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CEREBRAL APOPLEXY—*Continued.*

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Other points for consideration in Cerebral Apoplexy.

Bowels. Constipation frequently precedes or accompanies the attack. Or, on the contrary, where there is deep unconsciousness or prolonged stupor, and especially if drastic purgatives are given, involuntary discharges may occur.

Urine. At the onset the urine is usually acid. Transient glycosuria is an accompaniment of hæmorrhage in any part of the brain. The sugar usually disappears in from a few hours to a couple of days. Presumably it originates from shock to the so-called sugar centre. When the spot in the floor of the fourth ventricle is directly involved, the sugar may persist longer, though it usually subsides even then, in a week or two.

As a part of the same manifestation there may be a polyuria simply. That is then even more fleeting in character.

Albuminuria is a frequent and more serious accompaniment. Like the preceding symptoms, it may be but transcient in character, but its presence is always a cause for anxiety. Many cases of apoplexy are due to Bright's disease, and an examination of the urine should be a routine procedure in all cases.

Case in which Albuminuria occurred as a Symptom of Meningeal Hæmorrhage.

The case was that of a woman of 54 who complained of sudden malaise with general chilliness and severe head-ache but no loss of consciousness, merely some torpor. In the litre and a half of urine that was voided in the 24 hours 20 Gm. (5 drams) of albumen per litre (quart) was found but there was no tendency to Oedema or any bruit de galop of mitral stenosis although there was slight fever. Lumbar puncture revealed a typical and abundant meningeal hæmorrhage; all the symptoms gradually diminished and by the ninth day there was no trace of albumen.

The discovery of large amount of albumen without other signs of nephritis may aid in the differentiation of meningeal hæmorrhage.

Ophthalmoscopic changes are not sufficiently marked in the early stages to be of any value. After development of the full apoplectic state there may be some choking of the retinal veins especially on the side of the lesion. Miliary aneurisms have been observed in the retina but are quite unusual.

Hæmorrhages of the retina may indicate nephritis; but only to that extent suggest the cause of any cerebral condition.

DIAGNOSIS.

A diagnosis between cerebral hæmorrhage and cerebral embolism and thrombosis should be considered.

Against Embolism. The absence of any distinct mitral or aortic lesion the presence of head-ache or other prodromal manifestations; deep coma especially late development; vomiting pronounced anisocoria and advanced age.

In favour of Embolism.

Most commonly associated with valuable lesions of the heart, aneurysms, or a suppurating thrombus chiefly in young adults.

Onset sudden—no prodromal symptom.

Convulsions rare.

Paralysis.—A sudden hemiplegia on the right side with aphasia.

The left middle cerebral artery is frequently plugged.

Consciousness not usually entirely abolished.

Against Thrombosis.—Youth unless the patient be a syphilitic, coincident or early rise of bodily temperature, early and deep coma, vomiting great inequality of the pupils, beginning of attack when the person is under effort or excitement, a pulse of high tension, the absence of prodromata, and the existence of vigorous general health.

In Favour. 1. Arterio Selerosis, Atheroma, and syphilitic disease of arteries. Bright disease and alcoholism therefore predispose.

2. Pressure upon the veins or arteries (tumour or abcess).

Age.—In the old from vascular de-generation ; in the young from Syphilis.

Onset. Gradual—with prodromal symptoms.

Convulsions.—Convulsive attacks are not uncommon as the cortex is frequently affected.

Paralysis is more gradual. Aphasia is not so common.

Consciousness.—Often not lost. Coma may come on, gradually after the hemiplegia.

Diagnosis of cerebral Hæmorrhage.

When the patient is first seen in a profound coma the difficulties are great, especially when there is no history, as in many cases found unconscious and brought to the hospital. The question to be first settled should be—Is the case one of real apoplexy caused by some cerebral lesion, or is it one of uraemic or diabetic coma or is it the result of poisoning by alcohol, opium, or chloral, or is it a case of head injury, epilepsy, hysteria, or syncope, etc. ?

