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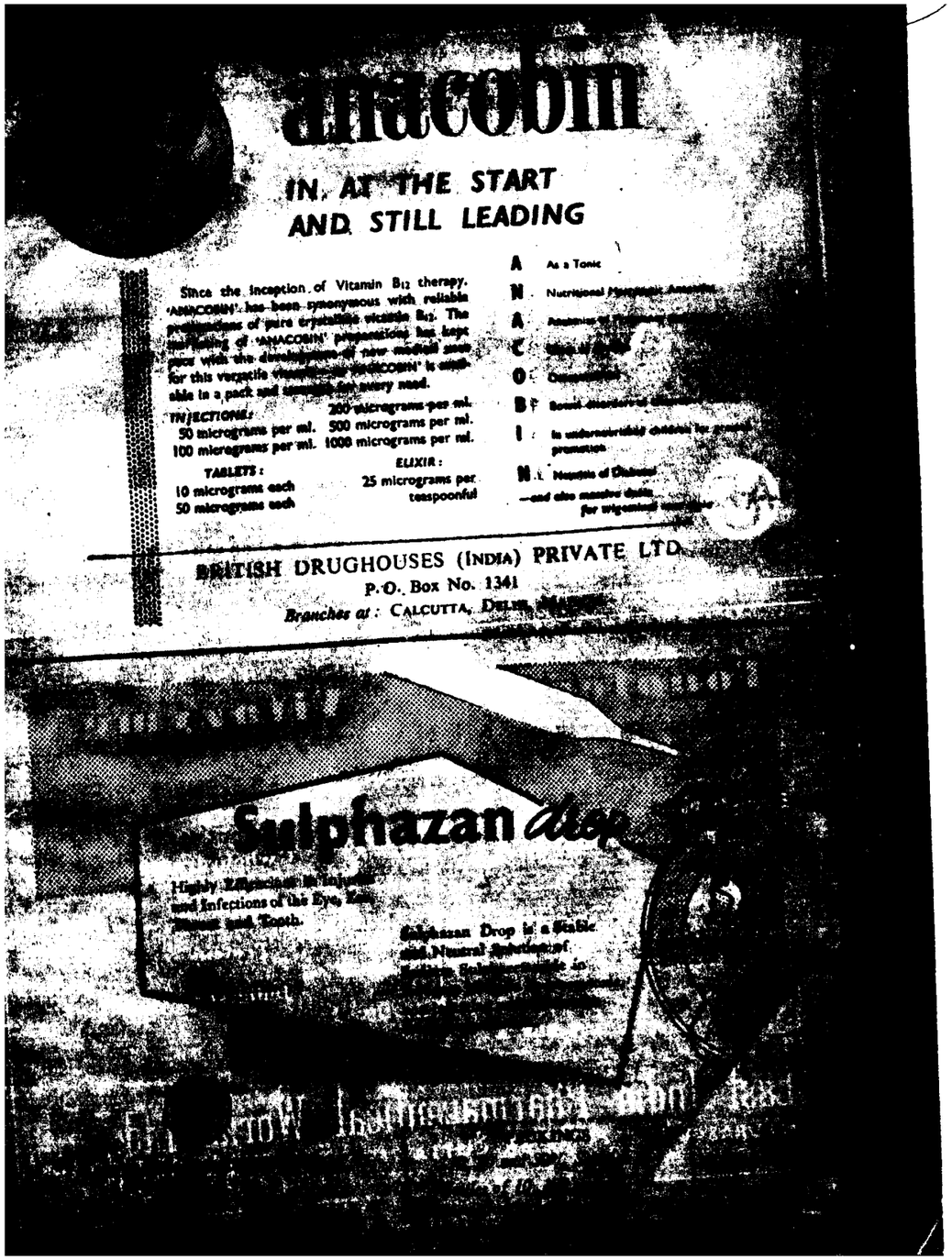

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Ocular Headache with Special Reference to Heterophorias and Convergence Insufficiency*

By

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Headache is undoubtedly the commonest and one of the most distressing and least well understood ailments within entire realm of medicine. It is an annoying symptom of many diverse derangements of human body so because of its diverse and varied pathogenesis, may present in many guises. I do not propose to bother you all with the long list of the causes of headache but I will only discuss in brief some of the important ophthalmic causes of headache and facial neuralgias, because ophthalomologist receives more than his share of patients complaining of head pain or "Mur Pirata Hai"

Headache accompanied by blurring or running together of letters, more marked in the evening after day's work and associated with state of irritability and mild congestion of eyes may be due to refractive errors, anisometropia, anisokonia and insufficiency of accommodation and convergence. The mechanism of this headache associated with eyestrain is probably muscular in origin and a close parallel can be drawn between it and cardiac pain. Headache due to eyestrain is a referred pain and may be localised to frontal, temporal, vertical or occipital regions; while overstrain of cardiac muscle produces angina which spreads over the shoulder, down the arm and up the neck. Moreover the pain can be boring dull, sharp, shooting and lancinating in character.

* Presented before the M. P. Medical Conference held on 26th Oct. 1958 at Allahabad.

Out of refractive errors—hypermetropia, anisometropia and astigmatism are most potent causes of eye strain and Headache. It is always the small error where eye tends to compensate and rectify by muscular effort, bring on nervous and muscular fatigue with its attendant train of reflex and referred symptoms. So examination under a mydriatic is very essential and prescription of suitable glasses will give an unexpected and dramatic relief.

I must impress upon you all that prescription of glasses should not be merely a matter of placing lenses in front of a patient and ordering those which give him the highest visual acuity as is done by many general practitioners and all the opticians. It is indeed an admitted fact that more harm is done to the eyes by using wrong glasses than by avoiding them. It is a great pity that there is no legislation to bar these unqualified opticians from prescribing glasses. Their duty should only be to dispense glasses correctly and have proper centring. The prescription of glasses should be the duty of the qualified eye specialist as it is a delicate and complicated operation and demands not only sound knowledge of the optical system of eye but also one has to take into consideration the individual's re-fractive peculiarities, accommodative power and condition of its associated muscles.

Anisokonia or inequality in the sizes of the images on the two eyes is another cause of eyestrain and headache. Recently it has been determined by a simplified apparatus called Eikonometer

that nearly 2% of the unexplained cases of headache and eyestrain with normal refraction and muscle balance are due to this and can be duly relieved by special isekonic lenses.

Heterophorias particularly convergence insufficiency is a potent and important cause of eyestrain and headache and often overlooked by many eye surgeons. I have come across many such patients who have consulted various eye specialists but were given a new pair of glasses each time and some vitamin preparation particularly Vitamin B while their actual trouble was due to convergence deficiency and were relieved by a course of orthoptic exercises. It is probably the most satisfactory to treat of all the types of latent deviations and for which the patient is truly thankful.

A condition of absolute convergence insufficiency is said to exist if near point of convergence is greater than 9.5 cm. from the apex of the cornea or if there is difficulty in attaining 25° of convergence. But clinically more important than this is Relative insufficiency for a given distance because nearly 2/3rd of the total convergence should be in reserve.

The aetiology of convergence insufficiency is often not well ascertained. Anatomic conditions, delayed development of function, visual disturbances and general disease or debility have been implicated. Myasthenia gravis or history of head injury may be there to help the diagnosis. It can occur in the presence of orthophoria, Esophoria and more commonly with exophoria. In this condition the eyes usually are normal and although refractive error may be elicited but generally this is insignificant. Of the 50 patients treated herein 22 were myopic 11 were hypermetropic and 17 were practically emmetropic.

Marked anisometropia was not present in any case reported above. The patients usually complain of headache and discomfort on near work. The eyes may ache, the eyelids may feel heavy and there may be diplopia or blurring and running together of letters. The *on set* is not uncommonly associated with debility or during convalescence from febrile illness because of decompensation. Squamous blepharitis was noted only in one patient.

To have its proper treatment by orthoptic exercises, it is necessary to investigate the case fully by cover test, Maddox rod, Maddox wing, Major Amblyoscope, *Livingston* Binocular gauge (N.P.C.) etc. Moreover refractive error should be first corrected and attention should be directed towards any organic or functional disease or abnormality present. While prescribing glasses astigmatism and myopia should be fully corrected and hypermetropia or presbypia, slightly under corrected. If vertical muscular imbalance exists it should be corrected by either prisms or surgery. Orthoptic exercises twice a week supplemented by Home exercises of pin or finger nose test., PHYSIOLOGICAL Diplopia, and later on by stereograms will relieve the symptoms of many patients. If supression is present on major *ambly-oscope* it should be treated. Out of 50 patients 34 patients became completely symptoms free (8 Sittings) and six patients improved, while 10 patients did not improve because they did not make real and serious efforts at home.

If there is no improvement after an adequate trial of orthoptic exercises, prisms may be tried for near work. These really should be the minimum not usually exceeding 1/3rd. of the deviation. In one of the old and rundown cases I had to undertake surgery (Resection of one Medial Rectus muscle).

Glaucoma is an important ocular cause of headache and the diagnosis is quite obvious in acute congestive stage though it may be confused with Iridocyclitis and sometimes with conjunctivitis. It is not the place hereto describe various points of differentiation but error of mistaking iritis from acute glaucoma is the most serious which can be made because treatment of these two is diametrically opposite. Dilatation of pupil with Atropine which is absolutely necessary in iritis is the worst possible treatment for glaucoma. But more important than all this is to diagnose acute congestive glaucoma in its predromal stage or ch. simple glaucoma in its earliest phase. For all this we should have glaucoma clinics in every district to diagnose such cases by various detailed examination and provocative tests. Moreover we should make our innocent public conscious about it because any eyesight lost in this disease is lost for ever and we can only check further deterioration by medical or surgical

methods. A myth in our country that eye operations are performed during winter only has to be completely exploded.

Herpes Zoster ophthalmicus (Idiopathic or Secondary) causing severe neuralgic pain along the 1st. Division of trigeminal nerve *connot* be missed when accompanied by typical vesicular rash and ocular lesions (Conjunctival congestion, Keratitis, neuritis and Nerve palsies etc), but the diagnosis of the disease may be marked in the initial stages before these vesicles have appeared. I can easily recall a patient seen urgently for indefinite neuralgic pain and discomfort round and in the eye referred to me as a case of glaucoma which ultimately turned out to be a case of Herpes Zoster when following day diffuse areas of erythema on the forehead proclaimed the true diagnosis. This sequence of events I believe is well recognised by the physicians in infective Zoster affecting the chest wall. The initial pain usually lessens with the appearance of the skin rash.

