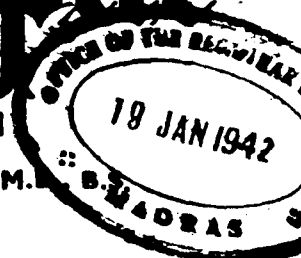


The Antiseptic

A Monthly Medical Journal

Edited by
Dr. U. Rama Rau, M.L.C. & U. Krishna Rau, M.L.C.



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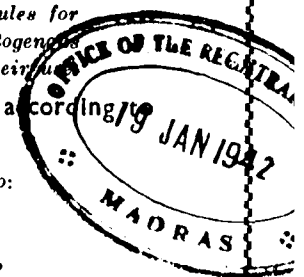
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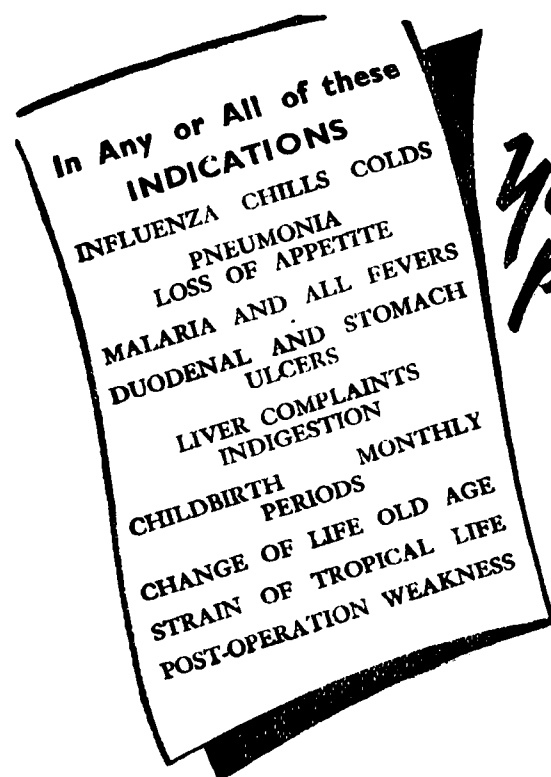
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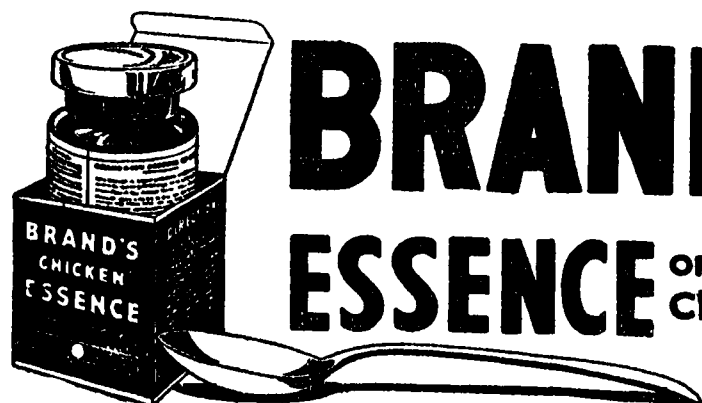


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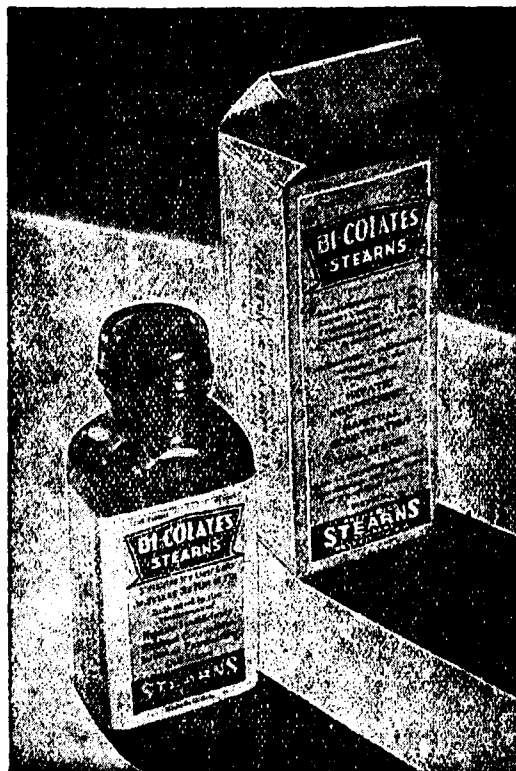
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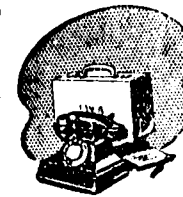
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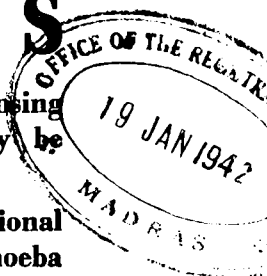
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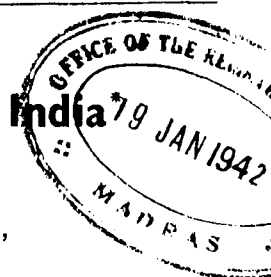
Surgical Tuberculosis in South India

Some Notes on 1,000 Operated Cases

BY

T. H. SOMERVELL, M.A., M.B., B.Ch., F.R.C.S.,

Neyyoor, South India.



It always seems to me an unfortunate accident that has determined the use of the word "tuberculosis" (or more often, in conversation, "T. B.") in referring to lesions caused by the so-called human Tubercle bacillus, such as phthisis and its complications, as well as to those caused by the bovine bacillus, such as bone, joint, and intestinal tubercular diseases. One must admit that the bacilli are both acid-fast, both look very much the same, and patients who are susceptible to the one are often, but by no means invariably, susceptible to the other. But such patients are also susceptible to leprosy, another acid-fast organism, and yet there should be no confusion in one's mind between tuberculosis and leprosy, however many cases of phthisis there may be in a large leper settlement.

The fact remains that the organism which causes phthisis and that which causes tabes mesenterica are two distinct organisms, and the etiologies of the two groups of disease are different. In some parts of South India, for instance, where phthisis is almost unknown, surgical tuberculosis is very common. In England, where phthisis used to be a very terrible scourge, tuberculosis of the intestinal tract is extremely rare. In Central Travancore, where phthisis is rampant, surgical tuberculosis is not common.

In South India as a whole, it is a widespread condition, probably owing to the scant attention that is paid to the disinfection of milk, from which it usually arises. I know a family of whom three members have three different kinds of surgical tuberculosis—of mesentery, bones, and small intestine respectively—although they are otherwise healthy people and never likely to get phthisis. Obviously, a cow, whose milk they drank unboiled, was the cause of the disease in all these three cases.

* Read before the District Medical Association, Trichinopoly, in October '41, and specially sent to 'The Antiseptic' for publication.

The big problem the medical profession is up against in its fight against surgical tuberculosis is undoubtedly the problem of rendering milk free from infection, by inspection and control of cows and by insistence on the boiling of milk in all places where surgical tubercle is known to exist. In this attack on disease, Governments and the medical and veterinary professions must work hand in hand. But, as surgeons, we are more often called upon to treat cases which have already fallen a victim to the milk they have ingested, than to arrange for their milk supply to be properly dealt with.

In South India, surgical tuberculosis is fairly widespread, but has a very different distribution among the various organs from that which obtains in Europe. In England, laparotomy in cases of tumour of the caecum reveals a carcinoma twenty times as often as a tuberculous caecum.

In my own experience, I have performed the operation of excision of the caecum for tuberculosis on 54 occasions during the last twelve years, as against 2 excisions of large intestine for carcinoma. Relative to carcinoma of the colon, then, tuberculosis is more than 500 times as common in India as it is in England.

Tuberculosis of the spine seems to be less widespread here, but stricture of the small intestines is very often found—I have operated on 128 cases of this condition in 12 years, in every case resecting the diseased intestine and performing anything from one to four anastomoses. Yet, in Europe, it is a rare disease.

Surgical tuberculosis is best divided as follows:—

Particulars of Operations.		No. of Cases.	% of Series.	Deaths.		
T.B. of Neck. Particulars		52	5%	14	27%	
Stomach		2	0.2%	—	—	
Small Intestines		95	10%	5	5%	
Caecum		54	5%	4	7%	
Rest of Large Intestine		8	1%	—	—	
Rectum		1	0.1%	—	—	
Anus. (T.B. fistula)		20	2%	—	—	
Total		232	23%	23	10%	
GLANDS: Neck		272	34%	1	0.29	
Axilla		44				
Glands		24				
JOINTS: Limbs (including Pelvis and Scapula)		...	273	27%	4	1.5%
Spine		...	47	5%	1	2%
Ribs		...	46	5%	—	—
Other: Tibia 2. Skull 4. Clavicles 4		...	10	1%	—	—
Genito-urinary—Epididymis and vesicles		46	52	5%	1	2%
Kidney		6			1	17%
Total Cases Operated.		...	1000		31	3%

(Tuberculous abscess of skin)

(Tuberculous ulcers of skin, tongue, etc., and lupus, not included).

It is too long a list to permit of an exhaustive description of every class of case, but I shall content myself with making such observations as seem to be useful about each type.

Tuberculous Peritonitis.—Diagnosis fairly easy by the semi-resistant feel of the abdomen, wrongly described as "doughy" which it is not. Oedema is doughy. Tuberculous peritonitis is elastic and has a semi-solid, gelatinous

feel, unless it is of the ascitic type in which case it simply feels like any other ascites unless lumps (e.g. of omentum) can be palpated.

Treatment is always laparotomy, however ill the patient may be, because of:—

1. *Curative Effect of Laparotomy*:—This does not always occur, but it does often do so, and in surprisingly bad cases.

Case.—1929. C. Age 22. Temp. 101-103°, remittent, for several weeks. Case appeared hopeless. Incision, Right lower paramedian. Intestines matted together with gelatinous nodules; only one small coil of small intestine could be exposed. Temperature came down at once, and in 3 weeks patient was completely recovered and remained so. (Later had tubercular glands neck, and tuberculosis of both epididymes, all of which reacted well to operations, but died recently of tuberculosis septicaemia from glands in the axillae, having been persuaded by a friend to trust himself to Ayurveda).

2. The possibility of finding a removable tubercular focus, as caecum, glands, omentum, etc., on the principle that if there are two or more foci of tubercular disease, they should always if possible be reduced by excision to one focus, which may then improve by medical treatment (S. F. Iodide, Thisin, Codliver oil and malt, sunlight, ultra violet rays, X-rays) but is unlikely to improve in the presence of other foci.

Incidentally, it is taught in medical colleges, and printed in text books, that one of the commonest causes of tuberculous peritonitis is a tubercular appendix, the removal of which is advocated in order to bring about a cure. Personally, I have never seen a tubercular appendix in South India, and I doubt if such a thing exists. When I started work in India, I always looked for the appendix in these cases, and usually removed it, but never found it to be tuberculous, although the caecum and small intestines often are, and occasionally the Fallopian tube. I have a suspicion that this statement about tuberculous appendices is one of the many statements which are repeated from text-book to text-book without having been verified by the writers of these books. Has any one here ever seen a tuberculous appendix? (Only specimens in museums were mentioned as answer to this question).

Stomach.—A great rarity. I have had 2 cases, both diagnosed before operation as cancer of stomach. One was inoperable; the other was resected by a subtotal gastrectomy.

Small Intestine.—(In this series, nearly half of all cases of abdominal tuberculosis and 10% of all cases of surgical tuberculosis). Tuberculous ulceration of the small intestine is common in this country. The symptoms are those of intestinal colic. The patient may retain good general health, but is made miserable by the ever-recurring attacks of colicky pain, felt around the umbilicus (to which area small intestine pain is always referred). In all such cases—and there must be thousands in South India, where so much unsterilised cow's milk is drunk at the present time—a sedimentation test must be done, and a screening with Barium meal, preferably by the surgeon himself, who can elicit the relation of tender points in the abdomen with portions of the intestine which are obviously dilated. In all cases that I have seen, at least one piece of small intestine is broadened to about twice its normal width, and at the distal end of the broadened portion is a tender

spot corresponding to the ulcer itself. There is also delay in emptying of the distended loop or loops of intestine.

The physical signs are thus:—(1) Tenderness in one or more very localised points in the central abdomen. (2) High rate of sedimentation. (3) Delayed emptying of small intestine. (4) Radiographic evidence of distended loop.

There is only one treatment; *viz.* laparotomy, excision of ulcers and entero-anastomosis. Personally, I always do a lateral anastomosis, iso-peristaltic. I fear that the quicker end-to-end anastomosis may become the site of further disease leading to a stricture and I have seen evidence of this in cases operated elsewhere. In all cases, the entire intestine must be searched for ulcers or strictures; portions of intestine containing several ulcers that are close together should be excised, but not more than one-third of the entire intestine can be taken away without endangering the digestion, if not the life, of the patient. In one case, I have performed five resections, and anastomoses and four in several cases, all at the same sitting. Novocain regional anaesthesia should be used, and an adequate incision made to avoid pulling on the sensitive mesentery.

Tubercular enteritis is very often complicated with pulmonary tuberculosis. Ulceration of the small intestine on the other hand is very seldom complicated by a pulmonary lesion. I think it is extremely probable that ulceration of the intestine is due to the bovine bacillus, whereas enteritis is due to the human bacillus. The statements in text-books about the ingestion of tuberculous sputum as a cause of ulceration of the small intestine are probably quite incorrect.

Cæcum.—(One quarter of all abdominal cases and 5% of all cases of surgical tuberculosis). This disease is also very common, and is often diagnosed as chronic appendicitis, resolving appendix abscess or a tumour, or even as duodenal ulcer. Owing to the size of the cæcum, tubercular ulceration of it may progress to a serious extent without stenosis occurring. The symptoms, are therefore, usually very different from those of small-intestine tuberculosis. Patients tend to become cachectic before they show signs of stricture or obstruction; in small intestine disease, they are obstructed before they have become cachectic.

Diagnosis usually depends on the following signs and symptoms:—

- (1) a palpable mass in the right iliac fossa, slightly tender.
- (2) high sedimentation rate.
- (3) deformity of outline of cæcum with Barium meal—not always evident by screening alone; a film must be taken.
- (4) no leucocytosis unless a peri-caecal abscess has formed.
- (5) slight evening temperature, sweating etc.

A few cases of tubercular cæcum have been diagnosed before operation as duodenal ulcer with chronic appendicitis; but I have only made that mistake in cases which have not had X-Ray examination. For some years now, I have done a fluoroscopy on every single case of duodenal ulcer before operating upon it, and as a result I have not recently made that mistake. If there is no deformity of the duodenal cap, and no localised tenderness of it, one's attention is directed to other parts of the abdomen including the cæcum, and a sedimentation test is done.

When tuberculosis of the cæcum is diagnosed, there is only one

treatment; laparotomy (preferably under spinal Percain anaesthesia), with excision of the whole ascending colon and terminal ileum, and either end-to-end anastomosis of ileum into transverse colon, or end-to-side or side-to-side, according to the surgeon's judgement in the particular case. If the small intestine is not at all distended, lateral anastomosis (ileotransverse) should be done. Anastomosis without resection of the colon may prolong a life for a year, but never saves it, as the disease progresses and the patient becomes less and less able to stand the operation of resection which alone can save life. During the last ten years, I have never attempted a two-stage operation, but have done resection and anastomosis at one sitting. It can be done in well under an hour. Three layers of sutures are recommended for the anastomosis, or leakage may occur and lead to fatal peritonitis. The middle layer can be made continuous right around the anastomosis, not being knotted, for a knot is a weak point in a suture. No suture under tension in anastomosis of the large intestine will heal well. Hence, the purpose of the outer suture is primarily to relieve the tension of the other two sutures which really do the work of keeping the two portions of gut together.

Tubercular stricture of *colon* (transverse) is rare; in this series, there are only eight cases, and very few were diagnosed before laparotomy. Excision and anastomosis is the treatment, with preliminary caecostomy if there be obstruction.

I have only one case of tubercular *rectum* to report; permanent colostomy was done, as the patient was never fit enough to stand the excision of the rectum. A course of A.O. Vaccine, the Japanese product sold by Thakore of Madras, might be useful in this type of case, and should be tried in cases of abdominal tuberculosis which do not submit to operation. 20 injections are given, at 10 days' intervals. A reaction after a few weeks, with worsening of the symptoms, does not signify failure; it may be that it is actually a good sign. Gold injection therapy may be beneficial.

Tubercle of the *anus* takes the form of very chronic and painful fistulae. The treatment is wide excision of the fistulous track, and general anti-tubercular measures, and the track usually heals.

Tubercular Glands.—(In this series 340 cases=34%). In general, it may be said that one of the objects of surgery in a case with several tuberculous lesions is this; that tuberculosis is always worse in the presence of more tuberculosis. A single tuberculous lesion has a better chance of healing if all other tuberculous foci are definitely removed by operation.

Nowhere is this principle more evident than in the case of tuberculous glands. Accessible glands, in neck, axilla or groins, should always be removed, however bad they may be. A caseating gland will never recover, and should be taken away. A non-caseating gland can recover with medical treatment. Ultra Violet Light, Mercurial ointment or Iodex, Thisin injections, Syrup Ferri Iodidi, Gold preparations, and Cod Liver Oil. But caseating glands should be removed radically and completely even though operations in several stages are necessary.

A short time ago, I operated on a case in which there was an immense mass of caseating glands, some of them as large as a hen's egg, on both sides of the neck and in one axilla. The patient was desperately ill on admission, with a pulse of 136 and general cachexia, anaemia, and consequent oedema. Five operations were performed on the neck under

Paraldehyde by the rectum, and Novocain locally. The patient gradually recovered, and a hectic temperature of 102° and 103° daily for many weeks went down little by little after each operation, till finally the patient had only three palpable glands left, and a normal temperature. I am convinced that a general inhalation anaesthetic, at any rate for any of the first three operations, would have killed the patient.

In operating on tubercular glands of the neck in one stage, an incision is made along the whole length of the sternomastoid muscle, and the skin and platysma dissected off for $1\frac{1}{2}$ inches along either side. The sternomastoid muscle is divided transversely below the spinal accessory nerve which pierces its upper half; the two halves of the muscle are dissected up and down respectively and retracted up and down. Thus, the whole interior of the neck is laid bare, and a very complete operation can be done with the minimum of shock to the patient, and visibility is so good that nerve roots, vessels etc., which might otherwise be damaged are spared. At the end of the operation, the muscle is sewn together again with Cat-gut stitches of a Lembert or mattress type; this gives strong, firm union which has not in my experience ever broken down. The wound is completely closed, with a rubber drain for 24 hours only.

The three types of tubercular glands which do not necessitate operation are:—

(1) Those in which nature has already got rid of tuberculous matter, the caseous substance having reached the surface and extruded itself. Beyond a little scraping, no operation is usually advisable for this class of case, unless it is decided to do a complete dissection of the neck and to remove *all* tuberculous glands; drainage must be free or mediastinitis may result.

(2) Those in which all glands are small, soft, and non-caseating; medical treatment may be successful here.

(3) Those in which there is a large matted mass of innumerable small glands. X-ray exposure is valuable in this class of case, and operation is difficult, tedious, and causes shock to the patient owing to its lengthy nature and the difficulty of preserving nerves etc.

Apart from these exceptions, I make it a rule to remove all tubercular glands, even at the risk of producing oedema from imperfect lymphatic drainage of head or limbs. Ultraviolet Rays and complete medical measures will then have a chance of restoring the patient to good health.

In one case with tubercular glands of the neck, both sides, and tuberculous peritonitis, the patient did not do at all well on medical treatment, and laparotomy failed to clear up the peritonitis. Complete removal of all glands of the neck caused oedema of the face, but as soon as it was done, the peritonitis began to clear up and the patient started to make a thorough recovery.

Tuberculosis of Joints.—(In this series 320 cases = 32%). I shall not say much about these as the better text-books describe the details of the treatment of these joints, and every single case has to be judged on its own merits.

Briefly, it may be laid down as a general rule that tubercular joints, if there is no fluctuation and no sinuses, must be immobilised in unpadded plaster splints. If there is fluctuation, abscesses must be aspirated, or

better, actually removed *including the wall* of the abscess cavity, in which are many tubercular foci, and then they must be treated *either* by being completely sewn up and immobilised in plaster, *or* by being left *completely* open to granulate, the limb (if they are on a limb) being immobilised by plaster or extension, but the cavity being laid bare to the surface by the cutting of an ample window in the plaster. If sinuses exist, choice has to be made between: (1) treating the limb by immobilisation in plaster and paying no attention to the sinuses, which will very likely close up, and, (2) erasing the joint, hoping for its cure by ankylosis, or, (3) excising the joint, aiming at ankylosis in the case of hip, knee, or ankle, and at a movable joint in the case of elbow and, sometimes, also of the wrist.

The golden rule is this: ankylosis is the only way of securing *absolute* rest to a joint and thereby healing the tuberculous process. Ankylosis should, therefore, usually be aimed at primarily, except in the elbow. Arthroplasty may be possible later, when the tubercular disease is completely cured and has remained so for a year or more. Bone grafts as in the spine, or knee, sacro-iliac joint, or ankle, may aid the ankylosing process, but must be done in such a way as to involve no exposure in the field of operation of any tuberculous tissue at all. If a closed plaster is used for a case with sinuses, there must be a dressing inside the plaster consisting of lint or gauze soaked in a strong glucose solution (20%) which will almost entirely prevent the smell of a plaster soaked in pus which is so unpleasant.

One of the chief difficulties, we all encounter in treating tuberculosis of joints, is the extreme unwillingness of patients (or of their parents) to submit to immobilisation for many months; we all know that a tubercular joint ought to be immobilised for six months; but if ankylosis is not produced in that time, it may have to be kept at rest for a year or more—even two years. Everything fights against this. The patient wants to get moving again: or his parents want to put a stop to the inconvenience of having to feed or to carry a child who appears to be fairly able-bodied or at any rate is not acutely ill. The unqualified practitioners and Ayurvedic experts get busy persuading the patient or his relatives that they can bring about a cure in a few weeks; a business friend tells of a little girl he knew who went to Rameswaram and there was cured in a few days of an exactly similar complaint. Against all this, the patient has very little chance for rest and immobilisation which can save limb or perhaps even life; the plaster is removed; quacks have their way, the joint is violently moved or a potent and sacred oil is applied to the top of the head, and if eventually the patient returns to his original practitioner, the joint is in far worse condition, and requires two years of immobilisation, instead of a mere 6 months more, to give it a chance of cure.

In view of all this—a sad picture which we must all have seen time and again—one has to consider very carefully whether radical surgery is not more often required for tubercular joints. On the whole, I feel that in people who are prepared really to be a patient, major surgery should be avoided in favour of plaster and immobilisation. But there is no doubt that a patient will obey the instructions of a surgeon who has operated upon him much more readily than those of one who has merely put him in plaster. Why that is so is a matter for the psychologists to decide. But it is true; and it provides the chief justification for the otherwise doubtful surgical procedure of *eration*. Eration is sound surgery only if *all* the tuberculous tissue is removed, a very difficult thing to do; or if ankylosis can be

produced which will put an end to painful movements and provide that rest which alone can cure (or enable nature to deal with) the tuberculous process. If erosion is done, then, it must be thorough, and it must include removal of joint cartilage so as to encourage ankylosis. It must be followed by immobilisation in plaster (unpadded), best applied a few days after the operation is performed.

The following are a few instances of the importance of removing the joint surface bones; all similar lesions of the knee joint. All kept in touch with for 2 or more years after operation.

1. Girl, 12 years. Femur and tibia full of holes near joint surface. *Limited Excision.* Plaster. Is now healed and fit.
2. Boy, 18 years. Do. Do.
3. Boy, 18 years. *Erasion only*, main joint surface not exposed or deprived of cartilage. Plaster 1 year. Ultimately required *amputation*, and is now very happy.
4. Brahmin (Pusari), 56 years. *Erasion only*, joint surface not touched. Only fibrous ankylosis produced, still painful and unable to walk without both crutches and *pain*.
5. Man, 41 years. *Erasion only*, cartilages removed but joint surface not sawn. *Unsatisfactory*. Still some pain on walking.

As for excision of joints, this depends very largely on the particular case. If the surgeon is about to embark on an erosion operation, he should always be prepared to change his mind during the operation and perform an excision. Personally, I very often perform what might be described as a limited excision, together with erosion of all traceable tuberculous tissues, making it a rule to remove joint cartilage with a saw, so that the surfaces of the joint can be in apposition over a wide area. My failure in tubercular joints have nearly always been traced to doing too little. I can remember only one failure due to doing too much, and that was an elbow which became a flail joint, which is quite useful if a permanent splint is worn, and in any case is free from all active tuberculosis.

In excising a joint, then I would urge:—(1) Thorough exploration of the extent of the tuberculous process, through an adequate incision. (2) Excision with knife and scissors (not spoon) of all accessible tubercular soft parts, while the joint is intact and anatomical positions preserved. (3) Sawing of the bones at appropriate places, not too far from the joint. (4) Excision of tuberculous tissue that is now revealed when the bone-ends are removed (*e.g.* back of knee, front of elbow etc.). (5) Drainage for 24 hours, on a splint. (6) No antiseptics—only dry dressing or external Eusol, or Flavine. (7) Plaster 3 or 4 days later, unpadded with no windows if possible, and a dressing of 20% glucose (in 1 in 100 Dettol) if any discharge is anticipated. (8) Re-application of plaster every 6 weeks for 3 to 6 months. Make for ankylosis of all joints except the elbow.

In the spine, ankylosis can very seldom be satisfactorily obtained by any apparatus which is worn while walking. There are really only two methods—first, the application of a good plaster in hyperextension, and while the patient is lying on this another “plaster bed” can be applied to the front of his body, including a rest for the head. Lying in these alternately, real rest for the spine can be obtained.

The second method is that of bone grafting. After a plaster bed has been made, two good strong tibial grafts, 6 inches or so, must be cut; and

one applied each side of the spinous processes. Operation—Paraldehyde with Chloroform if and as required: (1) Prepare the spinous processes and laminae to receive the grafts and pack the wound. (2) Cut the grafts and sew up the leg wound. (3) Apply the grafts using no fixation except the catgut to keep them in place.

The patient lies on the plaster bed face upwards for 1 day, face downwards for 4 weeks, upwards for 4 weeks, and is then allowed to sit up with the back plaster on, but not to walk, for another 4 weeks.