The characteristic features of cerebral apoplexy have been described and the observer by watching for a few moments, may be able to determine that the apoplexy is cerebral if he sees that there is evidence of hemiplegia. The patient in his coma may move the limbs on the sound side of his body only, and the physician by lifting up the arm of the suspected side may find that it is absolutely powerless ; compared with the opposite member it has lost all tonus, or it may be rigid or it may be

the seat of twitchings or of a unilateral convulsion. The cheeks may puff out only on one side and some other irregularity of the face may be noticed ; upon lifting the eyelid conjugate deviation of the eyeballs may be noticed, when the diagnosis will be certain. If the coma has lasted for several hours, the reflexes may have returned to the sound side and the plantar and cremasteric may be demonstrated on one side and not on the other.

Head-injury is then looked for if the above rapid survey fails to reveal hemiplegic symptoms, and an examination of the scalp may show a wound or a depressed fracture. It must not be forgotten that many apoplectic patients brought to hospital present contusions on the scalp or temples caused by stones in the streets, the result of the apoplectic stroke. Rupture of the middle meningeal is often caused by violence, and the site of the wound or contusion should be considered.

Alcoholic Poisoning should always be suspected in cases of difficulty ; but grave mistakes are constantly made in arriving at a conclusion from the odour of the patient's breath, as he may have taken alcohol upon feeling the first symptoms of his attack, or he may be suffering from cerebral hæmorrhage, induced by his being overstimulated by the drug. The pupils are generally dilated ; and though this is a valuable point in the diagnosis from opium-poisoning it is of little value in excluding brain hæmorrhage, because the pupils may be found dilated in this condition also-success in rousing the alcoholic patient need not be considered, as this is impossible when the coma is profound. There is, however, a valuable aid, generally overlooked which is especially reliable in the class of a drunken case picked

up on the roadside or found comatose in bed. This consists in the marked drop in temperature which may amount to 5° or more Farenheit. It may seem a serious matter to use the stomach pump on a patient with cerebral hæmorrhage ; but it is really a very serious matter to permit a case of severe alcoholic poisoning to die from the effect of an overdose, which might have been removed with ease ; and in all cases of grave doubt the passage of the soft rubber tube into the stomach without changing the patient's position, should be resorted to.

Opium poisoning is recognised by the odour of the breath when especially landanum is the form ; the pupils are contracted to a pin's point and the breathing is slow ; of course, all the symptoms are bilateral and the coma, at first incomplete becomes profound. It resembles hæmorrhage into the pons, but the temperature is below the normal, and there are no convulsions.

Chloral poisoning closely simulates apoplexy ; the pupils are contracted, but not to the extent seen in opium poisoning ; the pulse is weak and slowed ; the depression of the temperature is characteristic ; there may be a fall of 10' F. or more.

Uræmic poisoning is a common cause of unconsciousness, though it is, perhaps rare for the coma to be very profound. It very often follows or alternates with, convulsions, and these are never unilateral. The high tension pulse, hypertrophied heart, and œdema may generally be observed and albumin is found in the urine ; but these are very often present in cerebral hæmorrhage, as they are indications of diseased vessels. Notwithstanding, also, the fact that a cerebral hæmorrhage may cause albumin to appear in the urine of a patient without

kidney disease, an examination of the urine often may settle the diagnosis. If the catheter removes only a very small amount of urine of very low specific gravity, the diagnosis of uræmia is almost certain to be correct. A bladder distended with a high specific gravity urine, though highly albuminous, is not indicative of uræmic poisoning.

Diabetic coma has very frequently more the aspect of a complete collapse with stupor than of a profound coma ; but in cases where the onset is very sudden it may resemble apoplexy ; the breath has a peculiar sweet odour ; the urine is saccharine and the blood sugar may be determined. In some cases the characteristic slow dyspnœa may be present. Even with all these characters present, there should be a search made for hemiplegia, as cerebral hæmorrhage is not uncommon towards the late stages of diabetes.