More important than this is intense post-herpetic Neuralgia which may persist indefinitely and may lead to severe depressive and even psychotic manifestations. Fortunately this occurs only in a minority of patients usually the elderly and paticularly those with severe scarring of the skin. The affected skin area also shows varying degrees of sensory depression presenting the characteristic picture of anaesthesia dolorosa. Occasionally this post-herpetic pain may be of a more spasomotic nature, in this way resembling painful tic.

Sinuses which are our immediate neighbour are very often responsible for headache which is worse in the morning and improves as the patient moves about. Charlin's syndrome due to inflammatory involvement of Naso Ciliary nerve causing unilateral Rhinorrhoea, pain in the same side of the nose and in the skin and conjunctiva round the medial canthus and occasionally conjunctivitis and iritis (IRITIS) must be kept in mind. It may be said that diagnosis can be clinched and pain relieved by cocainization of the anterior nasal fossa. Similarly Sluder's neuralgia probably due to inflammation of the spheno-palative ganglion in pterygo-palative fossa possibly secondary to infection of the sphenoid or ethmoid sinsues causes diffuse neuralgia located mostly around the orbit, root of the nose, jaws and even mastoid region presenting the picture of lower half headache by which the condition is sometimes known and can be relieved by sensory block of spheno-palative ganglion.

Temporo-mandibular Nauralgia (Costen's syndrome) is a rare and ill defined condition thought to be due to changes of traumautic origin in the joint following for example extraction of molar teeth or insertion of an anaesthetic, gag etc., causes Neuralgic pain felt throughout the distribution of the trigeminal nerve initiated by the movements of the Jaw.

Temporal arteritis an inflammation of temporal, cerebral and retinal vessels manifests itself by general symptoms and headache which may be dull, sharp or even stabbing in character mostly along the occipital and temporal vessels.

The temporal vessels are usually tender. So it is always advisable to palpate temporal arteries in all cases of occlusion of retinal vessels and in acute Retrobulbar Neuritis. The treatment is of course Symptomatic and local injection of Procaine or excision of a part of the inflammed temporal vessel may very often relieve the pain. While cortisone and PREDINISONE therapy have somewhat changed its gloomy outlook.

Recently I came across a rare case of pulseless disease or Takayasu's disease in a young woman who was having of severe headache with absence of radial pulse arterio-venous aneurysms round the pale optic discs and attacks of vertigo and syncopy etc. This case presented all the features of this disease and is still under our observation. She has now developed cataract in one eye while other fundus is pale and vessels can't be traced beyond a little distance from the optic disc around which are many small fine, Arterio-venous dilatations.

Migarine or Hereditary hemicrania attributed to spasm followed by vasodilatation of cerebral arteries is quite common cause of headache about which you all know so well. It is of ophthalmic interest not because that it causes prodromal aura with visual disturbances (Fortification spectra and Homonymous hemianopia etc.) but sometimes refractive errors and eyestrain act as a predisposing factor and worsen the attacks, so prescription of glasses may relieve the strain and lessen the attacks. Before giving treatment, this hereditary form must be differentiated from symptomatic and opthalmoplegic *migraiene* which is always on one particular side and may show transitory nerve palsies. Among other ocular causes of headache and neuralgic pain are acute infections of the lids and lacrimal sac in the initial phase, episcleritis, particularly when hidden by the lids, daoryoadenitis and orbital periostitis.

Space occupying lesions of the cranium leading to increased intracranial pressure are indeed very important causes of headache and ophthalmologist very often has to deal with these cases because of many ocular and visual symptoms of Cranial palsies with diplopia and changes in the visual field etc. Quite often we have to draw the attention of the physician and send the cases to Neurosurgeon for operation. Lesions around the chiasma particularly intra, supra and perisellar tumours and aneurysms of internal carotid artery are all of particular ophthalmic interest. Cases of arteriosclerosis and malignant hypertension are often first seen by us and referred to physicians for treatment.

It is quite obvious from the above description that eyes constitute an important cause of headache, so no case of headache should be treated empirically unless we have ruled out the possibility of its ocular origin.

RESEARCH & LABORATORY NEWS.....

IMPORTANT BREAK-THROUGH IN PENICILLIN FIELD

Many thousands of new penicillins, which can be "tailor-made" to cure a wide range of specific diseases, are likely to be evolved from a new British scientific discovery just announced in London.

The discovery is 6-aminopenicillanic acid-a basic molecule of penicillin. This substance, says Professor Ernest B. Chain, Nobel Prize winner and co-discoverer in Britain (with Sir Alexander Fleming) of penicillin, could lead to "the most important break-through in the penicillin field since the discovery of penicillin's curative power."

Four young scientists at the research Laboratories in Surrey of the Beecham Group Ltd. have been working for the past three years to isolate this molecule. Leprosy and tuberculosis are among the many diseases which are now being experimentally attacked with this new antibiotic—at present on live animals.

The director research at the Beecham Laboratories, Dr. J. Farquharson, said in London on March 6 that his company had decided to concentrate its research upon one proven antibiotic—penicillin—and, by marrying the sciences, of microbiology and chemistry, to produce new penicillins. The nutural fermentation method which had been generally used hitherto in the search for new antibiotics could never produce this result, he said.

After much research, he said, and following an unusual result during the scientific investigations, a brew was made which was found to contain intact a penicillin-like material.

The company's scientists had managed to extract this material—"a raw material from which we could get almost any penicillin we cared to make."

PROMISE OF SOLUTIONS

There were three urgent problems for which the new development offered promise of a solution, said Dr. Farquharson. These were: the widespread existence of staphylococci resistant to known penicillins; finding a weapon to combat Gramnegative organisms causing intestinal disorders, typhoid, and other such diseases: and the development of new penicillins which could be used to treat patients who were allergic to existing ones.

Because of the relative non-toxicity of the known penicillins, "the field seems wide open to our researchers and, in fact, we have some very promising leads in these directions." said Dr. Farquharson. He added that further research of considerable magnitude would be needed to exploit the discovery as quickly and as fully as possible.

The Chairman of the Beecham Group, Mr. H. B. Lazell, added: "We have received direct approaches from—with two exceptions—every large American pharmaceutical company engaged in antibiotic production and research. When one remembers the vast American research effort in microbiology, it is, indeed, gratifying to know that we have achieved something that has eluded them." He hoped to make an announcement soon about research and development collaboration with a large American company in the antibiotic field.

An article on the discovery in the British scientific journal Nature concludes: "The substance is...a most useful chemical intermediate for the preparation of new penicillins and related compounds which are not easily accessible in other ways."

The current issue of the medical journal Lancet describes the discovery. and adds: "The consequences could be far-reaching."

(Courtesy Indian Express, Madras)

*Cardiac Emergencies in General Practice

'SYNCOPE'

By

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A sudden decrease in the volume of blood in active circulation produces acute circulatory failure which includes from the mildest form of simple syncope to the most profound degree of collapse, shock, coma and sudden death. The failure of circulation is caused either by loss of blood or plasma or by disorganisation of vasomotor mechanism producing failure of peripheral resistance there-by stagnating large quantity of blood in the arterial or venous reservoirs. This reduces the venous return to the heart and decreases the output per beat, arterial pressure falls, producing acute cerebral anoxia leading to syncope. If anoxia persists longer other syndromes like shock, collapse, coma and even death results. So syncope, shock or collapse differ from each other only quantitatively (Weiss). The treatment is directed to the correction of exciting cause and restoration of an adequate blood volume in active circulation to the vital centres.

Syncope is defined as a transient loss of consciousness due to acute decrease of cerebral blood flow for a few seconds.

Physiology of Syncope: Cerebral blood flow depends on the supply pressure and the vascular resistance of cerebral blood vessels. The latter factor is unimportant except in cerebral syncope. When supply pressure or arterial pressure is below 50 mm. Hg. for few seconds syncope occurs. The arterial pressure depends directly on the cardiac output and the total peripheral resistance. To produce syncope either the cardiac output or the peripheral resistance must decrease or both decrease simultaneously to reach syncopal level. In normal subject, however, these two factors work in opposite direction by means of baroceptor reflexes (Carotid sinus, aortic arch and other regions classified together under the term baroceptors), so that within the range of normal living the supply pressure to vital organs including brain is kept constant. With the nervous system intact, increased pulse pressure causes peripheral vasodilatation, while decreased pulse pressure causes vasoconstriction. The time of these responses, however, is little different, while a single large beat can be shown to cause vasodilatation, a single small beat does not cause measurable vasoconstriction. Several beats are needed and the time 5 to 6 seconds is long enough to result in syncope.

* Paper read before the I.M.A. Sitapur Branch.

Classification of Syncope: The syncope may be of the following types:—

1. Cardiac Syncope,
2. Vasomotor or Vasovagal Syncope,
3. Cerebral Syncope,
4. Anoxic Syncope.

Before discussing the cardiac syncope I shall lay below few lines about the other types of syncope.

In Vasomotor Syncope: The cerebral blood flow fails as a result of sudden fall in arterial blood pressure due to collapse of the peripheral resistance operating through the nervous pathways i.e. due to disturbances of the autonomic nervous system. This includes the common faint. It may be initiated by the following causes:—

(a) Producing critical fall in central venous pressure e.g. Haemorrhage loss of plasma outside or inside the tissues; orthostatic hypotension and postural hypotension (Fainting in soldiers on Parade, fainting after lumbodorsal sympathectomy, fainting in pregnant women while lying on their back due to compression of inferior vena cava.

(b) Chemical agents causing sudden profound vasodilatation i.e., acetyl choline, histamine, tetraethylammonium or hexamethonium compounds, nitrites etc.,

(c) Stimulation of receptors exciting vaso-vagal reaction e.g., acute emotional distress, hypersensitivity of carotid sinus, extreme pain, sudden visceral catastrophy like pleural shock.