Finally, a word about tuberculous ulcers, which are not included in the present statistics but which must be mentioned for the sake of completeness. These may be of any size, from the smallest up to a foot across. They are usually superficial, and, therefore, permit of the following treatments. If attempts to cure with exposure to sunlight, or to ultraviolet rays are not successful, there is only one thing to do—the removal of *all* tuberculous tissue—and there are two ways to do it.

1. The edge of the ulcer is the active part, and must be excised with an incision keeping in the normal skin, so as to cut away *all* abnormal tissue. The ulcer is then excised complete, and except in the very largest and most chronic cases, this can be done without disturbing the deep fascia, muscles, or essential nerves and arteries. The ulcer bed is then allowed to granulate, and eventually Thiersch's grafts are applied.

CASE. Man, aged 25. Ulcer 14×8 inches on the external aspect of thigh for 10 years. Excised completely.

Granulating well in 2 weeks. Grafted in 3 stages from the other thigh and abdominal wall. Went home completely healed, 5 weeks after admission.

2. The ulcer is scraped vigorously with a spoon in such a way as to remove all tuberculous tissue, and finely powdered Potass Permanganate is placed thickly upon it. Bleeding stops instantly. A dry dressing is applied and kept there for 3 or 4 days. At the end of that time, the dressing is removed and reveals a healthy-looking, granulating ulcer ready very shortly for skin grafting. This method is very useful for lupus of the nose etc., and is only unsuccessful if tubercular tissue is left behind, in which case it must be repeated.

Finally, we may sum up the treatment of surgical tuberculosis using 4 words all beginning with R. First, we have *Rest*, which must be absolute if possible, such as is given to a joint by bone grafting or by a really good plaster. Next, we have *Rays*. The rays of the sun do a lot of good, and Ultra Violet Rays still more, as they can be properly controlled. On the whole, “general” doses are preferable to “local”, and more effective. If tuberculous tissue will not react to these two, there is still a method at our disposal which must not be unduly postponed—*Removal*. And this must be real removal. No more scraping or messing about, but a radical removal of the diseased parts, followed in its turn by rest, and possibly Rays again. All tuberculous cases *must* be under continual supervision until they are finally cured. There is no disease in which this is so important as tuberculosis; nature cures many ills, but *not* tubercle unless aided by the surgeon (with his enforcement of rest to the diseased part, and his well-timed removal of infected tissues if rest fails, if rest cannot be adequately obtained, or if there are more than one tubercular focus). Rest, Rays, Removal—and the fourth “R” is that all treatment of tuberculosis must be Radical.

South Travancore Medical Mission,
Neyyoor.

Chrysotherapy in the Treatment of Pulmonary Tuberculosis*

BY

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Ever since the bacterial origin of tuberculosis has been proved, attempts have been made to find out a pharmacological substance possessing a selective affinity for the cytoplasm of the Tubercle bacilli without harming the cells of the host.

The chief hindrances in finding out such a substance have been due to the covering of Tubercle bacilli with a fatty capsule, which makes it impenmeable to the action of bactericide, the avascular structure of the tubercle, and complex etiology of the disease.

It was Koch, who, in 1890, observed that a solution of cyanide of Gold and Potassium, even as weak as 1 in 1,000,000 would prevent the growth of Tubercle bacilli in culture, but as it did not hinder the growth of the organism in the presence of blood serum, it could not be used in the treatment of tuberculosis. So indefatigable researches were carried out by various workers in this line to find out some suitable Gold compound which could be useful in the treatment of tuberculosis in human beings, and it was Mohnmark of Copenhagen who, in 1924, found out such a compound ((Gold-Sodium Thioculphate) and named it Sanocrysin.

Since the introduction of this compound for the treatment of pulmonary tuberculosis, quite a large number of Gold compounds have been prepared and put up in the market by various firms of Europe and other countries and extensive and varied literature is available on the subject.

Many workers from England and India consider Gold compounds to be of a definite clinical value in suitable cases, while others, notably Americans and French clinicians, have reported strongly on the therapeutic inefficiency of Gold salts and the danger involved in their usage.

Opinions differ even on the merits of different Gold salts. For example, FARMER, MONROE, FARMER and some others prefer Sanocrysin while SACKEN and SUGEMAN of Bombay Sanatorium report favourably on Solganal-B-Oleum. KORRISON believes in the superior merit of oily preparations as compared with watery solutions.

Although it has not been proved that Gold salts have a definite anti-tuberculosis value, the results have been found satisfactory and encouraging in suitable cases; and so, Gold salts have a place in the treatment of tuberculosis and are worth a trial. During the last seven years, I have treated 98 suitable cases with different Gold compounds and the results in about 70% of cases have been quite satisfactory.

There are a number of Gold compounds in the market, but broadly speaking, these can be divided into three groups:—(1) Those soluble in water, (2) Those soluble in oil, (3) Those insoluble in oil.

Although I have only used four or five preparations in the treatment of my cases, there is a large number of such preparations, and the commonly known compounds are as follow.

* Specially contributed to "The Antiseptic"

1. *Sanocrysin* (Sodium and Gold Thioculphate):—It is the first Gold compound which was used in the treatment of pulmonary tuberculosis and it contains 37% of Gold. It is a crystalline substance occurring in fine, white needles which are readily soluble in water. When dry and stored in a cool place, it is highly stable but decomposes in the presence of moisture, when it becomes yellowish in colour and unfit for use. It is administered intravenously, dissolved in distilled water or even in Calcium Gluconate solution and is available in 0.01, 0.025, 0.05, 0.1, 0.15, 0.20, 0.25, 0.30, 0.35, 0.4, 0.5, 0.75, and 1.0 gramme doses. I have used it in 23 cases and the results have been on the whole quite satisfactory. Great care should be taken about the initial dose as it causes a good deal of reaction, even if slight carelessness is made in seeing to the proper dosage.

2. *Oleo Sanocrysin*:—It is the same preparation as number one, but it is suspended in oil and is available in 1%, 5% and 10% suspension in ampoules of 2cc., 5cc. and 10cc. Before using this preparation, the ampoule must be thoroughly shaken to suspend the precipitate.

3. *Solganal and Solganal-B* are described as disodium derivative of P-sulpho methyl amino-o-toluenesulphonic acid containing 36.5% of Gold. The injections are given intramuscularly.

4. *Solganal-B-Oleum* (SACKEN):—It is an oily suspension of Aurothio-glucose and contains 50% of Gold. Being an oily solution, it is absorbed slowly and thus avoids toxic bye effects. It is injected intramuscularly and is available as—

Dose 1—0.01 gramme in 1.5 cc of oil.	Dose 4—0.2 gramme in 2 cc of oil.
Dose 2—0.05 gramme in 1.5 cc of oil.	Dose 5—0.5 gramme in 3 cc of oil.
Dose 3—0.1 gramme in 2 cc of oil.	Dose 6—0.8 gramme in 4 cc of oil.

I have treated 50 cases with this preparation. Reaction is not severe and the results have been fairly good, except in few cases in which it did not suit the patient causing severe untoward reactions and, in these four cases, this line of treatment was given up after two or three injections. I did not use a 5th or 6th dose of this preparation in any of my cases.

5. *Crystalline* (MAX AND BAKER):—It is a crystalline substance containing 37% of Gold. It occurs in white, silky needles and is easily soluble in water forming a colourless neutral solution. It is administered intravenously, dissolved in distilled water, but I always dissolve it in Calcium Gluconate solution and give Gold and Calcium in one injection. I have tried it in 15 cases and the results have been fairly good. It does not cause much reaction and is available in single ampoules of 0.05, 0.10, 0.15, 0.20, 0.25, 0.50, 0.75, and one gramme and in boxes of twelve.

6. *Myamisin*—Sodium Aurothiomalate (MAX AND BAKER). It is a pale yellow powder easily soluble in water and contains 50% of Gold. It is administered intramuscularly and is supplied in aqueous solution and oily suspension in ampoules of 0.01, 0.05, 0.1, 0.2, 0.3, 0.5, and 0.75 grammes. The injection should be given very slowly.

7. *Lopion*—Sodium Aurothiosulphate (BAKERS). It contains 43% of Gold and is a highly detoxicated preparation. It is administered intravenously and is available in ampoules of 0.01, 0.025, 0.05, and 0.075 grammes. The injection should be given very slowly.

8. *Aurophos*—Sodium p-N-dimethylamino-o-tolylphosphonate. It contains 25% of Gold.

9. *Krysolgan*-Sodium p-amino-o-auriomercaptobenzene Carboxylate (SCHERING). It is a fine golden yellow substance which is readily soluble in water. The drug is dissolved in 1/2 to 1 cc. of distilled water and is administered intravenously. The injection should be given very very slowly, drop by drop. It is available in 0.0015, 0.0075, 0.015, 0.075, 0.15, 0.375, 0.75, and 1.5 grs.

10. *Triphal*-Sodium 2-aurothiobenzemina-zole-4-carboxylate. It contains 47% of Gold.

11. *Neosolganal*-said to be a Gold keratinate containing 14% of Gold.

12. *Lipochrysol*-double campho-dithiocarbonate of Gold and Sodium (RAPTAKOS). It is an oily preparation containing 33% of Gold. It is supplied in ampoules of 1 cc. and 2 cc. and each cc. of this preparation is said to contain 1/2 a grain of pure Gold. It is administered intramuscularly.

13. *Prominal* (BAYER):—It is a Gold preparation newly introduced in the market. It is administered intramuscularly and is used in the treatment of pulmonary tuberculosis and rheumatoid arthritis.

14. *Aurogonal-B-Oleosum-Oro-theo-glucose Auro-mercapto-benzol* (ORIENTAL CHEMICAL WORKS LTD.). It is an organic compound of Gold and has only been recently put up in the market. It is available in ampoules of 1.5 cc. 2 cc. and 3 cc. containing from 0.01, 0.025, 0.05, 0.1, 0.15, 0.20, 0.25, and 0.3 gramme. It is administered intramuscularly, and the ampoule should be shaken in a horizontal position before use to ensure even distribution of the medicine and if it does not mix properly even on shaking, the ampoule should be put in warm water for a few minutes. I have given this preparation a trial on one of my patients but I found it very difficult to dissolve its Aurogonal part into its oleosum part.

15. *Auroform*-Auro Sodium Thiosulphate (CONTINENTAL DRUG CO.):—said to contain 37% of Gold and is administered intravenously.

16. *A. K. B.* (RAPTAKOS). It is a polyvalent compound of sterols of Gold, Copper, Molybdenum, and Cerium and sterols irradiated soluble in oils. It is supplied in 2cc and 5cc ampoules and is administered intramuscularly. It is not stated as to how much Gold it contains. I have used it in ten cases. It did not cause any reaction but reduced the amount of sputum in nearly all of them.

17. *Calgosal* (INDIAN HEALTH AND LAB. LTD.):—It is a blood-red, watery solution stated to be a Gold complex with a trace of Copper and is said to contain 2% of Gold. It is available in 2, 3, 4, and 5cc. ampoules.

Pharmacological Action of Gold.—Gold, like all other heavy metals, is a protoplasmic poison and its soluble salts possess bactericidal properties. Gold salts are not much absorbed from the gastro-intestinal tract, but are well absorbed when administered by injection. Elimination takes place largely during the first two days but continues for several days. It occurs through the kidneys, large bowel, bronchial mucosa, skin, salivary glands and lachrymal glands, and out of all these routes, 90% is excreted through the kidneys and 10% through all the other routes combined together. The toxic phenomenon occurs when any of the system through which it is excreted is not healthy or the dose administered has been a large one. The general toxic effects resemble those of arsenic and both acute and chronic forms of poisoning are known.

Mode of Action of Gold Salts.—The mode of action of Gold salts in the treatment of pulmonary tuberculosis is obscure. There are two theories, the first one is that Gold actually killed the Tubercle bacilli and liberated endotoxin, and this has not been supported by any of the recent workers and the unpleasant symptoms which were attributed to the flooding of the system with endotoxin were really due to the poisonous effect of the heavy metal on the system during its excretion. The second and the modern theory is that the Gold salts are stored in the cells of the reticulo-endothelial system and stimulate phagocytosis and other defence mechanism and also favour the formation of connective tissue. As such, these salts cannot be regarded as chemotherapeutic agents and their action can be considered as that of catylitic stimulation of the reticulo endothelial system or general defence mechanism.

Mode of Administration.—As Gold salts are not absorbed from the gastro-intestinal tract in colloidal form, but only in small traces when administered as soluble compound, these are administered by intra-muscular or intravenous injections depending on the particular preparation used. These compounds are never injected subcutaneously.

Selection of Cases and Dosage.—The whole success or failure of Chrysotherapy depends on the selection of cases and proper dosage. The following types of cases are found to be most suitable for this line of treatment.

1. Early cases of infection with recent exudative lesion are most suitable and give the best results.

2. It is also indicated in bilateral cases where there is a small degree of involvement in the contralateral or good lung. I have seen that in such cases it has a distinct value in turning the balance from a bad to a favourable result when used in conjunction with collapse therapy.

3. In chronic fibrotic cases it is of no use except when used to check the acute exacerbation that is in acute progression of chronic cases.

4. Another type of cases in which Gold compound can be used is where there is continuous regular pyrexia.

Gold compounds can be used equally successfully in delicate patients and women but correspondingly small doses are to be given. Children on the other hand tolerate it well.

As regards dosage, it is only by individual care and attention to the details of the dosage, and constant clinical supervision that the best results are obtained, and these are always more favourable with reasonably small than with heroic doses. It has also been observed that reactions to Gold are commoner in patients with achlorhydria or hypochlorhydria; and so acid should be given to such cases, and intake of vitamins should also be kept adequate.

The dose and interval should be modified according to (a) general condition of the patient (b) weight of the patient (c) extent and type of the disease (d) and reaction of the patient towards the treatment. When a suitable dose has been found for a particular case the treatment should be carefully pushed in gradually progressive doses.

A mild reaction always occurs after an injection of Gold salt, but severe reaction only occurs when the dose is a big one or the patient has got an idiosyncrasy or sensitiveness to Gold salts. In such cases, the dose should be

decreased and if the reaction is still severe, this mode of treatment should be given up.

In all cases—(a) proper attention should be paid to the hygiene of the teeth and mouth and appropriate treatment carried out when necessary.

((b)) The patient should be kept in bed for two or three days after an injection and his four hourly temperature noted down, and further injection should not be given until any eventual rise in temperature caused by the last injection has disappeared.

((c)) A careful examination of the urine should be made before starting Gold therapy and after each injection.

((d)) The dose should not be increased unless the patient has shown good tolerance to the dose last administered.

In cases in which the Gold treatment suits the patient, reduction of temperature, a regular gain in weight and healthy appetite frequently occur.

Contraindications.—Chrysotherapy is contraindicated in:—1. Cases manifesting high temperature or marked cachexia and emaciation regardless of the degree of tubercular involvement.

2. In advanced tubercular disease showing marked toxic symptoms.

3. Cases suffering from pronounced intestinal tuberculosis.

4. Cases having organic disease either of the heart or of kidney or liver (especially fatty liver.)

5. Cases with recurrent attacks of hæmoptysis and those suffering from agranulocytosis, purpura, etc.

6. Cases with severe diabetes.

7. Cases having had a previous attack of exfoliative dermatitis.

8. And lastly, it should be used with extra caution in pregnancy and in cases with high blood pressure. Injections should not be given to women during or in the days immediately preceding the menstrual period.

Treatment with Gold Salts Should be Discontinued.—1. If there occurs very severe reaction even with the first small dose.

2. If the rise in temperature goes more than two degrees above the previous level and continues so for more than one week.

3. If a rash appears and lasts for more than three days.

4. If definite albuminuria lasting for more than four days occurs.

5. If diarrhoea starts after two or three injections.

6. If on blood examination there be found a rise of polymorphonuclear count and eosinophilia and diminution of blood platelets.

Complications.—Complications and toxic reactions result from over dosage, idiosyncrasy, or sensitiveness to Gold salts. The chief complications that might occur during chrysotherapy are:—

1. *Cutaneous*.—Skin reactions are described as quite common and many, including pruritus, erythema, macular or pustular eruptions, exfoliative dermatitis, lichen planus, herpes zoster, purpura, erythema nodosum, and urticaria. In my cases, I did not meet with more than five patients who showed skin reactions due to Gold treatment and even in

these cases the reaction was a mild form of erythema or urticaria. On the other hand, Chrysopigmentation or the deposition of Gold pigment in the dermis which is described as rare occurred in one of my cases at Summ. She is the wife of a police constable, to whom I gave Gold injections three years ago but the pigmentation is still present.

2. *Gastro-Intestinal*.—Metallic taste in the mouth, glossitis, gingivitis, and ulceration of the mouth is another common complication which I meet with among my cases but it passes off when proper attention is paid to the hygiene of the mouth. Stomatitis and anal ulceration may also occur in rare cases.

((b)) Loss of appetite and jaundice may occur in some cases having sluggish liver, but it passes off if glucose and fruit juice are freely given during the treatment. Vomiting may take place in some cases immediately after the injection, but it is never of a serious consequence.

((c)) Diarrhoea does not commonly occur, but if it occurs, and persists, the treatment with this drug should be stopped. I had one case in which diarrhoea started after second injection of Solganal-B-Oleosome. I stopped giving him Gold and his diarrhoea got better. Next year, I again started giving him injections of Solganal-B-Oleosome and the diarrhoea again started after the second injection and I had again to stop this drug.

3. *Renal*.—Slight form of albuminuria occurs in a large number of cases but if it persists or the percentage of albumin be large, the treatment should be discontinued.

4. *Febrile Reaction*.—It is common in most of the cases, but it usually subsides after a few hours. A long febrile reaction is generally due to over dose and the next injection should not be given until the effect of the reaction from the previous dose has passed off.

5. Varying degrees of conjunctivitis and phlyctenular ulcer may also occur in a few cases.

Results of Chrysotherapy.—It is extremely difficult to assess the value of chrysotherapy, unless a follow up of the treated cases is kept for a period of at least five years which is very difficult in this country.

For the sake of convenience the results may be divided into immediate and remote ones. Immediate results are in the form of:—

- (1) The effect on the general conditions of the patient.
- (2) Diminution in the amount of the sputum and rendering it free from Tubercle bacilli.
- (3) A retrogression in the clinical signs and tendency towards fibrosis of the lesion.
- (4) Favourable effects on the radiographic appearance of the skiagram and,
- (5) Improvement in blood sedimentation rate seen in all good cases.

Regarding the remote results or the permanency of the good immediate results of Gold salts, we are up against a great difficulty, as the results of treatment are not always uniform and as much depends—

((a)) On the defensive forces of the body. In one case, they may take

advantage of the check in the disease caused by a Gold compound and make a permanent arrest of the disease while in others they may not.

(b) and on the status and home condition of the patient to which he returns and these conditions are so vastly different in each case that comparisons become almost impossible.

It can be said with confidence that the remote results are only encouraging in those cases which lose their Tubercle bacilli and become clinically arrested after treatment.

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Malaria and the Mosquito*

With Particular Reference to Eradication of the Pest
On Economic and Popular Lines

BY

F. D. BANA, M.B., M.R.C.S., D.P.H., D.T.M.&H., J.P.,
Administrative Medical Officer, Bombay Port Trust.

MALARIA in man is a protean disease killing many and wounding many more.† Col. COTTER, I.M.S., the present Sanitary Commissioner with the Government of India, says, "Malaria continues to be the most important public health problem in India. Although absence of medical certification makes it impossible to secure correct figures regarding the incidence of the disease, all the available evidence seems to suggest that malaria is responsible for more deaths than any other infectious disease prevalent in the country‡". It is not only a tropical disease but prevails in the sub-tropical and even temperate regions as in some parts of Europe and America. It is carried by the mosquito, a seemingly flimsy defenceless insect composed of cellulose, air-tubes and connective tissues. Biologically, the species of Anophelene which carries the parasites of malaria and transmits the disease to human beings is a Diptera and belongs to the classification of insects, intermediate between the molluscs and the vertebrates. It has the distinction of carrying its respiratory organ in innumerable breathing tubes distributed to all parts of the body unlike separate breathing organs as the lungs and not having an endoskeleton as in vertebrates. The former is his triumph as it is difficult to catch on the wings and also his

* Specially contributed to 'The Antiseptic'.

† Latest Annual Reports of the Indian Sanitary Commissioners reveal that out of a total approximate population of between 38-39 crores, about 15 lakhs die yearly from malaria. These are mere recorded figures. It is presumed that about ten times this number suffer from loss of working capacity or disability such as anæmia and other complications. As most of the figures are statistics entered by laymen such as the village headman or chowkidar, the actual figures must be very high indeed. One can thus imagine what potential loss in money and vitality to the Nation the incidence of malaria costs to India.

‡ Page 2, Annual Report of the Public Health Commissioner with the Government of India, 1939, fully bears out the above remark.



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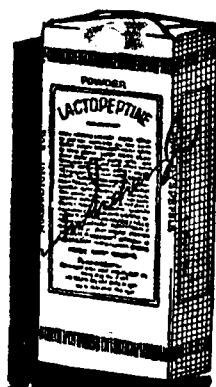
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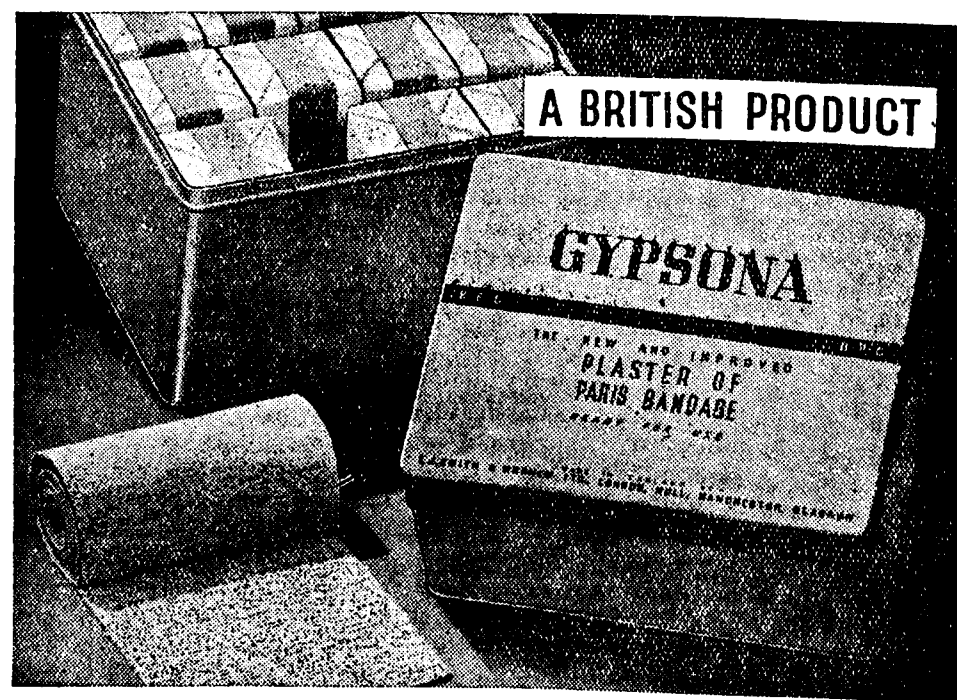
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weak point is vulnerability in as much as it is through this that one can easily suffocate and kill him, both in the larval and the adult stages. He has an exoskeleton of cellulose to make his body rigid which helps him to fly and get away from the grasp of man.

It breeds in collections of water, feeding on matter which is made up of cellulose such as fresh or decaying vegetables and organic matter found in weeds, leaves, branches and animal substances. All these biological factors come into prominence and have to be counteracted in the war we have to wage against it for carrying in his body the "Million murdering Death" as pointed out by the late SIR RONALD ROSS.

These points which are not stressed in text books, I am bringing to your notice as the growth of the insect from the egg to the Imago or adult stage depends in different species on the prevailing conditions of moisture, heat and on collections of water having clean or decaying matter. Some are clean feeders like the Anophelene and breed in the clear water of streams, ponds, cisterns, fountains etc. Others are dirty feeders like the Culex which breed tremendously in drains, cesspits, sewages and septic tanks requiring a rich mixture of decaying organic matter. Thus, one species of Culex in particular is a big fellow and is so vigorous and predacious that the big larva eats the small one and has a very vicious sting. But like the barking dog who seldom bites, the bite of Culex is not so injurious to man as that of the small dapple Anophelene, which works havoc, imparting malaria with its hundred horrors.

Now, let us see what species of mosquito carry diseases to man and how the vicious circle can be broken.

Anophelene :—Malaria ; Tertian ; Quartan and Malignant.

Culex :—Filaria ; Dengue.

Stegomyia :—Yellow Fever virus.

In malaria, the cycle is well known to you, how from an infected person, blood is sucked up by the adult Anophelene, the parasitic zygote develops in its stomach, breaks up and passes via the salivary glands into fresh human beings as merozoite. To eradicate malaria in man, he must be treated by Quinine or by Atebrin and Plasmochin. This may be done by the citizen if he can afford it in his private capacity or by Government and Local Bodies supplying Quinine free or with grants-in-aid, to the public especially in rural areas where people are infected with malaria, but the community as a whole can ill-afford this expense. Secondly, the vicious circle of development caused by the mosquito which transmits malaria and breeds in water collected in streams, ponds, cisterns, roof gutters etc., must not be allowed to do so. It is up to the citizen to see and also a duty of the local authority or Government to prevent this by adequate drainage and good sanitary supervision and by destroying the larval stage of the species. That part of the cycle of development occurring in water is the most practical and vulnerable part. This can be broken up or easily effected en masse, with maximum benefit at minimum cost. It involves 3 factors, viz. (1) Money, (2) Labour, and (3) Co-operation. As a rule, the value of these principles in killing and treating the breeding of mosquito larvae is hardly considered adequate or appreciated in proportion to the eradication and relief of the disease and consequent saving of life, suffering and economic loss to the population ; for results in cash values are not apparent immediately to the laymen. But, the benefit of these in the long run is unquestionable. To give you an instance, the new city of Delhi is tried so

as to free it of the scourge of malaria which has decimated the city from time to time in former days, so that the site of the capital has had to be shifted several times. A grant of nearly 40 lakhs of rupees is being made by the Government of India, spread over a number of years, for this scheme of anti-malaria measures and adopted at the instance of the Director of Malaria Survey of India, COL. COVELL, I.M.S., who, with the co-operation of the local authorities, has already started the work.