Epileptic coma is less profound than apoplexy, as a rule and soon passes off ; it resembles deep natural sleep, and the reflexes rapidly return. Transient hemiplegia and fleeting aphasia are, however, not very rare after epileptic convulsions.

General paralysis of the insane is often characterised by a series of attacks of coma resembling cerebral hæmorrhage, and the diagnosis in the absence of a history is difficult ; but soon the attack passes off leaving the patient in his former condition, with grandiose delusions, tremors of the face etc.

Congestion or cerebral hyperæmia sometimes produces a condition approaching apoplexy ; but the unconsciousness is never so profound, and it rapidly passes off without hemiplegic symptoms.

Hysterical coma is recognised by the other symptoms of this diseased condition; consciousness is not really lost, the pupils readily respond to light, stertor is absent, and the symptoms rapidly yield to external stimuli.

Syncope is easily eliminated, the cardiac failure being the chief feature, as evidenced by the absence of pulse and the feebleness of the heart sounds; the sphincters do not give way, and the reflexes are rarely lost.

Assuming the diagnosis of cerebral apoplexy with hemiplegia to be demonstrated, the question then comes to be whether the cause is one of hæmorrhage, embolism, or thrombosis; complete loss of consciousness may characterize each, and the history of the case is generally necessary before the lesion can be determined.

The following points may be repeated. *In embolism* the coma will be less profound; as a rule the patient will probably be under forty; there will be probably heart disease or arterial plugging elsewhere present; the onset is rapid and the symptoms may pass of quickly. Should the question of diagnosis only arise during the hemiplegic state, the fact of a sudden seizure without unconsciousness is in favour of embolism.

In thrombosis the differential diagnosis is most important, as it seriously influences treatment. It may be very difficult as evidences of diseased vessels are to be found in both cases and age affords little help. The history of the onset is of vital importance. It lacks the suddenness of hæmorrhage and of embolism; there are premonitary headache, vertigo, and tingling in the limbs on one side; consciousness if lost is lost slowly; in syphilitic cases it is often retained. The state of the

pulse is important; the strong heaving impulse of hæmorrhage is absent, and the pulse is weak and of low tension. Local convulsions at the beginning of the attack are more likely to occur in thrombosis, and general convulsions in hæmorrhage. The temperature, unless the thrombosis be very extensive or basilar is not influenced, as it is in hæmorrhage, in which latter condition there is usually a sudden moderate fall.

Abscesses, tumours and aneurisms may produce hemiplegic symptoms with more or less interference with conciousness.

Prognosis. This must be based on the following factors. However, there are two separate questions in the matter of prognosis; one has regard to the continuation of life and the other to the extent of recovery from the attack.

The age of the patient. In childhood the rare cases that do occur are usually severe, but if the attack itself is outlived, the natural recuperative power is so great that the person will live on indefinitely. Improvement may be expected for some years, but entire removal is unusual.

In middle life the outcome depends on the causal trouble and the severity of the apoplectic attack. Where the motor involvement is not great or is due to indirect pressure, obsorption of the clot permits a practically complete restitution of all functions. More often some impairment of the involved area remains. If the primary cause still obtains (=subsists) this also interferes with recovery and the general outlook.

In senile conditions (tortuous or calcified arteries, dry and wrinkled skin, arcus senilis etc.) but limited

recovery is to be expected. Life may be prolonged, but most depends on the promptness with which the attack is checked. The subsequent length of life depends much on the kindness and care with which the chronic invalid is surrounded.

Nephritis. Real kidney disease, when present, limits recovery and determines the eventual duration of life. Even with the complication, however, if the site and extent of the effusion be favourable the paralytic condition may be fully recovered from.

Syphilis. The existence of this systemic infection is principally of ætiological importance. It may constitute an indication for treatment, but otherwise has little significance.