TREATMENT

Prophylactic: The attacks may be prevented by avoidance of emotional strain, correction of the psychological conflicts, stripping of the coats of carotid artery on the side of more sensitive sinus or by adequate employment of local anaesthesia when surgical measures are contemplated. In postural hypotension, attempts are made to prevent the aetiological causes. When this is irremediable, permanent symptomatic relief may follow the use of 8 to 30 mgm. of Ephedrine (1/8 to 1/2 gr.) thrice or four times a day. Mechanical support to the splanchnic area in the form of suitably padded elastic belt, and development of increased muscular tone by a course of abdominal exercises are measures employed to supplement drug therapy. Force of gravity puts a burden upon the fine adjustments of the circulatory mechanism. Individuals prone to faint discover this fact and when the attack threatens, they frequently lie down or adopt a sitting posture with trunk flexed and head thrown forward between the knees. Hot stuffy rooms; long hours in the erect posture without food; exposure to cold and damp are factors which predispose to syncope in susceptible persons and should be avoided.

In actual faint loosening of clothes about the neck and chest and placing the patient prone or on his back, elevating the feet some inches from the ground and massaging from periphery towards the heart thus fascilitating the venous return, are effective measures

Profound bradycardia and hypotension of reflex origin in severer cases may be corrected by the subcutaneous administration of 0.25 to 0.5 ml. of adrenaline. When consciousness is regained hot drinks are serviceable.

Cerebral syncope occurs either when there is cerebral vascular spasm or transient occulsion. The fault is local. The hyperventilation syncope is due to lack of carbon dioxide producing cerebral vasoconstriction. This induces dizziness within a minute in most normal individuals undergoing forced breathing and if it is maintained long enough syncope occurs. Spontaneous attacks are seen in hysteria and sometimes in encephalitis lethargica.

Anoxic Syncope: Most causes of anoxia leads to asphysia or coma, but syncope however may occur in congenital heart disease with right to left shunt especially in Fallot's tetralogy. This is attributed to diminish peripheral resistance, increased pulmonary resistance, or a fall in total cardiac output.

Tussive Syncope results after a prolonged attack of coughing and is due to failure of cardiac filling by an extremely high intrathoracic pressure which obstructs the venous return at the thoracic in-let.

Cardiac Syncope: Occurs when heart either due to its own defect or defect of bigger vessels fails to maintain adequate cerebral blood flow. It is caused by the following conditions:

1. Cardiac stand-still vagal inhibition,
2. Ventricular asystole-(Stokes-Adams fit),
3. Ventricular fibrillation;
4. Ball valve thrombus or pedunculated myxoma,
5. Paroxysmal rhythm change with extreme rapid ventricular rate.
6. Cardiac compression from haemopericardium,
7. Massive Pulmonary embolism,
8. Aortic Stenosis,
9. Low Cardiac output states.

In first four conditions, the loss of consciousness is abrupt without any warning. The patient is grey or white, flaccid, pulse-less. Heart sounds inaudible, respiration may continue. In 10 to 15 seconds anoxic twitchings begin and may develop into convulsions if attack lasts long enough. If no recovery within two minutes, death usually results. Cardiac stand-still and Ventricular asystole usually recover within 5 to 20 seconds, but ventricular fibrillation is usually fatal. Ball valve thrombus and pedunculated myoma are rare. Return of consciousness is abrupt and complete followed by a vivid flush.

In paroxysmal tachycardia with ventricular speed over 300/m, the heart has no time to fill or empty properly so both cardiac output & B. P. fails. This may lead to syncope.

Cardiac compression must be gross to reduce the cardiac output sufficiently to cause loss of consciousness. This is present in haemopericardium due to rupture of an aneurysm, dissecting or saccular or perforating wounds of the heart, or rupture of the ventricular aneurysm.

Massive Pulmonary embolism cause syncope when more than 2/3 of circulation is blocked. The onset is sudden and may be preceeded by pain or tightness in the chest. The unconsciousness may last for hours and recovery is only partial, extreme faintness persisting. During the attack patient is limp, grey, sweating and breathless. Pulse is thready, heart sounds faint and B.P. low.

Small emoli may cause reflex syncope. In aortic stenosis the syncope is vasomotor nature.

The cardiac output varies with B.P. and inversely with the total peripheral resistance (C. O. = $\frac{B.P.}{R}$). So in low fixed cardiac output states e.g., severe aortic, mitral, pulmonary, or tricuspid stenosis, and severe pulmonary hypertension as the cardiac output cannot rise, the B. P. must fall producing syncope. Syncope on effort in certain obstructive lesions of circulation e.g., primary pulmonary hypertension and severe pulmonary stenosis with closed septa is due to acute right ventricular failure (Howarth & Lowe 1953).

Stokes-Adams attacks: Syncope due to ventricular asystole occours in 50% of cases of complete Heart Block and is especially common when partial block becomes complete. The clinical history is very interesting. The loss of consciousness is abrupt, without any warning. Patient lies limp, still and pale, unconscious with or without convulsion. On examination there is no pulse, pupils widely dilated, not reacting to Light. Breathing is present. The severity depends on the duration of ventricular asystole. If only for few seconds patient feels a "light Wave" and comes over it. If longer he becomes unconscious. After 10 seconds twitchings commence leading to convulsion and if the ventricular asystole continues for mor than 2 or 3 minutes recovery is rare.

As a rule, ventricle starts beating after a few seconds and consciousness returns abruptly and patient may feel perfectly well and be ready immediately to resume his former state. Patient does not remember what has happened. His skin is flushed. When the attacks occur in bed, patient only exhibits flushing of the skin. The events of these attacks are so typical that the diagnosis can be made on the history alone.

Second cause of this condition as reported by S. Robin and others is where the underlying mechanism is ventricular tachycardia or ventricular fibrillation or ventricular asystole alternating with rapid ventricular rate.

PHYSIOLOGY :

The stokes-Adams attacks are due to depression of the idio-ventricular pacemaker in cases of complete heart block. Ventricles stand still while auricles continue to beat. They are likely to occur when partial block becomes complete either due to depression of pace-maker by the depressive influences on the conduction or due to the initial sluggish nature of the idio-ventricular pacemaker. When complete heart block is well established attacks do occur but are less common.

Abrupt loss of consciousness is due to sudden total failure of cardiac output. Twitchings are due to cerebral anoxia, convulsions are due to (1) anoxia (2) Co_2 depletion in the blood stagnant in the lungs during asystole, and vasodilatation produced by tissue metabolites. On resuming the ventricular beating blood rich is O_2 without CO_2 is thrown abruptly into the widely dilated vascular bed.

Complication: Attacks of paroxysmal ventricular tachycardia or ventricular fibrillation (Parkinson, Papp & Evans. 1941).

Prognosis: It is much worse, majority of them dying suddenly.

Treatment: The most effective prophylactic treatment in cases where ventricular asystole is the cause, is oral administration of Ephedrine 1/2 gr. (30mg.) t.d.s. If attacks are frequent and patient is bed ridden, adrenaline 0.5 mg. (0.5 ml. of 1: 1000 solution) injected subcutaneously and repeated 2 to 6 hourly.

In one of my cases the underlying cause of the attacks was ventricular asystole, the patient got an attack of coronary thrombosis and developed heart block. He was treated with anti-coagulants and ephedrine. He showed good recovery and the attacks ceased on the continuous treatment with ephedrine 1 tab. (gr. 1/2) t. d. s. orally; He continued taking ephedrine tablets for six months and on check up it was found that he had developed hypertension. He was given Serpina tablets along with ephedrine. This treatment he continued for about two years. Ultimately he got a severe attack at home and died. Intracardiac adrenaline did not help.

Sublingual Isoprenaline 20 mgm. is helpful, (Nathanson & Miller 1949) but noradrenaline has very little effect (Nathanson & Miller 1950). Both ephedrine and adrenaline prevent undue depression of ventricular pace-make and encourage the heart to beat a bit faster. Barium chloride is poor substitute for ephedrine. There are rare instances in which adrenaline has to be injected directly into the heart during a prolonged period of asystole, as under such circumstances a subcutaneous injection is useless blood flow being ceased. A fine needle about 3 inches long attached to a syring containing 0.25. ml of adrenaline (1:1000 solution) is inserted into the fourth intercostal space about one finger-breadth from the sternal border. This site avoids the mammary artery and coronary-artery branches. About 2" of the needle inserted. Needle is in the ventricle, & blood is withdrawn to be sure. Adrenaline is injected into the ventricle.

When the attacks are severe and occurs in quick succession adrenaline may be given intravenously 0.5 ml. of 1:10,000 solution (0.5 ml. of 1:1000 diluted by addition of 4.5 ml. normal saline and taking 0.5 ml of this) slowly.

When repeated seizures are partly due to paroxysmal ventricular tachycardia or ventriculr fibrillation the problem arises whether the unconsciousness is due to asystole or to fibrillation. Adrenaline is hazardous as it

encourages the latter rhythmchange. Both quinidine and procaine amide should be avoided as they are likely to cause ventricular stand-still (Miller et. al. 1952, Schwarz et. al. 1952-1953). Sublingual Isoprenaline 20 mgm. is recommended by Schumacher & Schwock 1954. S. Robin & others (1955) reported two cases of stokes-adams syndrome in which the underlying mechanism was ventricular tachycardia or fibrillation and consider that this is the drug of choice. Treatment of the primary cardiac condition may help.

In special clinics external *electric pacemaker* is used to tide over the critical period. The machine delivers an electric shock of 75 to 150 milliamps at 45 to 100 volts for two or three milli seconds, 60 to 90 times per minute. The negative electrode is placed over the apex and the positive on the opposite side of the chest posteriorly (Zoll, Linenthal & Norman 1954.)

Other measures are inhalation of 1:100 adrenaline solution; full doses of atropine, thyroid extract and complete digitalization.

