, especially noted in moving the eyes from side to side, when the palpebral fissure is wide opened with the finger and thumb, is due to a loss of elasticity in the deeper layers of the conjunctiva. Diminution

or loss of lachrymation also occurs early in the disease, causing a peculiar dryness of the conjunctiva.

2. *Night Blindness*:—Hemeralopia, of continental authors, and Nyctalopia, of English authors, is the inability to see in the dark especially after sunset. During the day the vision is clear. But at night, or in a dark room, even with artificial light, the vision is lost. The patients are unable to walk, read, or write, or see objects in front of them after sunset. There are no ophthalmoscopic changes in the eyes. This inability to see at night without any obvious lesion in the fundus of the eye is known as *Essential Night Blindness*. It is due to a disturbance in the regeneration of the visual purple contained in the outer end of rods in the Retina.

3. *Skin Eruptions*:—Small, hard, dark, sharp pointed, epidermal papules on the skin of the limbs, buttocks, chest, back, and abdomen are very characteristic of nutritional deficiency. These skin eruptions are generally accompanied by either night blindness, or *Xerophthalmus epithelialis*, or by both. It might occur alone as an early symptom of deficiency of the Fat-Soluble Vitamin A. This hyperkeratinisation of the epidermal epithelium is known as Lichen spinosum or phranoderma, and is similar in appearance as Pityriasis rubra pilaris, and Lichen ruber acuminatus. Like *Xerophthalmus epithelialis* of the conjunctiva, and Cornea, this also is due to an epithelial disturbance caused by the Fat-Soluble Vitamin A deficiency. It is remarkable to note that these symptoms of epithelial Xerosis (called in Malayalam as Ithel=line in the eye), night blindness (in Malayalam Rākuruḍu or Mālakannu), and lichen spinosum (in Malayalam Kāra), were recognised in Malabar from very ancient times by Ayurvedic Physicians, occurring singly or in combination as symptoms associated with nutritional deficiency, and were effectively treated by the administration of chicken soup, black chicken cakes, and medicated cow's and goat's milk, butter and ghee. It is interesting to know that our ancients discovered the nature, probable cause, and treatment of this disease without the facilities of the modern research worker with his costly experiments on rats, and mice, and other animals.

4. *Diarrhœa, and Gastro-intestinal disturbances are common occurrences in Keratomalacia*:—Diarrhœa is a very disturbing condition to deal with in Keratomalacia, especially as the oral administration of nutritional diet is made difficult. Diarrhœa, if not tackled carefully in the early stages, leads to emaciation, anæmia, dropsy, and finally marasmus, and death. Here also

the mucous membrane of the Gastro-Intestinal tract is affected, showing that the disturbance in the epithelial cells is brought about by the Vitamin A. deficiency more than the deficiency of other nutritive factors in the diet.

5. The epithelium of the respiratory tract also gets affected in Keratomalacia as shown in the early occurrences of Catarrh of the mucous membrane of the Nose, Pharynx, Larynx, Trachea, Bronchi, and Bronchioles leading to capillary Bronchitis, and Bronchopneumonia which may end fatally.

6. In Keratomalacia it is significant to note that the eyes are free from the disturbing symptoms of pain, lachrymation, photophobia, redness and chemosis which are the most prominent symptoms of other acute affections of the eye.

Course.—It is convenient to describe the course of the disease in three stages.

1st stage:—Epithelial Xerosis or discoloration, and pigmentation of the Conjunctiva may be the earliest symptoms to appear. Conjunctival lesions may be accompanied by night blindness, or skin eruptions. Night blindness or Skin eruptions may be the earliest symptoms to appear. Either of these may be followed by the other and also by Conjunctival Xerosis. If any of these symptoms are detected and treated early, the disease is cured without the occurrence of later symptoms.

2nd stage:—If untreated, the epithelial Xerosis of the Cornea will begin manifesting itself by slight loss of lustre of the Cornea, and loss of sensibility of the Cornea, developing into the appearance of irregular white patches scattered in the centre, or the periphery of the Cornea. Treatment of this early Corneal lesions also is attended with good result.

3rd stage:—If not treated properly, degeneration, disintegration and desquamation of the entire Corneal epithelium takes place; and this is the condition to which the name Keratomalacia has been originally applied. Keratomalacia of the entire Cornea very seldom yields to treatment but results in the entire peeling of the whole Cornea with anterior staphyloma of the Iris, or in the loss of the whole thickness of portions of the Cornea with prolapsed Iris, resulting in adherent leucoma, or Staphyloma Cornea.

Pyogenic infection of the Cornea may set in, causing severe ulcers of the Cornea with hypopyon. Perforation of ulcer with prolapsed Iris results in adherent Leucoma or Staphyloma Cornea, with partial or entire loss of vision. Secondary glaucoma

is quite usual in these eyes with the result that the Staphyloma attains an enormous size that it bulges outside the Palpebral fissure giving rise to considerable discomfort and ugliness to the patient.

Diarrhoea, which occurs during any of the three stages in the course of the illness, if not properly controlled, and cured, leads to emaciation, anæmia, dropsy, and marasmus, with a fatal termination.

Bronchitis and Broncho-pneumonia also occurring during any of the three states in the course of the disease, if severe and if the patient is emaciated, will also end fatally.

Age and Sex Incidence.—Infants in their first two years of life are the greatest number of sufferers. The percentage diminishes as age advances. The number of sufferers between 2 and 6 are less than before that age, also still less between 6 and 14. Adults are not free from the disease, but are very much less frequently attacked than children. In my experience in children, the disease is almost equal in both sexes; but in adults the female sufferers are about 3 times as many as the males. Females, during pregnancy and lactation, have a great susceptibility for the disease. I get many cases of this type every year at my clinic, most of them too far advanced to do any good by treatment.

Although poverty is undoubtedly the cause of Malnutrition and the largest number of victims are found amongst underfed children, and women of the poor, the rich cannot be entirely excluded. I have had Keratomalacia amongst the rich Nambuthiris, the Zamindars of Malabar.

Ætiology.—It is proved beyond doubt by research workers by experiments on animals, and by clinicians that Keratomalacia is caused by the deficiency in the diet of the fat-soluble Vitamin A. Vitamin A deficiency has been proved to cause characteristic abnormality in the mucous membrane cells and the nerve cells. It is significant to note that in Keratomalacia, the epithelial cells of the Conjunctiva, the Cornea, the Epidermis, the mucous membrane of the Gastro-intestinal tract, and the respiratory tract are affected.

There are certainly other contributory causes. The deficiency of other Vitamins and active factors in the diet may help the disease. Intestinal parasites, especially round worm and hook worm, by their deleterious effect on the patient, undoubtedly help the disease. So also dysentery, both amœbic, and bacillary.

Similarly other debilitating diseases also help the disease. In Malabar especially in children round worm and dysentery have been frequently associated with Keratomalacia.

Treatment.—Early recognition of the disease and early treatment is attended with complete success. General Practitioners in cities, towns, and rural districts, are the first to come across these cases. They will be consulted for the diarrhoea, the skin eruptions, the night blindness, or the Xerotic patches in the Conjunctiva, the Catarrh of the nose, or the respiratory tract.

An adequate supply of Vitamin A in the food is the principal treatment of the disease. Pure Cod Liver Oil is the cheapest, and the best for administration by mouth, or in severe cases of diarrhoea, it may be rubbed on to the axillary, or abdominal skin. Concentrated forms of Cod Liver Oil, and Halibut Liver Oil containing Vitamins A and D of various concentrations are available in the market for oral administration. Concentrated preparations of Vitamin A are available for injections also. Healthy mother's milk contains all the Vitamin A necessary for the infant. Nursing mother should be given pure Cod Liver Oil, milk, butter, ghee and green Vegetables in abundance.

Children left off the breast should have an abundance of goat's milk, cow's milk, butter, and ghee mixed with a sufficient quantity of Cod Liver Oil. Green vegetables as Carrots are also useful.

Intestinal parasites should be got rid of at the beginning of the treatment. Santonine, Oleum Chenapodium for round worms, Carbon tetrachloride for the hook worm. I have a child 2 years and 2 months old just now under my treatment with Keratomalacia of the 2nd and early 4th stage with diarrhoea, and slight fever. Parents told me that the child has passed over 50 worms under Homeopathic treatment. Santonine followed by an emulsion of castor oil and Oleum Chenapodium was administered by me the first day the child was brought to me and the child passed over one hundred and eighty round worms, and the diarrhoea stopped immediately. With the continuation of Syrup Ferri Iodide m. xv and Adexolin m. xv thrice daily in milk, and the routine treatment for the eye, the child is improving rapidly.

Dysentery also should get treatment from the beginning, if amœbic, Emitine, and if bacillary, serum, should be injected.

Local treatment for the eye lesions in the 1st stage is not required, and in the 2nd stage mild antiseptic lotions, and Argyrol 5% solutions for washing, and instillation are all that is required. In Corneal affections in infants, and young children, Eserine $\frac{1}{4}$ th to $\frac{1}{2}$ % instillation is preferable to Atropine 1% which is used in older children and adults.

The Treatment of Corneal Opacities

BY

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THE blindness resulting from corneal opacities is very common in India and Burma. It is said that the general estimate that 30 per cent of all blindness is caused by corneal opacities would seem conservative. Corneal lesions, if untreated, produce gross visual disability resulting often in blindness of varying degree.

Much has been written of the treatment of corneal opacities and much advance has been made in recent years in medical and surgical procedures for the treatment of corneal scar and yet this condition still continues to baffle the best of ophthalmic surgeons. How often he is conscious of his inability and how limited are his knowledge and methods of removing even a tiny speck of deep corneal opacity lying in front of the pupil greatly interfering with the vision of the patient!

There are several varieties of opacities of the cornea. The two chief varieties are:—(1) Inflammatory opacities and (2) Non-inflammatory opacities. The Inflammatory variety may again be divided into two types, *viz.*, (a) Opacities due to Keratitis and (b) Opacities which are the result or residua of an injury or an inflammation of the cornea. The Non-inflammatory opacities are due to increased intra-ocular tension. The opacities vanish and subside with the fall of tension.

In dealing with the treatment of corneal opacities, I would confine myself to those permanent opacities which are the result of an ulceration.

It is important to remember that an opacity whether it is due to an inflammation or an injury tends to clear and improve as time goes on. This tendency is more marked and noticeable in the case of children.

The anatomical structures of the cornea are:—(1) Anterior Epithelium; (2) Bowman's membrane; (3) Corpuscular layer (Substantia Propria) (4) Descemet's membrane; (5) Endothelium.

The epithelium and endothelium regenerate when damaged but the intervening layers cannot. Bowman's membrane cannot

regenerate itself because it has no nuclei. Once damaged, it is replaced by foreign tissue, so there is a definite amount of opacity. The substantia propria, if injured, is replaced by the corneal corpuscles and so the scar tissue remains. If the Descemet's membrane is damaged as in the case of a perforating wound of the cornea, the two ends of the membrane curl away and the endothelial cells fill up the gap and lay down thicker Descemet's membrane. Corneal opacity also results from rupture of Descemet's membrane by forceps at birth.

The opacity of the cornea is called nebula, if thin and ill-defined; a macula, if localised and a little dense; and a leucoma, if very dense and white.

The cornea receives its nutrition from the limbus vessels, the aqueous and the tears. It can get sufficient nutrition from the limbus vessels and the tears and does not suffer from lack of nutrition following a complete peritomy and following section of three-fifths the corneal circumference or following the complete corneal grafts. This also shows that the cornea can obtain sufficient nutrition from the aqueous and the tears. It is important to know the source of the nutrition of the cornea as the treatment of corneal opacities to a certain extent aims at increasing the supply of nutrition to the cornea by causing local irritation and congestion in order to hasten the cells to do their work of regeneration.

The most important part of the treatment is, of course, the prevention of the corneal ulcers from spreading or perforation. In the treatment of recent opacities various remedies are employed. It may be possible to modify to some extent, the opacity of the resulting scar after the corneal ulceration has healed by adopting such measures as, (a) insufflations of calomel powder (b) gentle massaging with yellow oxide of mercury ointment, 1 to 5 per cent or dionin drops 5 percent, (c) subconjunctival injections of sterile 2 to 10 per cent solution of sodium chloride. Some advocate subconjunctival injections of 1 per cent Sodium Iodide in varying doses, 0.12 c.c. to 0.85 c.c. Two percent quinine bisulphate ointment (a protoplasmic poison) applied twice daily has also been found useful.

Sometimes relatively slight keratitis causes not only corneal opacification but also alteration of the refraction of the eye on account of the yielding of the outer coat of the eye. Suitable glasses will greatly improve the vision then. But if the scars are thick and permanent, optical aids are of small value. In

cases of dense opacities, the use of stenopæic apertures with strong lenses may be required to enable the patient to read.

Physical methods of treatment such as finger massage, electric vibro massage, electrolysis, ultra-violet light, x-rays and radium, have also their advocates. But on the whole, the use of drugs and the employment of physical methods are rather disappointing.

If medicaments and physical methods prove to be of no avail in removing the opacities, operative measures should be adopted. Optical iridectomy may be done if the opacity does not cover the whole cornea and if there is a clear cornea left at the periphery. In cases of unsightly leucoma, tattooing may be done for cosmetic effect. Tattooing may also afford better vision in cases of nebulae by eliminating their dispersive properties. The aim of tattooing is to render the leucoma opaque by introducing a colouring pigment which produces chemical reaction in the corneal tissues. The colouring pigments usually employed are Indian ink and platinum black. If the scar is superficial, an excision of the anterior layers of the cornea may be tried to improve the appearance.

Lastly, corneal grafting operations may be performed. These operations, known as keratoplasty, are usually carried on dense scars covering the whole cornea where other methods are of no avail to regain sight. Tudor Thomas in England, Castroviejo in the United States of America, Filatov in Odessa and Ortin in Spanish America have obtained satisfying results out of their operation of corneal grafting.

With improved methods and improved technique, Keratoplasty is full of promise for the future. Out of the several methods of Keratoplasty, such as Total Keratoplasty, Superficial Lamellar Keratoplasty and Penetrating Circumscribed Keratoplasty, the last one which consists in replacing a portion of the entire thickness of the opaque cornea by transparent corneal tissue gives the best results.

Tattooing of the Cornea

BY

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COLOURING or tattooing of the cornea (*Tinctio cornea*) was in olden days practised by Arabs, to permanently colour larger or smaller parts of the corneal tissue.

During the last century, this process, was again revived by de-Wecker, who punctured the cornea to be tattooed with a needle dipped in some dyeing material. Taylor used a bunch of needles to accelerate the process.

To imitate the colouring of the iris, Holth and others have recently experimented with different pigments, *e. g.* Uveal pigment from animals (Nieden), Metal colours in powders and organic dyes (Holth), Indian and Chinese inks (de-Wecker), Lamp-black and Candle-soot (Blaskovics). The pigment is directly introduced into the corneal tissue, mechanically.

The alternative method of staining the cornea by chemical process, though easier, is not so long lasting in practice. This is accomplished, by the application to the denuded area to be tattooed, of Goldchloride, or Platinumchloride, and reducing these with Tannic acid and Epinephrine, or Hydrazinehydrate.

Tattooing serves twofold purpose:—

I. Optical. II. Cosmetic.

I. Optical.—It is known that a transparent corneal scar disturbs the vision more than an opaque one. If we make this part of the cornea quite opaque, sharpness of the vision can be improved. (Always remembering that the remaining part of the optical zone is clear, and will remain so.). Unfortunately this process usually fails, because of this last reason. A whitish blur develops around the part tattooed, which disturbs the optical effect. The process is more effective in the case of small central nebulae. Tattooing if later is combined with iridectomy, gives better results, than tattooing alone.

II. Cosmetic.—The aim is easier, and, more certain to attain. Thick corneal scars are very disfiguring, but with the colouring of the part, a great service is done to the patient. However well the colouring has been accomplished, it does not last for ever, in time it starts fading, in 5-10 years very few traces of the colour remain. Nothing prevents us however from repeating it.

The tattooing is usually done for the following conditions:—

- (1) Making the transparent scar opaque. This is done for optical reason but, not much advantage is gained by this.
- (2) To improve the appearance. It is desirable to make the leucomata cornea invisible by resorting to colouring. If the scar extends over the whole of cornea, then we tattoo a central deep black circle corresponding with the pupil, as compared with the surrounding lighter colour of the iris. Thin scars are first transformed into thick ones, by deep cutting and then coloured, as this is only suitable for firm smooth scars in perfectly quiet eyes.
- (3) To cover the "Star" in the eye (Opaque white shine of a cataract in a blind eye). This is done by tattooing the central clear cornea, with black colouring, to a diameter of 6 mm. The results are not very encouraging, as sideways the grey pupil shines through.

The usual procedures are:—1. *Colouring with an Ink (Chinese or Indian, powdered, dried and sterilised, or fluid ink made germ-free by boiling):*—None of these inks can be guaranteed germ-free, also because of some unknown ingredient used in the making of these inks they cause irritation in the eye.

Instead of pricking with the needles, it is advisable to make small cuts in the cornea. The cuts be made in half thickness very close together and parallel (first horizontal and then diagonal). The distance of the cuts, one from the other, should be at the most $\frac{1}{2}$ mm. On the worked surface, a drop of the ink is dropped and worked thoroughly with the spatula. The eye is bandaged for a few days. After two or three days the surface is usually, smooth, shiny and beautifully black.

2. *Colouring with candle-soot, or Lamp-black:*—The method is very convenient, the colouring material is easily obtainable and is obviously germ-free. It consists of fine-grain chemically pure carbon, which does not irritate the eye. The candle-soot is obtained by simply placing one or two glass slides on the candle flame, without disturbing the wick.

After thoroughly washing and anæsthetising the eye, lid-retractor is applied. (The eye must never be held with toothed forceps otherwise the conjunctival punctures will be tattooed as well). From the area to be tattooed, the epithelium is very

carefully scraped with the aid of a sharp spoon. To get a good round circle we can mark the region first with the 4-5 mm. compass (Elliot's). The scraping of the epithelium is important, if not done, the cuts will close up, and hinder the soot from penetrating. The cutting of lines in the corneal tissue is done with a rounded lancet, or a small cataract knife. One makes single row of cuts, the surfaces of which overlap like tiles. When these cuts are deeper, one can introduce more soot into them, and there are more black coloured surfaces behind each other. When one cuts on the slant, one must not cut any further lines in a different direction, otherwise the corneal lamellæ would drop out.

By means of the iris spatula, one takes the soot from the glass slide, and heaps it on to the cornea, and rubs it in carefully. The moisture mixes with the soot, and a thick black substance is formed, which can be rubbed in. If the moisture is not enough, one can take a tear drop from the lower conjunctival sac. We rub for a few minutes, and if necessary add more soot. To finish with, we remove the surplus soot if any has come on the sclera etc with a dry swab. The eye is then bandaged, which is changed every other day, until the surface is smooth, black and shiny (Usually 6—8 days).

3. *Colouring with Gold-chloride*:—It would be the best method, if only the colour will last longer. Unfortunately it disappears completely in from 1-2 years. It is a success to start with, although the colour is dark-brown and not black. Instead of Gold, Platinum and Silver have also been tried, but with the same drawback i.e; the colouring does not last longer.

The mixture is to be freshly prepared, this consists of 3% Gold-chloride exactly neutralised by sodium-bi-carbonate. Epithelium is scraped from the area to be tattooed. The absorption of the liquid can be helped, by superficial cuttings as explained previously. Cotton-swab is twisted on a glass rod, then dipped in the mixture and squeezed lightly. This is then applied on the denuded area, for a couple of minutes. The process is repeated once again. The area is coloured lemon yellow. The result is noted next day, when the tissue impregnated by the Gold-chloride, has been reduced to "Koloidal". This reduction can be immediately brought about, if one uses 2% Hydrazine-hydrate, or adrenalin, but the final result is not good.