This object can only be achieved where enlightenment can be brought to bear upon this vital branch of Public Health by the popular representatives voting more money for adequate services and seeing that they are properly utilised to advantage when got. In big cities and Presidency towns like Bombay, such facilities exist and are provided by the Sanitary and Malaria staffs of the Municipality or Local Bodies, and it is the duty of the enlightened citizen to help in these matters to deal effectively with the nuisance, *i.e.* to deal with the pest wherever breeding occurs or is found in the light of experience gained. Chief amongst these measures in a city like Bombay, with independent water and carriage systems, should be to close all unnecessary wells and to make them mosquito-proof by providing suitable pumps if water is needed. All cisterns for storage of water must be made mosquito-proof and all stray collections of water drained off or treated with larvicidal agents. Open, useless ponds and pools should be drained and those that cannot be so treated must be examined periodically and treated by larvicides or stocked with *Gambusia* fish which thrives well in India. Above all, full co-operation and encouragement be given to eradicate all other possible sources of infestation. In Bombay, one source of breeding of *Anopheles*, so far not dealt with, was the hidden breeding in water of roof gutters in the monsoon. I am glad to say that with the co-operation of big bodies like the Port Trust, Municipality and Mill Owners, this problem is being rapidly tackled now. In towns, besides the *Anopheles*, breeding of *Culex* which carries dengue and filaria occurs extensively in dirty waters, especially of gutters, storm water entrances, cesspits and stagnant waters. This can, to a certain extent, be attended by covering their entrances or openings by suitable means and treating them with strong larvicides periodically on repeated examinations.

Now, what about the rural areas or villages? It is supposed that there is no bundobast at all. The village authority, if it has power, has no money to spend over such matters as malaria prevention. At most, if the village is being ravaged, the inhabitants have to shift for themselves. May be a little Quinine is sold at the village Post Office which the people are reluctant to buy or cannot afford to. Here, most of the breeding occurs in wells and tanks or collections of water along the neighbouring stream, river or canal. This the village authority alone cannot tackle. It requires expert advice and help from the local or Provincial authority. If the incidence of malaria or of filaria rises, this surely invites attention of the Public Health authority who on due report sends a representative to look into the affair and the Director of Public Health deals with it. Apart from this, what about a chronic condition of prevalence of this pest, the annoyance and ill health of the populace, and loss of vitality and money value to the agriculturist and labour due to sickness and disability. This condition could only be remedied if 3 definite modes of actions are undertaken. (1) Education of the young and old that mosquitoes breed in water and carry malaria and other preventable diseases. Therefore, all potential sources for storing water or its remaining stagnant for eight days, *i.e.* the

period of the cycle of development of the pest, must be cut short by emptying out water at least once a week. This can only be effected for small and temporary collections of water in or near the house in water jars, utensils, barrels and empties of all sorts containing water. (2) The importance of the word "Slope" and "Drainage" for all constructional work must be impressed not only in the lay-mind but the Engineer, the Contractor and the Maistry building a house, a drain or railway. Most of the epidemics of malaria in India and elsewhere were started by the neglect of this main principle whenever a new project was started. Thus, it is well known to you that the first success of Suez Canal, the Panama Canal and the building of the premier dock in India, Alexandra dock in Bombay, was marred by intense epidemics of malaria and yellow fever. Workers died like flies in the Suez. Panama and Bombay got an unprecedented taste of malaria, all of which incidents are of historical record. (3) A systematic anti-mosquito campaign. This can be undertaken in a rudimentary way tackling the nearest source of breeding such as the village cesspit which could and should be covered up, so as not to let the mosquito develop in it by depositing the eggs. In towns, drains, cesspits and septic tanks with loose and irregular covers and open empty tins etc. cause great annoyance from breeding to the immediate neighbourhood. All empties must be removed or placed upside down in the monsoon if needed. If the cesspit cannot be cleared once a week, it can be sealed up in such a way that the mosquito cannot have access to it. Temporary collections of water as in the monsoon should be drained away or prevented by filling in depressions; larger tanks, pools, canals and sides of the river cannot be dealt with effectively in the absence of help from the authorities and are costly affairs. But there is a way to deal with minor points at first and then tackle major ones. And that is to start on a small voluntary scale by interesting young boys and girls who can form small Mosquito Clubs or Associations, thus improving the sanitary condition of the village or district. This can easily be done under the leadership of the school master or headman who can employ their spare time to go round just with a small net and a tin plate to find out the wriggling larva which once shown to the young mind would interest them. It will serve as a hobby and, instead of their wasting, their leisure time will be profitably employed in getting rid of the nuisance to themselves and to others. They can examine their own pots, pans and barrels and empty them out if found breeding, or draw the attention of the neighbours or the authorities to stop this breeding in other places which do not belong to them. Thus, a small co-operative circle can do a lot to check much unnecessary suffering and avoidable annoyance to the community at practically nil or very little cost. It is for this that I appeal to those who are popularly and sanitarily minded to get this going in their own localities or districts, either rural or urban, if they are so inclined, keeping in mind that one helps not only his own interest but of local authority supplementing its work.* One will thus gain the blessings of the poor and innocent people who, suffering through lack of knowledge and effort to ameliorate the local conditions, often blame too much their own proverbial 'Kismet'.

* The Bombay Municipality has provision for such work for which help can be given when doing so.

Cases and Comments

Mixed Infection of Cholera and Typhoid

BY

AMAR NATH GULATI, L.M.P. (C.P.); L.S.M.F. (BENGAL),

Sargodha.

ON the 4th June, 1941, I was called upon at 2 P.M. to attend an urgent case who was sweating and passing watery motions with vomiting. On examination, I found clinically a case of cholera. He was a Hindu, male, aged 20 years, unmarried, getting severe cramps in legs, profusely perspiring, with cold and clammy skin, eyes sunken, voice practically inaudible, eye balls rolling with cornea covered by dropped upper lid, reflex of cornea slightly present. Pulse was not perceptible at the wrist. Respiratory rate was slightly increased and was of sighing character. On auscultation, the heart sounds were heard very feeble. The rate of heart beats was 130 per minute.

History was elicited from his relative and the conclusion reached that patient took infected sweets from the neighbouring town and developed symptoms of cholera 18 hours after the exposure to infection (after taking sweets). Patient duly passed through the stages of invasion and evacuation and was then in the stage of collapse.

Treatment was started as follows:—(1) Normal Saline with Adrenaline Hydrochloride .5 c.c. per pint, intravenously. The amount given was 2 pints. One pint of the normal Saline was given subcutaneously under both the axillæ. (2) 'Coramina' and 'Musk-in-ether' were given hypodermically to save further sweating and collapse. (3) Hot water bags were put all round the body. (4) Liberal supply of water was given by mouth which was previously abandoned by the attendants for fear of vomiting. (5) Foot of the bed was raised.

The whole process took about 1 hour. After this, patient vomited again and passed a watery motion in bed: (motion and vomit were soon covered with lime. Therefore, no time was available to examine the specimen of motion for vibrio-cholera).

'Essential-oil' mixture by mouth was started. Again, Saline treatment was adopted starting from one pint of 'Alkaline Saline' I. V. followed by Normal Saline 1 pint I. V. Patient gradually showed signs of improvement. Pulse became perceptible at the wrist and its rate was 140 per minute but very feeble. Patient came to senses and was always whispering for water which was unhesitatingly given. Uptil 12 at night, patient did not vomit again, passed no motion, pulse was the same as in the evening and no further improvement was seen.

On 5th June, 1941, at about 2 A.M., patient again started profuse sweating. Stimulant dose and an injection of "Musk-and-Camphor-in-Aether" was given. Hot water bottles were kept to keep the patient warm. Constantly, the palms and soles were being rubbed to increase the body temperature. Passed no urine. At about 6 A.M., rectal temperature was taken and found to be 103° F. Pulse perceptible, patient was in senses, voice slightly improved, 3 motions passed (semi-solid), since the previous evening, no urine and no vomiting: 20 ozs. of *Alkaline-Saline* was given I.V. and was soon followed by 20 ozs. of *Hypertonic Saline*. Patient showed a little restlessness, shivering and catching in the chest. 'Atropine

injections' subsided the whole trouble. Extremities became hot, pulse sufficiently improved, general condition—"full possession of senses." Rectal temperature 103° F. He could speak now and there were no motions and vomiting. At about 1 P.M. again, Hypertonic Saline was given, patient gradually entered into the stage of reaction. Loins were constantly kept warm to prevent the patient from the post-choleraic kidney complication. Bladder was filled with Alkaline lotion to reflexly stimulate the bladder. About two hours later, patient passed urine in a small quantity. In the evening, patient again became restless. Again, 1 pint of Hypertonic Saline was given I.V. and everything was O.K. Along with this, stimulant mixture was given by mouth: So far, the patient passed urine 3 times but no motion, no vomiting. Pulse rate 120 per minute. Temperature by mouth 99.0 patient did not complain of any more cramps in legs.

6th June, 1941. Patient keeping temperature 100.5°, pulse rate 120 per minute. Passed 5 semi-solid motions along with large quantity of urine. One pint more of Hypertonic Saline was given and he recovered from the attack. He started running temperature which was gradually increasing in the evening everyday. Patient did not complain of any other trouble and no "post-choleraic complication" was seen whatever except little pain in the lumbar region which subsided by the local application of fomentation and massage. Temperature remained between 101° and 103° in the morning and evening respectively from the 13th till 18th.

"Mumarkhi Pearls." Sudamina (Said to be *Pearls of Typhoid*) appeared over the whole body in abundance. Other clinical findings such as tongue and pulse rate were in favour of enteric fever. Temperature remained for about 28 days after which the patient started recovery and was in convalescence after that. Patient left the treatment and was walking about not caring for the warnings against the danger of relapses. He got continuous fever again *i.e. relapse* after a week and was bed-ridden for another 18 days showing all the symptoms of typhoid *i.e.*: (a) Dry coated tongue, red at the margins and tip. (b) Excessive headache, extreme thirst, pulse rate comparatively slower. (c) Pains and aches in the body.

Alkaline Saline constituted:—Soda Bicarbon gr. 160; Nacl gr. 90; Distilled water 1 pint.

Hypertonic Saline constituted:—Hypertonic Saline Tablet (P. D. & Co.)

Diet of the patient throughout the attack was (a) Barley water and simple boiled water; (b) Glucose water; (c) Albumin water; Sago and Dahi.

Interesting Points.—Mixed infection of cholera and enteric fever seen in one case; though it is not the least surprising regarding the source of infection—because, the "*common-house-fly*" is the infective agent for both the infections—yet, such cases are, however, rarely seen in epidemics.

(b) After the stage of recovery, the pyrexia was presumed to be one of the post-choleraic complications, but was excluded with the final diagnosis of enteric fever.

(c) Diagnosis was bacteriologically confirmed by the M. O. H. to whom the report was sent about this infective case.

Reference:—Antiseptic—March, 1941.

Block No. 11,
Sargodha,

Treatment of Nephritis with Sulfanilamide

BY

K. KUPPUSWAMI, M.B.B.S.,

Masulipatam.

I. Child, aged 2 years. History of passing scanty, bloody urine generalised anasarka with ascites and cough.

I saw this patient 8 days after getting these symptoms. The child was having cough and the voice was very hoarse. The child was very restless and was having very laboured respiration. When I examined the child, it had not passed urine since 18 hours. Heart was very rapid and the child was slightly toxic. It had no interest in the surroundings.

I examined the throat and there was acute tonsillitis. I straight away gave the child 5 cc. Sulphanilamide intramuscularly, taking smear of the throat. It was negative for diphtheria and there was gram+cocci in chains and in groups and also Pneumococcus. To my surprise, next day the child was more or less alright. The child passed larger quantities of urine. Oedema gradually disappeared within 48 hours. Child regained consciousness and began to take interest in the surroundings. Heart came to its normal speed. In short, the child came to its normal condition within a week.

II. A boy, aged 14 years, came to me with puffiness of the face. Oedema of eye lids. History of passing scanty urine, red in colour and heaviness in the chest. The patient came to me one week after he noticed the symptoms.

I made a routine examination and this is my report. A fairly nourished young boy. Puffiness of the face and oedema of eye lids present. Slight oedema of both legs present. Scabies which was secondarily infected present on both knees and both wrists, not anaemic. No P.A. present. Tongue coated. Temperature 100°F.

Circulatory System:—Pulse slightly rapid, 98 per minute—regular, good volume and tension. Heart boundaries:—Left border, nipple line, accentuation of aortic and pulmonary 2nd sound. Suspicion of a systolic murmur in the mitral area. Blood pressure is 170/120.

Respiratory System:—Normal.

Alimentary System:—Slight distension of the abdomen. Spleen and liver not palpable. No free fluid present in abdominal cavity.

Examination of Urine.—Specific Gravity. 1020. Acidic. No sugar loaded with albumin.

Microscopic Examination.—R.B.C. Casts. R.B.C. W.B.C. few granular casts.

Blood.—Leucocytosis. A diagnosis of acute nephritis was made and the orthodox treatment started.

On the 5th day after I started the treatment, the patient one night suddenly developed heart failure. Both the lungs were congested and the patient was gasping for breath. Heart was very irregular. Temporary treatment for combating the heart failure was given. Next morning, the patient was slightly better. I gave him 5 cc. of Sulphanilamide intramuscularly. The next day, to my great surprise, I found the patient in an entirely different condition. Oedema had almost disappeared. Patient was passing large quantities of urine and the urine was free from albumin to heat test. Microscopically, R.B.C. were seen. Blood pressure was 140/100. Next day, I gave another injection of Sulphanilamide. Urine was absolutely clear. Blood pressure came down to 130/90. The picture of the patient

was entirely changed. Oedema completely disappeared and he became almost normal. The slight rise in blood pressure shows that there is still slight damage to kidneys which is not completely resolved. The patient is advised to take rest completely. Oral administration of Sulphanilamide was given up to a total of 60 grams, 5 grams every 4 hours, maximum 20 grams a day. I have put him on rice and buttermilk.

He has almost recovered. I am still trying Sulphanilamide on some other conditions. After taking certain statistics, I shall send you the report.

III. On 27-12-40, I was asked to attend a boy, aged 18 years, with puffiness on his face with the history of passing scanty urine and a heaviness in the chest. I made a detailed examination. The patient had 100°F. This boy also had skin infection over both hands and knees and ankles. Moderately nourished individual. Oedema of both eye-lids, puffiness of the face, slight oedema over legs and feet present. No anaemia. No P.A. present. Tongue clean and moist.

Circulatory System:—Heart was slow, 66 per minute. Boundaries normal. Slight accentuation of the aortic 2nd sound. No murmurs heard. B. P. systolic was 130 and diastolic 100. *Respiratory System*:—Few moist sounds heard over the bases on the back. *Alimentary System*:—Normal. *Examination of Urine*:—Loaded with albumin. R. B. C., W. B. C. and casts are seen under microscope. The history was the patient had not passed urine for the last 14 hours and so they came to me. This was one week after they had noticed the symptoms. A small sample of urine collected and the urine was examined. 5cc. Colloid Sulphanilamide was given on the 1st day. After 24 hours, I saw the patient again in the same condition. Another 5cc. of Sulphanilamide was given. After this, the patient passed larger quantities of urine, and patient was feeling very much better. On the 3rd day, another dose was given. The urine was clear. No albumin. No R. B. C. and no casts. No more Sulphanilamide was given, and the B. P. came down to 110/70. This patient responded well to the treatment and showed no damage to the kidneys. The patient was put on rice and buttermilk and was advised to continue a bland diet for some time.

A Case of Ludwig's Angina

BY

G. KRISHNA, P.M.S.,

Civil Hospital, Mirzapore.

ONE, K, 50 years, widow, vegetarian was brought to the hospital with the following complaints:—1. Fever. 2. Swelling in upper part of neck, in front and sides—Duration 2 days. Her past illness was of no significance.

History of Present Illness.—She had severe pyorrhoea alveolaris for the past many years. Gums swollen and tender with purulent discharge for the past 15 days. I noticed fullness below the mandible 4 days back; pain in the submandibular region and slight fever. Some paint, given by a quack, applied. No relief. Swelling increased rapidly within the previous 2 days.

Present Condition was extremely toxic, dehydrated and exhausted with marked constitutional disturbance in the form of high temperature (102°F.), pulse 112 p.m., tension and volume low, regular; respiration 30 p.m., shallow and rapid. Respiratory embarrassment present, more marked in the recumbent posture. Unable to speak and swallow fluids.

Locally, there was foul smell from mouth which opened about 15 degrees; and could not be opened further. In between the upper and lower teeth, the tongue was swollen, protruding and dirty looking and gums too were swollen, dirty red and tender. Many teeth were loose with purulent discharge therefrom. Pharynx could not be inspected due to the intervening swollen and protruding tongue. Submandibular region, swollen was also with swelling ill-defined, extending below upto the thyroid cartilage and laterally to the sides of neck—dusky red, tense, hot and tender. No blister nor fluctuation was detectable.

Systemic Examination.—*Respiratory and Circulatory Systems*:—Nothing abnormal was detected save as noted above. *Digestive System*:—Mouth and pharynx were as noted above. Liver and spleen were not palpable. Abdomen was full. Colon was loaded with faeces. *Nervous System*:—Nothing abnormal was detected.

Pathological Examinations.—(a) *Urine Examination*:—Reaction—Acid; Specific Gravity—1015; Albumin—absent; Sugar—absent; Microscopically, no casts detected, Uric acid crystals present. (b) *Blood Examination*:—1. Total W. B. C. Count:—13,850 per cmm. 2. Differential W. B. C. Count; Polymorphonuclear leucocytes—80%; Large Mononuclear leucocytes—1%; Eosinophile leucocytes—1%; Lymphocytes—18%.

Treatment Adopted.—12-7-41, Afternoon. (At the time of admission). Propped up in bed. Loose teeth extracted. Mouth well syringed with H_2O_2 lotion (1 in 10) and painted with 1 per cent Mercurochrome in water. This to be repeated every 4 hours. Enema Saponis given. Scobulae and much faecal matter passed. Hot boric compress applied externally to neck. This to be repeated every 4 hours. Soluseptasine, 10 cc. injected intravenously very slowly. Glucose-saline with Soda Bicarb (one pint) given per rectum. Tracheotomy things kept ready all the time. 10 p. m. Condition same. Glucose solution 20%, 20 cc. injected intravenously and to be repeated twice daily. Soluseptasine 5 cc. injected intramuscularly. Repeated after 4 hours.

13-7-41. General and local condition same. Temperature 102°F. Pulse 115 p. m. Respiration 30 p. m. Pulse weaker. Same treatment followed. Strychnine hydrochloride gr. 1/60, and Digitalin 1/100 hypodermically stat and 4 hourly. Glucose Saline with Soda Bicarb (one pint) put in per rectum. Soluseptasine 5 cc. I.M. 6 hourly.

14-7-41. General condition slightly better. Tongue swelling less. Respiratory embarrassment less. Swelling neck slightly less. No localisation. One motion—semisolid. Temperature 100°F. Pulse—95 per minute, Respiration. 25 per minute. Nothing could be given by mouth. Same treatment followed.

15-7-41. General condition much better. Tongue retracted in. Foul smell from mouth disappeared. Swelling in neck much less. Marked respiratory relief. Could speak with difficulty. Temperature 99°F. Pulse 90 per minute, respiration 24 per minute. Could drink fluids. Milk with glucose and brandy started in 4 ounce feeds every 4 hours. Rectal Saline discontinued Soluseptasine, 5 cc., I.M. twice daily. Rest of the treatment same.

16-7-41. General condition much improved. Tongue normal size and colour. Could speak well. Swelling in neck reduced 75%. Temperature 98.4°F. Pulse 78 per minute. Respiration 20 per minute. No respiratory embarrassment. Soluseptasine injection stopped and the patient shunted to Proseptasine tablets, 2 tablets every 8 hours. (6 tablets in 24 hours.) Strychnine and Digitalin injections discontinued. Otherwise, treatment the same.

17-7-41. Temperature normal throughout the previous day. No complaints except extreme weakness. The swelling of neck completely reduced. Felt hungry. One motion in the morning. Intravenous glucose and local boric fomentations discontinued. Proseptasine tablets 2 every 12 hours. (4 tablets in 24 hours.)

18-7-41. No complaints. One motion. Temperature 98.4°F. Pulse 75 per minute, volume and tension good. Respiration 20 per minute. All treatment stopped except mouth toilet. Sago added to diet.

19-7-41. No complaints. Cheerful. 20-7-41. Left the Hospital.

Some Noteworthy Features of the Case.—(1) The clinical features and the pethological findings left no doubt as to the correct diagnosis. (2) Comparatively prompt response to the Sulphonamide therapy. (3) Syringing of the mouth proved highly efficacious. (4) Tracheotomy was not necessitated. Had it been done in the beginning at the time of admission, patient's stay in the hospital would have been much prolonged. All the same, close watch was kept on respiratory embarrassment and tracheotomy things were kept in readiness all the time so that necessary operative procedure could be resorted to without a moment's delay, should such interference have become imperative. (5) Complete resolution without localisation.

Status Typhosus

Treated with 25% Glucose—Intra Venous

BY

AMAR NATH GULATI, L.M.P. (C.P.); L.S.M.F. (BENGAL),

Sargodha

S. D., female, aged 16 years, got gradual rise of temperature reaching upto 105°F. on 8th August last. Pulse rate ranging between 96 to 100 per minute, but no variation between the morning and evening temperatures. Respiration counted to be 25 per minute. Bowels moving regularly. No headache, restlessness or thirst was complained of. General condition good. Lying quietly in bed. No other trouble. Treatment started with diaphoretic mixtures, 3 doses daily; but no response was seen. Antipyretics also failed to bring down the temperature. Blood was examined for M. P. & D. count, but got negative results regarding the nature of high fever.

Foreign protein therapy was tried by giving "Omnadine" injections every sixth hour, but it also did not affect the temperature at all. Urine was examined. It also did not throw any light on diagnosis. From clinical appearance of the tongue and pulse rate, it was labelled as a case of enteric fever having entered stratum into "hyperpyrexia stage".

'Ice-cold compress' over the head and 'sponging' started but without any effect. Ten days after this range of temperature i. e., on 18th Aug., temperature at once shot up to 107°F. Hair was shaved. Ice was directly applied over the vertex and nape of the neck.

Patient gradually passed into the stage of coma: later on, muttering developed which resulted in imperceptible speech and unconsciousness: Picking at bed clothes and dreadful appearance of the face greatly frightened the relatives of the house to a great extent. Pulse gradually increased to 150 p. m. Pupils dilated and eye-sight fixed. Vigorous sponging and ice very slowly brought down the temperature to 104°. But as soon as little rest was given to the patient, it at once rose to 107.2°F. Patient started having convulsions, became absolutely senseless. Eyes closed. Passed stools

and urine in bed. Breathing was hurried and pulse became feeble and rapid as if failing. 'Cardiac stimulants' by mouth were given. Injection of Cardiazol Coramine and Camphor-in-Aether given with $\frac{1}{2}$ an hour's interval in each. Room temperature was kept below normal by placing large amounts of ice around the bed of the patient. Ice was directly put over the head and nape of the neck. 'Ice-cold-Saline' with Glucose 'drop method' was started per rectum after an ice-cold Saline enema. Rapid warm sponging was done for ten minutes only to increase the functional activity of the skin to put out some toxins by means of perspiration.

On 19th August:—Glucose 25% 10 cc. (HEALTH RESEARCH INSTITUTE) heated upto body temperature was injected I.V. This was again repeated after 6 hours. This acted like a magic, and all brain symptoms were soothed down. Patient became quiet and restful. Did not show any sign of delirium at all. Digifortis (P. D. & Co.) m. 10 was given every 6 hours by mouth. Ice-cold-rectal-Saline with Glucose continued three times daily. Temperature remained at 105°. Pulse improved and was decidedly better than the previous day. Constant application of ice and warm sponging continued twice daily.

On 20th August:—Temperature remained between 104° and 105°. Patient lying quiet. General condition did not show any definite sign of improvement, though pulse became full and strong, and brain symptoms become less severe. Glucose 25% 10 c.c. and Saline treatment continued. Patient developed stomatitis; whole mouth and pharynx got ulcerated. Patient did not take in any liquid, medicine or food. Nasal feeding with No. 10 Rubber Catheter was adopted and found to be very satisfactory. Milk and fruit juice was given with this method thrice daily. Mouth was carefully attended to. Lungs showed a few wet sounds, due to œdema, which very quickly subsided to local embrocations.

On 21st August:—General condition remained as such. Fever did not go beyond 105°. No restlessness delirium or picking at bed clothes. Glucose 25% was increased to 20. cc every 6 hours; the rest of the treatment was the same as previously adopted.

On the 22nd August:—Fever was found to be down by one degree. Pulse full and strong. Patient lying in restful condition. Respiration resumed normal character. Lungs became clear. General condition a bit better. Nasal feeding continued. Rectal Saline with Glucose reduced after every 12 hours. Glucose I. V. continued (20 cc. every six hours).

On 23rd August. Morning temperature came down to 102° F. At noon 20 cc. Glucose 25% I.V. was given with usual precautions. About 20 minutes later, a good deal of shivering appeared. Fever went up to 106° F. This was rightly considered to be "Glucose-shock". Reaction lasted for about an hour after which fever came down to 104°. Patient showed definite signs of improvement and gradually recovered senses. Expression of the face and general look became brighter. Mental condition resumed normal features.

On 24th August. Temperature touched 101°. Patient started taking fluids by mouth. Pulse was full and bounding. I. V. Glucose was withheld. Nasal feeding stopped. Patient nicely entered into stage of convalescence as temperature was daily found to be half a degree lesser than the previous day. After a fortnight, patient became perfectly all right with no doubt some weakness. Patient totally recovered in a month.