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Treatment of Myocardial Infarction*

By

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Treatment of myocardial infarction is undoubtedly of interest to any physician and is still one of the most difficult topics in medicine. Before discussing it, we should define which are the aims of this treatment. They are the following. First of all, obviously, *the physician tries to remove the pain*, not only for humanitarian purposes, but because pain may have unpleasant repercussions on the cardiovascular system. Second, *he tries to put "at rest" the heart.* Obviously this would be attained only by extremely deep hibernation or anesthesia. Neither of them is done routinely, but attempts have been made in this direction. Even though "complete rest" of the heart is impossible, a "relative rest" is useful. Therefore, we try to avoid any unnecessary cardiac strain for a certain time. Third, *he tries to decrease the severity of reflex stimuli* arising in the infarcted area and in the heart in general and reaching the vasomotor center and other centers of the medulla and various centers of the middle brain. Several changes of temperature, blood pressure, heart rate, G-I function, respiration, and metabolism are due to these reflexes. Most important, ectopic rhythms may by caused by them. Fourth, *he tries to decrease the excitability of the myocardium* surrounding the infarcted area, again trying to avoid ectopic rhythms, particularly ventricular fibrillation. Fifth, *he tries to prevent the formation of clots* because mural thrombi, common in myocardial infarction, may be the source of thromboembolic phenomena. Sixth, *he tries to prevent shock*, one of the most common causes of death, and *to treat failure*, especially if complicated by *pulmonary edema.*

What is the purpose of prevention of pain and anxiety? As already said, pain should not be tolerated for humanitarian purposes, and because the patient is seeking its relief. The stimuli causing pain are possible causes of reflexes. This is why removal of the pain is actually useful to the patient, apart from sedation. This aim is obtained routinely with the injection of *morphine sulfate*, to which often is added *atropine sulfate*. Usually 1/6 to 1/4 of a grain every four hours plus 1/250 of a grain of atropine are given in the first one or two days. The dose depends partly upon the severity of the pain and how effective is the relief. If the pain is relieved by the first injection, the patient can wait for the next injection 6 to 8 hours. If the pain is not immediately relieved, a second should be given within an hour. On the other hand, patients with myocardial infarction are at times treated with such large

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doses of morphine that they reach the toxic stage, something which should be carefully avoided.

Instead of morphine and atropine, one can give *Demerol*, which has both morphine-like and atropine-like effects. The dose of Demerol is between 50 and 150 mg. it can be given by injection or by mouth. However, Demerol is far less effective than morphine in relieving anxiety.

How long should sedation be continued? According to several cardiologists, when there is no more pain, sedation should be discontinued. On the other hand, sedation has other purposes, in addition to relief of pain, and should be continued even after pain has subsided.

Rest has the purpose of decreasing the danger of rupture of the heart muscle of decreasing the incidence of heart failure, and of decreasing the possible occurrence of ectopic rhythms. There are statistical studies which show that untreated myocardial infarcts, like those occurring in Insane Asylums, where they often are not diagnosed, are followed by a much greater number of deaths than treated episodes. It is likely that death of patients which were not put to bed was caused by either ventricular fibrillation or rupture of the heart muscle. In regard to arrhythmias and heart failure, there is no doubt that when greater work is performed, the left ventricle fails more often and presents ectopic rhythms with greater frequency. This is why rest should be as complete as possible and prolonged.

Is it advisable to obtain the most complete possible rest of the heart muscle? In the late 30's, a Viennese surgeon tried to keep patients with a myocardial infarct under deep *anesthesia* for several days. The results were said to be very good. One might consider that the danger of producing shock is increased because obviously there will be a poor venous return, but apparently this is compensated by the lower requirements in oxygen by the tissues. A few years ago, French researchers advocated *hypothermia* for patients with myocardial infarct. and even treated cases in shock. Excellent results were claimed. The patients were kept under hypothermia for two to three days, then gradually rewarmed. After recovery, apparently shock had disappeared. Unfortunately, both the studies of the Viennese surgeon and of the French authors are based on small numbers of cases and have no controls. There is no information on how many were severe cases, how many recovered, and so on. Therefore, we should consider these studies as preliminary observations, which should be followed by extensive and well-controlled observations.

The usual accepted therapy is that of *bed rest*. In this regard, it seems strange that this type of therapy has given rise to such discussions that nowadays the subject cannot be discussed in an impersonal, objective way. The reason for this is that, in the past, the patients were kept completely immobile in a supine position; they were not allowed to move their limbs or to feed themselves. At present, some physicians advise

to take the patient out of bed, put him on easy chair, and keep him there, either most of the time or all the time. Both of these extremes are exaggerations. The immobile patient will experience more frequently congestion of the lungs; he will present more often a dangerous slowing of the circulation in the venous system, due to inaction and atrophy of the skeletal muscles, and this may favor phlebothrombosis, a possible cause of pulmonary embolism. For this reason, even those who advocate bed rest either ask the patient to turn from side to side several times during the day or request the nurse to change the patient's decubitus at periodic intervals. They invite the patient to move his legs and arms whenever he wishes. Sometimes, if the course is favorable, the patient can be propped up at meal time and, instead of being fed he can feed himself.

The *chair treatment* is often impractical for the following reasons. First, if the patient is in severe pain and receives sedatives, he is only semiconscious and cannot sit up without being held. If the patient is in shock, obviously the best treatment is to have him lying down with the head at the level of the rest of the body. If the patient is in failure and has orthopnea, he obviously should be sitting up in bed. However, sitting in a chair may require greater muscular work and increase the dyspnea. Psychologically, in nine-tenths of the cases, it is bad to have the patient sitting in a chair, because it is difficult to convince him that he is not allowed to move about and to take care of his needs. This is even more serious if the patient is treated at home and there is less control over his activities. On the other hand, there would be considerable advantage to use the armchair treatment in those cases who have severe mental depression. It should be remembered that, if the armchair system is adopted, the patient should be lifted from the bed and deposited in the chair in the morning, taken from the chair and put back into bed at night. He should not be allowed to walk from the bed to the chair or to jump into bed.

In regard to bodily functions, it has been proved that the *bed pan* represents a severe strain for most patients while *the commode* represents a moderate one. Again, the patient should be lifted and placed on the commode and vice versa. He should not be allowed to do this unassisted.

How long should the bed rest period last? An individualized decision should be adopted. Patients with a severe infarct with failure, shock, or other complications, should be kept at extreme rest for at least three weeks; then, they should be mobilized very gradually according to tolerance and on the basis of possible changes of heart rate. Patients with a milder clinical picture, with a smallar infarct, and no complication, should be kept in bed only for about ten to twelve days, then in an easy chair for ten to twelve days, and then gradually mobilized.

In regard to *early mobilization*, the patient should not be placed sitting on the mattress with his legs dangling:

This causes more strain than if he sits comfortably in an easy chair. As soon as the patient can be mobilized, he should be lifted from the bed and placed on a chair while the nurse is fixing up the bed, and he can take his meal in the chair; then he can be lifted again and put back to bed. Further mobilization should consist of walking around the room once, twice, or more times according to instructions; the pulse should be taken before and after exertion in order to see whether arrhythmias arise and whether there is any lasting tachycardia.

Another point concerns *visceral reflexes*. We know that these play an important role in the first few days; for this reason, it is essential to decrease the excitability of the autonomic nervous system for several days. In the first two or three days usually this is obtained by morphine and atropine or Demerol, so that no other drugs are needed. However, as soon as the amount of morphine or Demerol is decreased, it is wise to start administration of *phenobarbital* or any other slow acting barbiturate. These, in addition to decreasing the excitability of the nervous system, will also decrease the wish of the patient to move about, get up, and increase his activity. Many of these patients, especially in the younger age group, refuse to submit to any long period of rest and wish to resume a normal activity. Morphine and atropine should be given in large doses for two to three days; then, in smaller, gradually tapering doses for another three to four days. As soon as the dose of morphine is decreased, one should start giving phenobarbital and continue it for 10 to 15 days. Sedation should not be so deep as to put the patient asleep.

Another problem is represented by the increased excitability of the myocardium. *Oxygen* by inhalation has proved to be of use and is given routinely to patients with myocardial infarction. *Rest and sedatives* help in preventing extrasystoles, paroxysmal tachycardia, atrial flutter or fibrillation, and ventricular flutter or fibrillation. Routine prophylactic use of *quinidine* has been recommended by Levine. This can be questioned because quinidine is a depressant of the myocardium and favors tachycardia. For this reason, quinidine should be given only in cases who exhibit extrasystoles and, therefore, have a greater likelihood of developing paroxysmal tachycardia or flutter, or who start presenting atrial flutter or fibrillation since the beginning. In these cases, usually one tablet of quinidine (3 gr.) t.i.d. is given, but the dose should be increased, even to twice as much, if severe arrhythmias develop. Larger doses should not be given if there is any evidence of heart failure. In cases with ventricular tachycardia or even ventricular flutter, one should prefer *procaine amide* (*Pronestyl*) given by the drip method to quinidine. If Pronestyl fails and the patient had not received digitalis, then one can try digitalization. This, because, even though digitalis increases ventricular excitability, in some cases it may be definitely of help. If, on the contrary, the patient is fully digitalized and develops ventricular tachycardia, it is wise to stop digitalization, at least for a certain number of days and try i.v. potassium with extreme care. If necessary, digitalization will be resumed after a few days.

Another problem which is still debated concerns the use of *anticoagulants*. Those who advocate the anticoagulants point out the sharp decrease of thromboembolic phenomena and the lower mortality in treated patients. A contrasting opinion maintains that the quoted figures are too high and that no important difference can be found between treated and untreated cases. It is possible that studies made in different hospitals may not be exactly comparable because the mortality of the cases studied in a City Hospital is probably higher.

In addition to those advocate anticoagulants in all cases of infarct (and even in cases of severe coronay insufficiency) and those who advise against anticoagulants, there is a third group which accepts the use of these drugs, but only in severe cases. Anticoagulants should be given, according to them, in patients above 60, in cases with massive infarcts, shock, pulmonary edema or peripheral congestion; they should be given in cases with atrial fibrillation, pulmonary infarcts, or history of thrombophlebitis. Therefore, only mild cases myocardial infarct in relatively young or middle aged patients should not be treated with anticoagulants. The difficulty arises in the fact that some cases with apparently mild episodes then present thromboembolic phenomena. Previous anticoagulant treatment might have prevented this dangerous complication.

Another important point concerns the level at which the clotting activity should be maintained. Theoretically, a very slow clotting is the ideal, but undoubtedly it may cause some complications. For this reason, the ideal level is between 25 and 30% of coagulation, as revealed by the prothrombin time and other laboratory tests.