None of these methods solve the problem. When the eye no longer reacts to light, and there is no hope of its regaining its function, it must be considered, whether it would not be better (for cosmetic reason) to provide an artificial eye.



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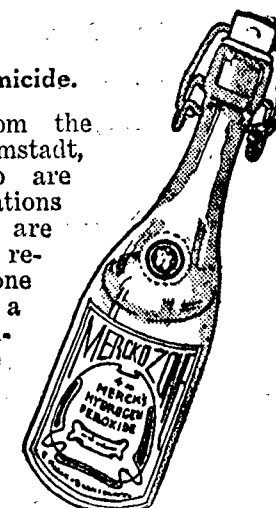


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Prevention of Blindness in Cases of Small-Pox

BY

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SMALL-POX is one of the commonest causes of blindness in places where it occurs either in endemic or epidemic form. The easiest way to prevent blindness from this very dangerous disease is to prevent the disease itself by vaccination and re-vaccination. The ideal of getting every person and child vaccinated and re-vaccinated will, most probably, never be reached, hence there will be cases of Small-Pox.

The eye complications resulting in blindness, in Small-Pox, as described by the various observers, are Hypopyon ulcer, Spreading ulcer of the cornea with perforation and Iris prolapse, Iritis, Iridocyclitis and Secondary Glaucoma.

The conditions, which one sees in the eyes which are already blind from Small-pox, are (a) large thick leucoma covering practically the whole of the Cornea, (b) adherent leucoma, extending over a large part of Cornea, (c) anterior staphyloma, (d) atrophic condition of the eye-ball, (e) Secondary Glaucoma.

It is quite interesting to study the genesis of the various conditions in different types of Small-Pox. The eye complications are most common in the confluent type. (a) The vesicles form in conjunctiva and sclero-corneal region within the first week of the disease usually the 4th or 5th day, and on the Cornea after the acme of the disease is passed i.e., when the patient is on the way to recovery. (b) Breaking down of old Corneal Scar Leucoma—this is very frequent; the epithelium over this scar is raised (vesicle formation?), then it gives way forming an ulcer. (c) Iritis and Iridocyclitis—very rare. (d) Sub-conj. hæmorrhage common in hæmorrhagic type of Small-pox but occurs in other types too. (e) Keratitis with Lagophthalmos, designated by us as "Exposure Keratitis". This is not a complication of the disease, but it is due to bad nursing and it can be prevented by good nursing.

None of the above conditions should cause Blindness. Chief factors in the causation of Blindness have been observed to be two:—first, presence of infection in the conjunctival sac, specially, Pneumococcus, Streptococcus, Staphylococcus aureus and

Koch Week's Bacillus and secondly, ulceration of the cornea due to any cause, *e.g.*, Keratitis with Lagophthalmos, breaking down of Sclero-corneal and Corneal vesicles, marginal ulceration of the cornea as a complication of Acute Conjunctivitis, or in other words Corneal ulceration + the infection in the Conjunctival sac.

From the above observations, it is evident that the Blindness can be prevented if any or both of the above factors can be either prevented or controlled.

Prevention of Corneal Ulcer.—Corneal ulcer due to breaking down of vesicles in Sclero-corneal region or on the Cornea cannot be prevented. But those cases are very few. Corneal ulcers due to Lagophthalmos and conjunctivitis can be prevented. By far the largest number of corneal ulcers are due to lagophthalmos. When a patient is either unconscious or semi-conscious, the eye-lids remain half open exposing the lower part of the cornea. The epithelium of the exposed part of the cornea gets dry. Then the desquamation and exfoliation of the epithelium leads to corneal ulceration (Keratitis with lagophthalmos or "Exposure" Keratitis). This can be quite easily prevented by keeping the lids closed—which require constant attention to the lids. In a large Hospital where one nurse has to look after a number of cases, the lids are closed over a liberal amount of sterile Boro-vaseline which prevents the epithelium from getting dry. The corneal ulcer due to acute conjunctivitis can be prevented by energetic treatment of the conjunctivitis.

Control of Infection of Conjunctival Sac.—There are two sources of infection, the most dangerous, Pneumococci, being in the Lacrimal sac, and in the conjunctival sac itself. Infected Lacrimal sac is the cause of Hypopyon ulcer. In a case of Small-Pox, there is no question of removing an infected Lacrimal sac and Hypopyon ulcer, in a small pox case, is almost sure to lead to Blindness. So, when a patient is admitted, his Lacrimal sac is first examined, and if there is any suspicion of infection, the puncta are cauterised with pure carbolic acid. This is repeated if required.

Regarding the infection in the conjunctival sac itself, quite a large number of normal looking conjunctival sacs, as previously worked out in this Hospital, are infected either with Staphylococci or streptococci, a mixed infection. The conjunctival sac of every case, on admission, is considered to be infected. His conjunctival sac is washed out with Lotio Hydrarg Oxyeyanide (1 in 15,000) and 4% argyrol drops are instilled and this is

repeated 3 times daily. If there is any sign of discharge from the conjunctival sac, 10% argyrol drops are instilled.

Summary.—Most of the blindness in Small-pox is due to infected corneal ulcer. Iritis and Iridocyclitis are very rare and easily respond to treatment. As soon as a patient is admitted his Lacrimal sacs are examined and if there is any regurgitation, the puncta are cauterised with pure carbolic acid with a very fine pointed probe and repeated if required. The conjunctival sacs are washed out with Lotio Hydrarg Oxyeyanide (1 in 15,000) 3 times daily and Gutt argyrol 4% is instilled 3 times daily. If there is any secretion, Gutt argyrol 10% is used 3 times daily. If the patient is unconscious or semi-conscious, and the eyelids are half open exposing the lower part of the cornea, a liberal amount of sterile ung. Acid Boric is put into the conjunctival sacs and the lids are closed down and constant attention has got to be given so that the lids may not remain open.

This routine method was introduced in this Hospital during the big epidemic of 1936—when only one man went out cured with one eye blind. The Epidemic of 1939 is almost over—and no person has gone out blind even of one eye. This is mentioned to show the efficacy of the above routine treatment.

The Intra-capsular Extraction in Senile Cataract by Tumbling of the Lens

BY

Rai Bahadur Dr. Mathradass Pahwa,

*Late Hony. Asst. Surgeon to H. E. The Viceroy, Retired Civil Surgeon,
Late In-charge, Mathradass Hospital, Moga, Kaiser-I-Hind Gold Medalist.*

THE reason which has prompted me to write this article is the fact that since the year 1903, when I first started in the profession, I have performed 147,461 Cataract Operations almost all of them by the Intra-Capsular Extraction. The technique, which I have followed throughout, was devised by me shortly after I first began Ophthalmological Surgery. The popularity of this operation can, to some extent, be imagined by the magnitude of the figure given above. Although I have trained many a surgeon in this operation, but never before, due to some reason or other, I have published its details.

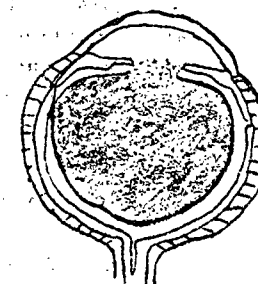
Before describing the technique of the operation, I shall like to recapitulate the reasons as to why I prefer an Intra-Capsular extraction to an Extra-Capsular one. The controversy between these two operations is always going on in all countries, the Indian Surgeons, on the whole, specially the Ophthalmic Surgeons in the Punjab have always favoured the Intra-Capsular method, while elsewhere Extra-Capsular operation is the rule, although Elschmig, Sinclair, and Barraquer have adopted the Intra-Capsular operation as a routine in their clinics. I need not discuss in detail the advantages of one operation over the other, as they are dealt with in most of the Text-Books on Ophthalmology, but there are certain points which deserve greater attention and emphasis. Briefly the advantages offered by the Intra-capsular operation are:—

- (1) That it does not require two operations as the extra-capsular operation invariably does; this is specially the case when one is dealing with Indian patients, as they do not like to come back for a second operation.
- (2) That the operation can be performed even when the cataract is not completely mature, thereby saving the patient a lot of suffering which is usually his lot, when he has to wait for a slowly progressing cataract in both eyes to mature. With Indian patients it is almost a matter of necessity to operate on them when they come out long distances from their homes.

(3) As there are no shreds of capsule or lens matter to be caught in the edges of the wound, so the chances of subsequent iritis and cyclitis are reduced to the very minimum.

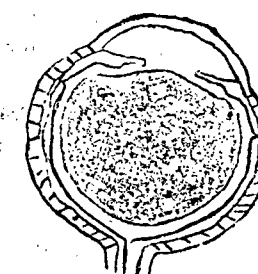
(4) The ultimate vision after an Intra-capsular operation is very much better than after the extra-capsular extraction. There are two reasons to account for this, one is that as the lens and its capsule are removed in toto there is no chance of an opaque capsule to obstruct the passage of light. The

second reason, a more potent one, is that when after a capsulotomy operation subsequent discission is performed to tear the capsule, the anterior surface of the vitreous is also torn, and if after some months this surface is examined with a Slit Lamp an appearance something like this will be seen, fine strands of the vitreous body emerging through the pupil. These thread-like projections in course of time tend to



become opaque, so interfering with the visual acuity.

On the other hand, in an Intra-capsular operation properly performed there is no tear in the vitreous, and the appearance of its anterior surface is quite regular, even, untorn slight bulge through the pupil as illustrated.



Against these advantages in an Intra-capsular extraction, there is one very great disadvantage, which if it does occur is a serious matter indeed, that is, the prolapse of vitreous, it is this accident which has led many surgeons to abandon this operation altogether. To combat this complication, I have perfected the method of delivery of lens by tumbling. All the ophthalmic surgeons who have visited my clinic are struck with the rarity of this accident.

In a later article, I shall give the exact figures as regards

the incidence of this as well as other complications; here it is sufficient to mention that in all the cases operated upon by me prolapse of vitreous has not occurred in more than $\frac{1}{2}$ to 1 per cent.

Technique of the Operation.—This operation is the method of choice with me and many surgeons have been trained in my clinic because of its remarkable simplicity, freedom from complications, and little instrumentation.

Indications:—I perform this operation in all types of senile cataract, mature or not, hard or morgagnian cataract. The technique in morgagnian cataract differs from that of hard cataract.

This operation is contraindicated in juvenile, traumatic, complicated, and secondary cataracts. All cases of senile cataract are dealt by me with this operation, and apart from the varieties mentioned above I do not select my cases. It is obvious from this statement that this operation has a very wide applicability.

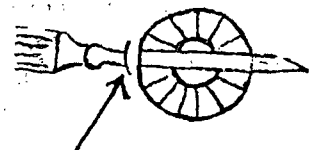
Instruments required:—I have indicated above that this operation is very simple and requires few instruments, they are, one eye speculum, one pair of fixation forceps, one Graefe cataract knife, one iris forceps, one pair of Iris scissors, De Wickers are preferable, and two squint hooks.

A trained assistant who knows what is expected of him at each stage, and who is well versed in the idiosyncrasies of the surgeon is necessary.

Anæsthesia:—2% Butyn solution one drop three or four times at five minutes interval before the operation is the anæsthetic used by me, it gives a fairly complete anæsthesia. I do not use facial block as a routine, this is only necessary in a few nervous patients. It is perhaps advisable for the beginner to employ facial block in all cases.

Pre-treatment:—Patient is laid upon the table, and his eye is irrigated thoroughly with 1/5000 perchloride of mercury. The eye-lashes of the upper lid are cut with a pair of scissors dipped in sterile vaseline. Cutting of the lashes is perhaps not necessary.

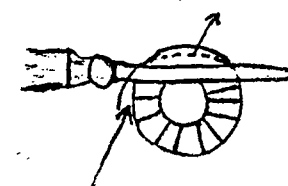
Corneal section:—After placing the speculum in the eye, the patient is asked to look towards his feet, and the eye is fixed there with the fixation forceps. The Graefe knife is inserted at 9 O'clock (right eye) about one millimeter outside the limbus, this forms a scleral bridge to provide nourishment for the corneal



Point of Entry of Knife in Sclera.
Formation of Scleral Bridge.

flap. The knife is carried straight across the anterior chamber, and is brought out just at 3 O'clock (right eye, left eye—the section is from 3 to 9 O'clock). With gentle sawing movements knife is carried straight upwards, and the section is finished about 1 millimeter below the upper border of cornea.

Site of Finishing the Section.



Scleral Bridge.

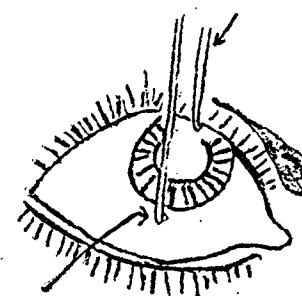
It will be obvious from above that I believe in low section, the scar is hidden by the upper lid, therefore it is no disadvantage, on the other hand with a low section the delivery of the lens is considerably facilitated.

With the beginners it is a common fault to press the eyeball with the fixation forceps; this should always be guarded against, and just before finishing the section the fixation forceps should be removed.

Iridectomy:—I perform a fairly wide iridectomy in all cases, this again makes the delivery of the lens easier without requiring undue pressure on the lens to be pushed through the uncut pupil. Through the corneal wound iris is pulled out with the iris forceps, and it is cut with the iris scissors.

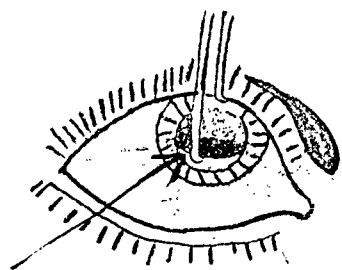
Delivery of the Lens:—At this stage the speculum is removed from the eye, and the assistant lifts up the upper eyelid with a strabismus hook held in his right hand, he pulls the lower lid down with his left hand. The surgeon places his left thumb just above the eyebrow, he slightly pulls the upper eyelid upwards and steadies the eyeball, and in the other hand he takes hold of the other strabismus hook. This hook is now applied on the sclera a few millimeters below the sclero-corneal junction at about six O'clock. It is slowly drawn upwards up to the limbus exerting

This Hook Lifts up the
upper Lid.



To show the site where the Hook
is First Applied,

very slight pressure backwards. This movement dislocates the lens, it is a mistake to apply too much pressure, this is not necessary, a gentle touch is quite enough. If with the first attempt lens is not dislocated from the zonule, this manoeuvre should be repeated. As soon as the lens is dislocated, the lower pole of the lens is gently pushed upwards with the same hook, and now the lower border

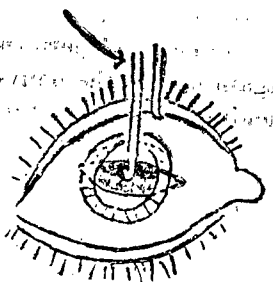


Lower Pole of the Lens Supported and Gently Lifted By the Hook. Cornea Intervenes Between Lens and Hook.

of the lens will be seen in the anterior chamber. The hook lifts up the lower pole of the lens still further until it presents in the corneal section. This step is difficult to describe without the aid of photographs, which unfortunately are not yet ready, I hope to publish them at a later date, but these drawings might help to demonstrate this step.

It will be gathered from the above description that the lens turns a complete somersault before it is delivered, that is that the lower pole of the lens comes out first through the corneal wound, and the upper pole last, and that when the lens comes out, it is the posterior surface which is looking forwards and its anterior surface is lying backwards. This manoeuvre is called 'Tumbling of the Lens', it ensures that the lens with its capsule is completely free from its attachments before being delivered through the corneal wound, thereby the chances of prolapse of the vitreous are very few indeed.

This Hook Lifting the Lower Pole of Lens still further. Now Lens Has Turned Complete Somersault.



Previously delivery of Morgagnian cataract used to be carried out by a somewhat similar procedure (the movements of the hook are of course different), but I was the first to attempt the extraction of the ordinary hard senile cataract by tumbling the lens, and now I have trained many workers from all parts of the world who follow this method, and I have no doubt that this operation will gain still further in popularity.

Reposition of the Iris:—After the lens is delivered, Iris is carefully freed from the edges of the corneal wound and replaced either with the strabismus hook or with an Iris Repositor.

The upper lid is lowered gently, the patient is warned in advance not to close his eyes tightly or in any way squeeze his eyes. The eye is covered with a piece of cotton-wool smeared in sterile vaseline, and both eyes are bandaged.

After-treatment:—Patient should lie prone on his back, and

remain quite still for the first 4 to 6 hours, after which he is permitted to turn towards his un-operated eye.

Unless there are any indications which demand examination of the eye earlier, I generally examine the eye on the 7th day, when the unoperated eye is left uncovered.

On the 10th day the operated eye in the absence of complications such as hyphæma, prolapse of the iris, ununited corneal wound, sepsis etc., is unbandaged and covered with a green shade.

Summary.—Like any other surgical procedure a little patience and considerable practice is required before the art of neat delivery is mastered, perseverance is essential, but if it is mastered once, the surgeon will find it a great aid in his cataract operations, he will have acquired a simple, efficient, and a safe method of doing his Intra-capsular cataract operations. I have mentioned above that up to the present, I have performed 147,461 cataract operations with this way of delivering the lens, and many more have been performed by the various surgeons who have visited my clinic, and all of them have found that it has lived up to their expectations, thereby they have been able to render a great service to humanity as well as to ophthalmic surgery.

The Diagnostic Significance of the Pupil and its Reflexes

BY

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I SHALL first give a table of the various Normal and Abnormal Pupils and their Reflexes and then describe them separately under their headings:—

I. Normal Pupil:—

- (1) At different ages.
- (2) In different refractive errors.
- (3) In sleep.
- (4) Mechanism of the pupil movements.

II. Normal Pupil Reflexes:—

- (1) Light Reflex:—(a) Consensual Light Reflex
(b) Galvanic Light Reflex.
- (2) Near Vision Reflex.
- (3) Reflex dilatation of the pupil or Cilio-Spinal Reflex.
- (4) Oculo-Pupillary or Trigeminal Fascial Reflex.
- (5) Lid-closure Reflex (also called Orbicularis Reflex or Westphal-Philtz Phenomenon of Gifford Galassi Reflex).
- (6) Cortical Reflex of Haab (also called Attention Reflex).

III. Abnormalities of the Pupillary Reaction.—

1. Disturbances of the Light Reflex:—
 - (1) Amaurotic Rigidity of the pupil (Bach) or Reflex Deafness (Heddens).
 - (2) Hemipic Pupillary Reaction (Wernicke).
 - (3) The Neuro-tonic Reaction.
 - (4) Absolute Rigidity of the Pupil.
 - (5) Mytonic Reaction.
 - (6) Reflex Rigidity of the pupil or the Argyll Robertson pupil.
 - (7) The Paradoxical Light Reaction.
2. Disturbances of Near Vision Reflex:—
 - (1) Failure of the Near Vision Reflex.
 - (2) Perverse Near Vision Reflex.
3. Disturbance of Reflex Dilatation of Pupils:—
 - (1) Loss of the Cilio-Spinal Reflex.

- (2) Absence of the normal pupillary oscillations.
- (3) Undue activity of the Cilio-spinal Reflex.

4. Hippus.

5. See-Saw Pupils.

IV. Abnormalities of the Size of the Pupils:—

1. Abnormally Small Pupils:—

- (1) Spastic myosis.
- (2) Paralytic myosis.

2. Abnormally Large Pupils:—

- (1) Spastic mydriasis.
- (2) Paralytic mydriasis.

3. Inequality of the Size of the Pupils (called Anisocoria).—

- Due to:—(1) Benign causes.
(2) Pathological causes.