Severity and nature of the attack. Coma, stertor, vomiting, prolonged semiconsciousness, extensive and complete paralysis etc., indicate a large effusion with much damage to the brain both in local destruction and local shock. Consequently there is immediate danger to life and much less chance of functional recovery when life is prolonged. In proportion as these features are less prominent the chances for preservation of life and for recovery are increased. Prolonged high temperature, or a rise to 104° or 106° F makes a fatal prognosis probable. Especially about the fourth to sixth day a rise in bodily temperature, deepening of coma, increased muscular relaxation and the advent of cheynestokes breathing presage (forebodes) approaching dissolution the separation of the soul and the body.

General convulsions, as indicative of ventricular rupture (barring uræmia) are a particularly bad omen, death usually resulting in from a few hours to a few days.

Location and size of the lesion.

These two features are complementary. For, though much depends on the site, a large outpour by its mere volume may include temporarily all the effects of the smaller and certain general effects in addition.

Pontine hæmorrhages are more often promptly fatal doubtless from the importance of the local centres and passing tracts. The outpour is also more rapid because from relatively large vessels and close to the parent trunk. On the contrary hæmorrhages of the cerebral hemisphere above the central ganglia commonly become vast in size before inducing serious symptoms. Cerebellar apoplexy may be lethal from pressure on the respiratory and adjacent centres.

Inequality of the pupils developing as a part of the attack, especially where the larger is on the side of the supposed hæmorrhage, suggests a large focus, and hence points to a more serious condition. But this is indecisive by itself.

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After the acute stage has been tided over, the extent of presumable recovery is the main matter for prognosis. Here besides the points already presented, other manifestations have to be considered. The state of the tendon reflexes in the involved area must be determined ; if there is any increase compared with the other side, we can pretty safely conclude that some permanent injury of nerve tracts will remain though a slight local increase is not incompatible with recovery. Any marked increase of these reflexes—as ankle clonus or wrist clonus or a knee jerk—means lasting paralysis. The occurrence of

œdema or contractures in the paralysed part signifies so grave a lesion of the motor path as to preclude hope of full recovery.

The anæsthesias that are so frequently present in the early or acute stage rarely prove lasting. The occasional development of chorea in the affected extremities is so far a good sign as it indicates returning conductivity of the motor tracts.

So then, as regards recovery from the coma prognosis is always very serious ; it is grave in proportion to its depth and duration, and if the loss of power is evident on both sides death is probable and practically certain if cheyne-stoke respiration and profound coma are present at the end of twenty-four hours. A sudden and rapid oscillation of temperature is of evil omen ; so also is a bounding pulse and flushed face with much stertor.

From cortical hæmorrhage unless very extensive, the recovery may be complete without a trace of contracture. This is more common when the hæmorrhage follows injury than when it results from disease of the arteries. Infantile meningeal hæmorrhage is a condition which may produce idiocy or spastic diplegia.

Large hæmorrhages into the corona radiata and especially those that rupture into the ventricles, rapidly prove fatal.

The hemiplegia which follows lesions of the internal capsule, the result of rupture of lenticulo-striate artery, is usually persistent and followed by contracture. When the retino-lenticular fibres of the internal capsule are involved there may be hemianæsthesia, and later, especially if the thalamus be implicated, hemichorea or athitosis.

The rapid formation of bed sores particularly the malignant decubitus of charcot is a fatal indication. The occurrence of albumin and sugar, if abundant, in the urine is an unfavourable symptom.

When consciousness returns and the patient is improving the question is anxiously asked as to the paralysis. The extent of this cannot be determined for some weeks. If it is to get well, as a rule, it does so quickly, and if there be not marked improvement in ten or twelve weeks it will probably become permanent though the patient may be able to walk about ; but the upper extremity will be of very little use to him, and when contracture has developed, the prospect of further improvement is hopeless, some mental weakness often remains.

Treatment.