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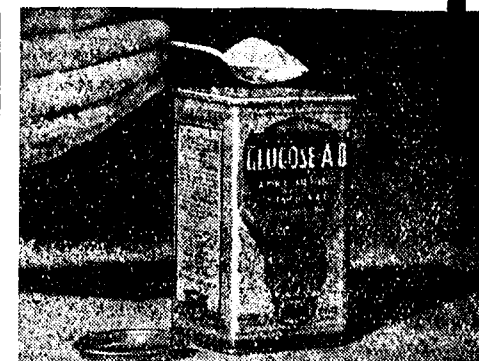


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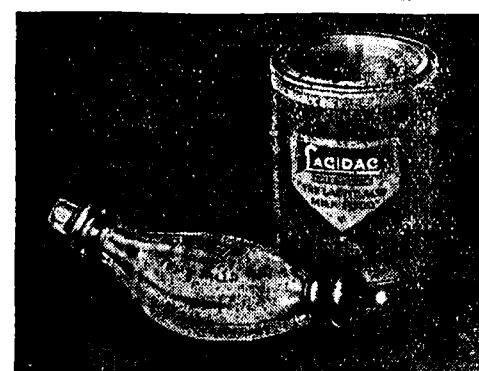
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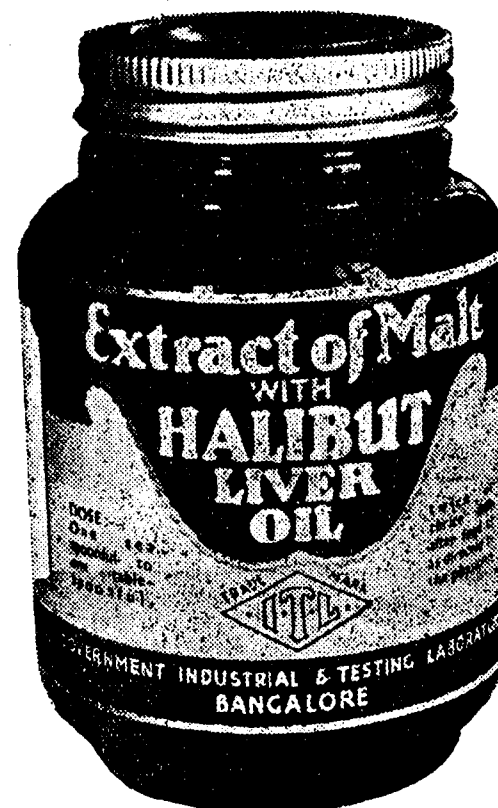
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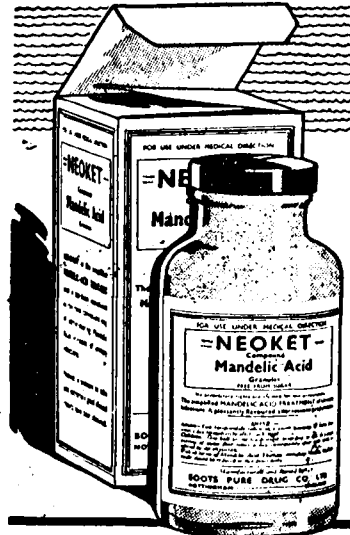
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Editorials

Effort Syndrome

EFFORT syndrome is a diagnosis seldom used in normal times, but very convenient in war-time to classify many cases under this heading. The syndrome is characterised by a certain group of symptoms which unduly limit the subject's capacity for effort and by a number of signs depending upon disturbance of the autonomic nervous system, when such symptoms and signs are not due to any known organic disease. The cardinal symptoms are breathlessness, palpitation, fatigue, left thoracic pain and dizziness; the cardinal signs are those of functional disturbance of the respiratory, vasomotor, and muscular systems. It occurs both among soldiers and civilian population, as a result of the stress and strain of war. In soldiers, the syndrome occurs in military age. In civilian life, however, it occurs among more elderly people. In civilian life, it seems to be more common among women. Although all occupations are represented, those employed in light work show a tendency to exhibit this syndrome. Although no race is immune, the emotional races are more susceptible than the stolid. During last war, there were about 60,000 cases of "effort syndrome" in the British Forces and of these, about 44,000 received pensions. In this war, however, the syndrome is not as much a military problem as in the last. Much work has been done on this disease. Our readers are referred to the classical article of DA COSTA with whose name this syndrome sometimes is called.

Among the symptoms, breathlessness, palpitation, fatigue, sweats, nervousness, dizziness and left thoracic pain are the most frequent, occurring in more than three-quarters of the patients. Minor symptoms, comprising headache, trembling or shakiness, sighs, flushes, throbbing, cramps, paraesthesia and dry mouth occurred in half to three-quarters of them. Less common were syncope and gastro-intestinal symptoms. Insomnia, anorexia and disturbances of the bladder were rare.

With regard to signs, although examination reveals no evidence of organic disease, these cases present signs of disturbance of the autonomic nervous system. They exhibit nervousness, cold blue hands, flushes, rapid pulse rate, raised blood pressure, hyperpnocia, visible sweat of palms, hyperalgesia, and other symptoms; all of them may be found in recognised forms

of psychoneurosis and may be seen in normal persons under the influence of certain emotions. These signs are roughly proportional in number to the severity of the case.

Effort syndrome is often diagnosed as rheumatic carditis, toxic myocarditis, weak heart, cardiac strain, anæmia, V. D. H., thyrotoxicosis, pulmonary tuberculosis, pleurisy, influenza, angina pectoris, or malingering. But, there really should be little excuse for these errors. The family history gives a clue in many cases. The patient's history is diagnostic in others.

There has been a good deal of discussion with regard to the explanation of the mechanism of these somatic manifestations. A complete review of the literature reveals the astonishing fact that not only the cause but also the site of origin of left inframammary pain is unknown. Various theories have been put forward to explain this origin. Some are of opinion that it is cardiac in origin. Others think that it is referred from faulty function of muscles or ligaments about the spine. Some consider that it is a myalgia, resulting from fatigue of accessory muscles of respiration, while a few others think that it is imaginary. A detailed description of the arguments in favour of or against one of these theories is perhaps out of place in this short note. We would refer our readers to the very excellent article on "Da Costa's Syndrome" by PAUL WOOD, M.D., F.R.C.P., in the May 24th issue of our distinguished contemporary—*The British Medical Journal*. Seeing the various factors, the author in that particular issue is of opinion that left inframammary pain arises in local muscular or fibrous tissue, that it may be due to fatigue or strain of respiratory muscles in cases with respiratory neurosis; to strain of certain muscular attachments involved; to incessant minimum trauma from the overacting heart of cardiac neurosis; it is predisposed to by poor physique and that it is maintained and exaggerated by the belief that it arises in the heart. The author is of opinion that several of these factors may operate together. Tachycardia is the sign which has drawn attention to the heart, yet it is one of the commonest psychomatic effects, occurring in every one of us under the influence of emotion. Variation of blood pressure, dermatographism, cold blue extremities, dizziness, syncope, headache, sweating, fatigue have all been explained as manifestations of emotional reaction. Similar explanations have also been given for the various respiratory manifestations such as breathlessness, headache, and irregular breathing, frequent dipping sighs etc. MAXWELL JONES, and AUBREY LEWIS, who discuss the same subject in a very interesting article in the June 28th issue of the *Lancet* are of opinion that the mechanisms of these syndromes can be viewed from the physiological as well as psychological view points. They are of opinion that this dualist attitude is justifiable, though the two types of mechanisms can be regarded as two aspects of the same complex happening, each capable of more or less separate and independent activation. These psychological and physiological explanations are, however, very complicated. It has been found that it is not effort alone but the situation in which effort may be required, and the emotional attitude of the man towards this situation that are often the significant factors. There are, at present, several psychological theories, e. g. the Pavlov theory of conditioned reflexes and the various hormic and libidinal theories of dynamic psychology in terms of which the mechanism of symptom formation in these patients can be described, psychologically, the problem of anxiety neurosis, hysteria, hypochondriasis and other states which are all

lumped together under this heading, and the mechanism is as little or as much known as the pathology of these familiar conditions. All observers are definite on the point that nothing peculiar to effort syndrome in the psychopathology of cases is seen.

The external or environmental causes which have set these mechanisms in action and the internal (constitutional) causes which have enabled external causes to act, have also been discussed very thoroughly by JONES and LEWIS in their article. First of the external causes to be examined is effort or the situation in which effort has been required. A considerable number of men reported that their first pain came on while they were attempting to make an unusual or exhausting effort. Only 47% of men in a group examined, reported that the work they were doing when their symptoms began was heavier than they were accustomed to do without discomfort before the war, from which it must be inferred that there were other important factors besides effort bringing on their symptoms in the first place. An examination among other groups of cases has compelled others to come to the conclusion that effort was not the precipitating factor, though many of the men had come to regard some effort as one of the original causes of their illness. These were linked up with the belief that the heart was affected and that effort is bad for the heart. Some of those who had this trouble blamed the weather and living conditions; with some, it was work they disliked that brought on acute symptoms; while a few reported that their symptoms came on even while they were at rest. Many of those with effort syndrome, had not their work to their liking especially, drill, physical training, digging and other essentials of army work. With many, it was not a question of deliberate pretence. A study of another large series of cases has led the observers to the conclusion that a mixture of external causes with exhaustion, infection and insufficient sleep as contributory factors was predominating. Along with others may be clubbed such as separation from the family, worry about financial and domestic troubles, uncongenial company, unsuitable work, painful experiences and added responsibility through promotion and the like. It was shown that more than one cause was ruling in these cases. So far as physical disease is concerned, 14% of the men had some organic disease.

Among the internal causes, the most important is constitution, which must be described in the physical aspects (physique, habitus, diathesis, idiosyncrasy). Though a certain degree of constancy or permanence is implicit in the usual notion of constitution, it does, in fact, appear to change in certain respects, so that one should properly speak of a man's constitution at a particular time of his life, rather than assume he has the same constitution from childhood to old age. A man's constitution is, in effect, his inherited tendencies, as inferred from the way they have hitherto manifested themselves under the conditions of his life. In practice, the manifestations themselves are largely taken as the measure of his constitution. Some people prefer to take bony, muscular and fatty structure as an index of physique. There are others who differ from this procedure. Others seem to think that a study of function would be more enlightening. There seems to be no unanimity on this question.

There has been a great deal of discussion with regard to the nature of this disorder, the syndrome being without any demonstrable somatic pathology, must be classified according to the functional changes manifested or

else according to its aetiology. As the causes of effort syndrome are numerous and no single cause is preponderant, no classification according to aetiology can be made. There are similar difficulties in the way of classification according to the functional changes. Many writers include this disorder under the category of anxiety-state or anxiety neurosis. This also is unsatisfactory.

The treatment of effort syndrome depends upon our understanding of the mechanisms involved in its development and must be largely prophylactic. Parents, child-welfare clinics and family and school doctors may prevent or provoke the condition. The military medical officer in the Services must be able to diagnose neurosis on positive grounds, and must be able to recognise personalities that will not stand up to military service. When the condition is discovered, the patient must be handled firmly but with understanding by the medical officer concerned. It is usually possible to decide at once whether the man is capable of adjustment and worth encouraging or whether he is better discharged from the Army. Only the doubtful cases and those in which a little more time is needed should be sent to hospital. Patients should be informed of the nature of their illness and be treated as psycho-neurotics. The psychotherapy is simple in its object and extremely difficult in practice. The patient must be induced to believe that he is suffering from the effects of emotional disturbance and not from any disease or alteration of visceral function. Reassurance coupled with adequate explanation in terms that he can understand, the object being to discover the truth and then to expound it. If emotional stimuli are due to false fears, the patient must be encouraged to overcome them. If it is too much for him, he is best removed from their presence after reasonable trial. If these men are to be of service to the nation, they must receive individual consideration. If they are expert in some particular line, they should be given employment in that line: if their civil work is of national importance, it would be wise to discharge them from the army; if anticipation of active service is the root of the trouble, give them non-combatant duty. Occupational therapy prevents introspection and adds interest to the day. Drugs are rarely indicated. Bromide or Luminal may be of occasional value. Benzedrine has been of some use as an aid to psychotherapy.

Import Restrictions

The Pharmaceutical and Allied Manufacturers' and Distributors' Association Ltd., Bombay, have addressed a letter dated 30th October last to the Hon. Sir A. Ramaswami Mudaliar, Commerce Member to the Government of India. The letter which is in the nature of a petition points out among other things:

1. That, as a result of the war, the Medical Profession and the public in India are unable to procure many of the drugs and preparations which were formerly supplied by Continental manufacturers and suppliers.
2. That, during the last two years, stocks of Continental preparations which were lying with importers, distributors, chemists and druggists in this country have been gradually depleted and that it would not now be possible for the English manufacturers with

their own war-time difficulties to face, to supply the numerous needs of the Indian market at very little notice. Even if the United Kingdom were able to supply them, the growing reduction in shipping facilities will naturally disturb the supplies.

3. That, as a result of this difficulty, the public and the profession have had to look more and more to the United States of America for supplying their needs. But, on account of the elimination of Continental supplies, the Americans themselves are now facing an enormous increase in the demand—mostly for their own domestic and State requirements, consequent upon their Government's Expanded Defence Programme, and the purchases by the Allied Purchasing Commissions, besides meeting the demands of Medical Missions sent in aid to China and Russia. Further, there has been shortage of raw materials, bottles, ampoules, packing materials and last but not the least, considerable curtailment in shipping facilities.

On the top of these, the association complains that the importers in India are confronted with new and drastic Import Restrictions on chemicals, drugs and medical and pharmaceutical preparations which are imported into India. We are told that most of these preparations are now placed on quota basis and that the quota has been fixed for a basic period—a period which has been arbitrarily selected, when import from the United States of America were then comparatively very low, the major items of the actual requirements of India still being met by the Continental suppliers.

Then they proceed to state that the growing Indian Pharmaceutical Industry to-day has to rely on more and more supply of raw materials from the United States and are of opinion that the restriction now placed on the importation of chemicals and drugs etc., will definitely reflect on the expansion of the Indian Pharmaceutical Industry itself. In view of the above facts, the Association, on behalf of its member firms, manufacturing and distributing drugs and pharmaceutical and medicinal preparations, requests the Government of India to relax the Import Trade Control regulations and to allow the much needed materials and preparations to be imported freely as in the past. Should this be not possible, they at least request that the basic year for the calculation of quota should be changed or the quota increased to allow larger importations from the United States of America, to make up for the Continental supplies which are no longer available.

As a result of restricted supplies from overseas due to war, and Export Embargo in some Empire countries, there has been greater demand for Indian productions. Though a great fillip has been given to the starting of new as well as to the expansion of the already existing Indian industries engaged in the manufacture of chemicals and drugs, yet, there is no doubt that, at least for some years to come, it will not be possible for the indigenous manufacturers to successfully cope with the situation by producing on a commercial basis, all those preparations which have been hitherto imported into this country either from the Continent or from America.

Thus, there are certain products and raw materials, which can be classified as absolutely indispensable and which are now not available and also not possible to be manufactured in India, and which, therefore, have to be obtained solely from the foreign manufacturers. Under these peculiar circumstances, there should, in our opinion, be certain relaxation of import

restrictions, on these absolutely indispensable items. As to what these are must be settled by a committee of medical men, importers and indigenous manufacturers, which the Government of India would be well-advised to appoint and investigate.

We would, therefore, in all fairness to the Indian manufacturer, as well as to the foreign distributor and the Government of India, suggest the appointment of such a committee immediately to go into this question of considering what preparations are indispensable to India and to relax the Import Regulation rules or in the alternative, to increase the quota provided for such preparations or to alter the period which was selected for the calculation of such a quota, as there is no doubt that these periods may have been periods when some firms had no importation from the United States at all, and/or when they might have had no need just then to import from that country during that particular period.

We are strongly of opinion that this is the time when important decisions have to be taken and, we hope, the Government of India will go into this question thoroughly and come to an early decision.

Gleanings From Medical Press

Medicine and Therapeutics

Coronary Embolism.—The common cause of coronary occlusion is thrombosis secondary to sclerosis of the coronary vessels. Embolism of the coronary arteries is rare. The first case of this type was described by Virchow in 1856. Saphir, in an exhaustive review of the literature, collected 11 cases, to which he added 3 observations of his own. Garvin and Work have encountered 3 cases of coronary embolism among 12,300 consecutive necropsies. Levy, Bruenn and Kurutz found 6 instances of embolism in a study of the records of 762 cases of coronary occlusion, Appelbaum and Nicolson 4 cases among 168, Kirschbaum 4 among 612 cases of severe disease of the coronary arteries. Coronary embolism, according to Hamman, was registered at the Johns Hopkins Hospital for the first time in a period of more than fifty years in 1931, and then ten times in the succeeding nine years. Hamman believes that the condition is not nearly as rare as it was supposed to be. He estimates that from 1 to 2 per cent of the cases of coronary occlusion are due to embolism and predicts that the percentage will rise as the interest and familiarity incite more thorough investigation.

The embolic occlusion, usually single, of a large branch of a coronary vessel with its sudden and dramatic sequence, rather than the largely asymptomatic, multiple occlusions of small coronary arteries, attracts the interests of the clinician. The embolus may consist of air, fat, bacterial vegetations from heart valves, particles loosened from atheromatous lesions or broken-up thrombi. The left coronary artery and especially its left descending branch is most commonly obstructed. The common sources of emboli are

endocarditic vegetations and cardiac intramural thrombi. The most frequent source of emboli is bacterial endocarditis. In the cases collected by Hamman, 50 per cent belonged to this group. The next most frequent source is to be found in the mural thrombi that occur in heart disease. Hamman points out that mural thrombi are found post mortem in 34 per cent of adults dying with heart disease, and, according to Garvin, in 25 per cent of all cases of heart disease there are mural thrombi in the left side of the heart, a condition which can give rise to coronary embolism. Other sources of embolism are a thrombus or atheromatous material in a coronary artery, a thrombus covering an arteriosclerotic plaque at the root of the aorta, thrombi in the pulmonary veins, and thrombi in the peripheral veins giving rise to so-called paradoxical embolism.

Why the coronary arteries are so rarely the seat of coronary embolism has been variously explained by the difference of the calibers of the aorta and coronary arteries, by the situation of the coronary vessels at the commencement of the aorta, the right angle at which they leave the aorta and the bulk and swiftness of the blood current at this portion, and by the fact that the larger part of coronary filling occurs in diastole. The clinical diagnosis of coronary embolism is difficult because death is usually dramatically sudden. Coronary embolism would be suggested, according to Hamman, if a patient with bacterial endocarditis was to die suddenly. Sudden death in bacterial endocarditis is usually due to coronary embolism or the rupture of a mycotic aneurysm into the pericardial cavity. However, gross cause for the sudden death may not be discovered

even at necropsy. The instantaneous death so characteristic of embolism, and contrary to the not unusual observation of recovery from a complete occlusion of a branch of a coronary artery, is probably due to the fact that in the former the coronary arteries were normal and a collateral circulation has not had time to develop. In the absence of a collateral circulation an embolus in a main branch of the coronary is apparently capable of shutting off enough of the nutrition of the heart to be responsible for sudden death.

In contrast to the dramatic symptoms which follow occlusion of large coronary branches, the obstruction of the small branches does not give rise to symptoms. Eventually symptoms of heart failure may supervene, but these are not distinctive of coronary disease. Multiple embolic occlusion of small coronary branches is observed most frequently in bacterial endocarditis and mural thrombi.—J.A.M.A.

Therapeutic Problems in the Treatment of Cardiovascular Disease.—Harely E. Kimble, M.D., Chicago, Illinois.

The therapeutic and dietary regimes in the treatment of cardiovascular disease and its concomitant symptoms, is at this time definitely established, and aside from the newer aids in the form of therapy is well defined. In the realm of drugs, sedatives, diuretics, iodine, digitalis, thiamin hydrochloride and calcium salts, perhaps embrace the therapeutic means at the disposal of the physician. Thus the therapeutic problem that presents itself does not pertain so much to the choice of the drugs as to the manner of their use, that is, their dosage, combinations, frequency of administration, etc. Again, although the combinations of the drug in various proportions do not necessarily encompass the complete treatment of the symptom complex of the cardiovascular or cardiorenal pictures our experience with a preparation containing phenobarbital, 1/4 grain theobromine sodium salicylate, 3 grains and calcium lactate, 11/2 grains, in enteric coated tablets diuretic Grant, gave results which were so satisfactory that we instituted the use of these tablets routinely, after diagnosis had been made.

Phenobarbital, theobromine sodium salicylate and calcium lactate, the three agents comprising this remedy, are amply documented in modern pharmacology, and the satisfactory therapeutic employment has been described by numerous investigators. Cushny states that phenobarbital is of its greatest value in inhibiting motor activity, and in that sense is a true sedative. Cushny explicitly states, that as a hypnotic, it has peers in the other congeners of barbituric acid. A state of semilassitude thus enforced upon a hypertensive patient is of definite aid in enforcing an amount of quietude, in addition to the objective sedative effects of phenobarbital. The cumulative toxicity of phenobarbital it was felt did not come into consideration, due to the small dosages in the combination (1/4 grains) which is easily

eliminated, even in the presence of concomitantly mild nephritis.

Theobromine sodium salicylate owes its choice as a preferable agent over alkaloidal theobromine to its solubility and its lesser irritating effects. Sollmann in a relative investigation of the methyl xanthines, states collectively that they are vasodilative, myocardial and circulatory stimulants, that their action is diuretic, and that the effect of theobromine is preeminent in this respect. As kidney pathology in some degree is usually present in hypertensive subjects, or those in whom cardiovascular symptoms are manifest, the auxiliary effect of theobromine upon the kidneys especially upon the glomerular epithelium is a matter of concern. No mention is made in the literature of damage to these structures due to theobromine. The contributory effect to already present albuminuria was also taken into consideration. Sollmann states that the methyl xanthines do not favour albuminuria, and our experience is similar, in that conversely albuminuria diminished or disappeared in most of the patients in our series. The advantage of theobromine sodium salicylate over mercurial diuretics is obvious; just in those renal conditions, as nephroses or nephritis where one would employ a mild diuretic mercury is contraindicated.

No special point was made by us of the presence of calcium in this combination although Schramm does not overlook the "tonic" effects of calcium on the myocardium. We evaluated the effect of this combination in the form of enteric coated tablets as a unit, as the synergistic effect of the ingredients was apparent to us, beginning with the first few patients in this series, as in previous instances, we had used phenobarbital and theobromine sodium salicylate, alone or in combination, in various dosages. Phenobarbital in this combination has not caused drowsiness and the theobromine sodium salicylate has not given rise to gastric upsets. Our observation inclines us to ascribe this favourable effect to the synergistic action of the ingredients, i.e. (1) To correlate non-cumulation of phenobarbital with the diuretic action of theobromine sodium salicylate, (2) the presence of calcium to potentiate theobromine sodium salicylate in lending myocardial tone and (3) the presentation of this combination in the form of enteric coated tablets which preclude gastric irritation by the theobromine sodium salicylate and facilitate solubility of phenobarbital in the alkaline chyle.

The paper considers the treatment of 35 cases which were divided into: (a) Cardiogenic, (b) Neurogenic, (c) Renogenic.

The principal clinical manifestations occurring in the order of their frequency were encountered in this order:

1. Hypertensive heart disease
2. Essential hypertension
3. Myocarditis
4. Chronic nephritis
5. Arteriosclerosis

Results obtained in the treatment of these

conditions with the remedy used and supplementary drugs were as follows:

Complete remission of symptoms	47.6%
Moderate improvement	33.4%
Only slight improvement	16.6%
No improvement	2.4%
	100.0%

Drugs used in the series included iodine, phenobarbital, thiamin hydrochloride and digitalis, one or more of which were given to some patients, and diurbital, which was given to all patients. The dose of the latter ranged from three to six tablets daily, which was continued for an average period of nine weeks.

SUMMARY AND CONCLUSIONS.—A series of cases is presented, consisting of 35 patients exhibiting various stages of cardiovascular disease, manifested by hypertension, myocarditis, nephritis and arteriosclerosis.

Treatment with an enteric coated tablet containing phenobarbital, theobromine sodium salicylate and calcium lactate was instituted in all cases, some of which however, received as supportive treatment one or more of the following: Phenobarbital, iodine, digitalis and thiamin hydrochloride.

Generally, good results were obtained; complete remission of symptoms of moderate improvement was noted in 81 per cent of the patients. Criteria of improvement consisted principally of clinical objective improvement, although those who reported subjective improvement were statistically accounted for in the "moderately improved" class. All the cases tolerated the drugs well and in none were nausea, vomiting or other gastric intestinal upsets apparent which would make withdrawal of the medication necessary.—*Medical World*.

Calcium Chloride for Rigors.—Rigors following the administration of serum, injections of typhoid vaccine, blood transfusions, and those due to malaria, have been treated by injections of a 10 per cent. aqueous solution of calcium chloride by P. B. Beeson and G. L. Hoagland (*New York State Journal of Medicine*, May 15, 1940, 40 803). The injections were given slowly in order to prevent recurrence of the symptoms. The usual quantity injected was 10 c.cm., although in some cases as much as 20 c.cm. was given. The disappearance of symptoms, indicated by relaxation of muscular spasm and cessation of tremor, occurred usually within about fifteen seconds of the beginning of the injection, the patients experiencing a feeling of warmth and comfort. Complete relief was obtained in thirteen out of twenty-one patients suffering from lobar pneumonia and to whom serum had been given: immediate relief in three patients with malaria induced in the treatment of neurosyphilis (in two instances the injections had to be discontinued on account of nausea). In a patient with rigors after blood transfusion the treatment successfully terminated one chill but not a second, and in two patients, to whom

typhoid vaccine had been given in the treatment of arthritis, complete relief was obtained in three rigors out of seven. It is stressed that the injections should be given carefully so that no extravasation of the calcium chloride into the tissues, with subsequent possibility of necrosis, may occur, and also that calcium chloride should not be administered intravenously to patients receiving digitalis, as it has an additive action. No untoward effects, apart from the occasional occurrence of nausea, are recorded.—*Parcitioner*.

Poliomyelitis and Vitamin B Deficiency.