I. Normal Pupil.—*Normal Pupil* should always be equal in all states of illumination, accommodation and convergence. Pupils, which in broad daylight are less than 2.5 m.m. or more than 5 m.m. in diameter, are abnormal.

Age:—Diameter greatest before 20; stationary between 20 and 25 and then declines; in the new-born and in the aged the pupil is very small.

Refractive Errors:—Large in Myopia, small in Hypermetropia.

In sleep:—Pupils are small and on waking up they dilate widely till the patient becomes conscious, when they regain their original diameter.

Mechanism of the pupil movements:—There are 4 theories, as to the causation of the pupillary movements, which are briefly as follows:—(1) changes in the fulness of the vessels of the Iris. (2) Increase in the intra-ocular tension, (3) Elasticity of the tissues of the Iris and vessels. (4) Inhibition and stimulation of the action of the Sphincter.

II. Normal Pupil Reflexes.—**1. Light Reflex.**—Gradual increase of light on the pupil produces no effect, but a sudden illumination causes contraction of the pupil. (Direct Reaction).

(1) *Consensual Light Reflex:*—In this, illumination of one eye causes equal contraction of both eyes. When testing an eye for Direct Reaction and if this fails to occur (*i.e.*, when the pupil

does not react when the light is thrown on the eye), the failure may be due either to defective light perception or to changes in the iris, such as paralysis, adhesions, etc. To decide which of these is the cause we try the consensual light reflex as follows:—(a) while alternately shading and illuminating the test eye, the pupil of the other eye remains immobile, this proves deficient light perception (or rather defective light conduction) in the former. (b) If on alternately shading and illuminating the other eye, the pupil of the test eye remains immobile this must be attributed to changes in the Iris in the test eye.

(2) *Galvanic Light-Reflex*:—In this, application of Galvanic stimulus to the eye produces a sensation of light as well as pupil contraction.

2. *Near Vision Reflex*—In this the pupils contract when the patient looks at near objects. This occurs even when the eyes are shaded or amblyopic, provided the patient is directed to look at a near object so as to converge; it always occurs in both eyes even when one is excluded or shaded. Errors of Refraction do not interfere with the contraction. Atropine abolishes it.

3. *Reflex dilatation of the Pupil (Cilio Spinal Reflex)*.—This reflex dilatation of the pupils is influenced by sensory and psychical stimuli, e.g., emotion, excitement, mental shock, fear, anger, strong effort of attention or any stimulation of the tactile, pain, muscular or special senses. This reaction is caused by stimulation of an anæsthetic area of the skin if the anæsthesia is due to a central lesion; but it cannot be produced if due to a peripheral lesion. A contracted pupil (due to light) dilates when pressure is made upon a tender spot which is due to some organic disease but if the tenderness is of functional nature, as in pressure spots of Hysteria, no dilatation occurs.

4. *Oculo Pupillary Reflex &c.*—Pressure with a Lachrymal probe on the skin at the External canthus or pricking the skin of the cheek causes dilatation.

5. *Lid Closure Reflex &c.*—Contraction of the pupils on closure or attempted closure of the lids.

6. *Cortical Reflex of Haab*.—It consists in a visible contraction of the pupil which occurs when the individual under observation looking straight before him in a darkened room, directs his attention but not his gaze to a small light placed in front and to one side of him.

III. **Abnormalities of the Pupillary Reactions.**—1. **Disturbances of the Light Reflex.**—(1) *Amaurotic rigidity of the pupil*:—In this, if one eye alone is affected, the direct reaction is absent, but the consensual reaction is present. i.e., illumination of the affected eye causes no contraction of either pupils while illumination of the other eye (sound eye) causes contraction of both pupils. The symptom indicates a complete interruption of the centripetal side of the reflex arc in front of the chiasma, i.e. a lesion of the Retina or Optic nerve. The other reflexes are not disturbed. Blindness will as a rule be present on the side of the lesion. As the Retina and Optic nerve are generally affected in this lesion the diagnosis can be confirmed by Ophthalmoscopic Examination. But in cases of injury to the Optic Nerve behind the point of entrance of the central Retinal Vessels and in Retrobulbar Neuritis in both of which the Ophthalmoscopic signs are absent or doubtful the diagnosis is confirmed by the above pupillary reaction.

This pupil symptom occurs in Syphilis of the Central Nervous System and disappears on anti-Syphilitic treatment; but if due to parasyphilitic diseases, the pupil symptom does not disappear on Anti-syphilitic treatment.

In the above description we have considered the lesion of the centripetal path in front of the Chiasma. A bilateral lesion of the connecting fibres between the Optic Tracts and the Sphincter Nuclei would give the picture of *Bilateral Amaurotic Rigidity without Amaurosis*.

(2) *Hemiopic Rigidity of the Pupil*:—(also known as Wernicke's hemiopic pupillary reaction). In this phenomenon, there is loss of the pupillary light reflex when light is thrown upon the blinded portion of the Retina.

EXPLANATION:—If the Hemianopia is right-sided, (i.e., blindness of the left half of each Retina), illumination of the left half of either Retina causes contraction of both pupils.

Hemianopia accompanied with the above pupillary reaction, is a characteristic symptom of a lesion of one Optic Tract, because the break in the conduction must lie below the spot at which the fibres to the oculo-motor nucleus are given off i.e., they must lie in the Optic Tract itself, but if Hemianopia is present without the pupillary reaction above described or in other words if the pupillary light reflex is intact, then the lesion is to be located higher up, e.g., in the external geniculate body, in the internal capsule or even in the Cortex of the brain itself.

(3) *The Neurotonic Reaction*:—It consists in a slow contraction of the pupil, coming on only after the illumination has been kept up for some time. No diagnostic significance. It indicates a lesion of the centripetal side of the Reflex Arc.

(4) *Absolute Rigidity of the pupil*:—If the condition is unilateral the affected pupil does not react on illumination of either eye nor does it contract on near vision; it also fails to dilate on sensory stimulation. It is moderately dilated and therefore appears larger than the normal pupil, but this irregularity disappears in dark. If the condition is bilateral both pupils are dilated and the light and near vision and Cilio-Spinal Reflexes are absent. The lesion which causes absolute inactivity of the pupil is situated in some part of the centrifugal side of the pupillary reflex arc i.e., between the Sphincter muscle and the Sphincter nucleus, so that we are dealing with a paralysis of the Sphincter.

The lesion will be nuclear (or possibly fascicular) if this symptom is uncomplicated with other paralysis and the same is true of the case in which there is at the same time paralysis of the ciliary muscle (Ophthalmoplegia Interna). If to these be added insensibility of the Cornea the lesion is situated in the ciliary Ganglion. Put a drop of Eserine to ascertain whether the lesion is in front of or behind the Ciliary Ganglion. If behind the Ciliary Ganglion then the Eserine will contract the pupil. If in the Ganglion or Short Ciliary Nerves, Eserine will have no effect. Among the Specific Causes one is Acquired Syphilis.

(5) *Myotonic Reaction*:—In this, there is a very slow recovery, to its normal state, of the pupillary contraction brought about by light convergence, accommodation or the orbicularis reflex. It indicates a *Centrifugal lesion of the pupillary reflex arc*. It only occurs in the presence of Organic diseases of the nervous system, e.g., Tabes, General Paralysis etc.

(6) *Reflex rigidity of the pupil (or the Argyll-Robertson Pupil)*:—The affected pupil does not contract to direct or indirect light stimuli nor does it dilate on painful or sensory stimuli; but it contracts in near vision (accommodation) and convergence. In this the reflex arc running from the Optic Nerve to the oculo-motor nucleus is interrupted, while the connections of the centres for the pupil, for accommodation and for convergence which adjoin each other in the oculo-motor nucleus are undisturbed. This symptom is generally found in Tabes and Progressive Paresis and is so very rare in other nervous diseases that it is almost pathognomonic of the above two diseases.

Resemblance and differences between Argyll-Robertson pupil and Amaurotic Rigidity.

If bilateral both resemble each other in the absence of the pupil reaction to light with preservation of convergence reaction.

DIFFERENCES BETWEEN THE TWO :—

<i>Argyll-Robertson Pupil.</i>	<i>Amaurotic Rigidity of the Pupil.</i>
(1) Pupils contracted.	Pupils dilated.
(2) Near vision reflex more active.	Less active
(3) No blindness.	Accompanied with blindness.
(4) Evidence of Tabes, General Paralysis &c.	Relatively little diagnostic value.

(7) *The Paradoxical Light Reaction*:—In this there is contraction of the pupil on shading the eye and dilatation on illuminating the eye. Of no diagnostic value.

2. Disturbances of near Vision Reflex.—(1) *Failure of the near vision reflex.*

(2) *Perverse near vision reflex*:—Pupils dilated on fixing a near object and contracted with relaxation of convergence and accommodation.

3. Disturbances of Reflex Dilatation of pupils.—(1) *Loss of Cilio-Spinal Reflex* may result from a lesion of the sympathetic supply to the Iris; but removal of Cervical Ganglion (for the relief of Glaucoma,) does not abolish it. It accompanies Argyll-Robertson Pupil.

(2) *Absence of the Normal pupillary oscillations.*

(3) *Undue activity of the Cilio-Spinal Reflex.*

(4) *Hippus*:—Consists in a constant and rapid change in the diameter of the pupil, occupying on the average a period of one to three seconds, independent of illumination, convergence, and sensory or psychical impressions and with an average amplitude of 2 to 3 millimeters. It is usually bilateral and is found in Multiple Sclerosis and Retrobulbar Neuritis.

(5) *See-Saw Pupils*:—This term is used in reference to three groups of cases:—(1) Cases with unequal pupils (2) Cases in which one pupil (the normal one) changes in size according to illumination &c., while the other remains fixed. (3) Cases in which one pupil (the abnormal one) changes its size independently of light, convergence etc.

IV. **Abnormalities of the size of the Pupils.**—1. Abnormally small pupils may result from irritation of the pupil contracting mechanism (spastic myosis) or from paralysis of the pupil-dilating mechanism (paralytic myosis).

(1) *Spastic Myosis* is found in local diseases, Iritis, inflammatory affections of the brain and meninges, in cerebral hæmorrhage, intracranial conditions causing irritation of the third nerve or their nuclei, in prolonged accommodative efforts and by alkaloids which contract the pupils (myotics); other poisons such as opium and morphine, chloral and nicotine also cause contraction of the pupil. In Spastic Myosis the light and near vision reflexes are abolished or are very slight.

(2) *Paralytic Myosis* is one of the most important symptoms of paralysis of the Cervical sympathetic. It very frequently accompanies spinal lesions and as a bilateral symptom is an important sign in Tabes Dorsalis, it also occurs as a spinal symptom in progressive paresis.

Paralytic differs from Spastic Myosis in that mydriatics fail in the former case to dilate the pupils.

2. Abnormally large pupils may be due to irritation of the pupil dilating mechanism (spastic mydriasis) or to paralysis of the pupil contracting mechanism (paralytic mydriasis).

(1) *Spastic Mydriasis* accompanies conditions of cerebral irritation of different kinds and continued irritation of the Spinal chord and its membranes, e.g., in the presence of intestinal worms, in case of psychical excitement and hysteria. There is slight contraction of the pupil's light and near vision but no dilatation on sensory stimulus. Mydriatics produce full dilatation. Action of myotics is limited.

(2) *Paralytic Mydriasis* is far more frequent. It is caused by paralysis of the fibres of the oculo-motor nerve, whose branches innervate the intrinsic muscles of the eye, i.e., the Sphincter pupillæ and the Ciliary muscle. It is found in compression of the brain, late stages of meningitis, during epileptic fit and sometimes in tabes and general paralysis and syphilis.

3. **Inequality of the Pupils (Anisocoria).**—(1) *Benign Causes* :—

(a) Slight degrees of inequality often occur in healthy persons, often congenital. The inequality in these cases is not associated with any disturbance of the reactions, and the difference in size persists in all states of illumination, convergence &c., (b) another benign cause is a difference in the kind or degree of Refraction of the two eyes,

(2) *Pathological inequality of the pupils* is an evidence of one-sided or bilateral but unequal miosis or mydriasis. Therefore we are dealing with lesions which interfere with the Afferent and Efferent sides of the light reflex arc or with the Sympathetic supply to the Iris.

(a) If the inequality is the result of one sided interference with the *Afferent path* for the light-reflex, the pupil on the affected side will be large and will present the characteristics of Amaurotic Rigidity. The inequality will diminish during the act of convergence. Miotics and Mydriatics will have their usual effects.

(b) When the inequality is due to a one-sided lesion of the *Efferent path* of the reflex arc there will be no visual disturbance; the difference in size may disappear in shade and it will be more marked in Convergence.

(c) Inequality due to a *paralysis of the sympathetic supply* on one side (paralytic miosis) will be seen best in the dark, the affected pupil failing to dilate. The dilatation on sensory stimulation is absent. Syphilis, being the most common cause of unilateral mydriasis, is therefore an important cause of inequality of the pupils. In cerebral hæmorrhage leading to compression of the brain, dilatation of the pupil may be observed on the affected side.

Treatment of Glaucoma

BY

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GLAUCOMA is a term applied to various conditions accompanied by pathologically increased intra-ocular tension. There are several varieties of glaucoma; etiologically, pathologically and clinically the picture differs so widely that they cannot be regarded as forms of the same disease.

Hippocrates did recognise hypertension and gave the disease the name of glaucoma from the sea green colour of pupillary reflex in absolute glaucoma. The following are varieties of glaucoma:—

- I. (1) Primary acute glaucoma.
 (2) Chronic inflammatory glaucoma.
 (3) Absolute glaucoma.
 (4) Chronic simple glaucoma.
- II. Secondary glaucoma.
- III. Buphthalmos.

The most important instrument for diagnosis is tonometer. The first and most important symptom is increased intra-ocular tension recognised either by palpation or actual measurement with tonometer.

The patient is rarely seen during the prodromal stage to enable the surgeon to abort the acute attack by prompt administration of miotics. If the patient comes when free from attack, the eye apparently looks like normal but by careful elucidation of history and comparison of tension of both eyes, a diagnosis of glaucoma can be made out. The patient must be explained the gravity of the attack and miotics regularly prescribed to control tension and recurrence of the attack.

The usual miotics are pilocarpine and eserine. Oily solutions are preferable to aqueous ones. The strength of solutions varies from .25 to 2% depending upon the intensity of tension; the higher the tension, the greater the strength.

Primary Glaucoma.—If the patient presents with acute symptoms *i.e.*, acute primary glaucoma, the diagnosis is extremely easy by the history of intense pain, œdema of the upper lid, congestion of the conjunctival vessel, lacrimation, shallow

anterior chamber and the dilated pupil and the impossibility of the fundus examination due to pressure, œdema of the media and the last but most evident and important symptom of raised intra-ocular tension by palpation, eye ball appearing stony hard under fingers if accuracy is desired by actual measurement with tonometer. During this stage 1% eserine by way of eye drops is the drug of choice used every quarter of an hour and asking the patient to lie on the same side as the affected eye to avoid the symptoms of absorption through lacrimal punctum and to avoid symptoms of severe diarrhœa and vomiting as a result of eserine absorption.

It is very imperative to institute prompt and efficient anti-glaucoma treatment to save the eye when the attack is definitely established. With the contraction of the pupil as a result of eserine administration, intensity of pain diminishes. Eserine is not specific, but it only frees the angle of the anterior chamber by causing powerful contraction of the sphincter iris muscle and pulls the iris from posterior surface of cornea, thus causing the angle of anterior chamber to work. Eserine causes reduction in thickness of the iris as a result of contraction of pupil. The iris is thickened in acute attack due to dilatation of pupil and thus causes mechanical obstruction at the angle of the anterior chamber. The generalized congestion may be removed by applying ice bag over the eye. The ice bag can be best improvised by a rubber glove tying the fingers and the free end and filling the pocket with small pieces of ice and putting two or three layers of gauze over the eye. Purgative is administered and the old procedure of blood letting is certainly helpful, (though personally I have never been obliged to do blood letting).

Medical treatment must be given a trial from three to eight hours and if it succeeds in causing contraction of the pupil to 1 mm. in size, all other symptoms of acute glaucoma, namely, pain, congestion, tension of the eye will be relieved. Morphia is another drug which acts as a synergistic of eserine because it relieves pain first and also causes contraction and thus helps eserine. If no relief is obtained within six to eight hours it can be presumed that no benefits will be derived from medical treatment. I have found intravenous injections of sodium chloride 10 % in 10 to 20 cc. often overcoming the acute attack in almost all of my primary acute glaucoma cases.

After six hours of medical treatment, it is advisable to continue eserine to maintain contraction of pupil, the medical treatment has to be continued for 48 hours before operation is

undertaken under favourable conditions. The forcible contraction of sphincter muscle of iris causing headache should not be mistaken for glaucoma pain. Headache is felt as if there is dragging of iris. The classical operation of iridectomy of Von Græfe affords best chances of permanent relief from glaucoma. If the anterior chamber is very shallow, paracentesis or posterior sclerotomy may be done. This procedure deepens the anterior chamber and relieves tension. Personally I have never done posterior sclerotomy nor do I like the idea of disturbing the vitreous. Iridectomy is the operation par excellence in acute condition and must be done either under general or local anæsthesia. I personally prefer local by retrobulbar injection of novocaine 4%. In acute attack of one eye always put once a drop of miotic in the other to prevent the attack becoming bilateral. If the patient is living at a distance from the surgeon or is too unreliable, preventive iridectomy of the other eye should be performed, the preventive iridectomy may be done purely peripheric to keep the chamberangle open.

In sub acute glaucoma, treatment has to be less vigorous and it is advisable to control tension by medicine and operation is not urgent. The treatment consists in eserine $\frac{1}{4}$ to 1% once or twice a day coupled with pilocarpine three or four times a day and watching the reaction of the eye will enable the surgeon to chalk out the line of treatment. Systemic treatment is of no avail. Miotic treatment may be tried from one to four weeks depending upon the reaction of the eye. Surgical interference will have to be done in the majority of cases. Iridectomy is not the operation of choice in this condition and the results of iridectomy are satisfactory to the extent of 50%. I have frequently observed results of iridectomy in chronic simple glaucoma by actual tonometric measurements and invariably found that it has failed to relieve tension in chronic simple glaucoma and filtering operation such as trephining or a iridosclerotomy or irisinclusion operation must be undertaken before long and under the most favourable circumstances in order to preserve vision and the visual field. Return of vision in contrast to acute glaucoma is not so great due to continued raised tension.

Absolute Glaucoma.—Medicine is utterly helpless and if the eye is painful and irritable, surgery offers permanent relief by enucleation operation in this condition.

Chronic Simple Glaucoma.—The treatment in this condition is always taxing the skill and judgement of an ophthalmologist.

Parsons states "as soon as diagnosis is made surgery must be resorted to". This statement holds good in cases of patients who cannot be trusted to carry out in thorough details the necessary treatment. It is certainly true that surgical treatment ultimately becomes necessary, but medical treatment must be thoroughly tried to enable the surgeon to assess the usefulness of medical treatment and determine the type of operation that may ultimately be required. The ideal treatment of an early case of chronic simple glaucoma consists in the administration of weak miotics with repeated measurement of tension to enable us to determine the strength and frequency of administration to control tension. Pilocarpine in early cases from .25 to .5 is ideal to control hypertension. If this is not sufficient, strength of the solution may be increased from 1 to 4% as the case requires, but on an average 2% is preferable. In the early part of medical treatment the patient must be observed by the surgeon daily to see for control of tension. There is no dragging in the eye and interference of accommodation or irritation of the eye. After regulating the treatment for a couple of days, weekly, monthly and bi-monthly examinations will suffice and lastly after thoroughly explaining the necessity of returning immediately if anything untoward happens in the form of increased pain, headache or diminishing of vision and the field, the tension is measured to stress here that it is pilocarpine and not eserine that is the drug of choice although eserine is a more powerful and more potent miotic than pilocarpine. It is almost an axiom that any case which needs eserine administration daily to control glaucoma tension and maintain it within normal range cannot be safely treated without surgical interference, *i.e.*, any case requiring eserine for control of tension has conveniently to be operated for relief of tension. Continued eserine therapy is prejudicial to the eye, because it leads to hypertrophy of conjunctival follicles and congestion of conjunctiva resembling acute trachoma. Continuous use of pilocarpine also produces similar results but it is more due to lack of sterilization, hence pilocarpine must be sterilized once a week. Patients should preferably be seen by the surgeon at one and at the same time of the day preferably in the morning. Patients must be impressed with the necessity for control of tension. Worries must be avoided at all cost and *moderation in all things* is the masterly advice, the use of eye for near work is not prohibited.