Prevention.—In general, the prophylactic management is indicated by the ætiological factors. If there are any suspicions of prodonmata, the patient must be warned against all lifting and straining, the bowels be kept free (calomel or salines) any overtension of the pulse be eased by mild depressants, and the patient kept in a warm atmosphere well protected from all chilling. Digitalis and cardiac stimulants of every sort should be carefully avoided. Any nervous overtension can advantageously be remedied with bromoids, and their use here is reguiarly in order. Gelatin foods are also recommended to favour coagulation.

Hence prevention consists in the detection of a special tendency to a persistent mean rise of pressure (blood pressure). Occasional rises occur in all persons but they disappear before damage to the vessels arises.

In others the rise is persistent and very marked, yet, if the tendency be detected early, it may be reduced and kept down. Therefore every adult of the age of 40 and upward should, as a matter of routine have his blood-pressure measured. This should be repeated every five years, say, up to 60; then, if there is no great increase of pressure, the danger of apoplexy may be put on one side.

During the attack.—Restricting the term apoplexy to the more or less sudden loss of consciousness and motor power arising from a vascular lesion inside the cranium, due either to the rupture of an artery in the cerebral tissue, with the extravasated blood ploughing up the brain substance, or to the lodgement of an embolus from the heart, or to thrombotic occlusion occurring in a diseased cerebral vessel, the treatment of these three conditions is widely different; nevertheless, owing to the difficulty of diagnosis, which is in many instances insuperable, the physician will be wise in the absence of differentiating factors to regard all cases as due to cerebral hæmorrhage.

It may be safely said that there are few conditions in whose presence the physician feels so powerless, but there are also few in which so much harm can be done by active meddling and unwise attempts at heroic treatment.

The first and main object is to stop further hæmorrhage. Our efforts should be directed to a lowering of the arterial pressure, and to a deviation of the blood current to other parts, *i.e.*, in general, to a reduction of the supply to the brain.

The first duty of the attendant is to insure absolute rest regardless of the wishes of the patient's friends who

are often anxious to have him removed from the place in which the seizure has taken place. He should be placed upon his back on a safe, or on a bed extemporised in the room in which he has fallen by laying a mattress upon the floor. His head and shoulders should be elevated slightly all constrictions about the neck being removed. If there be any urgent necessity for the removal of his clothing this should be effected in the most gentle and cautious manner by cutting up the seams and removing the garments piecemeal, whilst a reliable assistant takes charge of the head to prevent its being shaken. The next step is to turn his face to one side in order to prevent the tongue directly falling backwards and to permit of the saliva dribbling from the angle of the mouth. An ice bag is placed to the affected side of the head if there is any facial congestion. Nothing can be done if vomiting occurs except to wipe out the mouth and keep the head and tongue forward.

No attempt should be made to arouse consciousness by shouting into the ear, shaking the body, or flapping with towels or other methods of stimulation, and certainly nothing should be administered by the mouth of the nature of food or stimulating drinks. Rubber bottles filled partially with warm water should be placed at his feet and along each side of the trunk; and if bottles are used they should not be too hot, since blisters may be readily caused by a much lower temperature than in health. Hot water bottles covered with flannel had better be used. In their absence warm blankets may be used, but friction or massage of the cold limbs had better be avoided. Ice when available, should be applied to the head or an evaporating lotion to the forehead and temples. Ice to the head is a popular plan but also of

very uncertain value. Counter-irritants used with the view of determining a flow of blood to the surface of the body, are as a rule to be avoided. The best of these agents would be a sinapism applied to the nape of the neck but especially in heavy and muscular subjects, this can scarcely be carried out without shaking the cranium. At a later stage when all the risk of a fresh hæmorrhage is over, the patient may be moved from side to side to avoid undue pressure on one spot and thus avoid bed sores and to lessen the chances of hypostatic congestion of the lungs. The dangers of an aspiration pneumonia should be minimised by maintaining an aseptic condition of the mouth, any dental plates being removed, and the tongue and lips smeared with glycerine of Borax. The bladder must be attended to and distension avoided, catheterisation being carried out as required. Absolute cleanliness is only assured by assiduous care and the patient must be kept dry and clean, the limbs and body being washed, dried, and the points of pressure afterwards rubbed with spirit and then powdered. The bowels require evacuation.