According to most authorities, it is best to start treatment with a rapidly acting anticoagulant like *heparin*, which should be given intravenouly by the drip method. One dosage which can be recommended is 100 mg. of heparin in 500 cc. of 5% glucose, injected at the rate of 20 drops per minute for 12-48 hours. This slow rate is recommended in order not to overload the cardiovascular system of patients who have a potentially failing heart. Otherwise, 100 mg. can be given intramuscularly every twelve hours for 12-48 hours. Heparin prolongs clotting time, but fails to modify prothrombin time. Together with heparin, a slower anticoagulant is given by mouth, so that, when heparin is discontinued, the oral drug will start acting. In this regard, it should be kept in mind that *Dicumarol* initiates its effect after 48 to 72 hours, *Tromexan* after 18 to 24 hours and *Warfarin* after about 12 hours. According to which of these drugs is given, heparin is discontinued after 48, 24, or 12 hours, as soon as laboratory tests indicate that it is not needed any more.

Patients may present mild epistaxis or hematuria, or occasional petechiae. When this happens, it may be advisable

to decrease the dose of the anticoagulant.

If one is using Divumarol, he should keep in mind that any change in dose will be reflected by clinical and laboratory changes which take place 2 or 3 days later; therefore, there is no need for frantic adjustments which are sometimes done and which only reflect the impatience of the physician.

Tromexan is definitely more difficult to handle than Dicumarol. On the contrary, Warfarin is very easy to administer because, even though its effect starts much earlier than that of Dicumarol, it lasts as long as that of the latter.

The dose of Dicumarol is from 200-400 mg. the first day, then about 200 mg. on the second, and then, once the balance has been reached, it may vary as much as from 50 to 200 mg. With Tromexan, about four to five times larger doses should be given. On the first day, from 1,000 to 1,200 mg., then a daily dose of 200 to 600 mg. Warfarin requires a smaller dose, i.e., 50–75 mg. on the first day, while the maintenance dose is 5-15 mg. daily.

One of the contraindications to the use of anticoagulants is the existance of an active gastric or duodenal ulcer, a pulmonary cavity, or bleeding hemorrhoids. This is why it is always necessary to inquire about a previous history of gastrointestinal disturbance. In order to obviate possible dangers, one should have available vials of *Vitamin K*. From 30 to 80 mg. of this drug are injected whenever there is evidence of hemorrhage.

Another problem is represented by *heart failure*. There have been numerous discussions about treatment of this complication. There is no doubt that, apart from a different decubitus and from the administration of oxygen, these patients may require stimulants, in other words, digitalization. Knowing that *digitalis* in large doses or given too rapidly may cause ectopic rhythms, a gradual digitalization should be preferred, so that complete digitalization is obtained in 36 to 48 hours. Then, it is possible to see whether the heart tolerates a certain dose without reacting untowardly. Another precaution which is advisable, is to use a rapidly eliminated digitalis preparation like *digoxin, Cedilanid, or Lanoxin, or acetyl digitoxin (Acylanid)*. If, during the adminstration, one decides to discontinue the drug, this will be eliminated within a few hours and the undesired effect will rapidly disappear.

Whether or not digitalis should be given when the patient is in *shock* is a problem which has not been solved. Experimental studies in animals seem to prove that digitalis decreases the severity of shock. On the other hand, there is experimental and clinical evidence that, in the first few hours of digitalization, digitalis may cause a decreased cardiac output, a fact which might increase the danger of shock. If one patient requires both digitalis and quinidine, there is no contraindication and one can give the two drugs with safety.

Shock is one of the most dangerous complications. According to various statistics, at least 80% of patients with myocardial infarcts who go into shock die, and the best that has been obtained by means of prompt and wise therapy is to cut down this percentage to about 40%. *It is very important to start treatment as early as possible*. An interval of six hours in the onset of therapy may mean the difference between life and death.

The danger of impending shock is largely evaluated on the basis of the blood pressure level. This, again, should be compared to the habitual level of pressure of each patient. If the patient had normal or low blood pressure, a systolic level of 80 may represent the borderline; below this, one should start preventive measures. On the other hand, if the patient had high blood pressure, a systolic pressure of 100/110 should be accepted as evidence of impending shock. Two remedies so far have proved effective. One is the *infusion of fluids*, the other is treatment with *vasoconstrictor drugs*. In regard to the infusion with physiologic glucose solution, plasma, or plasma replacements, it should be kept in mind that, if the patient is infused too rapidly, he will present *pulmonary edema* which may be fatal. All the remedies which improve shock unfortunately favour pulmonary edema and vice versa. This is why, in case of shock, the physician should not leave the patient for several hours and should watch him every few minutes. As soon as evidence of pulmonary edema is obtained, the rate of infusion should be decreased or the latter discontinued for some minutes. In regard to the rapidity of infusion, it should be slower than for the average surgical patient in shock. In general, between 12 and 16 drops per minute are given.

Another effective remedy is represented by vasoconstrictors, the best of which seems to be *norepinephrine* (Levophed.) This drug still has some effect in increasing the excitability of the myocardium, but much less than epinephrine.

If the patient is not in shock as yet and one wishes to be sure to prevent it, then one can use *mephentarmine* (*Wyamine.*) This is a milder drug, has absolutely no cardiac action, and causes a moderate rise in pressure. In regard to doses, norepinephrine can be given in dose of 2 mg. per 500 cc. of fluid and nephentermine in dose of 30 mg. in 500 cc. The same dose can be repeated after four or five hours in both cases.

Once the shock has occurred, the chances of recovery are slim. This is why it is better to prevent it than to try to reverse the trend.

The studies which have been made a few years ago about *intra-arterial administration of fluids* have not been confirmed. Therefore, intravenous infusion is still used. In regard to other measures, it should be kept in mind that morphine, phenobarbital, venesection, application of tourniquets, and pressure respiration, all drugs and maneuvers which are used in pulmonary edema, decrease venous return and may precipitate shock. The same applies to mercurial diuretics. Therefore, if the patient is in shock, the amount of morphine should be decreased.

Another problem is represented by *paroxysmal dyspnea* and *pulmonary edema.*

Paroxysmal dyspnea may be treated in some cases with intravenous *aminophylline* as long as the injection is done very slowly. If there is no shock, morphine can be given in the customary doses. However, the patient with infarct usually already receives this drug, and an increase in dose might be detrimental.

Several studies were directed to the problem of what can be done to alleviate pulmonary edema without aggravating shock. *Intermittent pressure respiration*, even though it somewhat decreases venous return, seems to have been followed by good results in cases with pulmonary edema and shock. A physico-chemical method is called *antifoaming* or *defoaming therapy*. This was described by the author in 1950 and is based on the use of alcohol-oxygen vapor by inhalation. In the doses to be employed, it causes only moderate sedation and moderate vasodilation. The most important effect is modification of the surface tension of the foam which is followed by a decrease of its volume. It is a symptomatic treatment, which, however, may allow more oxygen to penetrate into the alveoli, prevent the dangers of anoxia, and interrupt the vicious circle by which hypoxia causes more pulmonary edema and edema causes more hypoxia. Enabling the patient to survive, more time is given to try any other remedy or procedure. Following alcohol vapor, other antifoaming agents have been advocated including *silicone aerosol*. In our experiments, we found silicone less effective than alcohol vapor and more difficult to administer.

Nitroglycerine and other vasodilators are without effect. The theoretical postulation that, by using them, we can favour an increase in the collateral circulation of the infarcted area has never been proved. More-over, there may be danger in their early use. Therefore, it is preferable not to use them until the patient is in convalescence; their use will be dictated by the recurrent precordial pain and not by a theoretical knowledge that it might help in collateral circulation.

How long should we continue therapy? Bed rest, if there are no serious complications, should last between two and four weeks and should be followed by a slow convalescence of about two weeks in the house and, if possible, two to three months before starting again full occupation. In regard to anticoagulants, as far as the complications are concerned, three weeks are usually sufficient. However, based on other considerations, the anticoagulant treatment should be continued for several months or years, according to the clinical picture, the age of the patient, the need for periodic blood tests, etc. In regard to sedation, two to three weeks is the usual treatment with phenobarbital after discontinuing morphine. However, in the following treatment, there is no harm in giving some sedative at night especially in hypertensive patients. Instead of barbiturate *chloral hydrate* might be used.

Among the possible precautions to take, these patients should know what to do in case of a new attack. They should carry two or three tablets of Demerol in their pocket and should take one if a new episode of pain should occur, lasting more than fifteen minutes. Then, they should lie down and call the doctor. If the latter fails to arrive for some time, the patient would take a second tablet after one hour.

GLEANINGS FROM THE MEDICAL PRESS

CHLORPROMAZINE JAUNDICE: Clinical Course, Hepatic-Function Tests, and Pathologic Findings—Summary of Twenty Cases. W. F. Gebhart, R. A. Van Ommen, L. J. McCormack and C. H. Brown, A. M. A Arch. Int. Med. 101: 1085–1093 (June) 1958 (Chicago). (via. J.A.M.A. Sept. 6, 1958–Vol. 168, No. 1)

The incidence of jaundice in patients treated with chlorpromazine has been reported to be as low as 0·2% and as high as 5%. The resulting liver disease is regarded as a benign and self-limiting condition that can be relieved merely by withdrawing the drug, although in some instances the duration of jaundice and of abnormal results of liver-function studies may be protracted. Concomitantly, there may then develop extreme changes in lipid metabolism, which, in turn, are capable of producing hepatohy-percholesteremic cirrhosis, or primary biliary cirrhosis. The mechanisms by which chlorpromazine produces a rather specific type of liver disease are not known, notwithstanding the resemblance in the liver to those changes caused by arsphenamine, thiouracil, methyltestosterone, and substances of similar chemical structure. The major feature of this entity is an intrahepatic obstruction to the flow of bile.

The likeliest explanation for chlorpromazine-induced jaundice would seem to be on the basis of an allergic mechanism, which results eventually in intrahepatic cholestasis and bileplugging. This explanation is supported by the presence of a latent period between the institution of therapy and the appearance of clinical jaundice or changes in the results of hepatic-function tests, or both. The not infrequent occurrence of a peripheral eosinophilic and of the periportal eosinophilic filtrate noted on biopsy lends favor to the possibility of an allergic mechanism. Shay and Siplet in careful studies on 47 patients treated with chlorpromazine found the serum alkaline phosphatase determination to be the most sensitive means of detecting early sensitivity reactions to chlorpromazine.