Some cases may show unfavourable progress in cases of chronic simple glaucoma inspite of a thorough and regular use of miotics as shown by fall in visual acuity, visual field and raised

intra-ocular tension. There are cases where though miotics can overcome the situation by violent reduction in tension, surgery is preferable. In chronic simple glaucoma it is not advisable to operate if the corrected vision with glasses is less than 6/60. My usual practice in cases below 6/60 is to give the patient a chance by medical treatment to increase visual acuity and visual field. I have succeeded in a number of cases by bringing the vision and maintaining it even after operation to 6/15 by the following lines of treatment. The patient's tension taken every day and kept below 20 m.m. of mercury or even lower by regular administration of eserine or pilocarpine, keeping the pupil contracted and then administering sub-conjunctival injection of origin of P. D. & Co., adrenalin in 4 or 5 m.m. doses every third day, the number of injections varying from 7 to 14 and they are supplied miotics at home for instillation, the frequency of instillation and percentage depending upon the degree of tension, increased directly with tension. The greater the tension the higher the frequency of instillation and percentage of miotic.

I had, in my practice by this treatment, met with two calamities setting up acute glaucoma immediately after injection of adrenalin. The acute onset was however overcome by immediate administration of intravenous hypertonic saline coupled with miotics. The attack in two cases was particularly severe, accompanied by signs and symptoms of acute glaucoma. It is very necessary to keep the pupil contracted to the size of 1 to 1.5 m.m. because adrenalin causes pupil dilatation due to stimulation of sympathetic.

Modified Iridencleisis for Simple (Non-Inflammatory) Chronic Glaucoma

BY

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THIS article is specially suited for those who practise in rural areas. It does not require many instruments but only the following, in order of sequence :—

Speculum, douche with mercury lotion (1 in 5000),
Conjunctival forceps, Iris scissors, ordinary needle,
Cataract knife, Iris forceps (seldom), one thin iron wire
and spirit lamp.

After cocainising, wash the eye well with mercury lotion, then separate a conjunctival flap with fixation forceps and scissors about 7 or 8 m.m. from the limbus towards the cornea off the sclera. Go on separating it upto the sclero-corneal junction, while nearing the sclero cornea separate it by means of the needle instead of scissors simply to avoid button holes in the conjunctiva as otherwise the operation will be unsuccessful.

Now pass the knife in the anterior chamber at one end of the conjunctival flap at the sclero cornea and make counter puncture at the other end of the flap at the sclero cornea and lastly complete the section at the sclero cornea. Soon after this a knuckle of Iris will be ballooned out of its own accord by the pressure of aqueous humour. In case it does not happen bring out the peripheral portion of the iris with iris forceps to balloon it out. The iris should be drawn out very slowly and lightly so that the central portion of iris remains inside. Now make the iron wire red hot on spirit lamp and make 3 or 4 holes in the ballooned iris with this. Lastly, put the conjunctiva back in its position and bandage the eye. There is no need of suturing the conjunctiva.

On the third day, open the bandage and put in atropine and argyrol. Do this for 4 or 5 days and then open the bandage.

Precautions during Operation :—(1) Avoid button holes in conjunctiva as mentioned above. (2) Avoid blood entering anterior chamber which may take time to absorb.

To avoid this before entering the knife in anterior chamber, make the operation field blood-free by dropping Adrenalin solution repeatedly.

*Precautions after Operations :—*Instruct the patient to massage the eyeball at least four times in 24 hours regularly, otherwise there is fear of conjunctiva adhering to iris and its holes.

Pitfalls for Beginners of Cataract Operation

BY

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Importance of Cataract Operation in India.—Cataract is so common in India, especially the Punjab, Rajputana, Sind and other parts where heat is excessive, that any medical man whether qualified or otherwise can earn the reputation as an ophthalmic surgeon, if he is able to perform the Cataract operation successfully. Even the quacks who roam about in various parts of India doing couching operation are styled ophthalmic surgeons or Eye doctors. On the other hand a young ophthalmologist who has passed the highest examination in ophthalmic medicine and surgery, who is quite proficient in the use of Ophthalmoscope, refractionometer, Ophthalmometer, Corneal microscope and Slit Lamp, Perimeter and Scotometer, who can correctly measure the degree of Exophoria and Esophoria, but who has not done a good number of Cataract operations successfully, will have to wait a long time before he earns the reputation as Ophthalmic Surgeon. In other words, in India a successful Cataract operator is an ophthalmic surgeon of repute. Such being the case, every medical man especially those practising in small towns and villages must try to be good cataract operators.

What are the difficulties in being a good cataract operator?

PITFALL No. I.

First difficulty is to diagnose whether the cataract is in a fit condition to be operated. For this, one must have sufficient knowledge and experience to diagnose a simple cataract from a secondary or complicated cataract. I have seen beginners operating on a Cataract which was secondary to chronic simple Glaucoma, with disastrous result for the patient and a heavy blow to his aspiration to be a well known ophthalmic surgeon. Therefore the 1st pitfall for a beginner is wrong diagnosis of operable cataract. To save himself from this pitfall he should examine :—

- I. Surface of the Cornea.
- II. Anterior Chamber.
- III. Margins of the pupil.

- IV. Reactions of the pupil.
- V. Colour of the Lens.
- VI. Projection of Light.
- VII. Intraocular Tension.

I. Surface of the Cornea should be quite clear if everything is normal. Dullness of the surface suggests Glaucoma. Opacities of the cornea should be borne in mind at the time of giving prognosis in the matter of regaining of vision after the operation.

II. Anterior Chamber if shallow rouses suspicion of Glaucoma. If it is shallow without the presence of Glaucoma the operator has to use a very narrow bladed cataract knife. If he fails to do so he cuts the iris at the time of doing incision of the cornea, with the result that the patient squeezes his eye with the utmost force, and lens and vitreous come out as soon as the *incision* is finished. Shallow anterior chamber also suggests swollen lens. Deep anterior chamber is generally seen in eyes with detachment of retina.

III. Margins of the Pupil should be minutely observed. They should be regular in normal conditions. If found uneven or irregular, posterior Synechia may be present which may be the result of old Iritis. If such an eye is operated, chances of Iritis or Iridocyclitis are very great and subsequent impairment or total failure to regain good vision after operation is quite likely.

IV. Reaction of Pupil. — This sign is very important, as good reaction means healthy fundus and chances of recovery of good vision; on the other hand sluggish pupillary reaction or total absence may be due to glaucoma, choroidal atrophy, diseases of retina or optic atrophy. All these conditions would account for unsatisfactory vision in spite of the operation being entirely successful. Absence of pupillary reaction is a contraindication for cataract operation. If the pupil is found dilated and immobile, it may be due to the use of some mydriatic or some fundus disease.

V. Colour of the Lens.—Black cataract is found by dark room examination and always suggests some retinal pathological changes. Recovery of good vision after operation in such cases is exceptional.

Muddy or brownish colour of the lens may be due to glaucoma or detachment of retina.

VI. Projection of Light.— This is to be examined very thoroughly and accurately in the dark room. If the case is

normal, projection of light should be very well marked in every direction. If it is limited or doubtful in inner and lower field, it is diagnostic of glaucoma. In these cases intraocular tension should be taken. If projection of light is defective or absent the best thing is to refuse operation.

VII. Intraocular Tension.—Normal intraocular tension from 15 m.m. to 25 m.m. of mercury by Schiötz and 22 m.m. to 40 mm. by Macleans Tonometer. If the tension is above normal maximum, presence of glaucomatous conditions are to be thoroughly ascertained before performing the operation. On the other hand if the tension is much below normal high myopia and detachment of retina are to be kept in mind. Cataract operation done in any of these two conditions will result in disappointment for the patient and the operator.

PITFALL No. 2

Conjunctiva and Lacrimal Sac.—For a successful cataract operation, it is absolutely necessary that the conjunctiva should be quite normal. In Europe and America, the ophthalmic surgeons are very particular about the healthiness of the conjunctiva. In fact, no case is operated for cataract unless a pad left on the eye for 24 hours, shows no pathogenic organisms from the conjunctival discharge. In India, it is impossible to be so particular in this respect as the majority of eyes that come for the cataract operation are either suffering from Trachoma or Conjunctivitis, or show cicatricial changes in the palpebral conjunctiva as a sequelae of chronic Trachoma. The beginner has to examine the conjunctiva and if it is found to be affected with Trachoma or Koch-Week's or Morax-Axenfeld conjunctivitis, he has to treat that eye thoroughly before undertaking any operation which involves opening of the eye ball. On the other hand if the eye is found to be suffering from Pneumococcal or Gonococcal conjunctivitis, the operation should be strictly refused, otherwise suppuration of the eye ball is quite likely.

Thorough examination of the Lacrimal sac is much more important than the conjunctiva. Even the slightest infection in the sac or simple stenosis, if neglected, results in severe infection of the wound. In every ophthalmic clinic thorough examination of the Lacrimal sac is a routine procedure for a patient being prepared for a cataract operation.

Every case showing even the slightest regurgitation is put aside, and every case in which the fluid injected into the sac does not pass easily in the throat is reported to the surgeon.

Any beginner who fails to be very particular in examination of the sac meets with disasters more often than one who is very particular about this point.

PITFALL No. 3.

I am purposely omitting to draw attention to the lotion to be used for cleaning the eye, Local anæsthetic which may be more suitable, the kind of speculum to be used, fixation of the eye ball and retraction of the lower eye lid, as all these points are fully discussed in the text books on ophthalmic operations.

The greatest pitfall that a beginner meets with and one which troubles him the most is the small corneal incision. Either out of nervousness or inexperience or false ideas of safety against vitreous losses, a beginner generally makes a small incision in the sclero corneal margins, the result is that the diameter of the incision is smaller than the diameter of the lens to be delivered, and it is impossible to deliver a normal size cataract through such an opening. The only cataract that can be delivered through a small opening is Nucleus of a Morgagnian or milky cataract. The advantages of a small incision are nil, on the other hand disadvantages are tremendous, while a fairly big incision is full of advantages and there are practically no disadvantages unless the knife is entered below the centre of the cornea. I have seen a number of beginners taking more than 15 minutes, trying to deliver a cataract through a small incision, with the result that instead of delivering the lens he delivers most of the vitreous, and the lens sinks down in the eye ball. In the end that eye gives pain to the patient for three or four months and is lost for ever.

In addition to this there is also a great danger of the flaps of the wound getting septic. When the operator tries to deliver a lens through a small incision there is great bruising of the margins of the scleral as well as corneal flaps of section. As cornea has practically no blood supply and as it depends for its nutrition on lymph supply only the result of bruising is loss of resistance to infection, and serious chance of infection with pyogenic organisms.

PITFALL No. 4.

As every operator for cataract knows so well that, after incision Iridectomy is the next step, inspite of good anesthesia of other parts of the eye, Iridectomy is always more or less painful, and if the iris is not handled delicately it gives severe pain to the

patient and the patient squeezes. At this time a beginner naturally gets nervous and instead of cutting the iris at once, he fails to do so and sometimes fails to open the iris forceps with which he has caught hold of the iris, and he draws out the iris forceps. Now if the iris is in the grip of the forceps, the whole iris is either torn from its root and taken out of the eye, or certain part of the circumference of the iris is torn or detached from its peripheral attachment resulting in a condition which goes by the name of Iridodialysis. If the eye escapes trauma to such an extent, even the traction on the fibres of the iris predisposes the eye to iritis or iridocyclitis. Therefore to escape from this pitfall the beginner should avoid nervousness on his part entirely.

Sometimes if the beginner fails to introduce the Iris forceps further up towards the margin of the pupil he holds the periphery of the iris, and the result is that instead of doing a good iridectomy he performs what is called a button hole in the iris. The consequence of this pitfall will be difficulty in delivery of the lens. The lens can neither come out from the pupil, nor a small coloboma in the peripheral part of the iris. To avoid the mishap, the beginner should be careful to introduce the iris forceps deep down in the anterior chamber, and to catch hold of that part of the iris which is nearer to the margin of the pupil.

PITFALL No. 5.

Difficulties in capsulotomy:—Under this heading, I omit purposely any remarks on the use of capsule forceps as I presume that a beginner always gives preference to a capsulotome. In the use of capsulotome one has to be very delicate in his manipulation. Usually a beginner either fails to cut the capsule or exerts so much pressure that the hyaline membrane of the vitreous gets ruptured, and vitreous presents itself in the wound before the delivery of the lens. The result is catastrophe, the lens deeps down in the vitreous and unless a vectis is used at once with dexterity the eye is lost.

PITFALL No. 6.

As every beginner knows, this is the most taxing part of the operation. An experienced operator, if he has done large enough incision, good Iridectomy and wide capsulotomy, knows fully well what amount of gentle pressure is required for delivery of the lens. At the same time he knows thoroughly in what part of eye pressure is most effective in delivery of the lens. A beginner either fails to exert the required amount of pressure or exerts

too much pressure. In the first case he fails to deliver the lens, in the second case he puts out vitreous instead of the lens. It is only by experience that one learns as to what amount of pressure is within the limits of safety. Sometimes a small lens, especially in a morgagnian cataract with sharp margins, sticks itself in the Scleral flap of the incision, and then any amount of pressure on the lower part of the eye ball fails to deliver the lens. The only technique that would succeed is to apply gentle pressure about 2 mm. above scleral flap and push the margin of the lens in the lower direction, so that the margin of the lens may present itself in sclero corneal wound.

After delivery of the lens all the lens matter should be removed, either by gentle pressure on the cornea or by use of a small irrigator and washing out the anterior chamber. Even a slight amount of lens matter or a piece of loose capsule left behind, would be a source of iritis, plastic in nature, or if there are other predisposing causes, constitutional or local Iridocyclitis may come off.

PITFALL No. 7.

Failure to replace pillars of the Coloboma of the iris in the angle of the wound.

Generally a beginner breaths a sigh of relief, when he succeeds in delivering the nucleus of the lens, and in a hurry omits to attend to the step of operation which goes by the name of "toilet of the wound".

Really speaking the most important step in the toilet of the wound is avoidance of any incarceration of the iris or lens capsule in any part of the wound. Failure to do this will result in prolapse of the iris in the first case, and onset of secondary glaucoma in the second case. Therefore, never fail to pass an iris repositor in the whole wound from one end to the other.

Ocular Manifestations of Diabetes Mellitus

BY

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OF all the metabolic diseases, the eye is affected most in Diabetes Mellitus. In it, practically all the parts of the eye may be affected. As is well known, Diabetes is a disease due to the altered carbohydrate metabolism where an increase of blood sugar or hyperglycæmia is produced. This hyperglycæmia probably acts as a toxin affecting the various parts of the eye. In addition to this, the normal metabolism of the eye is affected owing to the alteration in the composition of the intraocular fluid which is directly dependent on the composition of the blood which circulates in the orbit. In addition to the direct affection of the eye due to the above stated reason, the eye may be secondarily affected owing to vascular changes in the brain as a result of this malady and some very important ocular signs and symptoms may be produced.

Some of the ocular signs and symptoms in Diabetes are so prominent and so characteristic that it may not be difficult for the general practitioner to diagnose the disease from these signs and symptoms only even before diagnosing it by laboratory methods.

Patients generally come to the physician with the complaint of the affection of vision which may be of various degrees and kinds.

I. Moderate degrees of loss of vision, may be due to :—

- (1) Changes in the power of accommodation in which the near vision is only affected.
- (2) Changes in the refractive power of the eye.

II. Severe degrees of loss of vision sometimes leading to complete blindness which are generally due to :—

- (1) Retinal and vitreous hæmorrhage—sudden loss of vision.
- (2) Retrobulbar neuritis.
- (3) Cataract, when advanced or mature.
- (4) Retinitis—in severe cases, grave disturbance of vision.

- (5) Lipæmia retinalis—may sometimes be present without any symptom.
- (6) Sclerosing keratitis.

III. Seeing positive Scotomata in which the visual defect is noticed as a black or coloured spot or cloud which obscures vision.

- (1) Retrobulbar neuritis:—Central, paracentral or ring Scotomata.
- (2) Retinitis—Scotomata are produced by exudates in the retina.

IV. Symptoms such as Double sight and Hemianopsia.

- (1) Ophthalmoplegia.
- (2) Vascular lesions in the occipital lobe.

V. Besides the main complaints of the affection of vision, patients may come for painful affections of the eye in which vision may also be affected, *e.g.* :—

- (1) Iritis and cyclitis.
- (2) Relapsing scleritis and Episcleritis.

VI. Such signs may be found as :—

- (1) Epithelial dystrophy of the cornea.
- (2) Pigmentation of the cornea ; (3) Vascular changes in the conjunctiva.

VII. In addition to these complaints for which the patient may seek the advice of the physician, the physician himself may be called to see the comatose patient due to Diabetes in which the characteristic symptom is hypotonus or reduction of the tension of the eyeball (Krause's sign).

The tension is so low that even slight palpation with the finger should enable the physician to diagnose the condition.

It is very important for the physician to diagnose this coma, for the patient can be saved by injection of insulin (a minimum of 200 units) unless the case is a moribund one. It is also of practical importance to bear in mind that coma may be brought about by hypo-glycæmia in diabetic patients due to overdosage of insulin. It will perhaps be not out of place, if we mention here the distinctive clinical features between Diabetic Coma and Coma due to overdosage of insulin.

- (1) Diabetic Coma—Krause's sign usually present. Presence of the smell of acetone in breath. Absence of plantar response.
- (2) Hypo-glycæmic Coma.

Absence of Krause's sign. Absence of the smell of acetone in the breath. Presence of Plantar response.

It should be remembered however that while Krause's sign is usually present in Diabetic Coma, it may be absent or difficult to detect it in diabetic comas of short durations and according to some observers—it may be present even in hypoglycæmic coma and in some other conditions also.

We shall now try to consider each of the above stated ocular signs and symptoms due to diabetes, elaborately with respect to their practical importance and diagnosis.

Changes in the Power of Accommodation.—The patient complains of the difficulty of reading and writing and if he had already been using reading glasses, they no longer suit his eyes. On examination very high plus glasses may be necessary to bring the vision to the normal distance of reading. The affection in the ciliary muscle causing this symptom varies from a mere weakness to complete paralysis of accommodation. This weakness or even paralysis of the accommodation can be easily tested as follows:—

Remembering that with accommodation, the pupils contract and the eyes converge, the patient is asked to fix his eyes at a fixed object, say the point of a pencil held at about 3 ft. in front of his eyes. While the patient is still looking at it, the pencil is brought nearer and nearer to his eyes and the size of his pupils and the movements of his eyes are noticed. In weakness or paralysis of accommodation, the pupils will either contract very little at a certain time or not at all and the eye balls also will then either converge very little or not at all. Besides, the patient will himself complain of seeing 2 pencils at a distance where he should normally see one.