—Helms puts forth the view, for which she reviewed an extensive literature, that vitamin B deficiency is a predisposing cause of acute anterior poliomyelitis. She finds indirect evidence in the fact that poliomyelitis tends to occur with greater frequency when the demands for vitamin B₁ are heavy as in childhood, pregnancy, substandard nutrition and after excessive muscular exercise in children, or young adults, when poliomyelitis is prevalent. There are points of resemblance too between poliomyelitis and beriberi, which is known to be caused by vitamin B₁ impairment. The direct evidence, the author thinks, would be supplied if suitable tests could be devised. She suggests the investigation of the vitamin nutrition of patients suffering from poliomyelitis. These investigations should include the intake of the vitamin B complex, the conditions which increase the need for the vitamin or interfere with its absorption and, in adults, the amount of alcohol and narcotics taken. The presence of neutralizing substances in the blood may tend to reduce the occurrence of the paralytic form of the disease but does not seem to be a potent factor. The low incidence of paralytic poliomyelitis among those exposed to the disease may be due to the fact that this form occurs only when the biochemical state of the central nervous system favors the propagation of the virus. Vitamin B deficiency may constitute one cause of biochemical alteration. One investigator, reporting 5 cases of acute poliomyelitis, attributed the rapid recovery, with freedom from paralysis or rapid regression therefrom, to vitamin B therapy. Ordinary white flour, made from grain milled according to modern processes, consists entirely of starchy endosperm and contains neither the germ nor the bran, the former of which is nutritionally the best part of the seed. It is poorer than whole meal flour in vitamin B₁ (thiamine) and vitamin B₂ (riboflavin), both of which are concerned with tissue respiration. (Modern milling processes were adopted in 1870. Acute anterior poliomyelitis seems to have increased since 1880).—*J. A. M. A.*

Obscure Pulmonary Infections.—The pitfalls that beset the diagnosis of pulmonary tuberculosis in the absence of bacteriological confirmation have been enumerated by Scadding, and they are rendered prominent at the present time which caused an

unfortunate tendency in the Services to board a man out with the diagnosis of pulmonary tuberculosis solely on the evidence of a radiological opacity. In a recent paper Davidson and Ellman described five patients all of whom had radiological lesions in the lungs which pursued a benign course. The authors hold the opinion that these were examples of so-called "benign acute pulmonary tuberculosis," as described by Ornstein, Ulnar, and Dittler, though in no instance did the sputum contain tubercle bacilli. Many physicians would probably disagree with this diagnosis in some, at least, of the published examples, but the paper is of value because it directs attention to the difficulty of distinguishing between pulmonary tuberculosis and the various atypical forms of pneumonia. A certain amount of confusion has been caused by the indiscriminate use of the term. "Pneutypica pneumonia can be classified conveniently into four groups. The first is described as benign circumscribed pneumonia," a transient localized consolidation arising in the course of a catarrhal infection of the respiratory tract. This condition is of frequent occurrence; it produces few symptoms, and is commonly found on routine radiological examination. The second form is chronic suppurative pneumonia," a condition characterized by prolonged pulmonary consolidation and a tendency to abscess formation; the disease may spread, either directly or by aspiration, and when healing occurs it is accompanied by fibrosis. The sputum is purulent and may be offensive, and, the patient shows obvious signs of toxæmia. In the third type "chronic diffuse bronchopneumonia"—where is a widespread, patchy consolidation of the lungs with fibrosis, the radiographical picture closely resembling that of chronic fibroid tuberculosis: the patient is gravely ill, dyspnoea is the most prominent symptom and the disease pursues a prolonged but fatal course. The fourth form Scadding describes as "disseminated focal pneumonia," an acute condition not associated with upper respiratory tract infection in which fever, sweating, cough, and later, sputum are the principal features. Radiological lesions disappear in the course of a few weeks.

The etiology of these infections remains obscure, the sputum is the principal feature. Radiologically there is a mottling similar to that seen in miliary tuberculosis, though less widespread, and both the symptoms and radiological lesions disappear in the course of a few weeks.

The etiology of these infections remains obscure. The sputum commonly shows a mixed flora with no organism predominating. It is probable that the first three conditions are due to the bronchial tree. The recent work of Weir and Horsfall suggests that disseminated focal pneumonia is a virus infection. An epidemic of atypical pneumonia in infants, probably due to a virus, has been described recently by Adams. When faced with the need to distinguish between one of these conditions and pulmonary tuberculosis, the physician's lot is not a

happy one. On the one hand he is in danger of failing to diagnose an early case of pulmonary tuberculosis, while on the other, there is the risk of imprinting the stigma of that disease upon an innocent subject. Every recognized aid to diagnosis should be employed. A strong family history of tuberculosis may be a deciding factor. The sputum should be cultured and injected into a guinea-pig whenever possible and when none is being expectorated it is often possible to obtain a specimen through the bronchoscope. But every investigation may well prove negative or inconclusive and the search has to be relinquished until the disease either reveals its nature in its own time or remains a mystery. There is a need for new ways of approach to this problem, and the method of diagnostic lung puncture may deserve a more extensive trial.—*B.M.J.*

Prophylaxis and Treatment of Diphtheritic Myocarditis.—Albert G. Bower, M.D., Leon Rosove, M.D., and Y. Togasaki, M.D., Los Angeles, California write as follows:—

Circulatory failure associated with toxic myocarditis is a frequent cause of death in diphtheria. This problem is important because it contributes a great many fatalities and also because of the prophylactic value of prompt recognition with immediate and adequate therapy. Most workers feel that death from circulatory failure may be classified into two groups: early and late. Early circulatory failure is an essential part of the diphtheritic toxæmia while late circulatory failure is a complication of the disease occurring usually during convalescence associated with local inflammatory reactions incident to regeneration and repair. Recognition of these two syndromes is essential to adequate therapeutic management.

Early circulatory failure occurs from the second to the ninth day of the disease. The temperature is usually elevated; the pulse is rapid and thready; the heart is almost never enlarged; the heart sounds are surprisingly forceful; a systolic murmur is frequently present; there is usually no disturbance in cardiac rhythm except as a terminal event; a protodiastolic gallop rhythm may become manifest; the blood pressure which has remained normal may drop at the time of death. As the toxæmia progresses unconsciousness may suddenly develop and death occur.

Late circulatory failure usually occurs from the seventh to the fourteenth day or later. In contrast to the preceding clinical picture, the temperature may be normal or subnormal rather than elevated; vomiting is frequently present associated with epigastric abdominal pain and enlarged tender liver; increased cardiac dullness occurs; tachycardia is followed by bradycardia; the heart tones, particularly the first sound, become indistinct and change in character with subsequent development of a protodiastolic gallop rhythm; premature beats occur frequently and the blood pressure becomes progressively lower. There is

an anxious expression to the face and whiteness around the nose. As the severity of the illness progresses the patient becomes restless, cyanotic, lapses into unconsciousness and expires. Complete heart block, bundle branch block, auricular flutter, auricular fibrillation, premature beats, nodal premature beats, and other arrhythmias may occur in this type of circulatory failure. The electrocardiographic tracings may show, in addition, T wave changes, ST interval changes, and other abnormalities.

Therapy in the early stages of diphtheria consists of adequate doses of diphtheria antitoxin, complete bed rest and constant nursing care, adequate dietary and vitamin regimen, intravenous dextrose, and insulin. In pharyngeal and nasopharyngeal diphtheria the absorption of toxin is rapid because of the large vascular bed at the site of the membrane. Nevertheless, if treatment with adequate doses of antitoxin be instituted early, the fatality rate is low. When more than three days have elapsed prior to using antitoxin, the fatality rate increases rapidly; antitoxin, alone, will not suffice—other measures must be added. It is to these neglected patients that particular attention must be directed to reduce the fatality from circulatory failure.

There is overwhelming evidence pointing to marked abnormality in carbohydrate metabolism in cases of diphtheria. It has been known for many years that there is a transitory glycosuria in the early stages. Lereboullet showed hypoglycemia to be common in severe diphtheritic toxemia. Schwentker and other workers have advanced the opinion that the degree of hypoglycemia parallels the severity of the infection. It is also felt that intravenous dextrose aids elimination by its diuretic action, and pharmacological studies by Edmunds indicate improved tone of the myocardium in experimental diphtheria in animals. In 1927 Gordon first used intravenous dextrose solutions routinely in early toxic diphtheria. Toomey reported its use enthusiastically in 1928. Early in 1934 Hoyne gave routine intravenous dextrose of all diphtheria patients classified in the following groups: "(1) those ill more than three days prior to receiving antitoxin; (2) 'bullneck' types regardless of which day during the course of the disease antitoxin had been injected; (3) patients with marked albuminuria; (4) all postnasal cases and all malignant types of any character." By this procedure Hoyne reduced the fatality rate in desperately ill malignant cases to 9.8 per cent in contrast to a 30 to 60 per cent fatality rate otherwise expected—without dextrose. It was concluded, therefore, that to prevent diphtheritic myocarditis, dextrose should be administered without waiting for physical signs to appear. It has become good practice in these cases to administer insulin to facilitate the assimilation of this dextrose, probably through the usual glycogenic channels.

Treatment in late circulatory failure differs markedly from that of early circulatory

failure because its mechanism is different. Although myocarditis has consistently been demonstrated clinically and at necropsy, in late circulatory failure evidence has been offered that it is not of primary consideration. Romberg showed that stimuli effective on the blood pressure of normal animals was ineffective on animals with experimental diphtheria. However, in animals intoxicated with diphtheria toxin, Edmunds showed that the heart responded adequately when the blood pressure could be raised, repeatedly resuscitating them even at the point of death. From further experimental and clinical observations he concluded that the primary site of involvement lies in the peripheral vasomotor mechanism. Brody also showed that the perfused heart of an intoxicated animal beats many hours after death. Thus the vasomotor center as the structure primarily affected is eliminated by experimental evidence. The ineffectiveness of epinephrine in far advanced circulatory failure in animals suggests that the trouble lies in the receptive substance at the myoneural junction. Additional experimental evidence is furnished by observations with pituitary extracts in intoxicated animals, in which a good reaction occurs provided the blood volume is adequate. It is felt that involvement of the myoneural junction is not one of complete paralysis. On the basis of this evidence, Edmunds proposes the theory that diphtheria toxin unites with the receptive substance and this results in loss of control of the splanchnic circulation. Thus relaxation causes a drop in blood pressure and produces a deficiency in the amount of blood returning to the heart, and the subsequent oligemia causes anemia of the medullary centers and death.

Therefore, to prevent a fatal outcome in late circulatory failure heart stimulants such as digitalis are contraindicated: caffeine with sodium benzoate has practically no beneficial effect. An agent which will have a direct effect upon the blood vessels is needed. Epinephrine has been found slightly useful in a few cases, but pitressin, the pressor principle of pituitary extract, is most valuable. Doctor J. E. Gordon first used this substance in the treatment of late circulatory failure from diphtheria and we have used it since 1932 with most satisfactory results. Pitressin is of no value in cases of early circulatory failure where the blood pressure is fully adequate. This preparation raises the blood pressure and by furnishing an adequate supply of blood to the heart, promotes the general circulation, overcoming the deficiency in the peripheral circulation. It is very essential to supply the body with adequate fluids immediately preceding injection of the drug in order to avoid the reactions which otherwise follow, such as blanching of the skin, pallor, emesis and shock. While usually given in the form of intravenous dextrose, this fluid may be furnished by hypodermoclysis, or in suitable cases even by mouth. Blood pressure readings are taken and the drug is injected in amounts of 0.25 cc. to 1 cc. subcutaneously at suitable intervals until the blood pressure remains normal or higher than normal. Treatment must be strictly

individualized. The action of pitressin is much longer in duration than that of epinephrine and is not followed by the compensatory fall so commonly noted previously when epinephrine was used in circulatory failure in diphtheria. During the past few years we have added adrenal cortex (eschatin) in doses from 10 to 20 cc. in order to sustain blood pressure. This has not been used routinely for prophylaxis.—*Medical Times*.

The Lesion of Pneumococcal Lobar Pneumonia.—The criteria for the experimental production of pneumococcal lobar pneumonia were laid down by Blake and Cecil in 1920. The cocci must be deliberately placed in the lumen of the trachea or of a bronchus in a susceptible animal. Subcutaneous or intravenous injection gives rise to septicæmia with very occasional secondary lungs or is without effect; inoculation into the nasal or upper respiratory passages is usually negative. Blake and Cecil suggested that the infective traversed the lung from its hilum as a wave of acute spreading lymphangitis or the interstitial tissue, the cocci travelling in the peribronchial and perivascular lymphatics, the alveoli being invaded from without. On this view the peripheral parts of the lung should always show edema or early consolidation when the lesion at the hilum shows well-developed lobar consolidation. The conditions laid down by Blake and Cecil have become part and parcel of the experimental study of lobar pneumonia, but their conclusions regarding the evolution of the lesion have been strongly attacked. Loeschcke, in 1931, examined the lungs of forty-seven fatal human cases of lobar pneumonia and claimed to have discovered the primary focus of infection in them all. According to Blake and Cecil this should have been found near the hilum. Loeschcke always found it to be at the periphery, usually in a dorsolateral position. He satisfied himself that the spread of infection was from alveolus to alveolus through the pores of Kohn, that the infective agent was freely flowing edema fluid in which very large numbers of pneumococci were growing but in which cells were scanty, and that as this fluid increased in volume it was spread by coughing and movement, travelling in the small air tubes. Terrell, Robertson, and Coggleshall produced lobar consolidation of the lungs in dogs by introducing pneumococci suspended in a viscous starch emulsion into a terminal bronchus by means of a fine catheter. They found spread to take place radially from the primary focus and the pneumococci to be carried in large numbers in edema fluid which travelled along the small air tubes. Suspension of the inoculum in a viscous medium protects the pneumococci from phagocytosis, allows them to proliferate locally, and prevents their wide dispersion.

Hamburger and Robertson have recently published an account of experiments which give strong support to the contention that lobar pneumonia spreads from a primary focus in the lung by aspiration into the fine air tubes of pneumococcus-laden edema

fluid. They have taken the matter further, and offered a reasoned explanation, founded on accurate experiments in dogs, of the mechanism of spread of the disease from lobe to lobe and lung to lung. The first problem was to determine the exact anatomical disposition of the main bronchi and their main lobar branches. This was done by making metal casts of the bronchial tree. Primary foci of lobar consolidation were then established at different sites in the lung in a series of animals by injecting pneumococci in 6% starch broth into a terminal bronchus. For each primary site the location and order of appearance of secondary lesions arising from it were determined. For instance, with a primary focus in the right lower lobe the secondary foci appeared in the right middle, right upper, and post-cardiac lobes in this order. Having regard to the fact that the habitual posture of the animals was prone with the head and upper thorax slightly lower than the rest of the body and by reference to the metal cast, it was demonstrated that the factor decides where secondary lesions shall appear and in what order is the flow, purely under the influence of gravity, edema fluid from the primary focus. It was repeatedly observed in those animals with secondary foci that the primary lesion was much more oedematous than when secondary lesions did not arise. In a word, the spread from lobe to lobe depended in the first place on the degree of acute inflammatory edema in the primary lesion, and the situation of the secondary lesions in the lung on gravity. This is borne out by further experiments. Cultures from different parts of the lungs were taken from animals with a single lesion and from others with a primary and several secondary lesions. In the first group pneumococci were strictly limited to the primary lesion. In the second, the primary lesion being "wet" and oedematous, there was spread of edema fluid, and pneumococci were found throughout both lungs. An attempt was then made to induce a secondary focus of pneumonic consolidation in a part of the lung remote from the primary focus by plugging with viscid fluid the fine air tubes to a lobe. This was done by injecting mucoid by a fine catheter introduced through a bronchoscope into the bronchus to a lobe. In a series of nineteen animals no secondary lesion developed in the "plugged" lobe in sixteen. It is therefore reasonable to conclude that the site of a secondary focus is not determined by absence of the normal mechanism for expulsion of fluid from the air ways. A series of animals was then infected by injection of the freely flowing watery exudate taken from the air passages of dogs with severe lobar consolidation. Secondary lesions were readily produced by maintaining the animals in positions favouring the flow of fluid to the depths of the lungs, and lesions in various lobes could be obtained at will by varying the position of the animal. When viscid slowly flowing exudate from lesion showing less acute inflammatory edema was used consolidation did not appear.—*B.M.*

Treatment of Paralysis Agitans (Parkinsonism).—By Henry A. Davidson, M.D. (Condensed from "Diseases of the Nervous System," Dec., 1940).

Paralysis Agitans (parkinsonism) is a common disorder. There are four proved effective drugs available for the treatment of this condition: hyoscine, stramonium, Benzedrine and atropine.

Few of the individuals afflicted with the disease show the typical "pill rolling" tremor of classical description. What the practitioner usually sees is a slowed down, rigid, tense patient, with immobile face, stiff posture, wooden gait, lost or markedly diminished associated movements, and a monotonous voice punctuated by explosive stammering.

Treatment of parkinsonism should be planned for a long term. In the first year of treatment the physician should try all the drugs and combination of drugs for a definite period of 2 or 3 months, then at the end of the year the patient will be able to tell which drug benefited him most, and a selection of what will be most beneficial to the patient will be made.

Hyoscine (or scopolamine) is usually given as the hydrobromide. In my experience, at least, most patients ask for hyoscine when, after contact with all 4 drugs, they are queried as to which they prefer. A good starting dose is gr. 1/150 twice a day. This may be increased or decreased according to the patient's reaction. Blurred vision demands a reduced dosage, though not infrequently patients insist on the larger amount in spite of the complication. Patients with tremor and oculogyric crises are especially helped by hyoscine. For postencephalitic cases some of the other medications may be found more effective. Arteriosclerotic patients, however, generally report better results with hyoscine.

Atropine is always worth trying. It gives a patient the feeling of well-being not attainable with any other drug. It may be administered in tablet or solution form. Tablet should contain 0.25 mg. (gr. 1/250) of atropine sulphate. If a water solution is used (and this is generally preferred because it permits a more flexible dosage schedule), it should be in a 1/200 (that is about 0.5 per cent.) strength. One drop of this solution delivers

about 0.25 mg. of atropine, and is therefore the approximate equivalent of a gr. 1/250 tablet.

If tablets are used, start with 1 a day and increase gradually until the patient is taking as much as he can tolerate. Most patients prefer the solution to the tablet. Initial doses is 1 drop three times a day, increased a drop a day until the patient is taking 45 to 60 drops a day, unless tolerance to the drug requires a lower maximum. I have not found the Bulgarian or Italian preparation or the white-wine concoction any more effective than the ordinary 1/200 water solution of the standard belladonna alkaloid.

Tincture of belladonna is not effective.

Atropine may be given hypodermically, starting with gr. 1/300, increasing gradually to gr. 1/150, once a day. Since treatment must be continuous, it is inadvisable to use this parenteral route unless the oral medication provokes vomiting.

Benzedrine (amphetamine) sulphate is especially helpful in patients whose chief complaint is fatigue, drowsiness, or weakness. The 10 mg. tablet is used, with an initial dose of 1 tablet a day, and gradually increased a half tablet every 3 or 4 days. The drug is usually ineffective in the arteriosclerotic type of parkinsonism. Benzedrine should not be given in the late afternoon, as it may cause sleeplessness. The drug is especially useful in combination with stramonium.

Stramonium often dramatically relieves the feeling of stiffness. Either the pill or the tincture may be used, 1 minim of the latter being pharmacologically equivalent to a grain of the leaf or pill. It is necessary to start with a daily dose of about 1 dram (20 minim t.i.d.) and to increase it gradually—by 5 or 10 minims a week. Dosage must be pushed to tolerance before expecting results. The excessive dryness that results can be ameliorated by the daily administration of gr. 1/10 of one of the pilocarpine salts.

The management of the parkinson patient is dependent on a three-point attack, namely, psychotherapy, physiotherapy, and drug therapy. These various therapeutic agents used conjointly can give the greatest amount of relief and comfort to the parkinson patient who suffers from a pathetic chronic disease.—G.P.

Surgery

Surgical Importance of Plasma Proteins.—The extraordinary slowness with which practice follows knowledge in medicine is nowhere more evident than in the study of the body proteins. In a paper discussing the replacement of plasma protein lost in concentration in severe burns had already been recognised in 1885 by Buhl, and in 1881 Tappiener had suggested the transfusion of serous fluid for the treatment of these cases. Our knowledge of the functions of the plasma proteins in maintaining the fluid in the vessels was given a scientific

basis by Starling in 1896. Now, after nearly 100 years of haphazard research, mostly uncoordinated, we have at last reached an era where the body proteins are receiving some recognition; surely a powerful argument for some responsible body to correlate, verify and pass into practice as rapidly as possible the results of individual researches. Judging by the vast literature which has accumulated on the subject during the last few years, much energy, time and paper would have been saved by such co-ordination. The work done from the surgical aspect

has been excellently summarised by Ravdin, who discusses the various effects of hypoproteinaemia. This condition alone can slow down intestinal movements and delay the emptying of the stomach. After intestinal operations, the tendency to oedema all over the body consequent on the reduction of plasma protein becomes intensified at the site of operation, causing swelling at the suture line even to the extent of physical obstruction of the bowel. Further, the actual healing of wounds depends on adequate protein reserves, for a lowered plasma protein prevents normal fibroblastic proliferation, thus causing sutured tissues to fall apart when the stitches are absorbed—an accident well known to all abdominal surgeons. The effect of carbohydrate feeding in preventing damage to the liver due to some anaesthetics is possibly due to the protein-sparing action of carbohydrate, and liver protection could as well be brought about by adequate protein feeding. The pernicious practice of starving patients before (and after) operations must, in the light of this knowledge, have been the cause of much unnecessary mortality and morbidity. A rather more subtle point emerges. If, as Ravdin points out, it is possible to damage the liver by protein starvation, and if, as seems likely, the liver is chiefly concerned with protein metabolism and regeneration, might not a vicious cycle be started by the initial protein depletion? And to what extent may this cycle be broken by plasma-transfusion? It is clear that some rapid and accurate method suitable for universal use is required for the estimation of the plasma-protein percentage and the amount of the total circulating protein. The latter is the more important because in the presence of uncorrected anhydraemia (as in burns) the plasma-protein percentage may be normal though the total plasma protein may be greatly reduced along with the reduction of plasma volume.

One danger remains to be remembered during the intravenous infusion of plasma. The method at present much used for the production of plasma by the citrate method causes a serious dilution of the plasma by the citrate solution, with the result that the final concentration of protein may be halved. This citrated plasma, if run too quickly into a vein, may cause pulmonary oedema. If the danger of reactions can be removed from serum, this substance, which is undiluted, should be preferred.—*The Lancet*, Aug. 16th, 1941.

Non-Specific Ulceration of the Colon.

—Barlow has drawn attention to a little recognised form of acute abdomen, in which early operation is the only means of saving life, but which seems to baffle diagnosis. It is caused by the acute perforation of a non-malignant, non-specific ulcer of the colon. In many cases the ulcer is in the caecum, so that the clinical picture may suggest appendicitis: in over a third of the reported cases in the caecum and ascending colon this was the preoperative diagnosis. Evidence may

however be detected of free gas in the peritoneal cavity. The pathological findings suggest that the condition is one of chronic ulceration with acute exacerbation leading to perforation, and it is possible for symptoms to be sufficient to call for laparotomy before actual perforation occurs. The ulcer is funnel-shaped, thus differing from gastric and duodenal ulceration, which it otherwise resembles. Barlow analyses 52 cases of simple ulcer of the caecum and ascending colon, including 2 which he is personally reporting. Although these are the common sites, the sigmoid colon and other parts may be affected including the rectum. Barlow suggests that the relative stasis determines the incidence in different sites in the colon. The edge of the ulcer is hard, and the base at the end of the funnel shows active inflammation and is the position of perforation. Pain in the chronic stage, before perforation, is far less severe than in gastric or duodenal ulceration; in fact, 68% of ulcers on the right side of the abdomen, and 81% of those on the left, were already perforated when first seen by a doctor. Before perforation vague pains may suggest investigation of the intestinal tract, and a diagnosis such as carcinoma of the colon lead to laparotomy. Once perforation has happened, the urgency of operation seems as a rule to have been obvious. That some cases died without operation, Barlow attributes to the fact that the records go back more than thirty years. The two facts that stand out most clearly from the records is that no case was correctly diagnosed prior to operation; and that no case not operated on survived. Where the condition is suspected before the ulcer perforates the abdomen should be opened, because of the great risk of perforation. Simple drainage has been uniformly unsuccessful. Barlow advises resection and lateral anastomosis as the operation of choice in the absence of perforation. After perforation one can choose between oversewing the ulcer and draining the pelvis, and exteriorising the bowel with or without a caecostomy. Barlow carried out resection, as for carcinoma of the caecum which indeed he suspected at operation in his first case. The second had several ulcers of the caecum with acute inflammation of the caecal wall. Two of the ulcers had perforated. He therefore closed the perforations and exteriorised the caecum and ascending colon, suturing the bowel to the skin. Next day, he opened the caecum by diathermy. Both operations were successful. He suggests that barium-air contrast radiography might help in the pre-operative diagnosis.—*The Lancet*, Aug. 16th, 1941.

Roentgen Therapy for Rheumatoid Arthritis of the Spine.

—The authors write in the J. A. M. A. that 52 patients with typical rheumatoid arthritis of the spine were treated with local high voltage roentgen radiation over the spine. The results were excellent in the patients with early disease, in whom roentgenograms revealed abnormalities only in the sacroiliac

joint; in this group, 92% showed significant, sustained, subjective and objective improvement and some were completely relieved of all clinical evidence of the disease.

The erythrocyte sedimentation rate decreased significantly after roentgen therapy in 41%. Leukopenia was found in 3 patients, but all recovered completely within one month after therapy.

The technic used was as follows: Two hundred kilovolts (175 kilovolts constant potential equivalent) was used, with 0.5 mm. of copper and 1 mm. of aluminium filtration, a half value layer of 0.9 mm. of copper a 50 cm. skin-target distance and an output of 50 r. (measured in air) per minute, with the usual size of the field approximately 200 to 300 square centimeters. In the earlier part, each field was treated three times with 200 r (measured in air) each time. The treatment was given every other day; thus the total dose per field was 600 r.—by C. J. Smyth, M.D., R. H. Freyberg, M.D., and Isadore Lampe, M.D., Ann Arbor, Mich.—*Journal of the American Medical Association*.