Twenty cases of chlorpromazine-induced disease of the liver have been studied in which the daily patient dosage ranged from 30 to 150 mg. Seventeen patients received 75 mg. or less. The duration of administration of the drug ranged from 2 to 28 days, 8 patients having received the drug for less than 20 days, 9 having received it for 20 to 30 days, and 3 having received it for 42, 49, and 58 days respectively. In most of the patients, none of whom were known to have previously had liver disease, jaundice developed between 2 and 4 weeks after administration. Fourteen of these patients exhibited an "influenza-like" syndrome, with chills, varying degrees of fever, malaise, nausea, and generalized muscular aching prior to the onset of jaundice. Coincident with, or shortly thereafter, 36 patients experienced a moderate-to-severe degree of generalized pruritus· Eleven of the patients reported some degree of right upper quadrant pain or epigastric abdominal distress, although none described the "classic" pain of biliary colic. A relative eosinophil count of greater than 8% was found in 8 patients, the highest noted being 30%. Repeated cephalin-cholesterol flocculation tests in 19 patients were negative, the zinc sulfate turbidity test was normal in all 10 patients in whom the test was utilized, and the thymol turbidity test yielded slightly elevated values in 6 or 20 patients studied. Total serum protein, serum albumin, and serum globulin determinations obtained in all patients were normal in 17 patients, 3 of them exhibiting slight reversal of the

albumin-globulin ratio. The duration of jaundice and of abnormal results of hepatic function studies varied in length from 8 days to 10 months (2 cases).

In so far as liver-cell morphology is concerned, the absence of necrosis is 1 of the major features in differentiating chlorpromazine jaundice from viral hepatitis, necrosis having occurred in only 1 of the 15 patients evaluated. The absence of bile in the large ducts—found in all of the patients—is the major differentiating factor between chlorpromazine-induced liver disease and obstructive jaundice of large bile duct origin. Capillary bile thrombi were found among the liver-cell cords in all specimens, although bile duct proliferation of small hepatic ducts of small degree was found in only 3 specimens. Treatment in all 20 patients was the same as that administered for cirrhosis: high-carbohydrate and high-protein diet, bed rest, and large doses of supplemental vitamins, especially the B complex. Steroid therapy was found to be ineffective in 5 patients. Although it is difficult to evaluate the effectiveness of this therapy in the 20 patients, it is believed that treatment of this condition should parallel that of hepatitis, as it should at least minimize damage to the liver from the obstructive jaundice itself.

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THE CLINICAL SIGNIFICANCE OF SERUM CYANOCOBALAMIN (VITAMIN B_{12}) IN LIVER DISEASE. M. Rachmilewit, J. Aronovitch and N. Grossowicz. A. M. A. Arch. Int. Med. 101.1118-1128 (June) 1958 (Chicago). (via. J.A. M.A. Sept: 6 1958)

In this series the serum cyanocobalamin values were correlated with the common liver-function tests and particularly with the degree and duration of bilirubinemia, where it appeared that the estimation of serum cyanocobalamin might serve as an early and sensitive test for differentiating the various types of jaundice. There were 37 patients with viral hepatitis, comprising a heterogeneous group of adult immigrants from Israel with poor nutritional backgrounds, 1 patient with acute yellow atrophy of the liver due to phosphoning, and 14 patients with portal cirrhosis, 5 of whom showed clinical and laboratory signs of active hepatitis. Six patients were in the advanced stage of portal cirrhosis in decompensation, the remaining 3 having a compensated portal cirrhosis. The total serum cyanocobalamin value was estimated by the modified Escharichia coli assay.

In patients with acute viral hepatitis there was no definite correlation between the high initial serum cyanocobalamin values and the degree of bilirubinemia, as concentrations of cyanocobalamin 10-to-20-fold greater than normal were associated with moderately elevated serum bilirubin concentrations, while only moderate elevations of serum cyanocobalamin levels were found in patients with marked bilirubinemia. An exceptionally high serum cyanocobalamin concentration was found in a fatal case of actue yellow atrophy of the liver due to phosphorus poisoning. Five patients with portal cirrhosis, showing findings of active hepatits manifested by jaundice and severely altered hepatocellular liver function, had markedly elevated cyanocobalamin values. The 6 patients with advanced portal cirrhosis, who had clinical signs of portal hypertension but no evidence of active hepatitis, had cyanocobalamin values lower than those in the previous groups. Normal cyanocobalamin values were found in the remaining 3 patients with compensated portal cirrhosis, while higher values were demonstrated in 5 of the 6 patients with biliary cirrhosis who were all jaundiced, were in the advanced stage of the disease, and showed laboratory evidence of hepatocellular damage. The remaining patient in this group who was not jaundiced and showed no evidence of hepatocellular damage had normal cyanocobalamin values on 2 examinations. In patients with jaundice due to blie-stone obstruction with evidence of ascending cholangitis, the cyanocobalamin values ranged from 160 to 725 micromicrograms per milliliter, the higher value being found in a patient with carcinoma of the head of the pancreas. Two patients with chlorpromazine-induced jaundice, in whom the clinical and laboratory manifestations were characteristic of obstructive jaundice with hepatocellular damage, had normal serum cyanocobalamin values.

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VITREOUS IMPLANT FOR RETINAL DETACHMENT

By DONALD M. SHAFER, NEW-YORK.

Read on 26th Sept. 1958 in IInd. International course of ophthalmology held in Barcelona at Barraquer's institute.

The author discussed the indications, technique and complications of vitreous implant for Retinal detachment. He said that vitreous implant represents a logical surgical approach to the two basic dilemmas of detachment surgery to influence the vitreous and to mechanically bring retina against choroid. The technique described by him is simple and the complications are minimal. The visual results of the vitreous implant are favourable even in previously unsuccessful cases.

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SURGERY OF THE CONGENITAL CATARACT

By DERRICK VEIL, CHICAGO

Read on 22nd Sept. 1958 in IInd. International course of ophthalmology held in Barcelona at Barraquer's institute.

The surgery of the congenital cataract is not so simple as it looks because nearly 50 % of such cases have an associated ocular defects and remainder may present some early and late complications like glaucoma, iris Bombe, uveitis, retinal detachment, summerring ring and opaque cornea etc. The author is fully convinced that most of these complications are due to disturbance of anterior vitreous especially repeated needlings.

A review of the various techniques used with their disadvantages etc., has been discussed and the author has described a technique which is simple and quite safe.

(1) After preliminary dilatation of the pupil limbic based flap is made.

(2) One or two preplaced sutures of 6-0 catgut are inserted.

(3) Knife needle puncture (Optusely) through the cornea below as a track for air injection.

(4) Keratome incision enlarged with scissors to make a 60° opening.

(5) Full iridectomy in every case.

(6) Large bite of the anterior capsule with toothed forceps to remove most of the anterior capsule.

(7) Cystotome incision in the cartex and nucleus of the lens in all directions.

(8) Gentle irrigation of the lens material with the tip of the irrigator inserted directly into the lens substance, expression and possibly suction of the as much lens material as possible.

(9) Reposition of iris pillars, tying of sutures, closure of conj. flap with running 6-0 catgut sutures.

(10) Small bubble of air injected.

(11) Instillation of mydriatic and antibiotic. The author has found this method to be very safe and he advises that complete or full iridectomy should be performed in every Case.

CLINICAL TRIALS WITH DBI, A NEW NONSULFONYLUREA ORAL HYPOGLYCEMIC AGENT L. P Krall and R. Camerini-Davalos. A.M.A. Arch. Int. Med. 102: 25–31 (July) 1958 (Chicago) via J.A.M.A. Oct. 11, 1958)

The orally administered hypoglycemia-inducing agents used so far have been sulfonylurea derivatives with blood sugar-lowering ability in certain types of diabetes. Within the last year studies were started with a new group of compounds classified as formamidinyliminoureas. These drugs are biguanides. This report is a clinical evaluation of the effectiveness of N¹-B-phenethyl-formamidinyliminourea hydrochloride, referred to as DBI. One hundred twenty-one diabetic patients were observed for periods varying from 1 day to 6 months. They were hospitalised, were kept on a constant weighed diet, and were observed individually by 3 physicians. Patients previously receiving insulin had DBI substituted for the insulin. Newly discovered diabetic patients were treated with DBI immediately after the diagnosis had been established. One-third of the patients experienced side-effects involving the gastrointestinal tract. Of 104 patients who were observed for an adequate period, 90 (or 86%) experienced a blood-sugar-lowering effect, although in 19 (18%) treatment was discontinued because of side-effects. Although the primary object of the study was lowering of the blood sugar, "good" or "fair" control was achieved in 66 of the 88 patients evaluated in this manner. Sixty patients are still on maintenance therapy DBI. Only 6 of these take supplemental insulin, and these take less than 50% of their previous daily dose. Frequent liver-function, blood and other studies have shown no evidence of toxicity. There was an observable blood-sugar-lowering effect in every age group tested and in every type of patient, including juvenile diabetics, in this series. The new drug, despite its annoying side-effects, may prove useful in the future if no toxic effects are found.

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HEREDITY AND PERNICIOUS ANXMIA

On the basis of the findings in 308 close relatives of patients with pernicious anxmia, and 259 control subjects, the following concept of the pathogenesis of pernicious anxmia is advanced by Sheila T. Callender and M. A. Denborough (British Journal of Haxmatology, January 1957, 3, 88). The gene or genes responsible produce their effects only in middle life. By some mechanism, as yet unknown, they lead to a progressive lesion of the gastric mucosa, the first detectable sign of which is achlorhydria. This is followed by a failure to secrete intrinsic fact or and pepsin. The biality to absorb vitamin B 12 is therefore impaired, the body stores of vitamin B 12 are practically used up and the concentration of vitamin B 12 in the serum falls. The individual may remain at this stage for an indefinite period before the signs of anxmia or degeneration of the cord appear.

In a further study of certain inherited characters in pernicious anxmia, carried out in conjunction with Joan Sneath (Ibid., P. 107), no significant association was found between pernicious anxmia and the ABO blood groups, but when the results were pooled with those of several other centres there was a significant excess of group A. Such an excess of A, with a deficiency of group O; has been reported incarcinoma of the stomach. There was a preponderance of females, the male to female ratio being 1 : 1.5. There was a significant increase in the incidence of light eyes in the patients with pernicious anxmia, compared with the controls. (Via The Practitioner, No. 1067 Vol 178).