Sudden increase of presbyopic symptom in a patient especially when he is comparatively young, should arouse suspicion of Diabetes. After the proper treatment of diabetes, this symptom gradually disappears.

Changes in Refraction.—Persons suffering from unregulated diabetes mellitus may come to the physician with the complaint of dimness of vision. The refractive error is usually in the

direction of myopia. It should be the duty of the physician to examine the urine of a comparatively elderly patient who suddenly becomes myopic. Increase of blood sugar concentration or hyperglycæmia causes increase of sugar in the intraocular fluid which varies intimately and directly with that in blood and so the refraction of the eye is changed. This myopia may also be due to the beginning of swelling of the lens in diabetic cataract or even in simple senile cataract. These patients if they had been using reading glasses previously, prefer now either to have weaker glasses or no glasses. In such patients, glasses should not be ordered immediately but first the urine examination should be done and if the diagnosis has been settled, glasses should only be ordered if necessary until 2 or 3 weeks after proper regulation of diabetes has been completed.

The following case may be of interest which the author had in his private practice.

A Hindu male, aged 46 years, came to see me in order to get his eyes properly tested, for he lately found difficulty in seeing distant objects clearly; besides, he could see the near objects quite well without the glasses which he was using. He took reading glasses about 1 year ago. On examination of his distant vision, it was found that he had a refractive error of -0.75 Dsph. Substituting this amount from the presbyopic correction which was $+0.75$ D. S. he could naturally see his near objects quite well even without glasses. On examination of his urine, sugar was found and a course of insulin treatment corrected his distant vision so that he did not any longer require glasses for distant vision and his old glasses for his near work could serve his purpose all right.

In diabetic patients the refractive changes of the eye may also be hypermetropic. According to Duke-elder, a rise of blood sugar produces myopia and a fall of hypermetropia, through alteration in the crystalline concentration in the ocular fluids. Since insulin readily alters the blood sugar, such a disturbance has been thought to be due to the introduction of insulin.

Retinal and Vitreous Hæmorrhages.—This may cause sudden obscuration of vision in the affected eye. But fortunately such hæmorrhages are not very common in Diabetes. The hæmorrhages are usually of recurrent nature and frequently associated with retinitis.

Retrobulbar neuritis.—Retrobulbar neuritis often of both eyes with the symptom of central scotoma is a rare complication

of diabetes. This condition often resembles toxic amblyopia and may be associated usually with tobacco and sometimes with alcohol habit in a diabetic patient.

Patients generally complain of defective vision but in acute cases sudden loss of vision with pain on moving the eyes may also be complained of.

On examination, vision is not found to be improved with glasses and no visible change in the fundus except a pallor on the temporal side of the disc is seen. But by taking the field of vision, usually a central scotoma but sometimes a paracentral or a ringscotoma may be found. The reaction of the pupils to light is very important. When light is thrown on the affected eye, the pupil contracts at first but slowly dilates afterwards when it is still kept on the eye.

The prognosis in some cases is good if proper treatment is carried on immediately. In all cases of retrobulbar neuritis where no definite history can be found, the urine should be tested for sugar.

Cataract.—When a patient below 40 years of age comes to the physician with a history of gradual loss of vision in a rather short time owing to the formation of cataract in both eyes, then it must be the duty of the physician to think it first to be due to diabetes. This is usually the true diabetic cataract, the characteristic of which is that it begins as small milky opacities behind the capsule of the lens.

One of the peculiarities of diabetic cataract is often the association of swelling up of iris cells in the iris from which in many cases the pigments have been lost. This can be easily seen with a magnifying glass and a loupe.

In children diabetic cataract may develop within a few days' time only.

Senile cataracts may also be present in diabetic patients. Here the patients are elderly and the characteristic signs of diabetic cataract are not present.

In all cases of cataract, the urine must be examined.

Retinitis.—In our country, retinitis is perhaps the commonest complication next to the formation of cataract. This may be due to the fact that a large number of cases of diabetes in our country are left untreated and allowed to advance to this stage. The practitioner should bear in mind that a patient with diabetic retinitis has his span of life for a few years only. About 60% are said to live for not more than 2 years.

The diagnosis of this retinitis in spite of its prominent signs may be missed owing to the peripheral position of the lesions and the opacities of the lens. During Ophthalmological examination of the fundus, therefore, the pupils should be thoroughly dilated and the patient asked to look at various directions so that the whole field of the fundus may be seen. The characteristic is the presence of irregular, small and bright white spots around the macula. This should be distinguished from the albuminuric retinitis where the spots around the macula are brilliant white and have a characteristic stellate arrangement forming a star shaped figure around the macula. The earlier deposits are cloudy and look like "Cotton-wool" patches. Besides these, there may be changes in the Optic disc which is not to be found in true diabetic retinitis. The hæmorrhages which are often present are with sharp margins and not flat as in diabetic retinitis. But one must not forget that albuminuria is frequently associated with diabetes as one of its complications in its late stage and so in diabetes also all the characteristics of albuminuric retinitis may be present. It is of practical importance to the physician therefore to examine urine in all cases of retinitis.

Lipæmia Retinalis.—This is a very serious complication of diabetes. According to some observers, it is really the only lesion of the fundus which may be considered as pathognomonic of diabetes. It is often found in cases where diabetic coma sets in. The examination of the fundus, therefore, may be helpful in comas of doubtful origin. Lipæmia retinalis is frequently found in young diabetic patients.

The Ophthalmoscopic picture of the fundus is very characteristic. The retinal vessels containing lipæmic fluid look milky white. The arteries are of pale red colour and the veins have light violet tint.

Sclerosing Keratitis.—It is a complication of scleritis of diabetic or of other origin when the affection extends to the cornea. The opacities are grey and greyish yellow, becoming denser until they exactly resemble the sclerotic and hence the name. In our country where a large % of diabetic cases are either left untreated or not properly treated at all, it is possible to see this complication not so infrequently as supposed as a result of relapsing scleritis. The prognosis regarding the vision is very bad.

Ophthalmoplegia.—Partial or complete paralysis of nerves supplying the external ocular muscles are sometimes seen in

Diabetes. This condition is known as ophthalmoplegia externa and is distinguished from Ophthalmoplegia interna where the intrinsic muscles (sphincter pupillæ and ciliary muscles) are paralysed and which may also rarely occur in diabetes. In very rare cases Ophthalmoplegia totalis where the paralysis of both the external and internal muscles of the eye takes place, may be seen.

The three characteristic symptoms of Ophthalmoplegia externa are:—limitation of movement, deviation of squint and diplopia, all of which increase in the direction of the affected muscle. In addition to these, false projection, unsteady gait, vertigo, nausea and head tilting may be present. The 6th nerve is affected most and next to it is the 3rd nerve. Isolated paralysis of the 4th nerve may also occur.

The condition generally passes away after proper treatment of diabetes. In Ophthalmoplegia, in elderly patients, next to syphilis, one should perhaps think of diabetes when there is no particular history of any trauma or of any other disease.

Vascular Changes in the Brain.—Patients may come with such symptoms as Hemianopia *i.e.*, the inability to see objects in one half of the field of vision. This symptom is rare and when present may not be known by the patient himself until he meets with some sort of accident. The reason for its not being easily detectable by the patient is because of the fact that whichever field of vision is affected, the central vision remains good. The nature of accident or incident which enables the patient to detect it is either running into objects in bright day light, or when the lower quadrants of the field of vision is affected, then he may experience difficulty in reading and writing or seeing food in the plate.

In diabetes, the vascular changes are mostly arteriosclerotic and as a result of this, lesions of the blood vessels situated in any part of the visual tracts from the occipital lobe to the chiasma may take place.

In lesions of the optic tract, a characteristic pupillary reaction called the Wernick's hemianopic pupil reaction, may be present. In it, when light is thrown on the two sides of the pupil separately, one side contracts properly while the other side contracts either sluggishly or not at all, showing that the afferent pupil constrictor fibres which undergo partial decussation at the optic chiasma have been affected on one side due to lesion of the tract, the type of hemianopia depending on the site of the lesion.

Such lesions as thrombosis of the retinal veins, with or without hæmorrhages, proliferating retinitis and secondary glaucoma may occur due to vascular changes.

Iritis.—The characteristic of diabetic iritis is the presence of plastic exudates and occasionally a hypopyon in the anterior chamber. On careful examination, the presence of new or enlarged vessels in the iris is also seen. The iritis is usually of a chronic type. The author had a case of iritis in a diabetic patient with no typical signs and it was difficult to cure the affection until the presence of diabetes in the patient was discovered by examination of his urine.

Relapsing Scleritis and Episcleritis.—The author is of opinion that some cases of scleritis and episcleritis which are of diabetic origin, are usually misdiagnosed as rheumatic, gouty, syphilitic or tubercular and so it becomes very difficult to cure these conditions. In cases of relapsing scleritis or episcleritis in elderly people, diabetic origin which although is rather very seldom, should not altogether be lost sight of.

Epithelial Dystrophy of the Cornea.—Like the swelling up of the iris cells, in rare cases the epithelial cells of the cornea may swell up and form vesicles. Anaesthesia of the cornea may be present. The condition is easily diagnosed by means of a magnifying glass and a loupe. The cornea stains with fluorescein. It generally clears up with proper treatment of diabetes.

Pigmentation of the Cornea.—In old people, it is of frequent occurrence to find the presence of pigment cells on the inner surface of the cornea. This may either take place due to senile changes in the stroma of the iris cells or due to diabetes. In diabetic patients, the characteristic of these pigments is that these form a vertical spindle (Krukenberg's spindle) as a result of the disturbance of the aqueous humour.

The author has seen a diabetic patient who complained of seeing streaks when looking through a powerful light. On examining with a magnifying glass and a loupe the presence of such pigments on the inner surface of the cornea was found.

Vascular Changes in the Conjunctiva.—Several kinds of vascular changes in the conjunctiva have been described. These changes although they may help in diagnosis are not sufficient in themselves to establish a diagnosis of diabetes. In some cases a considerable enlargement of the venous branch of the capillaries may be present.

Lastly, if we consider from statistics, we find that about 20 to 30% of cases of diabetic patients have ocular symptoms. This is the statistics of European and American countries. In India, where a large number of diabetic cases are left untreated, the percentage of cases with ocular symptoms is perhaps somewhat more. Of the important ocular affections, Retinitis and cataract are the most common. Then come retrobulbar neuritis and paralysis of the ocular muscles. The more infrequent affections are, disturbances of accommodation and refraction and inflammation of the iris. Other affections are still rarer. These conditions may either be secondary to Diabetes or may exist independently of this disease.

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Eye Complications in Epidemic Dropsy

BY

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EPIDEMIC Dropsy has become a great problem in Bengal; because, both its prevention and cure are very imperfectly known. Although Bengal is the home of Epidemic Dropsy, outbreaks of this disease have occurred in all the big cities of Behar and United Provinces and the disease has also been reported from Madras, Bombay and outside India—from the Strait Settlements, Burma, Ceylon and some parts of Africa. The disease has frequently been confused with Beri-Beri but pathologically and clinically, Epidemic Dropsy is quite a different disease. Pathologically, enormous dilatation of the Capillary system has been observed, probably caused by a toxin, belonging to the Histamine group. Rice and mustard oil, both important articles of Bengalee dietary, has been held responsible for the disease but the etiology of the disease has not yet been definitely established. Clinically, the disease manifests itself with a swelling of legs, palpitation, gastro-intestinal irritation, a peculiar red mottling of the skin, fever (occasionally), hæmorrhages from mucous surfaces like gums, throat, lungs, etc., dilatation and impaired function of the heart and the onset of Glaucoma in a good proportion of cases. There are quite a large number of people in Bengal, who have become totally or partially blind due to Epidemic Dropsy Glaucoma. The percentage of Glaucoma in Epidemic Dropsy cannot be definitely ascertained but the incidence of Glaucoma is probably increasing in recent outbreaks and we see more cases of Epidemic Dropsy Glaucoma in Calcutta Hospitals now than before. It may be that the onset of Glaucoma is diagnosed much earlier now than before and the patients also seek relief for the eye complications much earlier than they used to do before.

The chief eye complication in Epidemic Dropsy is Glaucoma which shew contains characteristics, different from other types of Primary Glaucoma and differs clinically from the text-book picture of chronic Glaucoma. A good deal of research has recently been done on the ætiology of Primary Glaucomas and we know that the conditions of Vascular bed of choroid and ciliary body, the capillary pressure—both osmotic and physical, of ciliary blood vessels, the vasomotor control of choroidal and ciliary vessels,

the swelling of vitreous and pushing forward of the lens, the resiliency of the sclerotic coat, have each been proved to play some part in the causation of Glaucoma and we fully realise to-day in Bengal that Glaucoma is "a sick eye in a sick body". The predominant cause of Glaucoma in Epidemic Dropsy cases, is dilatation of ciliary blood vessels and their increased permeability producing more aqueous than can be filtered out through the canal of Schlemm and consequently producing a rise of intra-ocular tension.

It is important that an early diagnosis should be made in these cases, so that treatment may be started before any irreparable damage has been done to the eye by the high tension. Epidemic Dropsy Glaucoma might start in any age. I have seen this type of Glaucoma in a child of 6 years. This disease is more common in young than in old patients, as distinguished from other types of Primary Glaucoma and the largest number of cases would fall between 20 and 30 years of age. Several members of of the same family are usually affected at the same time. Condition of refraction of the eye does not produce any difference in the rate of onset—it occurs equally well in Myopics as in Hypermetropics. Both sexes are equally affected.

The earliest symptom which the patients complain of is seeing Rainbow halo round artificial sources of light with the red colour outside and blue or violet to the inner side of the halo. Although Glaucoma is not the only cause of Rainbow halo the occurrence of this symptom in an Epidemic Dropsy patient, should be taken seriously by the attending doctor and a full examination should be done to exclude the possibility of Glaucoma. This symptom of Rainbow halo is very variable in Epidemic Dropsy Glaucoma; sometimes it is very vivid and may even be seen in daylight, sometimes very faint or entirely absent.

Another common symptom is misty or foggy vision, generally in the morning which clears up after a few hours. There is no pain but occasionally headaches or heaviness in the eyes may be complained of, particularly after some near work. Diminution of vision is not complained of by the patient, except in very advanced cases but field of vision will show characteristic Glaucoma changes. Peripheral vision is gradually lost until a condition of tubular vision (like vision through a long tube) may be produced, ending eventually in complete blindness. Both eyes are affected always but one eye may be more advanced than the other.

On physical examination congestion of the eye is entirely

absent or may be slightly present in some cases. Ciliary congestion is only seen in some cases of sub-acute Glaucoma, occasionally seen in Epidemic Dropsy. Cornea is clear, or a faint haziness due to œdema of the epithelial layer may be seen with a corneal magnifier in very high tension cases. The Anterior Chamber is normal in depth or even deeper than normal (as distinguished from chronic simple Glaucoma). The pupil is also normal in size and reactions except in very advanced cases with optic atrophy. The most important sign is increased intra-ocular tension which can be felt by digital examination or recorded by Tonometer. The digital method gives very useful information in the hands of specialists but general practitioners may train their fingers to appreciate a moderate rise of pressure if they examine and compare with normal tension eyes. Great fluctuations of tension is observed by Tonometry in this type of Glaucoma but the tension is usually high. A tension between 70 and 100 m.m. as recorded by Maclean's Tonometer is commonly found.

The next important sign is, changes in the Visual fields. The fields of vision as recorded carefully by the Perimeter is the most important guide to future course of treatment of every individual case. There occurs a gradual peripheral contraction of the field of vision starting from the nasal side. A very rough idea of the field of vision may be obtained, without a Perimeter by the general practitioners, by the Confrontation Test. In this test, the observer and the patient must sit or stand facing each other, about one yard apart, each eye should be tested separately, by covering the other eye with the palm of his own hand. The observer and the patient must look at each other's pupil and the observer should close the eye which is opposite the patient's covered eye. A suitable test object or the index finger of the observer should be held midway between the two eyes and moved in various directions and the patient should be asked repeatedly if he can see the finger. If in any position, the observer can see the finger, while the patient cannot, his field is contracted in that position. This is a very crude method but gives useful information in advanced cases.

The next step of the examination is by means of the Ophthalmoscope. By Ophthalmoscopy, pulsation of retinal arteries could be seen in early stages and Glaucomatous cupping later on.

Treatment.—In the early stages the underlying cause—Epidemic Dropsy, should be treated and a constant watch should be kept on tension and field changes. Many cases are automatically

cured with the cure of Epidemic Dropsy. But if the tension continues to rise or field changes appear, operation should not be delayed long. Medicinal treatment is not of much use in this type of Glaucoma but Pilocarpine nitrous drops (1 to 2 per cent.) may be instilled twice or thrice daily and sometimes is of great help in mild cases. There are many operations for Glaucoma but we find in Calcutta the Elliot's Sclero-corneal Trephine is probably one of the best operations for this type of Glaucoma.

Other eye complications found in this disease are :—

- (1) Retinal hæmorrhages with or without high tension. This hæmorrhage may be quite small or big and may produce sudden obscuration of vision. The hæmorrhage may be situated on the optic disc or near its margin or along the course of a retinal vessel. The blood is gradually absorbed and full vision may be regained within a few weeks.
- (2) Sub-conjunctival hæmorrhages and extreme conjunctival congestion have been observed rarely.
- (3) B. N. Bhadury has reported some cases of swelling of optic nerve head (Papilloedema) in Calcutta Medical Journal, August 1937, which he thinks is caused by increased Intracrainal Pressure, produced by Epidemic Dropsy.

Headaches Due to the Eyes

BY

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It has been my experience over the last ten years or more to get patients referred to me in the Eye Department for headaches of long or short duration without their having undergone atleast a regular preliminary enquiry or physical examination to exclude causes of headache other than the eyes. When such patients come to us oculists, we cannot easily dispense with them saying that their headaches are not due to the eyes although a preliminary enquiry into the apparent cause, duration and situation prompts us to exclude the eyes. An oculist has, therefore, to go through the routine of testing for errors of refraction and examination of the fundus oculi without and with a cyclopægic involving loss of time and inconvenience to the patients who, in certain instances, are unwilling to go through the routine or can ill spare the time. They expect from us some ready cure which they did not demand from the general medical side whence they were sent to us. Moreover, in some cases, it is difficult to decide in a case of minor refractive error found in one of these patients how far that error might be responsible for the headache; and doubly it is difficult to assure the person that the prescribing of spectacles for such an error will cure his headache, for in practice, we find only in about 5 per cent of retinoscopy that we do in the Eye Department that there is absolute emmetropia. The rest do have an error, small or great and in many cases the small errors may not be the causal factor for the headaches. Particularly is this the case in working class men and women who never do any work to cause eye strain.

I had patients with definite supra-orbital neuralgia, bad sinus troubles, severe cold with slight fever, bad set of teeth, persons with obvious malnutrition, many other general or special local complaints in other parts of the body referred to the Eye Department as headaches due to the eyes, and I had to find out these outside causes and send them back for appropriate treatment.

It is with a view to make a strong plea to the general practitioner and to the medical officers of the general outpatient

departments in hospitals to devote at least reasonable care to investigate the causes of headache as far as it lies in them in the brief time at their disposal before sending a patient off hand to the oculist to exclude the eyes, that I chose this subject for my article. For it is the duty of the general practitioner to do a routine examination; and only in the absence of definite positive findings he should proceed to eliminate other causes; of course, the first one among them being the eyes and not vice versa.