Traumatic Edema of Hand.—According to Schorcher, traumatic edema of the hand develops as a rule a few hours after a slight dull injury to the hand. The back of the hand swells, and the thenar and hypothenar eminences and other soft parts of the hand become involved. The cutaneous rugae disappear; the skin becomes shiny and bluish pale and feels much cooler than the opposite hand. The swelling does not pit on pressure. Sweating may be increased or decreased. Motility of the fingers is restricted, so that making a fist is impossible. The hand is painful and hypersensitive to cold. The grave course of the disorder contrasts strongly with mildness of the injury. Operation offers the only cure. The cause is unknown, but from the effectiveness of surgical intervention on the nervous system it has been surmised that a constitutional inferiority of the neurovascular system is responsible. The author reports the history of a patient who developed edema of the right hand following a street car accident. Externally no injury was visible, but the hand became painful and two hours later presented an elastic swelling. Poultices, ointments, hot air, massage, exercises and roentgen treatment persisted in for ten weeks proved of no avail. Intervention on the sympathetic nervous system was decided on next. Several cubic centimeters of a 0.2 per cent solution of pontocaine hydrochloride was injected into the right stellate ganglion. This reduced the swelling of the hand but also produced Horner's syndrome. The swelling and pain recurred six days later. Anesthesia of the stellate ganglion was repeated with the same result, except that the improvement lasted only two days. Next, the stellate ganglion was resected, resulting in the disappearance of the swelling, which, however, recurred seven weeks later. A periarthral sympathectomy was now performed on the recurrent ulnar artery. The edema

disappeared within thirty-six hours but reappeared four days later. Periarthral sympathectomy was then performed on the ulnar artery at the wrist. Four weeks later the woman worked again as a milliner, but the edema recurred and local anesthesia was induced in the ulnar nerve at the elbow. The swelling subsided on the ulnar side of the hand. Three weeks later a silk thread drainage was introduced, and this controlled the swelling and pain after two weeks. The patient has since worked for eight months without further trouble. In the type of traumatic edema under discussion, the tissues show no inflammatory changes. Traumatic edema of the hand has been explained in various ways. The author is opposed to the assumption that the anatomic structure of the fascia of the hand is responsible. Those who regard traumatic edema of the hand as a reflex neurosis consider only the blood vessels to the exclusion of lymph vessels. The author argues that the lymphatic system must likewise have a sympathetic innervation and shows that in edema of the hand the pathologically increased peripheral sympathetic reflexes act not only on the blood vessels but on the lymph vessels as well. The fact that the silk thread drainage counteracted the edema is additional proof that the condition is due not only to the impairment of the capillaries but to that of the lymph vessels as well.—*J.A.M.A.*

Roentgen Irradiation of Cellulitis.—DeHollander reports 18 cases of acute cellulitis of tissues surrounding the alveolar process. In 12 it followed the extraction of teeth and intraoral surgery. In 8 of these roentgen irradiation was begun by the third day of onset. The remaining 4 had first been treated by hot compresses and the like, and they had the cellulitis for from seven to fourteen days before roentgen irradiation was instituted. The response of both groups was satisfactory and resulted in cure of all but 1 patient. Cellulitis of the other 6 patients followed slight abrasions or lacerations to the skin of the chin, face, eye, neck or ear or after an infected area was opened. Response to irradiation by all the 18 patients began within twenty-four hours. The patient first experiences an increase in pain, which persists for from six to ten hours, and at times an elevation of temperature. In twenty-four hours relief of pain is usually remarkable. The patient who was not cured died of the infection and complications. The amount of swelling decreases noticeably in from twenty-four to forty-eight hours. With this decrease the patient begins to feel an improvement and there is less toxicity. The swelling is not always absorbed. Absorption without suppuration occurred in only 7 patients; in 10 fluctuation occurred and opened spontaneously or was incised. It is best to treat the entire area at one time, but if the area of cellulitis is larger than 20 by 20 cm. it must be divided into two parts. When dealing with a cellulitis of the face or neck it may often be necessary to treat both sides. The initial dose is approximately

three fourths of a skin erythema dose. This is given in an attempt to destroy as many of the invading leukocytes as possible and liberate the greatest amount of protective substance. The author has given his patients an initial dose of 289 roentgens. After forty-eight hours he again treated the area of cellulitis but this time the dose was reduced to 193 roentgens, or about one half of a skin erythema dose. The total dose is given within forty-eight hours, and not less than thirty six hours should elapse between doses.—*J.A.M.A.*

Wounds of Knee Joint.—According to Ogilvie, immobilization of an injured knee until infection has been averted or overcome is the key to successful treatment. In a forward area local treatment is limited to cleansing the skin, applying an abundant sterile dressing and immobilizing the limb for transport. A prophylactic dose of sulfanilamide should be given if possible. Every wound of the knee joint is an urgent case; the most important cases are those seen within the "preinfective" or "grace" period of six hours. Through and through bullet wounds with punctured entry and exit holes do not usually require operation; wounds the result of low velocity projectiles often come in the same category. At time of stress these can be evacuated farther, temporary immobilization being replaced by something efficient and prophylactic sulfanilamide continued. These patients must be watched carefully; the joint must be aspirated if it gets at all tense, and immobilization must be continued until infection is overcome. The early motion of injured joints is as unsound as that of fractures. Most penetrating wounds of the knee joint and all those with injury to the patella or partial injury to the other bones should be operated on. Operation should be done under a tourniquet with the strictest aseptic precautions. Local injuries to bone are treated as radically as possible. A patella splintered by a projectile should usually be removed completely. Holes in the femoral condyles or the articular surface of the tibia may be scraped with a curet or trimmed with a small gouge until they are smooth cavities lined with apparently healthy bone. After excision, suture or partial suture should be attempted. The skin may be closed with interrupted sutures in favorable cases, but if one is in doubt it is better to pack the outer layers of the wound with petrolatum gauze down to the synovial membrane. The only apparatus that gives 100 per cent immobilization of the knee is a plaster spica. Sulfanilamide should be given for a full four days. Dressings are left alone for at least ten days. If evacuation is imperative before this, the surface stitches should be removed and the cutaneous wound packed lightly and left open. Complete fractures of the tibia or femur, seen within six hours of injury, demand primary amputation. After twenty-four hours bacteria have established their entry and the tissues their defences. The time for debridement is past and the alternatives are expectancy

or drainage. The wise policy is to continue immobilization and sulfanilamide, watch the condition of the patient and the wound, aspirate the joint if need be, and be ready to drain or to cut down on a foreign body. As soon as septic infection has been recognized, the knee should be drained, the drainage incisions packed with petrolatum gauze and the whole limb (including the foot and pelvis) immobilized in a closed plaster hip spica. If the infection is overcome within four days the joint is saved, as the cartilage has not been affected; if not, joint and cartilage are lost. If all goes well, the cast should be left undisturbed for at least three weeks; if the patient is not better at the end of four days it is best to amputate the leg. Only complete immobilization will insure success. Changes of plaster casts because of offensive discharge can usually be obviated by fluffed gauze applied over the petrolatum packs and covered by one or two layers of wool firmly bandaged. The cast should be so constructed that its strongest strut is away from the wound. These measures will protect the plaster and, if the rest of the cast is unpadded, will not interfere with immobilization.—*Lancet*.

Treatment of Penetrating Wounds of Brain.—Pilcher points out that most penetrating wounds of the brain in civilian and in military life are cared for by the general surgeon, who should remember that every compound fracture of the skull is potentially a penetrating wound of the brain. Treatment should not be attempted without adequate neurosurgical facilities. First aid should consist of superficial cleansing and shaving, control of hemorrhage and treatment of shock. Most important is rapid transportation to a point at which operation can be performed. During transportation the head should be moderately elevated (unless shock is present). Evacuation of the wounded by air-plane, as it is now practised by the German army, has met with considerable success. However, in open head wounds the low atmospheric pressure of high altitudes may increase cerebral herniation. In the author's experiments, immediate removal of foreign bodies in twelve hours resulted in 75 per cent recovery and their removal after twenty-four hours resulted in 75 per cent fatality. The complete removal of foreign bodies is not always feasible or wise. Great depth of penetration or lodgment in the opposite hemisphere from the point of entrance renders removal difficult, and the damage caused thereby is too great to justify the procedure. The bullet itself is not often a source of pathogenic bacteria. Contamination is much more likely to arise from fragments of skin, hair, clothing, dirt and other material that has been driven in. If cleansing is complete and damaged tissue thoroughly removed, and if the wound is closed within a few hours the danger of a retained projectile is minimized. The brain tolerates foreign bodies well. The author cites 2 cases which support the rational advice of Cushing that foreign bodies should be removed only

if their removal does not increase the existing cerebral damage. The location and depth of the wound influence not only the degree of residual disability, if the patient recovers but his chances of recovery. In addition to the high mortality resulting from laceration of large arteries or the great venous sinuses penetration of the ventricular system (particularly if early operation is not possible) and injury of the brain stem, midbrain or hypothalamic region are of grave prognostic importance; they usually terminate fatally. These structures may be unexpectedly involved by seeming distant wounds. For patients not seen until frank infection is present (or in the borderline period from twelve to twenty-four hours when infection may develop) immediate open drainage offers the only hope of avoiding a fatal outcome. The bony opening should be from 3 to 4 cm. in diameter, the dura left open and a soft tube or even small gauze packing inserted to the bottom of the cavity. The once dreaded cerebral fungus will develop. However, the danger from it is in reality small if drainage is adequate and the cerebral infection becomes localized. For the patient seen after healing of such an infection because of convulsions, paralyses or other disabling symptoms, excision of the cicatricial mass is indicated. The author states that, for traumatic neurosurgery, carefully trained and experienced operative teams and elaborate equipment, though desirable, are not essential. However, two devices, a strong suction apparatus and a high frequency, damped wave electro-surgical unit, are almost indispensable. Without them adequate debridement and control of hemorrhage are all but impossible. All macerated scalp tissue, free fragments of bone, devitalized brain tissue and accessible foreign material should be removed, but the injury already sustained should not be increased. Gentle irrigation will "float up" all but the largest foreign fragments, and careful suction will cleanse the tract and facilitate complete hemostasis. Temporary gentle packing with moist cotton may be useful; gauze should never be used on the brain. All wounds free of infection should be closed tightly with fine silk. A drain inserted into the brain is an open invitation to infecting organisms, and it increases the danger of cerebral herniation. If loss of skin has been extensive, it may be possible to close the defect by "relaxation incisions". The exact field of usefulness and the true effectiveness of the sulfonamide drugs in craniocerebral wounds contaminated by bacteria have not yet been finally determined. The Canadian and German armies are using them for prophylaxis and therapy. Extensive studies on the subject are in progress.—*J.A.M.A.*

Conservative Treatment of Habitual Luxation of Shoulder.—According to Heger, dislocation of the shoulder is a frequent sport injury. The relatively small glenoid cavity, a wide, loose capsule and trauma at the elbow or directly to the shoulder are among the factors responsible for the frequency of

luxation of the shoulder joint. The complicated ligamentomuscular apparatus is stretched or even torn during dislocations, and the capsular tear offers an insurmountable obstacle to reposition. The dislocation together with belated reposition is often responsible for habitual luxation of the shoulder joint, which the author defines as repeated dislocation in case of direct or indirect exertion of slight force or in case of extensive movement. The author considers only the traumatic forms, disregarding the pathologic luxations (injuries of the spinal cord, tabes syringomyelia and so on). The great number of therapeutic methods that have been suggested, such as plastic operations on bones, fascia and muscles, shortening of the capsule by puckering or excision, formation of capsular folds by means of the deltoid and more radical methods resulting in arthrodesis, indicate that the therapy of habitual luxation is difficult. Treatments by means of tapes and pressure pads are unsuccessful, because they limit the motility of the arm and produce atrophies. Prolonged immobilization is to be rejected, particularly that which forces fixation in a direction apparently opposite to that of the luxation, because it leads to ankylosis. Early movement has proved helpful. The method used by the author makes use of this factor. It attempts to produce shrinkage of the capsule by injecting around it the patient's own blood and attempts to strengthen the musculoligamentous apparatus by systematic exercises. The author thinks that failures following similar treatments were caused not so much by the methods as such but rather by their application, the choice of substances and the selection of cases. To select the cases roentgenoscopy is advisable, because all cases in which there are defects in the bony part of the joint are unsuitable for this treatment, as are also cases in which severe tears of muscles and ligaments have occurred. The injections are given once every week and consist of 6 cc. of the patient's own blood and 4 cc. of the cytozym of hog's lungs. They are introduced at several points through the deltoid muscle to the articular capsule. The author thinks that the cytozym makes the joint firmer by causing local irritation. Every injection is followed by twenty-four hours of immobilization. Beginning after the second injection, resistance exercises in the form of abduction and adduction and of pressing the arm forward and backward are begun and are gradually increased. At the end of seven injections the entire capsule has usually been surrounded by injections. The author does not think that this method will do away entirely with the surgical treatments of habitual luxation of the shoulder joint but that in suitable cases, particularly in athletes, the described treatment should be tried first.—*J.A.M.A.*

Vincent's.—C. Woodhead. The boundary between Vincent's Angina, which is the name applied to acute ulcerative stomatitis with swollen and tender lymphatics, and a Vincent's infection, which is applied to a

less severe ulcerative gingivitis, is very ill-defined, the one merging almost imperceptibly into the other.

The obvious sign, of course, is the ulceration, and it will be found on microscopic examination of smears taken from the ulcers that two organisms are constant—so constant, in fact, as to be probably causative. They are (1) the *Bacillus fusiformis* which predominates in the milder types, and (2) the *Spirochete Vincenti* which is more common in the more severe types, the two organisms probably living in symbiosis. Both organisms are practically confined to the mouth and both are gram-negative.

(1) A typical foetor oris, and (2) a marked ulceration. The ulcers themselves are covered with a yellowish slough, are ragged edged, bluish, with a tender, red and swollen surround. These ulcers may be confined to some small area if the case is seen early enough, or may involve the whole of the gingivae, and also include the cheeks, pharynx, tonsils, and contiguous parts; but as pain is a late symptom it is unusual to see the early stages. Pain becomes intense later, and there may be considerable difficulty in mastication. Other signs and symptoms include loosening of the teeth, swollen and tender lymphatics in the severe cases, and often a sore throat; temperature may be well raised, and increased salivation is a fairly constant sign.

A very common site for the ulceration to start is around the mandibular third molar. Of the causes of "Vincent's" much can be said. There is no doubt that a low general state of health conduces to infection, and in this respect dietetic shortcomings play their part; especially is this the case in diets deficient in the Vitamins B and C. Infection also plays its part, and this is no doubt responsible for certain groups of the community being particularly vulnerable. One other predisposing cause is the lack of oral hygiene, and this has shown itself to be a very constant factor in the cases seen and treated at the hospital, of which a very high proportion had neglected mouth, the teeth themselves being carious and the surrounding tissues dirty and unhealthy. Local treatment consists essentially of getting the mouth as clean as possible. Hydrogen peroxide should be used both as mouthwash and in the atomiser. Subsequently, gentle scaling of the teeth and application of tincture of iodine is needed. After the acute stage is passed, all badly infected teeth should be removed. It has been suggested that the organic arsenic compounds, such as Neocarsphenamine, may be used as a local application with beneficial effects.

All local treatment, however, serves merely as an adjunct to the general treatment. If, as is likely the patient, is in a low general state of health, this must be remedied by suitable tonic, rest, and fresh air. Attention to diet is most important, and the patients' diet should be examined thoroughly after which, instructions should be given regarding

the general lines to follow. Vitamin B and C deficiency has already been mentioned in this connection and it is wise, therefore, to see that the patient receives his full quota. The patient should be warned of the possible side-actions, which consist of flushing of the face and neck, and sometimes of the limbs, with a sensation of prickling in these sites. These effects usually pass off within half-an-hour of their commencement.

In conclusion, it should be borne in mind that "Vincent's" is infective, and therefore it is essential to warn the patient of its nature, so that timely preventive precautions may be taken through isolation.—*University of Leeds Medical Magazine.*

Tenosynovitis of the Tendo Achillis.

CLINICAL SIGNS AND SYMPTOMS.—The patient complains of pain in the heel along the achilles tendon. This is relieved by wearing light shoes, but reappears on wearing boots, particularly after marching. On examination slight swelling of the lower inch of the tendon sheath is apparent. There is usually one point extremely tender to pressure about an inch from the insertion of the tendon into os calcis. On movement of the foot, flexion, and dorsiflexion tendon crepitus is easily palpable in most cases. The crepitus is often felt along the whole course of the tendon from its origin in the muscle belly of soleus and gastrocnemius to its insertion. Clinically the condition is identical with simple tenosynovitis as seen in the extensor sheaths of the forearm.

This condition occurring in the achilles tendon has been described as a bursitis. Tendo achillis bursitis is a completely different clinical entity which generally follows some violent exertion—e.g. a cross country run; the painful spot is usually nearer the point of insertion of the tendon, and there is complete absence of tendon crepitus. Tenosynovitis of the achilles tendon is undoubtedly due to repeated minor traumata which are caused by an abnormal focus of pressure in the foot.

The following lines of treatment have been adopted: (1) Rest and the use of a counter-irritant—e.g. Scott's dressing. Most cases clear in five to ten days, but about 20% are more resistant to treatment. (2) Infiltration of the tendon sheath and surrounding subcutaneous tissue with 2% novocain. Three injections each of 5 cc. are given on alternative days. If there is no improvement the foot has to be immobilized in plaster-of-Paris for four to six weeks.—*B.M.J., 13th Sept. '41.*

The Physiologic Pathology of Shock as Applied to Treatment.—Alfred S. Giordano, M.D., South Bend, Indiana.

Shock may be defined as a condition of peripheral circulatory failure characterized by decreased blood volume, decreased volume flow and hemoconcentration.

Clinically, the syndrome is characterized by apathy, ashen cyanosis, rapid pulse, fall

in blood pressure and a scanty urine of low specific gravity.

The picture of shock must not be confused with the picture of cardiac syncope since treatment for each is distinctive. Cardiac failure is characterized by pulmonary congestion, dyspnea, orthopnoea, venous distention, liver enlargement, and some times a disturbance in cardiac rhythm. Circulatory failure is characterized by venous collapse and an absence of all the aforementioned symptoms.

Considerable experimental work has been done to determine the physiologic mechanism of shock. Cannon believes a histamine-like substance is elaborated from traumatized tissue and causes an increase in the permeability of the capillary walls. Blalock and Phemister demonstrated that the extravasated fluid in traumatized extremities extends to the groin and retroperitoneal tissues. Guthrie, Erlanger and Gasser and Rapport cite the importance of the sympathetic nervous system and adrenal in the production of shock. Evidence presented to date has shown conclusively that the initiating factor of shock is not due to toxemia, but to injury to capillary endothelium giving rise to increased capillary permeability. Plasma is lost and hemoconcentration results.

Diagnosis: The following diagnostic procedures are used to detect the onset of shock:

1. Complete blood count.
2. Total protein content of the plasma. Normally, it ranges from 6 to 8 gm. per cent; when it falls below 5 gm. per cent, cellular edema results. This determination can be carried out inexpensively and quickly by determining the specific gravity of the blood by the falling drop method.
3. Hematocrit determination: The hematocrit in men varies between 43 to 50 per cent and in women 38 to 43 per cent.

TREATMENT.—1. Oxygen: Inasmuch as anoxemia is the eventual result of peripheral vascular collapse, the use of oxygen in the prevention and treatment of shock is definitely indicated.

2. Whole blood transfusion: Should be given to patients who have suffered from loss of whole blood. Administration of fluids such as glucose only further dilutes the total number of red blood cells and plasma proteins.

3. Gum acacia: Use is discredited because it has been known to cause plugging of the small capillaries in vital organs producing permanent injury.

4. Crystalloids, salt and glucose: Should be used when hemoconcentration exists when the hematocrit is high and when there is hyperproteinemia.

5. Plasma and serum: Should be given when hemoconcentration and hypoproteinemia is present. Plasma has distinct advantages over serum in that it need not be typed, does not cause reactions and can be stored for months.

6. Adrenal cortical hormone: Useful in maintaining endothelial tonus of the capillaries; when administered with hypertonic salt solution, it facilitates the excretion of potassium.

7. Sodium solution dilutes inspissated blood in paralyzed capillaries, increases the velocity of flow, decreases the vasoconstriction and increases the elimination of potassium through the kidneys.

8. Drugs: Morphine is the drug of choice to relieve pain and restlessness. Caffein is beneficial. Digitalis is of no help.—*Medical World, Aug. 1941.*

Surgery of the Left Colon.—John M. Scannell, M.D., F.A.C.S., Brooklyn, N.Y.

It is not the purpose of this short paper to discuss the various malignant and benign tumors found in the colon but to present a procedure which has given the writer the best end results after operating for relief of obstructing lesions found in the left colon. The basic principle underlying the treatment of any organ or part is complete physiological rest.

Due to the anatomical, physiological, and bacteriological characteristics of the left colon, surgery in this section of the large bowel is difficult and the end results often unsatisfactory.

Many operations have been devised for relief of pathology found in the left colon but in my opinion credit for bringing to the profession the most valuable one belongs to Sir Hugh Devine of Melbourne, Australia.

The Devine colostomy is well known to most of us and the chief reason for presenting this subject is simply to emphasize its value and possibilities. It is made in the transverse colon and permits "dysfunctionalization" and "debacterialization" of the involved segment.

The operation described by Devine is a modification of Paul's method and consists of three vertical incisions placed in the upper right quadrant. A loop of the transverse colon is delivered through the large or middle incision and the edges of this loop are sutured together for a distance of four or five inches, the upper end of the loop being left free for a distance of one and a half inches. This loop is now replaced into the abdominal cavity, its division with cautery permitting the cut ends to be brought out under the skin through the small one inch incisions and sutured to the skin. The central incision is then closed. When healing takes place we have two openings in the skin two inches apart. The opening in the distal loop is used to wash this segment of the colon with saline solution or inject into it fresh, active cultures of *Bacillus acidophilus*, as the usual commercial preparations are ineffective.

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The procedure as originally described by Devine has been modified by some of us in an attempt to simplify the technique and

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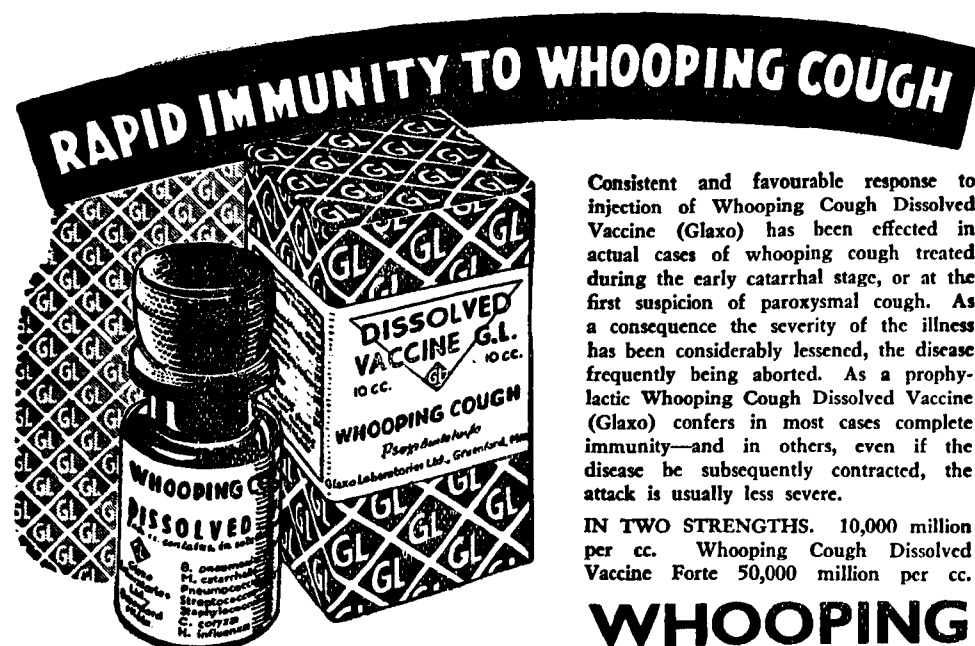
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diminish the possibility of wound contamination.

I prefer to make one transverse incision in the upper right quadrant over the outer edge of the right rectus muscle about two and one half inches long. The incision is carried straight through the abdominal wall, incising a few fibers of the outer edge of the right rectus muscle in order to get more room. After the hand has been introduced into the abdominal cavity and the exploration completed, a loop of transverse colon is delivered through the wound and treated as described by Devine.

If the obstruction in the left side of the colon is not complete the central part of the wound is sutured together in layers under the loop of the colon, and the gut is not divided for one week. (There is always a chance for contamination every time the gastro-intestinal tract is opened.) At the end of seven days the loop of colon protruding above the skin is removed, leaving two openings in the skin two inches apart.

In the cases where the obstruction in the

bowed is complete, the gut is divided between clamps and the cut ends sutured to the skin at the outer and inner angles of the wound. The central part of the incision is sutured together around the free ends of the gut.

The second and third stages of the operation are performed as described by Devine.

So far we have had no infections or herniations of our wounds following this procedure. Black silk was used for suture material.

One of the common errors made in operating on the left colon for obstruction is the removal of a segment of the sigmoid colon for carcinoma only to find out later that the correct diagnosis was diverticulitis. This mistake can easily be avoided by first doing a Devine colostomy.

In conclusion, I wish to state that the Devine colostomy, in my opinion, offers the patient the best chance for recovery, following operations for the removal of obstructing lesions found in the left colon, without a permanent abdominal anus.—*Medical Times*, August, 1941.

Obstetrics and Gynaecology

Induction of Labour by High Puncture of the membranes.—By Coralie Rendle Short, M.B., Ch.B., M.R.C.O.G.

Premature induction of labour has, during recent years, become a recognized treatment for many disorders of pregnancy, both obstetrical and medical. It is found that there are several methods by which induction may be performed, some obviously more efficient and practicable than others.

Many American writers, prefer the combined technique of pituitary, castor oil, with or without quinine, together with rupture of membranes in front of head. It was found that out of about 2,000 deliveries, personally dealt with at St. James' Hospital, London, 87 needed to be induced. The main indications were toxæmia, either severe or mild, certain medical conditions, and comparatively few for simple disproportion. Most of the patients were induced at 39 weeks and onwards, none before thirty-six. The method is quite simple and only on rare occasions was any difficulty encountered. In ordinary cases no anaesthetic was used, but in very nervous patients and in the severe toxæmic or eclampsia cases, a general anaesthetic was given. The patient was placed in lithotomy position and the vulva and the surrounding parts very carefully cleansed with dettol, and sterile towels arranged; a catheter was passed. Then a Sim's speculum was placed in the vagina, which again was washed out well with dettol. No volsellum was ever put on the cervix, and dilators were never necessary. A special point was always made of gently dilating the cervix with the finger at induction and the membranes where possible were separated from it by running the finger round inside the internal os. The special catheter was then passed and the membranes punctured above the head; as a rule about 15 to 20

ounces of liquor were drawn off. In some cases labour started at once; but in others, there was considerable delay.

It may be said that inducing labour by rupturing the membranes in selected cases is a commendable procedure. The high puncture method is simple to perform after a little practice and does not usually require an anaesthetic. Its only disadvantage is that labour may be longer in starting than when the membranes are ruptured in front of the head. It is important that the bladder should be empty and that the finger should guide the cannula all the time, otherwise it might theoretically be possible to damage the bladder, to push the cannula through the uterus or to dislodge a low-lying placenta. The mothers like it better than puncturing the membranes in front of the head because they are not always "wet" afterwards; in most cases where the head is engaged in the pelvis there is practically no leakage of liquor. The length of labour is actually shorter than normal. It is not prolonged in any way. The amount of operative interference necessary is also much less in cases that are induced and if Cæsarean section ever needs to be performed afterwards, provided the induction was carried out under strict aseptic conditions, the risk of sepsis should not be greatly increased. Probably it is less with high puncture than with low, because there is not likely to be ascending infection from the vagina.