Book Section

ANNOUNCEMENT

We are proud to announce that the 'Mediscope' dedicated to serve the Indian Medical Profession, has ventured to publish 'Text Books' for Medical Students & Doctors, thus affording our regular subscribers to possess 'Text Books' *without* paying specially for them.

The text books written by eminent authorities—Professors, Lecturers and Specialists—in Medicine and Surgery have graciously come forward to have their text books published in the 'Mediscope' serially, each author taking about eight to sixteen pages in each issue until the Text Book is completed.

From Feb. '59 issue the 'Mediscope' has commenced the valuable work of Dr. R. J. Maneksha, F.R.C.S. (Eng), on "PLASTIC SURGERY FOR MEDICAL STUDENTS & DOCTORS". From April '59 we are planning to publish concurrently the text book on "RETINAL DETACHMENT & ITS MODERN TREATMENT" by Dr. J. M. Pahwa M.B.B.S., D.O., Z.O., (Vienna M.S., Eye Hospital, Sitapur, U. P. Of course the 'Text Books will be in addition to the usual features of Articles, Case Reports, Gleanings, Notes & News etc.

As a few of our regular readers have observed, that the 'Book Section' not only affords an opportunity to possess text books, it also gives a very great incentive to the eminent Medicos of our country to come forward to write 'Text Books'.

Writing of a 'Text Book' suitable for the Universities is the hardest job, requiring the full time attention of an author at the sacrifice of his practice, not to mention of the financial risk.

Having risked time and finance none can be sure of the attitude of the Universities to recognise the merit of such costly works and prescribe them to the Medical Students.

Although the talent is not lacking in our country to write Text Books suitable to our Universities, many an authority on Medicine & Surgery, under whose coaching the Indian Medical Profession has today grown to a lakh and odd strong, have not ventured—rightly, to indulge in the highly speculative art of writing text books. Consequently this great Nation still depends on the resources of our brethern abroad to feed us with text books. It certainly does not stand in good stead of our National Prestige.

To fill up the lacuna in the field of Medical Text Books by our Indian Authors, the Mediscope has boldly ventured to publish Text Books absorbing the financial strain of the authors, thus giving them an impetus to contribute their precious knowledge and wide experience in the field of Medicine & Surgery for the benefit of our Indian Medical Profession.

We fervently hope that the Indian Medical Profession will appreciate our aim—laudable indeed, and make our efforts a success.

—Editor.

PLASTIC SURGERY

FOR

MEDICAL STUDENTS

AND

DOCTORS

BY

Dr. R. J. MANEKSHA, F.R.C.S. (Eng.)
SPECIALIST IN PLASTIC SURGERY

CHAPTER 3

Grafting

* * *

FREE SKIN GRAFTS
FLAPS AND PEDICLES

* * *

What is the meaning of grafting?

By grafting is meant the shifting of a particular tissue from a donor area to a recepient area where it is required. Since tissues require blood circulation, to remain alive, they are shifted in such a way, that one part is always receiving blood for its nourishment. This type of grafting is called Flap or Pedicle Grafting. Free grafting which is generally done in one operation depends on it's blood supply only from the recepient area.

What are the tissues generally grafted?

Most of the tissues of the body could be grafted but skin is the tissue most often grafted. Tissues taken from the same person are called autografts. When taken from another person it is known as homografts. Besides the skin fat, cartilage, tendons and nerves are usually grafted. Cartilage and bone are sometimes used from a recently dead person and stored in a tissue bank. Similarly the cornea of the eye is used from an eye bank. Skin homograft from the mother usually takes on the child whilst homografts from

other persons does not succeed. It is believed that certain antibodies are produced in the child's body which disintigrate the skin grafts if not taken from the mother.

What are the types of free skin grafts?
What are the advantages and disadvantages of each variety?

Free skin grafts are of three main types according to their thickness: The thin variety is called the Thiersch Graft whilst the full thickness is known as the Wolfe Graft. The third variety is called the Intermediate or the Split Skin Graft. The grafts develop their nourishment from the recepient bed. New blood vessles start invading the graft after four to five days of grafting. The graft must remain in firm contact with its bed during this crucial period. Hence any haematoma under the graft is sure to destroy it. The recepient bed should be free from infection. The thin variety of graft has a better take in the presence of slight sepsis in the bed, where a full thickness graft will usually fail under similar circumstances. The full thickness graft requires a bed with very good blood supply. Thin grafts contract by 25 per cent while the full thickness graft does not contract at all. Functionally the full thickness graft is the best.

How are the thin and the intermediate type of grafts obtained?

These grafts are usually cut with the help of a skin graft knife or with a Dermatome. Dermatome is an instrument which has a drum or roller and a knife attachment. The knife is worked to and fro against the drum surface for cutting the graft. At first adhesive is applied to the donor's skin and the drum surface. Both the surfaces are next approximated so as to adhere to each other and the required graft is cut with the knife. The depth of the graf-

can be regulated by altering the distance between the drum and the knife. Dermatomes worked by electricity are also now available.

What are Reverdin's (pinch) graft?

These are small islet grafts. They are easy to obtain and take well on the recepient area but leave behind as potted appearance on the grafted area. For this reason it cannot be used on the exposed part of the body. The area in between the pinch graft also heals up with scar tissue. Hence this variety of graft is not used. The split has replaced this method with very good results.

What are epithelial grafts?

When mucosa is to be replaced especially in the mouth cavity a thin skin graft is applied to the defect and tied over a guttapercha mould. The edges of the pocket are sutured over the mould. After ten days the wound is opened and the mould removed. After cleaning the skin grafted area, the mould is kept for further ten to fifteen days, the mould is retained in position by splints attached to the teeth. This method is very useful to increase the depth of the buccal sulcus.

What are dermal grafts? and where are they used?

These are very useful grafts for restoration of contour of non rigid regions like the nose, eyelids, lips and supra-orbital region. The graft consists of the dermal layer of skin. The desired area of the defect is marked out on the abdomen, thigh or arm and the epidermis is shaved off, the corium is desected out without any fat layer. The graft is folded on itself and inserted into the usually depressed area through a small incision adjoining to the defect. The graft is held in position by pull out sutures at the four corners.

A ten to twenty percent absorption in volume is anticipated and hence the defect is over-corrected. The operation can be repeated at the same spot if depression persists.

What are skin flaps and when are they used?

Skin flaps are usually taken from the adjoining part of the defect, to be remedied. They may also be used from distant parts of the body. Adjoining skin flaps could be advanced, rotated or advancedrotated to the defect. They are very useful on the face as they have the same colour and texture of the skin. (Free grafts on the face are avoided due to poor colour match). A good example of distant skin flap is the cross-leg flap used to cover a defect in one leg by a flap from the opposite leg.

What is pedicle grafting?

Skin pedicle grafting is the most useful procedure in plastic surgery. It was originally started by Filatov in Russia and by Sir Harold Gillies in 1917. A tube of skin is raised on the abdomen, chest or any other part of the body. The defect in the skin underneath is closed or skin-grafted. The skin tube has no raw area and hence there is no chance of infection. After three to four weeks one end of the tube is detached and is shifted either to an intermediate carrier like the wrist or directly to the deformed area. The main advantage of pedicle grafting is that the blood supply is constantly maintained all throughout. This method is not so quick as the free grafts or the transferred flaps but the results of pedicle grafting are far superior to free grafts. A thin layer of fat is usually transferred with the skin in this method.

CHAPTER 4

Scars

* * *

SKIN STRUCTURE—TYPE OF SCARS
SURGERY OF DELHI
BOIL &
SMALLPOX SCARS

* * *

What is the structure of the skin and why is it so important in Plastic Surgery?

Covering the whole body on the outside the skin has an approximate area of 10,000 to 18,000 square centimetere in an average adult. It consists of two layers, the superficial horny layer is known as the epidermis and the deep or the true skin is called the dermis. The thickness of the skin varies from 1.5 milimetere to 5 milimeters. It is thickest in the palms and the sole while it is thinnest over the eyelid and on the sex organs. The skin protects important structures like muscles, nerves and blood vessels from injury, it regulates the body temperature by evaporation, radiation and conduction. It is also provided with touch corpuscles for sensation which are highest developed on the fingertips, it excreates oily substance called Sebum through glands called sabacuous glands which are mostly found on the face, head and the back of chest. Majority of the defects amenable by plastic surgery are situated on the skin and hence it's importance to the plastic surgeon.

How does epidermis differ from dermis or true skin?

The epidermis is the horny, non vascular layer which is regularly peeled off and fresh layers are substituted from the deeper layers. The deeper layer of the epidermis also has pigment cells which gives colour to the person. When pigment cells are absent the person is called an albini. It is perforated by hair and the ducts of sweat glands. The dermis or true skin is the second or deeper layer. It is full of blood vessels, nerves and lymphatics. It contains the roots of the hair, the sweat glands, and the rich plexus of nerves which send out five filaments into the epidermis. It contains fibrous strands which are responsible for the crease lines on the body.

What is the proper care of the skin?

To have a healthy and fresh skin requires daily care. Daily washing with soap and water removes all the oily perspiration that collects with the dirt from outside. Avoiding diet containing fried foods, rich sweets and spices. The best tonics are sunshine, rest and exercise.

What are scars?

Scars on the skin are defects left when the skin layers are damaged either by injury or by disease. A scar will form when the true skin (dermis) is effected and not when only the outer skin is involved. The regular smoothness of the skin is disturbed as a result of scars.

What are the important causes of scar formation?

When the skin is badly damaged it heals up as a natural process but with a scar formation. When the skin is infected by such diseases as Delhi Boil, small pox, syphilis,

tuberculosis, leprosy, etc., the end result is a scarred skin defect. Similarly when skin is injured by accident and especially when not stitched and allowed to heal up, a very bad scar is left behind. Burns can leave behind very bad scars. Burns may be due to fire, acids or alkalies; even X-rays and radium give rise to burns when the dose is excessive. A bad scar can be the unwelcome monument of a surgeon's prowess—these are surgical scars!

What are the types of scars?

Usually the scars are flat, depressed or elevated to the surrounding skin. Again a scar my be linear, webbed or stellate or crablike. Scars may be hypertrophied or keloidel.

What is the difference between hypertrophied and keloidal scars?