Nothing should be done to combat any symptoms of shock or collapse beyond the above palliative measures. To administer hypodermically or by rectum powerful cardiac stimulants will only cause the heart to beat more vigorously and pump more blood into the ruptured cerebral tissue ; hence the use of irritating smelling-salts may be injurious through their power of increasing the general blood pressure.

The question of blood letting should be considered if the profound coma remains, with a high tension pulse, viogorusly acting heart and signs of asphyxia. Opinions

differ as to the wisdom of venesection in cases of cerebral hæmorrhage. It is well-known that a rise of intra-cranial pressure will cause a rise in the general arterial pressure. This rise in arterial tension may be regarded as Nature's attempt to maintain the supply of blood to the respiratory centres. Death ensues in most cases of cerebral hæmorrhage from anæmia of the respiratory centres due to rise of intra-cranial pressure Those not in favour of venesection consider that the lowering of blood-pressure is dangerous, because it further diminishes the blood supply to the already embarrassed respiratory centres. On the other hand we must not lose sight of the fact that the rise in intra-cranial pressure is due to the intra-cranial hæmorrhage and that the anæmia of the respiratory centres is primarily due to the rise in the intra-cranial pressure and not to any lowering of the arterial blood-pressure. Thus venesection seems to be indicated theoretically in cases of hæmorrhage with high pressure but practically it is of little or no value and is not advisable. Allied to the measure but sometimes very effective are puncture of the brain and lumbar puncture the loss of cerebro-spinal fluid in the latter case reducing markedly cerebral pressure. Of late years a bolder surgical measure has been advocated, and found acceptance in some quarters. This consists in triphining a large opening in the skull of sufficient size to afford complete relief of pressure ; this is particularly applicable in subdural hæmorrhage ; this is only admissible when the case is going on from bad to worse and when in the coma it can be confidently ascertained that the paralysis of motion is hemiplegic.

Before, however, considering such a serious step, the physician should resort to the use of other agents

possessing the power of reducing the general blood pressure.

(i) *By purgation.*—Owing to the slow action of purgatives in the apoplectic state, croton oil, on calomel should be administered without delay. Whilst it is true that remedies which reduce the general blood pressure are injurious in thrombotic cases and should not be used when the clinical symptoms or the history of former attacks of syphilitic thrombosis clearly indicate this condition, it is to be remembered that the diagnosis is almost impossible, and of all the agents used to reduce blood-pressure purgation is the least objectionable or dangerous. Hence under the condition now being considered it may be used as a routine with comparative safety. No good usually can be expected by enemata and much harm may be done by the necessary changes produced by the moving about and turning the patients body during their administration An enema is however clearly indicated if the rectum is impacted with scybalæ, which prevent the action of croton oil—one or two drops of croton oil with 5 grains of calomel mixed with a little butter or a few grains of moistened sugar should be placed far back upon the tongue and often this dose will require repetition. Stimulants are not necessay unless the pulse becomes feeble and signs of collapse supervene. During recovery the patient should still be kept entirely at rest, even in the mildest attacks remaining in bed for at least fourteen days. The ice-bag should still be kept to the head. The diet should be light and no medicine other than some placebo should be given at least during the first month after the hæmorrhage. There is often a febrile reaction which necessitates the removal of all external aids to the keeping up of the body heat. It is

a wise precaution in giving nourishment taught by the experience gained from surgical head injuries to withhold all animal foods, even in the form of soup, for a long period after convalescence.

It only remains to mention the treatment of an apoplectic seizure where the history of symptoms indicate thrombosis or embolism as the cause of loss of consciousness.

In thrombosis after the patient has been put to bed with his head and shoulders but slightly if at all raised and heat applied to his surface, the immediate indication is to check the coagulable power of the blood by the administration of citric acid or its potassium salt.