The main objections to induction are that the maternal morbidity and mortality are increased because of the risk of sepsis, the still-birth rate is higher, and a lot of premature infants are born. It has been shown that this fear is unfounded.—*The Bristol Medico-Chirurgical Journal*.

Leucorrhœa—Differential Diagnosis and Treatment.—By George H. Gardner, M.D., (Condensed from the "Quarterly Bulletin of Northwestern University Medical School. 14, 1940.)

In most cases a careful history and examination will reveal the source and cause of an abnormal discharge from the female generative tract. Intelligent treatment is dependent upon an accurate diagnosis.

Leucorrhœal discharges can be classified according to (1) the patient's age; (2) the manner in which the discharge occurs; (3) the character of the material discharged; and (4) the organ from which the leucorrhœa originates.

Age is of particular significance in that in children leucorrhœa is usually due to gonorrhœal vulvovaginitis, while after the menopause, leucorrhœa is most frequently due to senile vaginitis or, occasionally from periodic evacuations of a pyometra.

A continuous discharge usually is present with vaginitis and cervicitis while that which arises from a partially obstructed structure occurs periodically. The amount of discharge should be noted, as a Skene's duct abscess and a Bartholin gland have a slight intermittent discharge while that from a strictured cervix with pyometra or a large hydrops of the tube is gushing, profuse and intermittent.

The character is of importance. The discharge may be foetid and watery from a necrotic tumour such as a cancer of the cervix or thick and pasty with pruritus vulvæ, such as is seen with yeast infections. A mucous secretion or mixture of mucus and pus is characteristic of cervical discharge. A frankly purulent, continuous discharge is most often the result of a *Trichomonas vaginalis* vaginitis; if intermittent it may arise from a Skene's duct, Bartholin gland or pyometra.

Examination may disclose thickening of Skene's ducts bespeaking previous gonorrhœal infection. Surgical excision of the involved ducts is preferable to fulguration. A Bartholin-gland abscess with fistula is not necessarily gonorrhœal, as this condition may follow trauma. Usually, there is a history of a previous abscess which ruptured spontaneously or was opened surgically. One should be able to obtain discharge, by gently squeezing the gland, which is similar to the leucorrhœa noticed by the patient. Treatment includes thorough excision of the gland, with its cyst, chronic abscess or fistulous tract. The diagnosis of gonorrhœal vulvovaginitis in childhood is confirmed by finding typical organisms in the vaginal smear. Affected children must be isolated, as the disease is very contagious, and spreads like wildfire to other young girls. Treatment includes local hygiene and vaginal suppositories of ovarian hormone. With oestrogens in adequate dosage, infantile vaginal mucosa can readily be converted into the relatively immune adult type at which time the vulvovaginitis heals and the

gonococci disappear. The pH of the vaginal secretion as determined by nitrazine papers may aid in the diagnosis. A monilia infection does not change the pH from the normal of about 4. Trichomonads, however, cause an alkaline secretion of around pH 7.

A continuous thick pasty white discharge not accompanied by pruritus occasionally results from an excess in the normal desquamation of vaginal epithelium and occurs most frequently in tense highly emotional, over-sexed individuals. Local hygiene and sedatives usually suffice. With marked pruritus vulvæ, a moderate pasty vaginal discharge is usually due to a monilia infection. The diagnosis becomes more probable if there is a diffuse fiery red vaginitis and the discharge is strongly acid in reaction. Diagnosis is confirmed by culture on Sabouraud's medium. Yeast infections usually respond promptly to a few applications of 1 to 2 per cent. aqueous gentian violet on the vulva and vagina. I also prescribe daily weak iodine douches (1 dr. of the tincture in 2 quarts of water). Alkaline douches, although rarely indicated, do give symptomatic relief in patients with yeast infections.

Trichomonas vaginalis vaginitis is apparently becoming more prevalent. The typical clinical picture plus the fact that the flagellates are numerous and easily demonstrated by hanging drop preparation, make the diagnosis simple. It may occur in virgins or married women usually without apparent reason.

Rational treatment includes the use of a mild antiseptic, but is concerned chiefly with rehabilitation of the vaginal mucosa, i.e., a return of the normal bacteria, chiefly Doederlein bacilli, and a restoration of normal vaginal acidity. I treat office patients with trichomonas infections with vaginal insufflations of a preparation composed of lactose, anhydrous dextrose and diodoquin; the latter ingredient is a combination of iodine and hydroxyquinoline, the first two are fermented and broken down to produce lactic acid. I prescribe 1 tablet of this preparation high in the vagina each evening before retiring and daily lactic acid douches (3 teaspoonfuls of lactic acid in 2 quarts of warm water). Some patients have recurrences and others are extremely refractory to treatment.

Vaginitis is common among women after the menopause when the genital organs are atrophic. Treatment consists of sufficient oestrogen therapy to convert the senile vaginal mucosa into a sexually mature type, and the use of lactic and acid douches.

Leucorrhœa from inflammations of the cervix is more profuse before and after the menstrual period. It is mucous or mucopurulent in character from the mucus secreting endocervical glands. Acute endocervicitis is most often caused by the gonococcus, whereas chronic inflammations are often perpetuated by non-specific organisms. The chronicity of the lesion is due to the infection of the deep-seated, poorly draining cervical glands.

Acute endocervicitis, irrespective of aetiology, should not be treated actively. Douches are contra indicated as potentially harmful. Vaginal tampons are not advisable. The husband should be examined as the possible source of the infection, whether gonorrhœal or not. Rest to the inflamed structures, and general supportive measures are in order. After 6 to 8 week's local treatment is usually indicated. Topical applications are of little value because of the mucoid discharge. Cauterization of the canal with the electric cautery, however, establishes free drainage, overcomes gross and microscopic obstructions, opens infected crypts, and destroys some, but not all, of the cervical mucosa. The nasal-tip type of cautery blade is used in preference to either a cylindrical electrode or a conization outfit. One must avoid destroying all of the endocervical mucosa at any level in the canal for fear of producing late cervical strictures. Improvement because of increased discharge, and even hæmorrhages, does not begin for 3 weeks, but then is progressive over a period of many months. Periodic examinations with tests of the patency of the cervical canal should be made, and recauterization should not be done within a year.

An erosion of the cervix is a complication of endocervicitis; it may supplement and increase mucous endocervical discharge, and may ooze slightly when traumatized. An erosion is a fiery red symmetrical lesion of the external cervix, centred about the external os, and composed of granulations and endocervical mucosa. Erosions which are attended by leucorrhœa may be eradicated by cautery destruction of the eroded surface. Some are so extensive that amputation of the cervix is desirable. I prefer the bisection, with transverse wedge excision technique.

Following lacerations of the cervix, eversion of the endocervix may occur. It appears as reddened granulation tissue, and gives rise to a mucoid or mucopurulent discharge. Topical applications of strong silver salts or cautery treatments usually suffice for less extensive eversions. More marked lesions require surgery.

Cervical strictures or obstructions from whatever cause tend to perpetuate an endocervical inflammation and proper drainage must be instituted before cure will result. Occasionally in older women an obstruction becomes complete and the uterus fills with pus; giving rise to the clinical syndrome of lower abdominal pain, followed by evacuation of the uterus with gushing purulent leucorrhœal discharge and relief of the pain.

The foul watery discharge of cancer of the cervix is a late manifestation. Early diagnosis depends more on discovering friable tissue on palpation, which bleeds on contact.

The endometrium of the corpus of the uterus is rarely the source of leucorrhœa. A chronic endometritis from placental fragments, tuberculosis, foreign body, necrotic

tumour or cancer may, however, produce a discharge which is usually bloody.

The Fallopian tubes are affected by a rare clinical syndrome, hydrops tubæ profluens, or intermittent hydrops characterized by intermittent profuse gushing watery discharge. Vanishing adnexal masses strengthen the diagnosis. Present opinion suspects that many of these cases are due to cervical obstruction and hydrometra. Hyperæmia of the internal genitalia for a day or two both before and after menstrual periods occasionally causes a physiologic discharge. Menstrual congestion also aggravates most leucorrhœa. Emotional strain, physical fatigue, and protracted illness may cause leucorrhœas that do not respond to local treatment. Congestion from retro-displacement of the uterus will cause increased leucorrhœa from an associated endocervicitis. The retroversion should be corrected before cauterization because of the danger of pelvic peritonitis.

It is folly to attempt to cure a leucorrhœa when a chronic pelvic cellulitis continuously pours infectious material through the lower genital tract. First eliminate the trouble higher up.—General Practitioner.

The Value of Calcium in Labor and in Uterine Inertia.—G. D. Patton and R. D. Mussey (*American Journal of Obstetrics and Gynecology*, 41: 948, June, 1941) report a study of the effects of the administration of calcium during labor. This study was undertaken for two reasons: To determine whether calcium would relieve labor pains and to determine the effect of calcium on the contractibility of the human uterus during labor. Calcium gluconate was given intravenously to 26 women who were either in labor or "were undergoing attempted induction of labor." In 11 cases graphic tracings of uterine contractions were made by means of an air-filled balloon, placed on the abdomen over the region of uterine prominence, and a kymograph. A 10 per cent. or sometimes a 20 per cent. solution of calcium gluconate was employed; the rate of injection did not exceed 4 cc. per minute; the dosage varied from 10 cc. of the 10 per cent. solution to 20 cc. of the 20 per cent. solution. It was found that the calcium injection did not relieve labor pains in any case, and in some cases appeared to increase the pain. Definite stimulation of uterine contractions was observed in 23 of the 26 patients. Two of the 3 patients who showed no response to the calcium injection were not in labor at the time the calcium was given. The increase in uterine contractions was of short duration in 3 of the 23 patients who showed a response to the injection; in 4 others no progress of labor was noted, in spite of the increased uterine contractions; in one of these patients cesarean section was done, because of pelvic-fetal disproportion. In the remaining 16 cases, labor progressed "uneventfully" to delivery; 13 patients were delivered within four hours; delivery was spontaneous in all but one case, in

which low forceps were used because of slowing of the fetal heart. In 5 cases regurgitation of the gastric contents with little or no nausea occurred during the injection. After the injection, the blood pressure increased slightly and the pulse rate decreased slightly, on the average. The effect of calcium on the uterine contractions as observed with the kymograph was to increase the intensity of the uterine contractions but not their duration, and to decrease the interval between contractions. The administration of such analgesic agents as pentobarbital sodium and paraldehyde soon after the calcium injection counteracted the effect of calcium on uterine contractions; in cases of uterine inertia such analgesics preferably should not be given until after definite progress has been made. On the basis of reports in the literature, calcium should not be given if a drug of the digitalis group has been administered. As the injection of calcium tends to lower the blood sugar, glucose may be given intravenously if there is any evidence of hypoglycemia. From these studies, the authors conclude that the administration of calcium is of value in the treatment of uterine inertia in the first and second stages of labor, but it does not overcome severe dystocia. No evidence of any ill effect on the infant was observed in the author's cases. They suggest that further studies of the effect of the administration of calcium in labor should be made, in order that the therapeutic value of this procedure may be more definitely evaluated.—*Medical Times*.

Preoperative Management of Uterine Myomas Complicated by Pelvic Infection.

—C. A. Gordon and A. H. Rosenthal (*American Journal of Obstetrics and Gynecology*), have previously described a method for the treatment of pelvic infections by iontophoresis with a choline compound. As the association of inflammatory conditions in the adnexa with uterine myoma increases the difficulty of operation and the danger of peritonitis this method of iontophoresis has been used in 39 cases of uterine myoma in which there was evidence of pelvic inflammatory disease, degeneration of the uterine tumor, or both. In 32 cases there was abdominal tenderness or peritoneal rebound pain; in 27 cases a firm mass in the abdomen was palpable; there was pain in 32 cases; and tender

adnexal masses in 25 cases. The temperature was above normal in 25 cases; leukocyte counts were relatively low, being higher than 15,000 in only 13 cases. Sedimentation time was accelerated in all but 2 cases; this has been found to be a better index of "the presence and activity" of infection than the leukocyte count. In 28 cases the diagnosis of adnexal disease associated with uterine myoma was made, yet in 4 of these an unsuspected degeneration of the tumor was found at operation, and in one of these there was no pelvic inflammation. In 8 cases a differential diagnosis could not be made; at operation degeneration of the tumor alone was found in one case, pelvic infection alone in 4 cases, and both degeneration and infection in 3 cases. Hysterectomy was done in 32 cases, with removal of one or both adnexa in 27 cases. Preoperative treatment with iontophoresis of a choline compound was carried out in all these cases; the average number of treatments was six, given every other day; no patient was given more than twelve treatments. As a rule operation was done only when temperature, leukocyte count and sedimentation time returned to normal; if, however, temperature became normal, but sedimentation remained accelerated after one or two weeks, it was concluded that degeneration of the myoma rather than pelvic infection was the chief complication and operation was indicated. It was found that in those cases in which there was no degeneration of the tumor, but pelvic infection was present, the response to the iontophoresis treatment was prompt and favorable; pain was relieved in 75 per cent. of the cases; and the sedimentation time showed a return toward normal levels; usually to the normal of 80 minutes. In cases where degeneration of the tumor was present, the response to iontophoresis was not favorable; the degenerative process seemed to be accentuated, as indicated by the acceleration of sedimentation time. In cases of uterine myoma in which the differential diagnosis between a complicating pelvic infection and degeneration of the tumor is difficult, the use of iontophoresis may be of value in "preventing undue preoperative delay while awaiting satisfactory laboratory data." If pelvic inflammatory disease is present, the time of preoperative preparation may often be definitely shortened and the operative risk decreased by this method of therapy.—*Medical Times*.

Otology

Meniere's Syndrome: Recent Observations.—Meniere's syndrome has always been a perplexing problem for the otologist. While numerous contributions to the subject have been made in recent years, few if any have represented constructive material. Of the more prominent investigations, that of Furstenberg and his associates attracted the most attention. This study was based on the good results of Dida Dederling, in eliminating attacks of Meniere's disease by producing, dehydration. More recently the

work of Shelden and Horton gained prominence. They obtained striking results in patients suffering from Meniere's syndrome, by the intravenous administration of histamine. Through these newer approaches, solution of the problem under discussion seems closer than at any time heretofore.

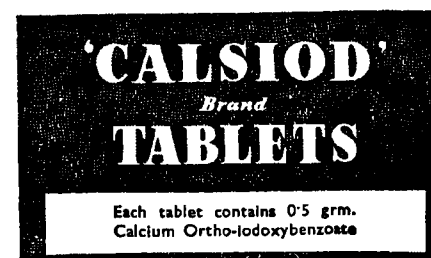
Now another report—that of Atkinson—is presented in the hope of explaining the mechanism of Meniere's syndrome and also classifying the patients for treatment. On the basis of criteria laid down, this writer

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believes that there is a group of patients with Meniere's disease who are unduly sensitive to histamine, whose sensitivity can be demonstrated by their cutaneous reaction, in whom attacks can be produced by injection of the drug and who can be relieved of their attacks by histamine desensitization. "It is therefore concluded that in this group the characteristic syndrome is produced by a reaction in the labyrinth caused by histamine or a histamine-like substance. The reaction is presumably the same as or similar to that produced in the skin-local vascular dilatation and increased capillary permeability."

Atkinson calls attention also to another group of patients with Meniere's syndrome, besides the allergic. In this other group patients give a negative response to histamine. Their attacks seem to be due to vasospasm. Evidence is presented to support this view. Therapeutically, of 19 patients treated with vasodilator drugs over a sufficient period to justify appraisal of results, 17 have been completely relieved or are steadily improving, while 2 who showed an initial improvement eventually had a relapse.

Atkinson claims no originality for the theory of a vasomotor basis for the phenomenon of attacks of Meniere's disease. Credit belongs to Portmann and Mygind and Dederig. Atkinson contends, however, that these authors did not envisage the dual mechanism which, if accepted, clears away many or all of the previous difficulties. It is also emphasized that the condition is not one of apparent simplicity. Further-more, while "it is well enough to say that attacks of Meniere's disease have been shown to be produced by vasospasm in many cases, it still remains to be shown why that vasospasm occurs."

Atkinson's investigation represents careful thinking and well-grounded research. The clinical characteristics of the allergic and vasoconstrictor groups are pointed out. On the rationale of the clinical symptoms, as well as the physiologic indications, a properly planned scientific regime, as carried out in the studies, is suggested.

The ideal treatment is to discover and remove or desensitize against the specific allergen. This procedure is not always possible, however. On the assumption, therefore, that allergic symptoms are the result of hypersensitivity to histamine, desensitization of the subject to this substance, according to the method of Dzsinnich and Galle, will relieve him of further attacks. In the vasoconstrictor group, nicotinic acid and thiamine chloride are the most effective remedies, the former in the young and middle-aged because of its vasodilator action even on the capillaries; the latter in the elderly, because of its sedate action and the fact that old people do not tolerate well drastic dilatation.

While it is realized that there remain to be investigated other far-reaching ramifications of the problem, Atkinson's work represents

a definite contribution to our knowledge of the etiology and treatment of Meniere's syndrome.—*The Eye, Ear, Nose and Throat Monthly*.

The Treatment of Sinusitis.—By Wesley Creveling Bowers, M.D. (Condensed from the *Virginia Medical Monthly* Nov., 1940).

All the sinuses drain downhill, except the antra and the sphenoid. Because of the uphill drainage in both these cases, ciliary action is of prime importance, and interference therewith renders drainage impossible.

The entire lining membrane of the nose and sinuses is supplied with cilia which wave toward the ostia and naso-pharynx. Over the cilia is a tenacious layer of mucus to catch foreign particles and invading bacteria.

The introduction of anything into the nose which interferes with ciliary action impairs the natural protective mechanism of the mucosa. Therefore, habitual spraying of the nose with so-called antiseptic is not recommended.

The tremendous absorptive power of the mucous membrane of an inflamed sinus is demonstrated experimentally by the recovery in the urine within 15 minutes of dye placed within such a sinus. This may help explain why a patient with sinusitis appears sicker than one with an ordinary cold.

When, in spite of all efforts, the ostia do not open, suppuration follows. Prolongation of this stage results in permanent hyperplasia of the mucosa, degeneration of the mucosa, polypoid and cystic formations—correctible only by removal of the diseased tissue.

Unobstructed ostia and free ciliary action are all-important and must be maintained or re-established at all times. In acute infections, swelling of the membrane in the ostia halts normal drainage; hence, anything which tends to shrink the membrane is advantageous. Thick pus retards or stops ciliary action, and should be evacuated by means of lavage. Ciliary action ceases at temperatures below 53 deg. F. and above 109 deg. F. Thus, the use of too hot or too cold vapours or solutions is contraindicated.

TOPICAL THERAPY.—The following drugs may be used in the nose and sinuses, particularly because none has deleterious effect upon ciliary action.

Ephedrine, 1% in normal saline, is excellent for the relief of congestion and aids sinus drainage. It interferes with natural function of the nasal mucosa as little as any known substance. It should be used, not too frequently, in the form of drops (½ inch of the solution in an eye dropper) placed in each nostril with the head well back.

Benzedrine inhalant may give considerable temporary relief, but continuous or repeated use may produce unpleasant constitutional reactions.

Cocaine in weak solution (not over 5 per cent.) is excellent, used alone or combined with ephedrine, for shrinkage of the ostia. Patients intolerant to the drug may complain of headache or malaise. In such cases, pontocaine (2 per cent.) may be substituted.

Habitual daily irrigation with normal salt solution does not interfere with ciliary action, but may produce changes in the mucosa.

Argyrol and neosilvol used frequently or continuously will produce argyria. Argyrol has no bactericidal properties, hence any beneficial results attendant upon its use must be due to osmotic effects.

Petrolatum—like any oily solution—does not interfere with ciliary action, but rests on the mucus and impedes its progress toward the naso-pharynx. Drugs dissolved in oil do not penetrate the mucous coat to reach the epithelium; phenol and iodine are so firmly held in oil that they are rendered inert.

The constant use of popular sprays for the nose, including menthol, eucalyptol, camphor, petrolatum, will produce definite changes in all layers of the mucosa, and is not to be recommended.

The author used chemotherapy in severe infections—which seemed to be escaping control—with miraculous results. Sulphanilamide appears to be specific for the hemolytic streptococcus. Sulphapyridine has a greater usefulness, in pneumococcal and staphylococcal as well as streptococcal infections. Sulphathiazole is useful in the same infections, but is not absorbed into the spinal fluid. Hence it is not of value in meningitis.

Heparid is a new remedy that may be of great value. Already proven useful in thrombophlebitis and thrombosis, it should be given in meningitis, preferably after operation, when the response to chemotherapy is unsatisfactory.

The X-ray treatment of sinusitis remains a method of debatable value. Diathermy is reported to be of value, but I have seen it increase the pain in acute frontal sinusitis.—*General Practitioner.*

Nasopharyngeal Tumors: Otolaryngological Aspects.—J. W. Babcock (*Laryngoscope*, 51: 451, May, 1941) notes that in nonmalignant tumors of the nasopharynx, the chief symptoms are obstruction and bleeding; secondarily deafness, tinnitus and otitis media and mastoiditis may result. Bleeding is especially severe in fibromas and hemangiomas. Such nonmalignant tumors are often difficult to remove surgically and are best treated by implantation of radon seeds; in hemangiomas this may be supplemented by injection of a sclerosing fluid. Malignant tumors of the nasopharynx often cause no subjective symptoms, as they do not grow to any great size at their point of origin. It is only when they invade the base of the skull or metastases develop in the cervical

lymph nodes that their presence is suspected. In a group of 42 cases of malignant tumor of the nasopharynx under the author's observation, there was only one instance in which the tumor was discovered in the course of a routine examination, permitting treatment to be instituted easily, but even in this case "it eventually caused the patient's death." Such tumors may cause nasal obstruction and bleeding, usually slight; these symptoms are not of sufficient severity to bring the patient to seek medical advice in the early stages. On examination, the tumor may resemble adenoids; a large tumor mass in the nasopharynx is found in only "a minority of cases;" sometimes only a small ulcerated area may be seen. Radiograms are useful chiefly for demonstrating any invasion of the base of the skull. Radiotherapy is the only treatment indicated; in most cases, the growth of the tumor is retarded, but cure is obtained only rarely. In the author's series only 14.5 per cent. could be considered cured for one to five years and a half. In one instance the patient had two recurrences and died nine years and a half after the primary tumor was treated. Including this case the average duration of life in those cases in which death was due to the malignant growth was nineteen months.—*Medical Times*, August, 1941.

Laryngotracheobronchitis.—From an analysis by Orton and his associates of 62 cases of acute laryngotracheobronchitis in children and 8 postmortem studies, it appears that more than half of the patients had previous colds or infections of the upper part of the respiratory tract in which the predominating organism was a streptococcus. The average duration of illness before hospitalization was usually three days. Forty of the patients were males and 22 females; 47 were less than 3 years of age. The ages of others extended over the first decade. There were 17 deaths. Acute laryngotracheobronchitis in young children is an important problem both for the general practitioner and for the pediatrician. Its proper handling requires early recognition. When signs of obstructive dyspnoea appear, valuable time should not be wasted by giving diphtheria antitoxin. When acute laryngotracheobronchitis is suspected, the patient should be transferred to a hospital where direct laryngoscopy and bronchoscopy and a more active treatment such as intubation and tracheotomy (the operation of choice) may be done at a moment's notice. If time permits, roentgen study should be made prior to operation; otherwise it should follow the tracheotomy. Intubation should be done only for immediate relief; it should be followed by tracheotomy. Patients should be in a room with the temperature at 70° F. and the air supersaturated. This provides a more comfortable environment than the old fashioned hot croup tent. The temperature and humidity have more effect on comfort than does the concentration of oxygen.—*Current Medical Literature.*

Dermatology

Etiology and Treatment of Impetigo.—By A. C. Roxburgh, M.D., F.R.C.S., (*The Practitioner*, May 1941).

Impetigo is an infection of the superficial layers of the epidermis, either by Streptococci primarily, with staphylococci as secondary invaders or by staphylococci from the start. In either case a vesicle is formed which ruptures in an hour or two in streptococcal cases, but not for a day or two in staphylococcal cases. In streptococcal infection thick, yellow translucent 'stuck-in' crusts are formed from the erupting serum, but traces of the extending vesicle can usually be seen at the external edges of the crust. In staphylococcal infection little or no crust is formed and the vesicles enlarge up to 1 to 2 inches in diameter, sometimes healing completely in the centre and extending at the edges when the crusts are removed the serum continues to pour out and soon reforms the yellow crust. The original lesion enlarges and new lesions soon appear in the neighbourhood.

The disease is highly contagious. The incubation period is two to three days. It is most common in children but occurs frequently in adults.

Course.—The disease has a tendency to clear up after persisting for about three weeks, although many cases last much longer and some may be cured much more quickly by skilful treatment.

Diagnosis.—The ordinary case is easily diagnosed, but, the circinate type is sometimes mistaken for ringworm. In the latter the ring is nearly always made up of a number of minute individual vesicles or pustules whereas in impetigo it consists of the continuous edge of one large vesicle the centre of which has disappeared.

A very acute impetigo involving practically the whole of both sides of the face almost symmetrically may be extremely difficult to differentiate from an acute weeping eczema of the face. Help can be obtained from the history: impetigo usually begins more at one side and spreads, whereas Eczema as a rule starts symmetrically on both sides.

ECTHYMA.—Sometimes the infection in impetigo penetrates deeper than the epidermis and involves the dermis. Ulceration then takes place and the crusts are of a dirty brown colour and have pus beneath them. Such lesions are called Ecthyma and take longer to heal, and leave permanent scars. The treatment is as for impetigo, with violet light being particularly helpful.

Treatment.—The essentials are to remove both crusts and vesicles as soon as formed and to sterilize the exposed surface as far as may be without any strong or irritating antiseptic which would only aggravate the outflow of serum.

STREPTOCOCCAL IMPETIGO.—The best method in a bad case is to apply hot fomentations of Boric acid or one in 4000 *hydrarg. perchlor* every quarter of an hour until the crusts are

softened and can be wiped away or starch and boric poultices may be used instead—being left on for four hours at a time. In mild cases merely bathing crusts with normal saline or 2% sod bicarb solution will be sufficient to remove them. The crusts having been removed a non-irritating antiseptic must be applied. The process must be done three times a day.

In Staphylococcal cases the blisters should be cut away as thoroughly as possible; and the raw areas painted with acriflavine 1 in 1000, brilliant green 1% or crystal violet 1% using a wisp of cotton-wool and wiping from the centre of each lesion outwards. The dye should be reapplied twice daily.