A hypertrophied scar is when there is excessive production of fibrous tissue within the limits of the scar. In a keloidal scar there is similar involvement of surrounding tissue. Hypertrophied scars are usually self-limited in duration and gradually disappear in the course of a few months.

What is the treatment of keloids?

On the whole the treatment of keloids is rather disappointing, as it very often re-occurs like the proverbial bad penny. Roentgen and radium therapy before and after excision have yielded the best results.

Can scars be prevented?

Yes, to a certain extent. Surgical scars can always be prevented by careful planning of the skin cut at operations, and careful surgery. Scars due to infection can be avoided by early treatment with present day antibiotics. Scars due to burns can be avoided by early grafting of the skin over the raw area.

What are the complications due to scars?

Due to a scar there is very often loss of movement of the part especially when the scar is across a joint line like the elbow or the knee joint. Secondly the scarred part is devoid of proper sensation and is thus liable to injury.

What is the treatment of Linear Scars?

Linear scars are often due to burns and are mostly found on the neck, axilla and front of elbow joint. When the surrounding skin is healthy linear scars are well treated by a simple Z plasty operation.

What is Z plasty and what is its main function?

Z plasty is a useful procedure to regain lost length of the tissue. With linear scar there is always a decrease in the length of the affected part. Two triangular skin flaps are elevated on each side. Each triangle has an angle of 60° degrees with the central scar. The apices of the triangle are transposed to increase the length at the expense of breadth.

What is the treatment of broad scars?

Broad scars do not respond of Z plasty. They have to be treated by other methods of skin replacement. This can be done by any of the following methods depending on the case in question.

(1) Free grafts—thin, intermediate or full thickness.
(2) Flaps—Either adjoining or distant.
(3) Pedicle grafting.
(4) Staged excision.

Can scars be shaved and skin grafted?

Yes. Dr. Hyues of Sheffield has tried this method in old burn scars. They are shaved with a knife and grafted with thin skin. The method is still under trial.

What is Delhi Boil?

Delhi Boil as the name so aptly suggests is a disease of the Northern parts of India which is caused by an infection by an organism called 'Leishmania Tropica'. It produces an ulcer due to the biting of a sand fly (Phlebotomus) which carries the small Leishmania infection in its saliva. The exposed part of the body like the face and the extremities are mostly affected. Many sores may occur on one person and many members of the same family are often affected.

What are the other names for this condition?

The other names for Delhi Boil are 'Lahore Boil' or 'Local Sire- or 'Bagdad Boil'. It is so called because it is frequently found in these places.

What is the result of a Delhi Boil infection?

The ulcer remains unhealed for a very long time if not treated and ultimately heals up leaving a very ugly scar which is depressed below the surface of the skin.

What is the treatment of such a healed scar especially when it is on the face?

The best treatment is to remove such a scar by scalpel surgery and bring the surrounding normal skin together with very fine stiches. If the ulcer is a very big one this may not be possible in one operation and repeated operations may be necessary or rotation-advancement flap from the surrounding area is the usual operation.

Is there any place for doing skin grafting in the treatment of Delhi Boil?

Taking the skin from another place and putting over the site of the ulcer is not advisable in dark skinned people

because that skin graft always shows as a patch. Hence skin grafting is not advisable in the treatment of Delhi Boil, as well as most other scars on the face.

What is the result of a small pox infection?

The effect of small pox infection is felt on the skin by the formation of blisters full of pus. This leaves behind multiple spots or depressed scars if the infection is very severe.

On which part of the body are the marks mostly left?

Unfortunately small pox marks are mostly left on the face and on the extremities. On the face also the marks are most prominent round about the area of the nose and the face. In some cases the marks are so many that they are very close to each other whilst in other cases the marks are few and scattered over the face.

Do the marks vary in their depth in different cases?

Yes, the marks are superficial in some cases whilst they are very deep in other patients depending on the severity of the infection. The marks also vary in a single person and unfortunately the marks round about the nose and face are somehow deeper than the marks on the periphery of the face.

Is a patient suffering from small pox marks at a handicap socially and economically?

Yes, a person suffering from marks on the face starts life with a definite handicap both socially and economically.

Is there any surgical treatment for small pox marks?

The treatment that is adopted since the last five to seven years is called 'Sand Paper Surgery' or technically dermo abrasion.

By whom was Sand Paper Surgery first started?

Sand Paper Surgery was first started in America by Dr. Iverson of Philedelphia and has been modified by different surgeons in America and the Continent.

What is the present method of doing dermo abrasion surgery?

The present method of doing dermo abrasion is by using circular stainless steel or stone burrs just like a dentist's brush. These are rotated on a dentist's machine at a very high speed of 15 to 30 thousand revolutions a minute.

What is the principle of dermo abrasion treatment?

The idea of treating is to level the surrounding healthy skin to the depth of the depressed scars so that the difference between the two areas is minimised as far as possible.

How many sittings are necessary before a patient is completely cured?

Usually four or five sittings are necessary when the marks are not very deep. If they are deep then seven to eight sittings are necessary.

At what intervals are these sittings undertaken?

Usually two months are allowed before a second sitting can be repeated but it can be done any time after that.

What anaesthesia is used?

Either local or general anaesthesia is used, The face is chilled by a special ice-bag applied for 20 to 30 minutes then ethyl cloride or Freon is sprayed on the skin so as to harden it and then the skin is abraded.

What post operative dressing is applied?

A layer of gauze with antiseptic ointment is applied on the face. The gauze falls out by itself on the 8th or the 9th day.

What is the ideal time for this operation?

Treatment commenced at an earlier age is better, since it is a prolonged treatment. But it can be started at any age after 7 years.

When is the Patient out of bed?

Patient can move about in his room from the second day onwards.

When can the patient wash his face?

The patient can wash his face with warm water a day or two after the gauze has separated.

How long does the patient have to stay in the hospital?

One or two days only.

Is there any special aftercare after sandpapering?

The patient is advised not to expose too much to the sunlight or sit near an open fire.

Are any complications likely to occur?

If the sandpapering is done to a deeper level there are chances of healing by scar tissue as well as darkening of the skin. This complication is best avoided by not going very deep.

When does the skin colour return to normal?

It usually takes 6 to 8 weeks before the skin again returns to its normal colour.

What is the result of one sitting of sand paper surgery?

There is usually about 25 to 40 per cent improvement in the marks depending on the skin texture. Hence multiple sittings are necessary for a complete cure.

Is there any risk of permanent damage to the face?

No. If the sandpapering is done carefully.

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CHAPTER 5

Hare-lip and Cleft-palate

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HARE–LIP

* * *

What is Hare-lip?

Harelip is a defect of either the upper or the lower lip. It is present from birth.

Is harelip more common in boys or girls?

Harelip occurs in approximately one in one thousand births. It is more common in boys than in girls and more often on the left hand side than on the right. Cleft palate by itself is more common in girls, whilst combined hare-lip and cleft palate is more common in boys.

What are the types of hare-lip defects?

Harelip may be unilateral, bilateral or rarely median. In the incomplete variety there may be just a notching of the vermillion margin and nonfusion of the muscles with a flat nostril base. The notching may extend higher up to the floor of the nostril. The alveolus is usually deformed, it may only have a deformed lateral incisor or the bone may be in two parts. The defect of the alveolus often varies according to the ratio of the lip defect but this is not constant. The maxilla of the affected side is not properly developed hence the nasal and the lip elements are at a depressed level on the face as compared to the opposite normal side.

What is the defect in a nose in a case of Hare-lip?

The deformity in the nose is most marked on the side of the hare-lip. In an incomplete hare-lip there is widening of the nostril floor. In a complete harelip there is saenceb of the nasal floor. There is flaring out of the lower alar cartilage instead of the normal rolled appearance. The dome of the nose is also at a lower level.

What is the treatment of this condition?

The only treatment for this condition is repair of the defect by Plastic Surgery.

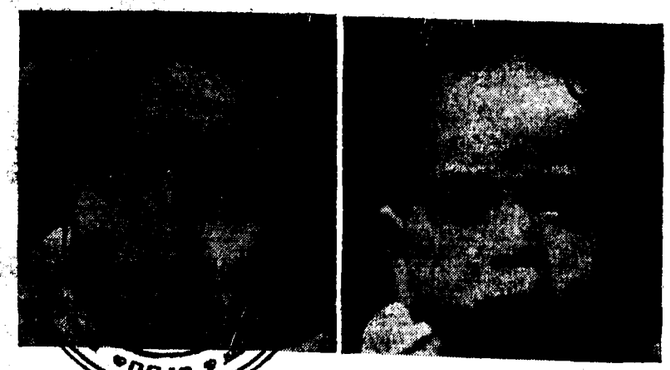
At what age should this operation be undertaken?

No sooner a child is born with a hare-lip it poses an immediate problem to the gynaecologist and the paediatrician with a plastic surgeon often called in for consultation. The parents are often very anxious for an early answer to this calamity. At some clinics repair at birth is undertaken within the first 48 hours, the author does not approve of such an early operation because it is very difficult to do full justice to the operation when the parts are so small, where the primary operation is so important and where the healing may not be so perfect and lastly where it seems to treat the parents wish rather than the infant's deformity. For this reason at many clinics, it is a rule to operate on the child during the second or third month and, when the child is weighing ten pounds or more. When it is thought that the child is not gaining weight due to the hare-lip defect preventing the intake of nourishment, then only an earlier operation is advisable.

Are secondary operations necessary?

Yes, very often. The parents are explained that this is a correctable deformity but that it may require a small

secondary operation when the child is four years and again when twelve to thirteen years of age. The surgeon would attempt to do all that is possible in the preliminary operation but it is not advisable to do too much especially to the alar cartilage of the nose, as it might restrict its further growth and hence a small defect may persist which can be corrected at the second operation. A frank discussion of this nature is much better for both parties concerned.



Unilateral or Single Hare-lip **"After" Unilateral Hare-lip corrected**

What are the requirements of a good Hare-lip repair?

A good hare-lip repair aims at a regular nose without any alar droop or widen floor of the nostril, a faint scar along the philtrum, an averted lip with a good cupids bow, absence of notching of the vermillion border and finally a lip that is not very tight.

Under what anaesthesia is the operation done?

Usually in infants and children general anaesthesia is advisable. In adults it can be done under local anaesthesia.

EDITORIAL

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★

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