In certain cases ocular headaches can be recognized in a way by the general practitioner, and then he may decide to send them to the ophthalmologist for diagnosis and treatment. For treatment of headaches due to the eyes should always be done by the eye surgeon; but what I request my brothers in the profession is, that they should start co-operation with the eye man when sending a case to him for headaches due to the eyes. Outside hospital practice this state of affairs is not so bad as I have mentioned; but even here, there is a little tendency on the part of doctors to shove away the patient with headache to the oculist when the treatment of an obvious general illness of which the headache is only one symptom, is not complete.

Before proceeding to consider the question of headaches due to the eyes, I shall briefly indicate how to investigate a patient when he comes with the complaint of headache. Sometimes even a detailed examination may be necessary : —

- (1) Enquiry into the actual site of headache is necessary *viz.*, frontal, occipital, round the eyes, etc.
- (2) Duration and time of onset; what part of the day?
- (3) Severity and nature (superficial or splitting, presence of tenderness of scalp, agonizing headache, boring, throbbing or continuous pressure, heaviness of head paroxysmal or continuous).
- (4) Accompanying symptoms; Dimness of vision, Vomiting, chills, vertigo.
- (5) Frequency.
- (6) History of previous illness: Malaria, lung troubles, a bad cough, constipation, etc.

Constipation is the commonest and simplest cause of headache.

After enquiry into the aforesaid points one must proceed to inspect the patient. A look at the patient's face and posture of the head may reveal the severity and even the nature of the

headache. Temperature and pulse may also give a guide to any general cause. Septic teeth, caries, and bad pyorrhœa, may also be the causes. High blood pressure and albuminuria may have to be detected. If a child, we have to note the state of nutrition, anæmia, presence of adenoids or tonsils, emotional condition of the child and even think of intestinal worms.

In women, history of uterine troubles is an essential point. Whether the patient is pregnant or lactating is also to be considered. There have been cases of women complaining of a persistent headache for which no cause has been found, and in such cases a prolonged lactation has been existing; and after asking the patients to wean their children, the headaches have disappeared.

In old persons, Insomnia, frequency of urination, backache and history of old syphilis, high blood pressure, have all to be considered.

Readers will pardon me for the suggestion of these methods of investigation, but in cases where a preliminary suggestion of headache by nature and history of symptoms not being due to the eyes, will make one think of going into all these points one by one or severally to fix up the right cause.

Now I shall proceed to the question of ocular headaches. A patient whose headache is due to the eyes, may very often complain of the symptom after reading or straining the eyes in close work. He may say that he gets headache after seeing a cinema show even though he is one of the upper class spectators, and though there has not been a crowded house.

A woman will say that she gets a pricking pain in the eyes when she sees. Generally this headache presents at the close of the day or after straining the eyes for some considerable time. Only in exceptional cases does it develop other than towards the end of the day. The percentage of such patients is only about ten in the hospital class. Some may complain of pain round the eyes or in the centre of the forehead. The headache is rarely temporal or occipital, and seldom vertical. A patient having iritis will complain of pain in the eye and over the eyebrow. A glaucomatous patient may complain of severe headache on one side from the eye up the head and throughout that side of the head. A case of orbital abscess or panophthalmitis will complain of pain round the orbit and on the side of the head and pain in the eye-ball itself.

A man with corneal ulcer will complain of pricking pain in

the eye and sometimes pain on that side of the head. He may have a sensation of even a particle of grit in the upper lid.

In cases of iridocyclitis and acute glaucoma the globe itself may be painful. In Herpes zoster-ophthalmicus, patients may complain of headache on the affected side of the forehead.

The mechanism of the causation of the ocular headache can be put thus: Excessive accommodation may explain the production of pain, a factor accompanied by unequal degree of refraction. Headaches may also be produced by photophobia due to any cause. The orbicularis oculi derive the nerve supply from the fascial by fibres not from the nucleus of the 7th nerve but from the oculomotor situated near the upper sensory nucleus of the fifth nerve and connected with it by reflex paths. In astigmatism, headaches may be caused by unequal degree of contraction in the ciliary muscles. The ophthalmic branch of the trigeminal taking origin from the upper sensory nucleus of the fifth nerve in the floor of the fourth ventricle is the communicating link for the causation of headache. The trigeminal nerve is the nerve supply of the extracranial tissues in front and also gives recurrent twigs to the meninges.

Headaches due to the eyes may be divided into (1) with obvious external signs; (2) without obvious signs.

I. With obvious signs.—Trachoma with thickening of the lids, multiple styas, panophthalmitis, tumours of orbit with proptosis, are easily recognized. In trachoma, headache is very often caused by photophobia. In styas and panophthalmitis, inflammatory pain radiating to the head may cause a coincident headache. Acute glaucoma, iritis, will cause severe headache where the eye condition will not be missed. In bad corneal ulcers, the tendency to perforation will also cause severe headache. Herpes zoster ophthalmicus is another cause where the eye also may be involved. Post operative complications in the eye such as prolapse of the iris, defective healing, hæmorrhagic and exudative iritis after operations will also cause severe headache. Tumours of the orbit and orbital cellulitis may cause headache with evident proptosis.

It is only with cases of ocular headache without external signs in the eye that it is difficult for the practitioner to suspect the ocular origin. These lesions are as follow:—

- (1) Chief among them are refractive errors, anomalies of accommodation and disturbances of muscle equilibrium. Headache of this sort occur especially after

undue or prolonged exertion of the eyes. They cease at night generally, but in some cases they attack the patient when he first wakes up in the morning if he has been straining the eyes the night before. Infrequently, in young children, these headaches occur between the ages of 10 and 20. Such headaches are often excited by looking at moving objects due to refractive or motor anomalies. Among refractive errors astigmatism and hypermetropia are the causes, simple myopia being seldom the cause.

- (2) Chronic simple glaucoma:—This is the condition rarely suspected by a general practitioner. Any history of occasional dimness of vision or flashes of light before the eyes complained of, along with the headache, will rouse suspicion of the practitioner.
- (3) Optic Neuritis, thrombosis of retinal veins, and retinitis, are other causes, where ophthalmoscopic examination alone will reveal the cause of headache.
- (4) Retino-bulbar neuritis is occasionally a cause for headache where ophthalmoscopic examination is even sometimes elusive. Here, inflammation is limited to the orbital division of the optic nerve.

Among the causes of headache due to eyes without obvious external signs in the eyes, the commonest cause is refractive error—Hypermetropia and astigmatism being the forms of refractive error causing ocular headache. Sometimes a person may not complain of any symptom owing to latent hypermetropia till after the age of 30 when the accommodation may get strained owing to the nature of occupation or constitutional changes including weakening of the ciliary muscle. Astigmatism of low degree in sensitive persons may cause headache when straining for close work. On the other hand, there are cases of high astigmatism particularly myopic, wherein no symptom of headache is complained of, and the patient takes the advice of the doctor when a casual examination of vision finds him defective. Cases of muscular imbalance in the eyes producing headache, may sometimes complain of giddiness and diplopia as well.

The general practitioner is not expected to recognize, and much less to know how to treat cases of headache due to the eyes; but a routine examination of the patient where no symptoms referable to the eyes as are suspected by attention to

the foregoing points, will have to be made by him before sending the patient to the ophthalmologist.

There are a few other causes of headaches not originating from the eyes, which the general practitioner has to bear in mind. In these cases, the eyes bear only a relation to the neighbouring parts and very often these causes are recognized at the eye clinic.

1. *Migraine*:—The pathology of the disease is very obscure and practitioners who are interested in the subject must have read a lot of literature about it without getting any definite enlightenment. In my opinion, the disease is a manifestation of allergy running down in families wherein other allergic phenomena such as epilepsy, chronic eczema, asthma, urticaria, are found in one or other individuals, of a pedigree. I know a family where the mother suffers from asthma, one brother suffers from chronic eczema for over 10 to 15 years, another brother suffers from migraine for over 20 years. The last named person being very sensitive to substances like gingelly oil, garlic and certain vegetables like 'pavakkai'.

2. *Sinusitis*:—The headache in frontal sinusitis is violent and hammering on dull pressure. There is pain in the morning when accumulation of secretion has occurred in the night; when the head is bent, the pain increases. Headaches may occur due to inflammation of the anterior ethmoidal nerve or nasociliary nerve. Headaches may also occur when the optic nerve is involved in sphenoidal or ethmoidal sinusitis accompanied occasionally by blindness, partial or complete. A slightly deflected inner septum may later in life give rise to what are called vacuum headache. This pain is enduring, of low grade, frontal, with inability to use the eyes.

3. *Neuralgia*:—The commonest being supra orbital neuralgia in the neighbourhood of the eye.

4. Tumours of the brain and pituitary body.

5. Constitutional causes such as uremia, nephritis, acute and chronic syphilis, arterio-sclerosis and even anæmia.

I shall now give records of a few cases which I have come across, where the patients have been referred to me for headache due to the eyes, and I was obliged to suggest other causes in the phase of negative eye examinations.

CASE. 1. A lady of 55 was brought to me three years ago with history of severe headaches, off and on, for over one or two years. Her doctor had suggested examination of eyes and fitting up glasses, or whatever cure the eyes may require.

Retinoscopy revealed no refractive error, her distant vision also being normal, but examination of fundus showed extensive atrophy of the retina, the condition commonly found in my experience among old people with obscure toxic causes in the general system. According to my surmise, general examination of the patient revealed no other cause except many loose teeth with extensive pyorrhœa. I advised complete extraction of all the teeth and this the patient was fortunately persuaded to get done. A fortnight later, up to this day, the patient has been free from headaches.

CASE 2. A lady, aged about 38, was telling me for some months past that she wished to have her eyes examined for change of glasses, if need be. One day, at about 11 A.M., she came up my stairs with a slight fulness of the face telling me that she came for an eye examination. There was a suspicion of blocked nose when she spoke. On enquiry, she was just coming from a physician who had been treating her for fever for the last four days and she was now free from fever, though a severe headache still persisted. Her physician directed her to go to me. I could see by mere inspection that she was not having headache, but that the whole head was heavy, as she corroborated the statement later. She was still having a bad cold, and perhaps was having a little invasion of the sinuses; I told her that she was not yet free from her general complaint, and that the trouble was still due to the cold in the head and directed her to go back to her physician to complete her treatment. I did not see her for a month afterwards, and then she came to my room and told me that she was laid up in bed with continued cold and bronchopneumonia during the interval, and that she was perfectly all right for the past one week only. Nevertheless she wanted to be examined for a change of glasses and accordingly I did the complete test with a cyclo-pædic and subjective examination. I found no necessity to change the old lenses that she was wearing and told her to continue the old glasses.

CASE 3. A girl of 15, attended the out-patient department of the Government Hospital about five years ago, complaining of a severe attack of headache of some three months' duration. General examination by her doctor was done previously but no cause could be found. She was examined by me after atropinizing the eye, retinoscopy showed emmetropia. But the examination of fundus revealed a mild swelling of the disc, with a certain amount of tortuosity of the retinal vessels. I told the patient

that her eye was not the cause of headache, but an investigation of the brain was necessary. She was taken to Madras sometime later and I learned afterwards that she underwent a decompression operation for brain tumour and it is interesting to hear that the patient is still alive and fairly well.

This case shows the importance of ophthalmoscopic examination in obscure headaches.

CASE 4. A girl of 14, the daughter of a physician, was complaining of frequent headaches and was reported to be running down in general health. She was examined a year and half ago, by an ophthalmologist for error of refraction. She was told that she needed no glasses. A year later, she was brought to me for the re-examination of the eyes and I also pronounced the same opinion that she had no error of refraction. But then the brother practitioner, being worried by the condition of the girl, asked me to suggest a cause of the headache, by a general examination which I did. The girl was having a fast pulse; occasionally had palpitation also, which was elicited only by enquiry. The headache was particularly manifest only in the evening when she returned from school. I was of opinion that the girl, during the adolescent stage had great demands on her general growth and the strenuous evening games that she had at her school caused her a great strain and this manifested itself in the form of palpitation and headaches, and a general run down condition. I advised accordingly. The girl is now all right after regulation of exercise and work.

CASE 5. A young man of 21, a Brahmin from Palghat, attended my private clinic, some one year ago, for severe headache. He gave history of an attack of typhoid some months previously from which he had recovered till about three months ago when this headache began. Examination and treatment by a physician at Palghat, for neuralgia, and coincident digestive troubles did not relieve the headache. He, therefore, came to me for examination of eyes with a view to putting on spectacles. His distant vision was normal and examination in the dark room after atropinization revealed no latent hypermetropia or astigmatism. But the fundus showed thinned, tortuous arteries corkscrew-like in the smaller branches, which is rather unusual in a young man. The condition of the arteries led me to take his blood pressure and make an examination of urine. His blood pressure was 190 systolic, 120 diastolic, and urine contained a good quantity of albumin. Evidently this young man suffered

from headaches owing to his arterial and kidney condition. I advised him to immediately put himself under the care of his physician with a note from me regarding the results of my examination. I have not seen the patient afterwards.

Numerous cases like the above can be cited to support the opinion that headache not due to the eyes very often come to the ophthalmologist. These patients have one advantage that, in some cases, a general case will be suspected by the examiner. I have mentioned some extreme cases but I have had numerous other cases where I have not been able to suggest a cause in the absence of any refractive error.

Recently, a lady of 26 years of age, who was examined by me for refractive error seven years ago and who was still wearing that correction, complained of severe headaches in front and side of the head for the past one year. She had great confidence in me and on my telling her after thorough examination of her eyes including retinoscopy, that her headache was not due to the eyes and that she could continue the old glasses, she insisted on my doing a physical examination for her, which I had to do. I could not find specially anything abnormal with her except that she had pain and tenderness in the left iliac fossa, which will mean nothing beyond a constipated bowel. A Lady doctor had examined her previously and said nothing was abnormal in her uterus or appendages. However, her menstruation was not regular and she was unmarried. She used to have attacks of temporary slight unconsciousness during these periods of headache. This information was given by her friends. I had to put this case down under the category of hysteria. I advised the patient to take some tonics, hip-baths and get her constipation relieved. Frankly, in this case, I was unable to suggest a cause for the headache.

In this connection, I would like to mention about neurasthenics having vague headaches, pain in the lids and round about the eyes, behind or in the head and complain of discomfort setting in within a few minutes of reading, while no refractive or muscle error can be found. In headaches due to refraction, discomfort does not very often set in without prolonged exertion of eyes. I would like to mention one more common finding in connection with headaches due to eyes and that is, an improperly fitting frame may also cause headache due

to eye strain. I had very many instances, where patients returned to me, after months of wearing a pair of properly prescribed glasses, with complaints of their headaches having re-commenced. In some of these cases, I have found that the frames were too loose, not setting properly at the bridge of the nose or far away from the eyes or not lying on the face horizontally. Sometimes a patient had changed the previous frame to one larger and more fashionable, but the optical centres of the glasses did not lie in front of the pupils. Remedying of these defects and wearing properly fitting frames have caused these headaches to disappear.

Readers will pardon me for a lengthy article which does not obviously contain much of a clinical investigation or even a systematic study of the subject. The subject was taken on the spur of the moment and it is only an attempt on my part to put down some views on a subject which I meet with in my daily practice.

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Squint

BY

Major S. Annaswami, M.B., B.S. D.O.M.S., F.R.C.S. I.M.S.,

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SQUINT is the condition in which there is a deviation of the visual axes from the normal.

This deviation may only be apparent and not real. An apparent squint is due to the fact that the visual axis very rarely passes through the centre of the cornea but usually cuts the latter a little to one or the other side.

The first step in the examination of a case of suspected squint is to exclude an apparent squint. This can be done by the Screen test. The patient is asked to look at an object and then a card (the Screen) is placed before one eye and passed quickly from one eye to the other. In apparent squint neither eye will deviate when covered and each will therefore remain steady when the screen is removed. In real squint, on the other hand, each eye when covered will deviate and when uncovered will turn back into the fixing position.

Clinical Classification of Squint:—

- I. Paralytic.
- II. Spastic.
- III. Concomitant:—(1) Manifest. (2) Latent (Heterophoria.)

I. Paralytic Squint.—This is due to paresis or paralysis of an external ocular muscle or a group of muscles.

Causes :—The commonest causes are Syphilis and Diphtheria. Other possible causes are lesions of intracranial blood vessels, tumour, diabetes, injury, etc.

Features :—1. Diplopia.

2. Vertigo.

3. Deviation of the eye affected : The deviation of the squinting eye behind the Screen in the Screen test is called the Primary Deviation and that of the other eye behind the Screen, the Secondary Deviation. The latter is greater than the former. This is an important point in distinguishing paralytic from concomitant squint.

4. False Orientation of Objects.
5. Head Tilting.

6. Limitation of movement. The normal range of movement of the eye is about 45 degrees up and 55 degrees down. In full abduction the cornea touches the external Canthus and in full adduction the cornea is partly covered by the internal Canthus.

Diagnosis.—1. From concomitant squint.

2. Of the muscle or muscles affected:—(a) Note the character of the deviation of the affected eye. (b) Test movements of each eye separately and note the direction in which there is maximum limitation. The movements should be tested in the six diagnostic directions namely, in, out, up and in, up and out, down and in, and down and out.

3. Of the Cause:—(a) History—Mode of onset, any previous or recent illness, any history of injury, etc. (b) Examination of blood for W. R. (c) Examination of urine for sugar, etc.

Treatment.—Treat the cause if detected. Otherwise treatment is symptomatic and palliative. Operation is seldom indicated.

II. Spastic Squint—is rare. It is caused by irritative lesions e.g., Meningitis, etc.

III. Concomitant Squint—In this the visual axes though abnormally directed maintain this abnormal relation to each other in all movements of the eyes. Hence the name.

It may be convergent or divergent, the former being much more common than the latter.

Concomitant Convergent Squint:—*Cause:*—The fundamental cause is unknown. There are several well-known ætiological factors. It is difficult to assess the importance of any of them individually. Often more than one factor is responsible.

The following are the chief factors:—1. *Error of Refraction* Hypermetropia with or without Astigmatism is very common. In uncorrected Hypermetropia the accommodation is always in excess of convergence, or in other words, the amount of convergence is not in proportion to the accommodation. This abnormal relation between these two functions leads to a constant tendency to excessive convergence, which, however, is kept in check in the interests of clear vision. This control may and does break down in the presence of one or more of the other factors mentioned below.

2. *Defective Fusion Sense.*—Fusion of the two images formed by an object on corresponding points of the two retinæ

into a single one is a central process and the degree of development of this faculty varies in different persons. A well-developed fusion sense has a powerful checking influence on the development of squint in a case where there is a tendency to it. This factor is therefore considered by some to be the essential one in the causation of squint.

3. *Physical Defect:*—(a) Lesions which interfere with the normal development of binocular vision, e.g., Opacities of the media, Intra-ocular disease, Congenital Amblyopia, Amblyopia due to injury, etc. (b) Congenital defect of the extra-ocular muscles.

4. *Psychological Factor:*—Shock, fear, imitation, jealousy, psychoneuroses, etc.

5. *Heredity.*

6. *Lowering of the general health due to any cause:*—It is not uncommon for squint to come on after measles, whooping, cough, etc.

AGE OF ONSET:—In the majority, before the end of the fourth year. Rarely after the sixth year.

Clinical Varieties.—*Unilateral:*—The same eye deviates always. It must, however, be remembered that concomitant squint is really a bilateral affection. The fact that one eye always squints does not mean that the affection is confined to that eye. For example, if there is a convergent squint of 30 degrees in the right eye, the real disability is 15 degrees in each eye. What happens is this. If both the eyes converge 15 degrees neither eye will be focussed for the object looked at. There will be diplopia and indistinct vision. To get over this difficulty, both the eyes turn laterally through 15 degrees so that the better eye may be brought into line with the object. The worse eye is therefore left with a squint of 30 degrees.

Alternating:—Either eye may deviate.

Constant:—The squint is present all the time.