As the extension of the thrombotic change is favoured by everything that tends to render the circulation languid stimulants are indicated. The best of these is Ammonia. The deep coma in thrombosis is due to the pressure of venous congestion consequent upon arterial blockage, venesection, leeching, and purgation in later stages may be tried. In syphilitic cases, the proper after-treatment will consist of full doses of the Iodides, always combined with large doses of Ammonia, whilst mercurial injunctions should be commenced without delay.

Embolism causing apoplexy must originate in the detachment of a fibrous mass from the endocardium or pulmonary veins or from a vessel between the heart and the cranium. The immediate effect is the shutting off of blood from an area in the brain and the tendency to further coagulation of the blood around the fibrinous focus brings the treatment of the case practically into the same category as that of thrombosis and Ammonia in

nornal saline solution hypodermically or by the rectum followed up by full doses of Citri acid is to be tried.

Very little can be done for the hemiplegia which remains. The damage is too often irrepairable and permanent.

The paralysed limbs may be gently rubbed once or twice a day, and this should be systematically carried out in order to maintain the nutrition of the muscles and to prevent contractures if possible. The massage should not be begun until at least ten days after the attack. The rubbing should be towards the body and should not be continued for more than fifteen minutes at a time. After tha lapse of a fortnight, or in severe cases a month, the muscles may be stimulated by the faradic current; faradic stimulation alternating with massage is of service even when there can be but little hope of any return of voluntary movement. The patient should perform simple movements and exercise himself. When contractures occur electricity at intervals may be of some benefit along with passive movement and friction.

In a case of complete hemiplegia the friends at the must onset be frankly told that the chances of full recovery are slight. Power is usually restored in the leg sufficient to enable the patient to get about, but in the majority of cases the finer movements of the hand are permanently lost. The general health should be looked after the bowels regulated, and the secretions of the skin and kidneys kept active. In permanent hemiplegia in persons above the middle period of life, more or less mental weakness is apt to follow the attack and the patient may become irritable and emotional.

And lastly, when hemiplegia has persisted for more than three months and contractures have developed, it is the duty of the physician to explain to the patient or his friends that the condition is past relief, that medicines and electricity will do no good and there is no possible hope of cure.

Drugs. The use of drugs in cerebral apoplexy I have reserved till the last merely stating about some place that should be administered for the satisfaction of the patient's friends and the patient himself, and that after some days after the attack. But during the attack the proper use of these remedies is our most valuable single resource.

Out of the vasodrugs. Ergot can be discarded. Adrenalin, digitalis, etc., are contraindicated. All stimulants, vascular tonics, morphine or opiates and, for the time strychnine should be carefully avoided.

The cardio-vascular depressants. Gelsemium, veratrum, or aconite—are sufficiently powerful and ordinarily safe drugs. Either of these can be administered hypodermically, though they also act promptly by the mouth Where the pulse warrants its use (and here accurate determination of the pressure is our best guide) it is well to begin with gelsemium. In adults the fluid extract can be started with an initial dose of 10 drops and followed by 5 drops doses at intervals dependent on the closeness with which the case can be watched.

Gelsemin which is more active can be used in doses of 1/10th grain or less. It should be pushed till its physiological action is manifest, whether little or much is required. The full effect of the drug is not obtained unless its paralysing effect is secured.

One of the most commonly used is Nitrite of Amyl. It is a good vasodilator and is practically the only drug available of this class, as it can be so readily administered by inhalation and it is innocuous (= harmless ; producing no ill-effect) unless asphyxia be already urgently threatening life. It is therefore worth a trial when the pulse is bounding and the tension high owing to its evanescent action, the effects must be kept up for some time before its trial is abandoned.

However 1/50th grain of Aconitine hypodermically is said to produce a moderate effect beginning in a few minutes and reaching its maximum in about thirty-five minutes.

An equal but quicker result (maximum in twenty minutes) can be gained by the use of 1/5th grain gelsemine hydrochloride.