Impetigo of the scalp must be treated in the same lines, after preliminary measures of cutting short the hair over the affected area. Impetigo of the scalp is usually the result of pediculosis capitis and if present must be treated if the impetigo is to be cured.

Mycoses of the Vulva.—Charles J. Druce, M.D., F.A.C.S., Chicago, Ill.

Vulval and vaginal mycotic infestations are due to fungi, more commonly the monilias and cryptococci. Endomyces and saccharomyces are not known to cause genital infection or oral thrush in the new born. The trichophyton and epidermophyton are less common. Rare organisms are actinomyces, blastomycetes, streptothrix, and others. These organisms grow especially well in the sugar-laden vulva of the diabetic and are often responsible for the so-called "diabetic vulvitis."

Woodruff and Hesselstine found the incidence of fungi to vary from 14 per cent to 33 per cent. The majority of infected patients had no symptoms. Benham reported that about 80 per cent of all patients carry these yeast-like organisms in the digestive tract so that this avenue appears to be the most likely course of spread to the genital canal. Occasionally the husband may have a mycotic balanitis and infect his wife, or she may infect him. Actual disease incidence is somewhat variable, but certainly the symptoms are not sufficiently severe to cause the patient to seek relief in over five per cent of the cases. This frequency of fungi in the genital passage explains sporadic oral thrush in the new born.

Symptoms. Pruritus is the only symptom consistently present. It may be so marked that the patient will be awakened from deep sleep to find that the tissues have been injured by scratching. Other symptoms are discharge, local pain, discomfort, local sense of heat, and congestion. The symptoms may vary considerably. The itching sensation is generally localized to the introitus and vulva area; discharge is ordinarily not abundant, but varies from a thin watery to a thick caseous exudate.

Pathology: When the infection is limited to the vulva, as it may be in the presence of diabetes mellitus, the area is reddened and has a whitish overcast and reddish blue background. It may at times present a clinical picture described as "boiled." As a rule, this condition is localized to the area moistened by the discharge, but sometimes it spreads to the adjoining moisture structure. The spread is usually more directly posterior to the anus and may encircle it. Vaginal mycosis may have an associated vaginal inflammation of lesser degree. The vagina shows the major change, especially with the accumulation of caseous material or thrush patches and a non-specific type of inflammation of mucosa. Only the surface of the cervix exposed to the vagina is involved; the interior of the cervix and the uterus appear immune. Biopsy studies have demonstrated the extension of the inflammatory reaction to the sub-epithelial structures in the more severe and the more chronic infections.

Diagnosis: Vulval mycoses may resemble certain stages of atrophic dermatitis or kraurosis. The infection in the vagina may resemble vaginal trichomoniasis, more than gonorrheal, "non-specific," or other infections.

Treatment: In diabetes mellitus, the vulval mycoses will tend to improve when the patient is under proper diabetic management, but local therapy is nearly always necessary. The patient should be instructed to avoid reinfection through vulval contamination by the bowel contents. The husband rarely may be the source of the reinfection. Daily applications of two per cent. aqueous solution of gentian violet is very satisfactory; Lugol's solution, diluted to one-fourth

strength, applied every three days is also useful; but douches are disappointing.

TRICHOPHYTONIS AND EPIDERMOPHYTONIS. The vulva and pubic areas are fairly common sites for infections by the fungi trichophytonis and epidermophytonis. The trichophytes and epidermophytes and related fungi behave on the pubic and genital area just as elsewhere on the body. They limit their activity largely to the external squamous epithelium and produce lesions of the ringworm type. The disease is called trichytosis or epidermophytosis.

Symptoms: There is pronounced and continuous itching of varying degree, and at times local heat and slight tenderness. The skin becomes reddened, slightly moist, and may crack. The reaction generally spreads to involve the pubic area, the genitocrural folds, and the labia majora. Occasionally there are finely granular, pigmented depositions on the hair. Rust, orange, yellow, and black colored materials are the most common.

Diagnosis: The diagnosis is established by adding a drop of 25 per cent potassium hydroxide to the skin scrapings and examining microscopically for mycelia or conidia. These are distinctly different from those of the yeast and yeast-like groups. Moreover, the former groups are actual epidermal invaders, whereas the yeast and yeast-like fungi are not.

Treatment: The treatment consists in promoting local dryness and the rubbing in of a preparation containing salicylic acid, thymol, or benzoic acid. These preparations are irritating but give the best results.—*The Medical World*—July, 1941.

Ophthalmology

Fundus Oculi in Diabetes Mellitus.—S. H. McKee (*Archives of Ophthalmology*, 25: 773, May 1941) reports a study of the fundus oculi in 2,360 patients with diabetes; 476 of these patients showed retinal changes that were more or less closely related to the diabetes; 205 showed arteriosclerosis of the retinal vessels and 105 retinal hemorrhages. Although evidence of lipemia retinalis was carefully sought, it was found in only one case. The remaining patients showed retinitis of various types, which was classed as diabetic retinitis in 41 cases and diabetic retinitis associated with retinal arteriosclerosis due to cardiovascular-renal disease in 26 cases. Diabetic retinitis is distinguished from renal retinitis by the following characteristics: In diabetic retinitis the patches of retinal exudate show "sharp-cut edges" and are often solid and soapy or waxy looking, and usually distributed irregularly; a star figure is uncommon and never so symmetrical as in renal retinitis; the cotton wool patches of severe renal retinitis do not appear in diabetic retinitis; retinal edema is never as marked and retinal detachment does not occur; retinal hemorrhages are present only in the deeper layers and therefore are not

flame shaped, but roughly circular in outline; circular retinal pigment spots that are found in the later stages of renal retinitis do not occur in diabetes. The patients showing arteriosclerosis of the retinal vessels and retinal hemorrhages were in the fourth or fifth decade of life or older; their diabetes was not severe but of long duration. In the last 1000 patients examined, there were 96 from six years up to and including thirty-one years of age; in this group all but 4 showed normal fundi. One had only two small retinal hemorrhages; 2 showed numerous retinal hemorrhages, associated with a few characteristic chalk-white patches in one instance; one, a woman thirty-one years of age, showed definite hypertensive retinitis with papilledema. Since the introduction of insulin, few diabetics die in coma; reports from large clinics indicate that cardiovascular disease is the chief cause of death in diabetics; the importance of ophthalmoscopic examination lies in the fact that arteriosclerotic changes in the retinal vessels may be detected before there is any other definite evidence of arteriosclerosis; and that by early diagnosis of this condition, it can be better controlled.—*Medical Times*, Sept. 1941.

Effect of Iontophoresis on Eyes With Special Reference to Intraocular Tension.—William Thau and Abraham Myerson. *Arch. Ophthalmol.* 24:4—761 (Oct.), 1940.

Iontophoresis exerts a general parasympathetic effect manifested as a slight narrowing of the palpebral fissure, a lessening of the size of the pupil, some anterior projection of the lens and a definite and durable dropping of the intraocular tension. These results were obtained consistently and repeatedly on four patients with tractable dementia praecox. When the positive electrode is applied over the eye, the effects last from five days to a week, while the negative electrode so placed produces a reaction lasting six to nine days.

The lowered intraocular tension thus obtained is much more durable than that obtained by medication, since any drug now in use has effects that last only a few hours. Repeated experiments on the same persons have shown no dangers from iontophoresis, if the electrode is kept from contact with the cornea. Necessarily, the electrode must be kept constantly moistened with normal salt solution.

With the positive electrode the ions of the salt solution discharged into the eye are the sodium ions. However, if the negative pole is used the chloride ions enter the local structure of the eye. Thus, negative and positive charged ions seem to produce the same general results. The effect may possibly be a stimulation of the ciliary muscle by the parasympathetic nerve and may be linked up with the increased blood flow, as evidenced by dilatation of the capillaries, or may have reference to some obscure and less ascertainable chemical changes within the reacting cells themselves. Whatever the mechanism, the effect seems worthy of extension to include the treatment of glaucoma. It is likely that iontophoresis will produce a more marked reduction in the pathologic than in the normal eye.—*The Eye, Ear, Nose and Throat Monthly*.

One Cause of Headache and Eye Trouble Often Overlooked.—Archibald Cary Lewis, M.D., F.A.C.S., *Professor of Ophthalmology, University of Tennessee*, Memphis, Tennessee.

The middle turbinate body is innervated by the superior maxillary division of the 5th cranial nerve. Prolonged pressure on this turbinate causes persistent headache, heterophoria and other eye symptoms. We frequently find this condition in chronic headache sufferers, who have had their eyes refracted and glasses changed a number of times, but have received no relief from the headache.

Many sufferers from chronic or frequent headache have a narrowness in the upper nose and an impingement on a middle turbinate, which could easily be relieved by a small surgical procedure, such as an infraction of the turbinate, a turbinotomy, or a resection of the offending nasal septum. These patients are often fearful of nasal surgery, because their friends and advisers have had unsatisfactory nasal operations.

Much unnecessary nasal surgery is done because of lack of judgment, conscience, or both, by the operator. These patients are not benefited, and are often made worse by the operation, and naturally condemn all nasal operations.

Heterophoria, or latent squint, is a condition where the extrinsic eye muscles are out of balance; usually spoken of as muscle imbalance. This is often associated with middle turbinate pressure and with ethmoiditis. Other eye symptoms commonly present are photophobia, lachrymation and asthenopia. It is a useless waste of time and effort to try to relieve these conditions by orthoptic exercises, monocular occlusion, eye rest, or a change of lenses until the off-ending turbinates are released from pressure.—*Medical World*, Oct. 1941.

Glaucoma.—By M. Carl Wilensky, M.D. Glaucoma is the name applied to a disease in which increased intra-ocular tension is the most characteristic sign. Three forms of glaucoma include, (1) primary glaucoma, (2) secondary glaucoma, and (3) buphthalmos or hydrophthalmos, the congenital or infantile type in which there may be absence of Schlemm's canal. In primary glaucoma there can be found no antecedent disease in the eye to account for the increase in tension.

Secondary glaucoma is characterized by the existence of definite pathologic changes—responsible for the increase in tension—such as (1) iritis or iridocyclitis, with adhesions between the iris and lens, or with blockage of the iris angle by leucocytes and iris pigment; (2) occlusion of the central vein; (3) severe ulcers of the cornea, especially if they perforate and anterior synechia results; (4) dislocation of, or injuries to the lens by accident or operation; (5) swelling of lens during cataract formation; (6) intra-ocular growth, and (7) intra-ocular haemorrhages.

Diagnosis:—Suspicion should be aroused by (1) the patient's history, (2) physical changes in the eye, and (3) typical changes in the visual fields.

History of trouble with glasses, frequent unsatisfactory changes of lens; (2) periods of dim or misty vision; (3) a history of gradually failing vision; (4) history of seeing rings, a halo, or a rainbow around lights.

Physical changes:—(1) shallow anterior chamber; (2) dull cornea; (3) dilated sluggish pupil; (4) elevated intra-ocular tension usually highest in the morning as evidenced by repeated tonometric readings; (5) glaucomatous excavations of the nerve head. Although an important diagnostic aid, it would be far better to diagnose these cases before permanent damage and excavation of the nerve head has occurred.

Changes in the visual fields. Early, there may be a comet-like scotoma, Seidel's sign. Later, there may be Bjerrum's sign, Ronne's step and a characteristic reduction first in the nasal portion of the field. The field may become constricted gradually with the patient unaware of it until he has a tubular field.—*General Practitioner*.

Proceedings of Associations

The Kistna Dist. Medical Association, Masulipatam.—(The Kistna Dist. Branch of the Indian Medical Association, Masulipatam). The monthly meeting of the above Association was held on Saturday the 29th November '41, at the Government Hospital, Bezvada, when 20 Doctors attended.

After Tea, the business meeting began at 5-30 P.M. with Dr. N. N. Sajeer, L.M. & S., President and District Medical Officer, Kistna in the chair.

Dr. M. V. Subba Rao, L.M.P., Secretary, read the minutes of the last meeting held at Masulipatam which was unanimously adopted.

Then read the letter dated 19-11-41, from the Honorary Secretary of the Andhra Provincial Branch, Masulipatam and resolved that the following members of the Kistna District Branch shall be elected delegates for the All India Medical Conference at Hyderabad. (In this connection the letter of the Secretary of 18th All India Medical Conference, Hyderabad, regarding the fees and other details for the information of the delegates was also read.)

Dr. N. N. Sajeer, L.M. & S., Masulipatam.
 „ M. Sesachary, L.M. & S., Masulipatam.
 „ S. Srirama Rao, L.M.P., Agiripalli.
 „ N. Rama Rao, L.M.P., Gollapalli.
 „ S. V. Ramanayya, L.M.P., Nuzvid.
 „ M. Venkatartnam, L.M.P., Jaggayyapet.
 „ P. Seshagiri Rao, L.M.P., Gudivada.
 „ M. Appalaswamy, L.M.P., Mylavaram.

Read letter dated 24-9-41, from the Honorary Secretary of the Andhra Provincial Branch, Masulipatam.

The following names have been nominated unanimously as Vice-Presidents for the Andhra Provincial Branch.

Dr. P. Veerayya, L.M. & S., Guntur.
 „ P. Gurumurthy, L.M.P., Rajahmundry.
 „ B. Tirumal Rao, L.M. & S., F.R.C.S., Vizagapatam.

Read letter No. C 72 from the Honorary General Secretary, of I.M.A. Calcutta, dated 15-11-41, regarding I. M. A. subscriptions with a list of members. The Secretary of Kistna District Branch of the I. M. A.,

Masulipatam, is requested to take steps to collect the arrears of 40-41 to the I.M.A. and also the subscriptions of the current year.

Also resolved that the contributions to I.M.A., be sent through the Provincial Branch, Masulipatam and not direct.

Resolved that with reference to C. 73 of I. M. A., dated 15-11-41, that the points raised 1 to 4 be left to the discretion of the President and the working committee and that the same subjects be moved in the open Session at Hyderabad. As regards No. 3 members of this branch are not in favour of increasing the subscription.

Resolved that the consideration of the Ethical Rules and the formation of Ethical Sub-Committee be considered at the next meeting.

Then the clinical meeting began :—Dr. M. V. Subba Rao, L.M.P., Secretary, read an interesting case on 'The Foreign Body of the Stomach.'

The president in his concluding remarks summarised the salient points of the above papers and cases read.

The meeting concluded with a vote of thanks by the Secretary at 6-35 P. M.

Bombay Medical Council :—The Executive Committee of the Bombay Medical Council has administered a warning to the following medical practitioners registered with this Council :—

Dr. Homi A. Choksey, M. B. B. S. (Bom.) M. R. C. P. (Edin.) Bombay, and Dr. Krishnalal G. Das, M. B. B. S. (Bom.), D. O. M. S. (Lond.), D. O. (Oxon.), Bombay, to be more careful in future and strictly conform to the provisions of clause 23 of Section II of the Code of Medical Ethics adopted by the Council for notifying change of address.

Dr. Viswanath B. Patil, Karachi, and Dr. Jaiwant D. Patil, L. C. P. S. (Bom.), Karachi, that they should be more careful in future and strictly conform to the provisions of clause (h) of rule 1 of Section II of the Code of Medical Ethics and clause 5 of the Warning Notice relating to association with unqualified persons.

Book Review

1. "THE THERAPEUTIC USE OF DEXTROSE"—By Gaston Levy.
2. "DEXTROSOL IN THE PATHOLOGY OF DIGESTIVE DISORDERS".
3. "ADVANCES IN TREATMENT WITH DEXTROSOL".

At no time has the need for careful prescribing been so great as it is during these days of war, and in no country is it more pressing than ours. The place of dextrose (d-glucose) in health and disease has of late received considerable attention in the medical press and the three booklets under

review are sure to prove informative and useful. The varied indications for dextrose are given in the publications among which special mention may be made of its use as a reliable antiseptic and hemostatic, remind the reader of Schwartzman's (J. Am. Inst. H., 26: 542' 1933) famous observation:

"Of the many new advances, one (dextrose therapy) stands forth in its unique brilliance from the standpoint of effective value and diversity of usage."

Copies of which can be had free of all charges from Corn Products Co. (India) Ltd., P.O. Box. No. 994, Bombay.

Preparations and Inventions

Cadminol.—Messrs. Union Drug Co., Ltd. take pleasure to announce the introduction of their latest product Cadminol, containing Cadmium Thio-Gluconate 1½% in oily suspension, marketed under the trade name "Cadminol" for the treatment of tuberculosis.

Reports on pre-introduction trials indicate that the preparation is useful. Trials are also being conducted in some of the T.B. sanatoria and Hospitals in this country.

Cadminol is issued in 10 and 30 cc. R/C phials:

For further details, members of the medical profession are requested to refer to the Manufacturers at 285, Bowbazar St., Calcutta.

Panliver 'Shiono'.—Ranbaxy & Company, Post Box No. 303, Bombay; Post Box

No. 34, Amritsar. A concentrated liver extract suitable for intramuscular injection. It ensures maximum concentration of hæmopoietic fraction of liver. As it is a polyvalent (containing the P. A. factor plus vitamin B complex and all those that contribute in the cure of secondary anaemia) and crude liver extract, this can only be used intramuscularly never intravenously or subcutaneously. Each ampoule of 2 cc. contains 900 grms. of best selected fresh liver, i.e. 450 grms. per cc. of Panliver. It is indicated in all anaemic conditions, delayed convalescence, sprue, to improve the coagulability of the blood, to protect the liver cells etc. In pernicious anaemia, 25-30 injections of 'Panliver' administered during a period of 4-5 weeks will give excellent results. Available in packings of 5 ampoules of 2 cc. and 50 ampoules of 2 cc.

Press Notes

I

I.M.S. PERMANENT COMMISSIONS.
WAR SERVICE PREFERENCE.

Government of India, Defence Department,
(Public Relations Section) Simla, 5th June 1941.

Officers holding King's and Indian Emergency Commissions in the Indian Medical Service during the present emergency will be given preference when permanent appointments are made after the war, provided they are qualified under the conditions then in force.

II

IMPROVEMENT OF GRATUITIES TO
EMERGENCY COMMISSIONED OFFICERS
OF THE INDIAN MEDICAL SERVICE.

D. G., I.M.S., Simla, 13th Oct. 1941:

Following upon a recommendation of the Indian Medical Service Recruitment Conference held in July, Government have now given their sanction to gratuities on a greatly enhanced basis payable to Emergency Commissioned officers.

The minimum gratuity will be Rs. 2,000 for those who obtained their basic registrable medical qualification before the 1st January 1940 and Rs. 1,000 for those who obtained this qualification after that date.

The minimum gratuity will be earned as soon as an officer completes his first year of service after which he will be entitled to an additional gratuity over and above this minimum, equal to one month's pay for each further completed year of service. The minimum guaranteed gratuity will be admissible to all Emergency Commissioned officers except those who on demobilization are appointed to permanent Commissions, who retain a lien on civil Government appointments or who on demobilization obtain permanent Government employment.

III

I.M.S. EMERGENCY COMMISSIONS—ADVANCE
OF PAY FOR SELECTED CANDIDATES

D.G., I.M.S.: New Delhi, Nov. 11 1941.

The Government of India have decided that candidates selected for Emergency Indian Commissions in the Indian Medical Service will be allowed to draw advance of pay at the time of appointment, not exceeding Rs. 300 if the candidate has been selected in India and equal to two months pay if in the United Kingdom.

The advance will be recovered from the officer's pay at the rate of one-third of his pay.

IV

D. G., I.M.S., New Delhi, Nov. 21st 1941.

Some weeks ago the Government of India intimated the concession of gratuities on a greatly enhanced basis to certain groups of Emergency Commissioned Officers of the Indian Medical Service.

Government now announced a further important war-time concession which makes employment in the Indian Medical Service for the period of the present emergency highly attractive. It has been decided to give an antedate, based on professional experience, to Emergency Commissioned Officers of the I.M.S. This ante-date, which is limited to 5 years, will be reckoned on the basis of half the number of completed years from the date of medical qualification to the date of appointment to the I.M.S. Thus ten years spent in active practice of the medical profession will qualify for the maximum of 5 years ante-date, six years in practice for 3 years ante-date and 3 years in practice for 18 months ante-date. Ante-date will not be reckoned in periods of less than 6 months and the minimum ante-date to be granted will be one year, earned by two years' medical practice.

This concession will be in addition to any antedate which may be admissible to an

officer of the Service on account of special postgraduate qualifications and specified hospital appointments, except that a period spent in hospital appointments will not count twice in reckoning the total antedate admissible. The whole period of antedate which an officer may obtain will count for pay and promotion on the time scale and for seniority in the Service.

*Director General, Indian Medical Service,
New Delhi, November, 21, 1941.*

In accordance with one of the recommen-

dations of the I.M.D. Recruitment Conference held in Simla on September, 23, 1941, the Government of India are pleased to announce that the emoluments of Assistant Surgeons of the Emergency Branch of the I.M.D. (Indian Cadre) will be raised as from December 1, 1941. The pay will be as heretofore, namely Rs. 75/- to Rs. 175/- but the former Civil Allowance of Rs. 50/- will be replaced by an Emergency Allowance of Rs. 125/- p.m. Thus the initial pay plus emergency allowance will now total Rs. 200/- p.m. instead of Rs. 125/- as before.

Circular

ECONOMY IN THE USE OF IPECACUANHA AND EMETINE.

Secretary of the Medical Stores Supply Committee, writes as follows:—

Amoebic dysentery is common in India and accounts for a fair amount of mortality and morbidity, especially amongst the members of the European communities resident in the tropics. In the treatment of this condition, emetine, one of the principal alkaloids of Ipecacuanha, is considered a specific and is very largely used.

Ipecacuanha is not indigenous to India. Attempts have been made to grow this plant in suitable localities in India but the indigenous supply has never been adequate to meet the local demands. India, therefore, remains largely dependent on foreign sources of supply.

Owing to conditions produced by the War, supplies of this essential drug have been scarce and irregular. In the interests of the sick, it is desirable that a general effort on the part of medical men should be made to conserve stocks of this important remedy, so that those who really need it will not be deprived of its valuable curative properties.

The extent to which Ipecacuanha enters into popular therapeutics is surprisingly large. Much of this is in the form of Pulvis Ipecacuanhae et Opii (Dover's Powder), which is almost an everyday prescription in influenza, chill, coryza and slight cough. It is often used to 'abort' an attack of cold and also to check mild diarrhoea. Its diaphoretic and astringent actions are mainly responsible for its popularity. Therapeutically there appears to be no particular advantage in using Pulvis Ipecacuanhae et Opii to bring about these effects. It is the Opium in the preparation, and not the Ipecacuanha, which brings about the diaphoretic and astringent actions. The astringent action can be very easily obtained, probably more conveniently, by the use of Pulvis Cretae Aromaticus cum Opio. For diarrhoea, practitioners have at their disposal a large selection of coal-tar remedies including

salicylates and their derivatives. The use of Dover's Powder can, therefore, be conveniently reduced by encouraging the use of various substitutes which may bring about similar clinical results.

A significant contribution towards economy in the use of Ipecacuanha may also be made by exercising a little more judiciousness and care in prescribing the galenicals of Ipecacuanha, of which Vinum Ipecacuanhae (now Tinctura Ipecacuanhae) is the most widely employed. Tinctura Ipecacuanhae is a common ingredient of 'Sedative Expectorant' mixtures frequently seen in the Pharmacopoeias of various Hospitals and Out Patients' Clinics. Tinctura Ipecacuanhae is neither a very rapidly acting nor always a trustworthy expectorant. In many types of cases of bronchitis, the use of such mixtures is not of any particular advantage and Tinctura Ipecacuanhae may be replaced by Vinum Antimoniale or Tinctura Scillae. Only in pediatric practice is there some justification for its continued use in wartime.

Pulvis Ipecacuanhae is gradually going out of fashion but prescriptions are still seen where this has been used either for its emetic or for its cholagogue action. Such uses are a relic of the old days and cannot be supported by rational therapeutics. Ipecacuanha has now been shown to be a poor emetic and its supposed cholagogue action is also doubted by modern pharmacological investigators. Its use in such conditions therefore need no longer be advocated.

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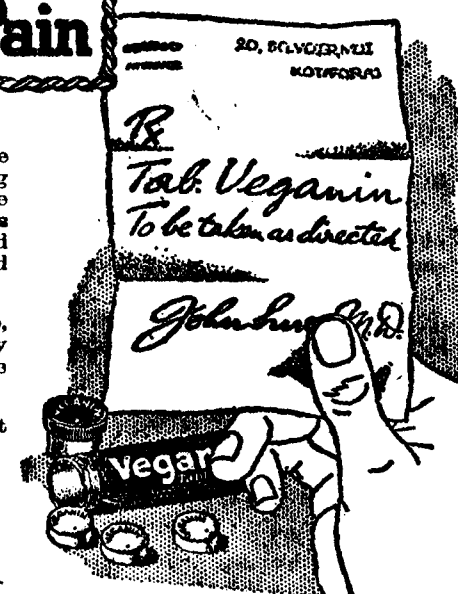
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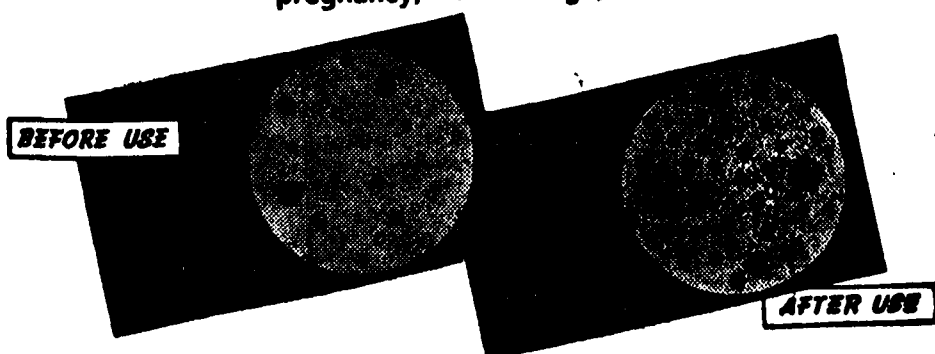
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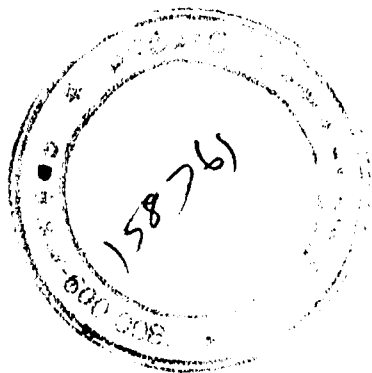
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