Periodic:—The squint comes on only at intervals. It may come on only when the child looks at near objects.

Characteristic Features:—1. The primary deviation is equal to the secondary.

2. The movements of the eyes are as a rule normal.

3. Diplopia is as a rule absent. Its presence may be elicited by careful questioning in about two per cent of the cases. Absence of diplopia is due either to psychological suppression of the image formed in the squinting eye or to the abnormal projection

of the image. In a very small proportion of cases it is due to true eccentric fixation by the squinting eye and formation of a false macula.

4. The squinting eye is usually amblyopic. This amblyopia is in the majority of cases acquired. Only very rarely it is congenital.

Investigation of a case of Concomitant Squint:—1. *History*:
(a) Age of onset. (b) Mode of onset. (c) Any recent illness.
(d) Family history and evidence of heredity, etc.

2. *Character of Squint*.

3. *Movements of each eye separately*.

4. *Power of fixation of each eye separately*. Note also if there is eccentric fixation by the squinting eye.

5. *Vision of each eye separately*.

6. *Binocular Vision*:—An infant at birth has no binocular vision. This is developed within the first few months of life and is perfected before the end of the sixth year. It can be tested and graded with the aid of an Amblyoscope and special object slides.

7. *Measurement of the angle of deviation*:—There are several ways of doing this. (a) Corneal Reflex. If this is half-way between the centre of the pupil and the corneal margin, the deviation is about 20 degrees. If it is at the corneal margin the angle is about 45 degrees. This is a rough but useful guide. (b) With Priestly Smith's Tape. This is a convenient method and can be used with children. (c) With the Tangent Scale. This is also a satisfactory method and can be used with children. (d) By means of the Perimeter. This method is not satisfactory and cannot be used with children.

8. *Refraction*:—Accommodation must be thoroughly paralysed by a cycloplegic so that the true state of refraction of the eye can be ascertained by Retinoscopy.

Treatment.—The objects of treatment are:—

1. To restore the vision of the amblyopic eye.
2. To straighten the eyes.
3. To train the fusion sense.

The means at our disposal to attain these objects are four:—

1. Correction of Error of refraction.
2. Occlusion of the fixing eye.
3. Orthoptic training.
4. Operation.

The first step is to correct fully an error of refraction present. Atropine should be continued till the glasses are actually begun to be worn. The glasses should be prescribed for constant wear. A child with squint is never too young for glasses.

The child should be examined frequently. At each visit the vision, angle of deviation, and binocular vision should be recorded.

If the vision in the squinting eye is very poor an attempt to restore its vision must be made through occlusion of the fixing eye. Total occlusion should be practised at first followed by partial occlusion. Later still, the fixing eye may be put out of action for near vision by instillation into it of $\frac{1}{2}\%$ atropine once daily. With the same object in view, namely, improvement of vision, home exercises by games with blocks and coloured pictures should be practised. The child, if old enough, may be taken to cinemas and should be encouraged to read books with graduated jaeger types.

Orthoptic training should be commenced early and persevered with. Training in a clinic should be supplemented by home exercises with the amblyoscope and stereoscope.

If after all this, deviation persists and is large or if the necessary time for the training cannot be spared, operation should be resorted to. Orthoptic training should be resumed after the eyes are put straight by the operation.

In alternating squint operation is often the only procedure as the error of refraction is usually nil and fusion sense absent. If amblyopia is present occlusion should be practised before the operation.

If the deviation is 10 degrees or less it can be corrected by simple tenotomy of the internal rectus. This operation can be done under general anaesthesia and can therefore be done at quite an early age. If the deviation is greater than 10 degrees tenotomy of the internal rectus should be combined with advancement of the external rectus. This operation should as a rule be deferred till the child is old enough to allow it to be done under local anaesthesia.

Concomitant Divergent Squint.—This is much less common than the convergent variety. It is usually associated with fairly high Myopia. Its treatment is regulated by the same principle discussed above except that operation is rarely necessary.

Non-concomitant Non-paralytic Divergent Squint.—This is seen under the following conditions:—

- (1) Very high Myopia. Here the divergence is mechanical on account of the abnormal length of the eyeball.
- (2) Divergence of a blind eye.
- (3) Divergence after tenotomy of the internal rectus.
- (4) Divergence of the worse eye when binocular fixation has been completely abandoned.
- (5) In anisometropia when the difference in refraction between the two eyes is great.

Latent Squint or Heterophoria.—This is due to imperfect co-ordination of the extra ocular muscles. It can be made manifest by producing artificial diplopia in which the images are so dissimilar that fusion is impossible. When this happens, the eyes give up the attempt to fuse the images and take up a position of rest. Here also it must be emphasized that the condition is bilateral as in manifest concomitant squint.

TESTS:—*Distant vision*: By means of Maddox Rod. With the rod placed before one eye, the patient looks at a light placed at six meters. The eye with the rod before it sees the light as a long line running at right angles to the axis of the rod. If there is no heterophoria the line passes exactly through the light which is seen by the other eye unaltered. In heterophoria, it is displaced to one or the other side of the light or above or below it according to the nature of the defect.

The deviation can be measured by means of a Tangent scale or by means of correcting prisms. The apex of the prism should be placed away from the direction in which the line is displaced or in other words in the direction of deviation of the eye.

Near vision:—Heterophoria in near vision can be detected by Maddox Wing Test. In this the character and amount of deviation can be read off from the position of the arrows as seen by the patient and can be confirmed by the prisms.

Varieties:—Esophoria ... Tendency to convergence.
 Exophoria ... „ „ divergence.
 Hyperphoria ... „ „ upward deviation.
 Hypophoria .. „ „ downward „

In all heterophorias it is not sufficient to measure the amount of deviation but it is also necessary to ascertain the limit of duction in the opposite direction as this latter is an

important factor influencing treatment. The normal limits of duction are:—

Super and subduction	... 1½—2½ degrees
Abduction	... 4 —5 „
Adduction	... Varies and can usually be much increased by practice.

The limits of duction can be measured by means of prism. If, for example, in right Hyperphoria the limit of subduction of this eye requires to be tested the maximum prism with its apex down before the eye which can be tolerated without producing diplopia is determined.

Esophoria:—In this condition when the eyes are tested with the Maddox Rod homonymous diplopia is found. That is, if the rod is placed before the right eye with its axis horizontal, a vertical line is seen to the right of the light.

Small degrees of esophoria are fairly common and produce no symptoms. The liability to cause symptoms is due not so much to the degree of esophoria but to the limit of abduction and binocular abduction. If the former is full and the latter is not less than 3 degrees no inconvenience is experienced.

In moderate degrees where there are no symptoms no treatment is required. If binocular abduction is very deficient, relieving prisms base out can be prescribed. The strength of the prism should depend not on the degree of esophoria, but on the deficiency of binocular abduction, the total prismatic effect being divided between the two eyes. In high degrees advancement of the external rectus may be necessary.

Exophoria:—Here there is crossed diplopia. That is, with the Maddox Rod before the right eye, with its axis horizontal the vertical line is seen to the left of the light. This condition more often produces symptoms than Esophoria, especially if the dynamic convergence of the eyes is deficient. A small degree of Exophoria is common in near vision even if there is none for distance.

No treatment is required in slight uncomplicated cases. Moderate degrees can be successfully treated by convergence training. In higher degrees relieving prisms base in may be ordered, the total effect being divided between the two eyes. It is important in these cases not to overcorrect presbyopia, for such overcorrection brings the near point too close to the eyes and consequently demands an excessive convergence. In high

degree of exophoria if other measures fail, advancement of one or both internal rectus may be necessary.

Hyperphoria:—Here when the rod is placed before the right eye with its axis vertical a horizontal line is seen passing above or below the light depending on whether the left or the right eye is hyperphoric. Vertical deviation is always expressed as hyperphoria of one or the other eye. Because deviation of one eye downwards is always associated with a corresponding deviation of the other eye upwards. Even small degrees of hyperphoria may produce distressing symptoms. Moderate degrees can be relieved by prisms prescribed for constant wear. The total effect of the prisms should be divided between the two eyes, the prism before the hyperphoric eye being placed base down and that before the other eye base up. If the defect is high *i.e.*, over four degrees, operation is usually required because the prisms necessary to correct the error are too heavy to be worn comfortably. In these cases advancement of the interior rectus of the hyperphoric eye may be necessary.

Insufficiency of Convergence.—This condition is diagnosed when there is more Exophoria or less Esophoria in near than in distant vision. It should be treated by exercises with prisms and by reading exercises. In the former, convergence of the eyes is exercised by gradually increasing strengths of prisms placed base out before the eyes. In the latter, the patient brings a book gradually closer to the eyes until the print gets blurred. When this happens he takes the book farther out and again brings it near the eyes. After every tenth line or so he looks away from the book into the distance thereby relaxing his accommodation and convergence. He reads two or three pages twice or thrice a day in this manner for a few months. If no relief is obtained by these methods prisms bases in should be ordered. Here also it is very important not to overcorrect any Presbyopia present.

In all cases where prisms have to be prescribed the same effect may be obtained by suitable decentering of the lenses which the patient may be wearing provided the lenses are of sufficient strength.

Presbyopia

BY

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THE correction of Presbyopia is not so simple as it is considered to be. It is not uncommon to meet cases where the Presbyope has obtained his glasses from a street-vendor or a way-side stall, which is not only unscientific but is actually harmful. Not less unscientific nor less harmful is the system of prescribing glasses for the Presbyope by the Rule of Thumb.

Presbyopia is NOT A DISEASE but a purely physiological process. The term literally means old man's sight. The eye is just as liable as any other organ to suffer from the effects of age. But just as all individuals do not age at the same rate or in the same manner, so does Presbyopia vary in its time and mode of onset.

The Presbyope complains that he is finding increasing difficulty in near work, for example, in reading small print, threading needles, knitting, sewing etc. He may also volunteer the information that he has to hold the work farther from the eye than usual in order to see well.

An emmetrope is a person who has no error of refraction in his eyes. His eye, with its accommodation at rest, is focussed for parallel rays *i.e.*, for an object at infinity. The rays from an object at any finite distance are not parallel but divergent. When such rays pass through an emmetropic eye with its accommodation at rest, they do not come to a focus on the retina but somewhere behind it. But an emmetropic eye is able to see clearly not only objects at infinity but also those at finite distances. This is accomplished by the process of accommodation. This is a physiological act in which the curvatures of the lenticular surfaces are increased by contraction of the ciliary muscle and consequent relaxation of the suspensory ligament. This results in the increase in the refractive power of the lens.

The nearest distance to which the eye can thus be focussed is called its Near point, and will naturally depend upon the maximum power of accommodation of the eye. It will also depend upon the state of refraction of the eye as explained below.

The power of accommodation of the eye diminishes with age due to increase in rigidity of the lens. At ten years of age the

total accommodation, the eye is capable of, is about 13 dioptries. That is, the eye can, by exerting the maximum accommodation, bring to a focus upon its retina, rays from an object at about 8 cms ($100/13$) from it, provided there is no uncorrected hypermetropia. In other words, the near point of such an eye is 8 cms. At 25, the accommodation is 10 D; at 35, about 7 D; at 40, 5 D; at 45, about 3.5 D; at 50 2 D; at 60, about 1.25 D; and so on. It must be remembered that these values are only approximate and that there is a wide variation between different persons of the same age.

Let us now consider the case of a hypermetrope, say of 2D. The eye of such a person when the accommodation is relaxed is focussed only for convergent rays. Such rays, however, do not exist in nature. Theoretically, the far point of such an eye is beyond infinity. Parallel rays meeting such an eye come to a focus behind the retina. To bring parallel rays to a focus on the retina *i.e.*, to see distant objects clearly, the eye has to make the rays convergent by exerting accommodation of 2D in this particular instance. The accommodation available for near work is therefore less by this amount. At 40, for example, he has only $5-2=3$ D available for near work. His near point at this age instead of being at 20 cms. as in an emmetrope, is pushed to somewhere about 30 cms. Therefore, a hypermetrope with his error uncorrected complains of presbyopia at an earlier age than an emmetrope; how much earlier will depend on the amount of the uncorrected hypermetropia. If however, the hypermetropia is corrected by suitable lenses such a person is placed in the same position as an emmetrope of a corresponding age, because the whole of his accommodation is then available for near work.

What about a Myope? Let us examine the case of a person with 2D of myopia. The eye of this person with its accommodation at rest is focussed only for divergent rays *i.e.*, for objects at a finite distance from the eye, the distance depending upon the amount of the error; 50 cms. in front of the eye in this particular instance. Parallel rays meeting such an eye come to a focus in front of the retina. The far point of this eye is not at infinity but at 50 cms. in front of the eye. The eye cannot see clearly objects beyond this distance. Unlike the hypermetrope, the myope cannot get over this difficulty in distant vision by exerting his accommodation. For, by so doing, he will only make matters worse. He, therefore, does not use any of his accommodation for distant vision. He has thus not only the whole of his accommodation available for near work like an

emmetrope but actually has an advantage over him. At 50, for example, the total accommodation available is 2D. The emmetrope by exerting the whole of this brings the anterior focus of the eye from infinity to 50 cms. which is, therefore, his near point at this age. The myope, however, has his eye already focussed for 50 cms. which is equivalent to 2D of accommodation. By a further 2D of accommodation, which he has at his disposal, he brings the focus to 25 cms. ($100/4$) from his eye. His near point is therefore at 25 cms. By a similar calculation it can be seen that at 60, such a person will have his near point at $100/3.25=30$ cms. approximately. The myope, therefore, complains of presbyopia at a later age than the emmetrope; how much later will depend on the amount of the myopia. By correcting the myopia, he is also placed in the same position as an emmetrope of a corresponding age, because, his far point has been pushed to infinity and he has to bring the focus of the eye to his near point from infinity instead of from a finite distance as before correction.

So long as a person is able to carry on his near work at his usual working distance without having to exert the whole of his accommodation constantly, that is, so long as he can keep a certain amount of his accommodation in reserve, he is quite happy. If, however, he has to exert the whole of his available accommodation to be able to do his near work, the eyes soon tire. This happens when his near point has receded to his habitual working distance. When this stage is reached, he is, however, able to carry on somehow. But when the accommodation drops still further, so that his near point recedes beyond the working distance, he finds difficulty in seeing his near work clearly. He at first tries the expedient of pushing the work beyond the usual distance if the work is of such a nature as to permit this. He may also try another expedient namely, having the light between his eyes and the work in order to make the pupils contract and thereby reduce the diffusion circles. (When an object is not properly focussed by the eye, diffusion circles are produced on the retina instead of a sharp image. The bigger the pupil, the wider will these diffusion circles be). But soon a stage is reached when these expedients do not help. He is then compelled to resort to artificial assistance.

Besides age and the state of refraction, there is another factor to be taken into account when discussing near vision, namely, the convergence efficiency. Normally, convergence and accommodation go hand in hand, but this relation may be upset in

disturbances of muscle balance. For instance, at 40, the accommodation is 5D. Theoretically, an emmetrope at this age, has his near point at 20 cms. But if his convergence is weak, or if he has exophoria, he will not be able to converge his eyes for this distance. At 50, a person with myopia of 2D, has, as explained earlier, his theoretical near point at 25 cms. But his actual near point may not be at this distance owing to his inability to converge for this distance.

We can now consider how to investigate a case of presbyopia and prescribe for him. The following plan should be adopted :—

(1) Take the history.—Any symptoms of eye-strain, age, occupation, distance at which he holds his work etc.

(2) If he is wearing glasses already, analyse them.

(3) Take distant vision of each eye separately and of both eyes together. First test without his present glasses and then with them.

(4) Take his near vision without and with his present glasses.

(5) Test his near point with his present glasses.

(6) Test the muscle balance for distance and near.

(7) Test the reaction of pupils and tension.

(8) Determine the refractive error if any, by Retinoscopy.

Although this can be done without dilating the pupils, it is more satisfactory to do it under a mydriatic unless the patient cannot spare the time for a post-mydriatic test, or there are definite contraindications to the use of a mydriatic.

(9) Examine the Media and Fundus.

If a mydriatic has been used, further examination should be postponed till the effects of the mydriatic have passed off. Instil $\frac{1}{2}\%$ Eserine into the eye and see that the pupils are beginning to contract before sending him away. Ask him to come again after a week. If no mydriatic has been used, the examination may be proceeded with.

(10) If Retinoscopy has revealed any error of refraction, test subjectively and find out the best correction for distant vision.

(11) With the distance correction before the eyes, test near vision of each eye separately, and of both eyes together. If it is subnormal determine by trial the smallest convex lens which, when added to the distance correction produces maximum

improvement in near vision. In an uncomplicated case, the near vision can be improved to J. 1. or snellen 0.5D.

(12) With the full correction thus obtained, placed before the eyes, investigate the near point of the eyes and also the range of clear vision.

The near point can, for all practical purposes, be determined as follows. Suppose the patient can read J. 1. with the correction. Let him take the chart gradually closer to his eyes till J. 1. gets blurred. Measure this distance from the eye with a dioptric tape. This tape is graduated in dioptries on one side and in centimetres on the other.

The accommodation card may be used if the patient is illiterate. This is a white card $1\frac{1}{2}$ inches square in the centre of which is drawn a thin black line 0.2 mm. in diameter and about 2 mms. long. The card is fixed in a handle about 2 inches long.

The range of clear vision with the full correction placed before the eyes should also be tested.

(13) Test muscle balance again with the full correction.

(14) Find out the suitability of the correction for the individual by testing under his usual working conditions, at least as far as the working distance is concerned. After all he is not interested in the actual distance of his near point. What he is really concerned about is, the ability to carry on his particular near work in comfort.

(15) Prescribe the necessary correction. The glasses prescribed should not only enable him to carry on his near work in comfort but should also provide him with a sufficient range of useful vision. That is, with the glasses on, he should be able to see well not only at his working distance but also at distances a little beyond as well as a little nearer.

It may be found that a myope wearing his distance correction prefers to do his near work without any glasses at all. The reason for this has already been, explained earlier.

It should be clear now that each case of presbyopia is a problem in itself. Never prescribe by the Rule of Thumb, that is, merely according to the age of the individual.

Do not overcorrect presbyopia especially if there is any exophoria or convergence insufficiency. For, overcorrection brings the near point too close to the eyes and thus puts a strain on the convergence.

Allergy in Ophthalmology

BY

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ALLERGY emphasises the characteristic and outstanding change of an altered reactivity of the body cells which in the usual sense is one of exaggerated susceptibility. There is a specifically altered state of the body, produced by previous exposure, and made manifest by subsequent exposure to the same, or some closely related substance. When a union between antibody and antigen occurs in the cell, some hypothetical histamine-like substance is liberated which damages the cell, and which in certain circumstances may produce a general musculo-spasmodic reaction called Anaphylaxis and in other circumstances a local necrotizing inflammation called Allergy. In an immunized patient anti-bodies are circulating in such a concentration as sufficient to protect the fixed cellular anti-bodies. In an Allergic patient, the antigen-antibody reaction takes place in the cells. Thus it is a deleterious reaction and not useful. Many of the destructive lesions sometimes ascribed to bacterial poisons are probably allergic responses to bacterial proteins. Allergy may be destroyed by repeated injections of the protein to which sensitivity has been acquired. A large number of substances act as exciting agents or Allergens. They are both of vegetable and animal origin.

The commoner methods by which human beings become sensitized are given below :

Placental:—Rarely foods partaken of in the latter days of pregnancy by a mother, whether allergic or not, may contain allergens which pass through the placenta and sensitize the foetus. Or the mother may be inoculated with some antigen as horse-serum to which the child may become sensitized.

Injection:—Foods may cause sensitization to particular individuals who may be susceptible to them.

Inhalation:—Air-borne substances like animal and insect emanations, moulds and fungi, house or occupational dusts, powders from toilet, insects and pollens.

Injection:—Animal serums, insect bites and stings and drugs from these allergens.

Infection:—includes bacteria and parasites.

Contact:—Wearing a woollen or artificial silk or natural silk garment or furs. Walking through a garden during pollination.

Generally, the most allergic states commence early in life, and the attacks tend to become less frequent with advancing years. Males are affected more often than females. They are more common in spring than in any other season. The onset is sudden. They vary in duration, intensity and length of the interval between attacks. Some occupations are more often the cause than others. It is necessary to note the various foods taken by the patient. Many allergic conditions commence when the appetite or digestion is upset and when bowels are irregular. Excess of alcohol or tobacco, or presence in a crowded theatre may bring on an attack. Fears, emotions and apprehension may precipitate an attack. Rapid or sudden changes of weather is also unfavourable to allergic patients.

Noon and Freeman made practical use of the conjunctival test in the diagnosis of Pollen sensitiveness. V. Szily showed that the conjunctiva had the same absorbent power as other mucous membranes. Nicolle and Alet, Sattler, Wesseley, Krusims, Kummel, Dold and Radas, Fuchs and Meller have proved conclusively that the tissues of the eye can readily be sensitized both locally and as a part of general sensitization. Seegal and Seegal in 1930 have definitely established the possibility of local allergic reactions in the eye.

Oedema of the lids without any cause occurs in Allergic individuals. It may or may not be associated with urticaria in the body. One eye is probably affected more often than the other. When no other cause can be found for oedema of the lid it may be attributed to Allergy. The swelling is usually pale compared to the redness of inflammatory oedemas. It appears suddenly and quickly disappears sometimes within a few hours. They are said to be more common in women especially at the time of menses.

Hyperæmia of the lid border and Blepharitis occasionally occur as an Allergic manifestation. I had a typical case of this variety which for a long time did not yield to the usual remedies. The patient, aged 35 and a man of active habits, had blepharitis. He did not get any relief after prolonged treatment. He had suffered from Duodenal ulcer three years previous to this an

was medically treated and cured for the same. The lid condition used to get better with application of adrenalin-chloretone ointment but used to recur when the treatment was stopped. I referred him to his usual medical attendant who gave him treatment for Duodenal ulcer again, on my request, though he did not have any symptoms of Duodenal ulcer. I gave him a course of six Aolan injections. He is now completely free from the recurrent attacks of Blepharitis for the last two years.

The conjunctiva shares with other mucous membranes and the skin, the liability to react in an abnormal manner to certain sensitizing substances. A sudden extreme vascular dilatation involving a boggy exudative inflammation, disappearing after sometime in acute cases and in more chronic cases by a cellular infiltration and finally by connective tissue new formation, is met with now in Allergic persons. Examinations of a Blood-film shows marked Eosinophilia. In the acute type the condition is usually seen about puberty. Onset is quite sudden and comes usually in Spring or early Summer. The eyes get bright red with slight oedema of the lids occasionally. A feeling of intolerable itching and burning is evident and lacrimation is profuse. Coryza may be a complication. Pollen, Corndust, flowers, feather, wool, or the dust from the skin of animals like cat or the horse cause this. Animal parasites or even Blood-serum may act as a cause. Drugs like atropine cause follicles to appear in the sub-epithelial tissue in large numbers and in rows. It usually passes off when the patient is removed from the irritating substance or the instillation of Atropine is stopped. There are no other symptoms of Atropine poisoning present.

In chronic simple allergic conjunctivitis, the conjunctival sac is usually sterile. There is hyperæmia, little secretion and very accentuated subjective symptoms. They are associated with a chronic focus of infection elsewhere. Onset is gradual, less severe in symptoms, runs a non-paroxysmal course persisting as long as the focus of infection is present. Clinically, there is chronic conjunctivitis with a red and boggy mucosa, with a little watery secretion, with constant irritation and itching. The most common sources of infection are Nose, Nasopharynx, and Tonsils and Teeth.

The abrupt appearance and disappearance of the symptoms in association with contact with the exciting agent and the negative bacteriological findings, the presence of Eosinophilia, in association with other allergic manifestations, the demonstration

of positive sensitization tests and relief by adrenalin, help in the diagnosis.

For treatment, wherever possible, the exciting substance should be traced and eliminated. Locally, astringent lotions and adrenalin ointment give relief. For the condition of the lids Zinc-Oxide ointment combined with adrenalin is useful. For specific treatment, discovery of the protein, by carefully going into the history of the case, tests of the skin sensitivity to the various pollens and animal proteins, can be made in some cases. If the patient cannot avoid these, systemic injections of the offending substance desensitizes the patient. In chronic bacterial types, the attention to focus of infection and in the absence of any cause, non-specific protein therapy may be attempted.

That Phlyctenular kerato conjunctivitis may be an allergic phenomenon was put forward by V. Szily in 1916. Phlyctenules are allergic phenomena—a reaction of the conjunctival and corneal hypersensitive epithelium to any protein. In the majority of cases, the protein is tuberculo-protein. The Epithelium is sensitized to some endogenous protein and subsequent contact with it results in the formation of the typical lesion. Exogenous proteins play no part in the ætiology. Sensitization occurs as a part of a general hypersensitiveness to tuberculo-protein from an early tuberculous infection of the lungs or glands. Intoxication follows by the introduction of Tubercular protein into the eyes either by the blood stream, following a recrudescence in the parent focus, by the tears, or by the direct exogenous inoculation in patients who are exposed to open tuberculosis in their surroundings. If Von Pirquet is positive, a course of Tuberculin injections gives good results.

The Theory that Vernal conjunctivitis is a manifestation of Allergy has the most definite evidence to support it. The clinical evidences in favour of this theory are:—tendency to attack the young, seasonal occurrence of attacks and the type of secretion with eosinophilia. The irritating agent is dust aggravated by dry heat, occurring preferentially in vagotonic individuals.

Jubianelle and Morris in 1932 put forward the plea that allergy can be suspected to influence trachomatous reaction in the cornea, though not as a primary cause, but only as a determining factor in the type of reaction in the cornea.

In recent times, much work has been done to elucidate the

influence of allergy in intraocular infections. There is liberation of foreign protein at the site of infection, either protein derived from the invading organism itself or elaborated in the reaction between it and the infected tissues. This is especially so in the recurrence of inflammatory conditions. By this method the tissues respond in a marked manner to bacteria, their toxins or other foreign proteins when these are present in concentration so small as would excite no inflammatory response in the non-allergic individual. Seegal injected into the anterior chamber of rabbits, a suspension of a strain of a hæmolytic streptococcus. Long after the reaction had died down in the eye, he found, on intravenous injection of the same organism, that a violent recurrence of Iritis became evident, conversely, he produced an ocular reaction after general sensitization by an injection into the eye. Not only an organism but a toxin can produce sensitization in the tissues of the eye, and recurrences of inflammation can be excited by a liberation of small quantities of that toxin into the blood stream. This is sometimes seen in cases of syphilitic Iritis. Long after the blood reactions have died down and the eye is normal, patients develop iritis, clinically similar to syphilitic iritis, though the blood Wassermann, reaction still remains negative. In such cases, when mere anti-specific treatment fails to bring about a speedy recovery, combination of the antisyphilitic treatment with general non-specific protein therapy gives permanent relief from recurrent Iritis.

In tubercular Iritis, a similar reaction based on allergy is evident. When the tubercle bacillus dies, the proteins which it contains are liberated and if the patient is allergic, they cause various reactions according to the sensitivity of the patient and the amount of protein liberated. The relatively bland products of the breakdown of tubercle bacilli are converted into powerful irritants and poisons, without any apparent gain to the organism in the way of immunity. Tissue necrosis is probably almost entirely the result of allergic hypersensitiveness to tuberculo-protein, though the formation of tubercles does not appear to be a reaction of this type. For production of allergy, it is necessary to use whole bacilli and it appears that it has not been possible to extract from the tissues sensitizing antigens. When the patient acquires immunity, the tubercle bacilli in their disintegration set free tuberculo-protein. If this patient is also allergic, he develops tuberculo-allergic Iritis, clinically termed non-tuberculous Iritis.

The development of Allergy seems to exercise a pre-disposing role in the ætiology of sympathetic ophthalmia. But it cannot be the single or determinating cause. In spite of the many theories of the causation of sympathetic ophthalmia, Elschnig and Bail think that something that is liberated by the destruction of the uveal tissue may be the cause. This conception is best adapted to build the ground-work for an understanding of the peculiarities of sympathetic inflammation through the absorption in antigenic form, through organ specificity, through local anaphylaxis.

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Hysterical Manifestations in the Eye with Case Notes

BY

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HYSTERIA assumes such deceptive and changeable aspects from time to time and stimulates such a number of organic diseases that it is apt to deceive the clearest brain. This fact is at once the explanation and the extenuation of errors which we have finally overcome. In the midst of this changeable symptomatology, the ocular manifestations are singularly constant and characteristic and these are of two varieties. Some are more or less life long while others are transitory and occur during convulsive attacks. Generally, the ocular manifestations of hysteria, are confined, either to the motor apparatus of the eye or the apparatus of special sense. The following case may be of interest.

CASE I.

Hysterical Amaurosis.—A married Hindu girl, aged 18 yrs., came up for consultation in August 1937, for loss of vision in both eyes. Some three weeks back, while lighting a spirit stove which had just gone out, the thing exploded and she sustained an injury on her right eye and lost consciousness. She was immediately placed under the care of a local doctor who attended upon her for about one week, when the swelling, redness, chemosis of the conjunctiva, etc. disappeared but the patient could not see with her right eye although there was no injury to the cornea or other vital structures of the eye. On the third day following this trauma, the girl complained of loss of vision in the left eye (the injured eye) and there was extreme hyperæsthesia of the skin over the right half of the face and neck. The hyperæsthesia began to spread along the whole of the right half of the chest, abdomen, right upper and lower extremities. The patient was evidently in great distress and would not allow anybody to touch the right side of her body and even the weight of her clothes and under-garments were painful to her. After a week, she was admitted into a hospital for purposes of investigation and treatment but unfortunately after a stay of ten days in the hospital the case was discharged as incurable.

Now, seventeen days after that trauma, I had occasion to examine the case. The girl was strongly built, well-developed and had no issue. Excepting a small scar about $\frac{3}{4} \times \frac{1}{2}$ inch over the right upper lid, close to the eyebrow, there was no other deformity. On attempting to open the lid, the girl shrank back and cried out in distress. I was not prepared for this and so felt a bit non-plussed. I asked her to open her lids, which she did, when I noticed that there was no sign of inflammation of the bulbar conjunctiva, cornea or the iris. Pupil was slowly reacting to light, media—clear, fundus—normal and there was no P. R. or P. L. Left eye presented a similar picture, there being no P. R. or P. L. in that eye too. All recognised tests failed to elicit evidence of any vision in the eyes. From the twentyfirst day the hyperæsthesia began to subside from below upwards, i.e. the skin sensation became normal in the lower limb first. On the twentyfourth day, the girl suddenly recovered her vision and it was found to be 6/6 in each eye.

During the time she was under my care I kept the patient confined in a dark room so as to avoid light (both natural and artificial) and asked her husband to continually suggest to the patient that she would recover her full vision in no time.

CASE 2.

Hysterical Spasm of Accommodation.—A Hindu boy aged nineteen years had been suffering from gradual diminution of vision since 1-10-31, when with (—0.50 Dsp. with —0.50 Dcyl. $\downarrow 90^\circ$) glasses he had 6/9 vision in each eye. On 25-5-32 he was once again examined by his old physician who noted that the vision had gone down to 6/24. He prescribed a fresh number (R. E. —0.25 Dsp with 1.0 Dcyl $\downarrow 90^\circ$; L. E. —0.50 Dsp —1.0 Dcyl $\downarrow 90^\circ$) and with this the vision was 6/12 in B.E. Disc-Hyperæmic. Three or four months after, the boy began to feel discomfort with his glasses, when it was changed once again. (Unfortunately the patient could not produce the record of this examination.) On 12-7-33 the boy was examined by another Ophthalmic physician who noted the following:—

R. E. V, with 1.0 Dsp with 1.0 Dcyl $\downarrow = 5/36$.

L. E. V, with 1.50 Dsp with 1.0 Dcyl $\downarrow 90 = 5/36$.

Fundus—Veins Engorged. Optic Neuritis.

He was given some nervine tonics, inspite of which the vision recorded by the same doctor on 3-8-33 was

B. E. Vision with own glasses — 5/?

Now things have come to such a state, that a thorough investigation was started.

Urine Report: Excess of phosphates—present.
Chlorides. —0·82%
Other things. —normal.

W. R. and Kahn Test. Absolutely negative.

Teeth, Tonsils and air sinuses were examined by specialists and declared to be healthy.

B. P. —120/90 m.m. of Hg.
Height,—5' 6". Weight 188 lbs. or 13 st. 6 lbs.

The boy was given an iodine mixture and asked to continue for three months.

On 12-8-33 the boy came to me for consultation, when the following were noted:—

Vision:—R. E. —1/60 with 10·0 Dsp = 6/24.
L. E. —1/60 with 9·0 Dsp = 6/24.

No further improvement of vision with glasses could be produced during subjective test.

Perimetry:—With 5 m.m. white object, at a distance of 333 m.m. the field of vision was mapped out and found to be contracted to 6° only on either side in each eye.

Retinoscopy:—Mydriatic used—2% Homatropine Hydrobrom Drops instilled every ten minutes for six times only. There was small hypermetropic astigmatism of 0·5 dioptré only, along horizontal axis, in each eye. Fundus—appeared to me, to be absolutely normal.

Under Mydriatic Test:—

R. E. V, with +0·50 Dcyl $\rightarrow$ 180° = 6/60.
L. E. V, with +0·50 Dcyl $\rightarrow$ 180 = 6/60.

I was much worried as I could not account for this loss of vision. Accidentally, I placed a dark blue glass in front of the right eye when the boy read upto 6/9 smoothly. In the left eye also the same phenomenon happened. For post mydriatic test, the boy was advised to come after fortyeight hours when with +0·50 Dcyl $\rightarrow$ 180°, vision was 6/24 in each eye. As it was found

to be a clear case of spasmodic myopia the patient was given Atropine for one week, at the end of which the vision improved up to 6/12. A further course of Atropine for fifteen days brought the vision up to 6/6 without any glasses. He was prescribed astigmatic lenses and now for the last six years he is absolutely free from all trouble.

Field:—Recorded on Lister's perimeter with 3 m.m. object at a distance of 333 m.m. was found to be normal.

I have forgotten to mention one curious phenomenon namely the inability of the patient to estimate the true size and form of objects which he had noticed one year back. Thus, while looking at a book, if it was moved some ten or twelve inches further, the small prints would appear bigger and bigger till they would get blurred and hazy.

Discussion.—In the cases mentioned above, the usual history of convulsions was absent and hence the question may arise whether they were truly hysterical or not.

In case I, we find a strong healthy girl suddenly losing her vision, following a trauma. Now, this injury was sustained in the right eye, then why should the vision in the left eye be lost? There was absolutely no injury to the vital structures of the eye, still there was no P. R. or P. L. in both eyes. Moreover, the pupil reacted to light, as it does not do in blindness from other causes. In other words, although the luminous impression is not perceived, it is carried to the brain in such a way as to produce the pupillary reflex. This fact alone, I consider, to be an important aid to an understanding of hysterical phenomenon. Again, the peculiar and acute hyperaesthetic condition of the right half of the body and its sudden subsidence on the twenty-first day, all serve to show that the condition was not due to any organic lesion but to some functional disturbance of the visual apparatus. I am afraid I cannot explain the pathogenesis of his hyperaesthesia, which I leave to my worthy colleagues for elucidation.

Case II presents a picture of amblyopia due to excessive spasm of accommodation and also the influence of light upon the development of that spasm.

In this case there is a double spasmodic myopia of 9 dioptrés (more so in the left eye), the eye being slightly hypermetropic with a visual field that is much contracted. With atropinised

pupil and a pair of dark blue glasses in front, vision improves upto 6/9.

This myopic spasm together with a contracted field, I consider to be hysterical in origin. It becomes more definite when we remember the other symptom, *e.g.*, perversion of the faculty of estimating the true size and form of objects held at different distance from the eye.

A healthy eye can estimate the true size of an object, as it approaches or recedes from the eye. But in this case, the objects appeared bigger as they were withdrawn from the eye.

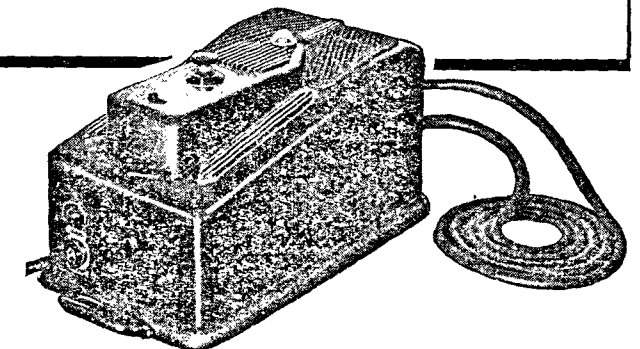
Conclusion.—I am at present investigating a case of dyschromatopsia, wherein, sometimes the red field becomes bigger than white or blue fields and at other times it is practically normal. For the last two years, I am following variations of the colour field as also the mood of my patient and as yet, have not collected sufficient evidence to declare it to be hysterical.

Another case of repeated attacks of ophthalmoplegia (lasting for three to four weeks during each attack) in a young woman, is under investigation for the last four years and during this period only three such attacks have taken place.

I hope to communicate the result of my investigation in a subsequent paper.

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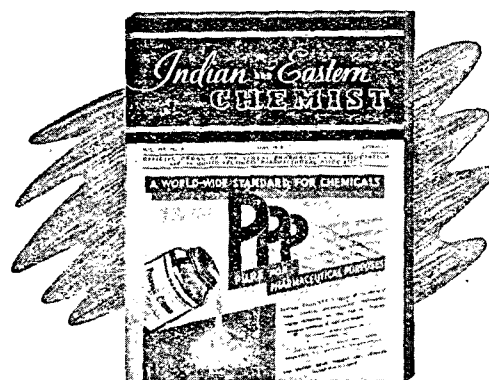
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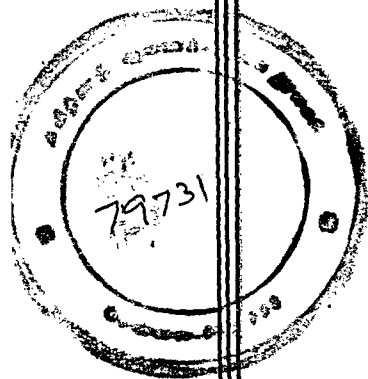
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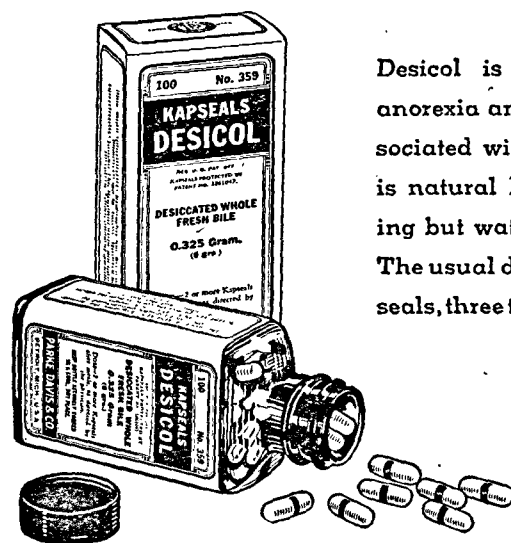
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