The usual hypodermic of aconitine 1/120th grain is said to produce the opposite effect.

When medication on this line has to be continued for any length of time it may be necessary to change from full doses of gelsemium. Then the Nitrites become useful. It is usually advisable to keep up some influence of this kind for from a couple of days to a week.

The possibility of a second attack should be remembered and the patient should have always a light simple diet, should abstsin from alcohol, take only moderate exercise, and keep from business or other mental worry as much as possible and in particular avoid sudden exertion.

PLACE OF MUSK IN THERAPEUTICS

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Musk is the dried secretion from the preputial follicles of the musk deer, *Moschus moschiferous.* This animal is a small, graceful animal about the size of the roe-buck and inhabits a large area in Central Asia, extending from the Caspian sea to the Eastern boundries of the Chinese Republic. It is found throughout the Himalayas at elevations about 8,000 feet. The male musk deer, which alone produces the musk, bears on its belly, a short distance behind the navel and just in front of the preputial orifice, a small sac produced by an infolding of the skin. This sac is the musk sac or musk pod, and it contains a treacly or soft, unctuous brownish substance, *musk*, which is remarkable for its intense, penetrating, and persistent odour. Very young animals do not secrete musk and old animals but little. Most of the musk of European commerce is obtained from Tibet or from the Chinese province of Scechuen—Tonquin musk, which is the best variety. Smaller quantities are obtained from South China and some finds its way *via* Nepal or Assam to Calcutta.

Musk yields by distillation with steam and subsequent purification a small percentage of a viscid, colourless oil with a very powerful and agreeable odour of musk; this oil appears to be a ketone and has been termed muskone. The drug contains moisture, fatty matter, resin, proteids and inorganic substances. It should not yield on incineration, more than 8 % of ash or contain more than 15 % of moisture. Musk is liable to gross adulteration, which is in some cases easy to detect, but in others exeedingly difficult. Inorganic substances, such as small stones, leaden shot etc., are comparatively easily detected, and so are such adulterations as scraps of leather or horn. Dried blood yields a red ash, whereas the ash of genuine of musk is whitish. An interesting popular test for musk has been reported from the Punjab. A thread is passed through asafetida and then through musk pod. If after this, the smell of asafetida remains the musk is not genuine.

Musk has been in use in India for a very long time as a cardiac and general stimulant and as an aphrodisiac. Its fame as a cardiac stimulant is so great that it is almost the last resort when everything else has failed to support the heart. Musk was once official in the B. P. but has since been removed. It was official in U S.P. IX but has been deleted from U.S.P. X. It was formerly regarded as a powerful medullary stimulant and good results were claimed in cases of collapse. It has been used in the treatment of Hysteria, hiccough and other nervous manifestations, also in spasmodic asthma, and as a stimulant in Typhoid fever, pneumonia and bronchitis.

Modern pharmacological research has however not been able to substantiate the claims made for this drug.

There is no experimental evidence that could justify its use as a cardiac stimulant. But there may be some slight stimulation of the nervous system presumably by olfactory and psychic reflexes, due to the strong and penetrating odour. Experiments were done by us on intact and isolated mammalian heart using a sample of Tincture of musk prepared by Southall Brothers and Barclay Ltd., Birmingham, and no stimulation either in dilute or concentrated solutions was noticed.

A series of observations were made by us on human beings and it was noted that the pulse, respiration and blood pressure were not affected. But there was an unmistakable change in the cellular composition of the blood; there being an increase in the total number of white blood corpuscles. The subjects were patients in the wards of the senior author at the Madras Government General Hospital, a few controls among healthy medical students and doctors were also taken. The details of the experiments are given below:

The subjects were not given any food for at least 2½ hours—often 3 hours—before taking the blood for the preliminary examination. The blood for this was taken at the time of administration of musk.

During the whole time the observation on blood was being made, the subject was kept fasting, and at rest in bed.

For each prick a different finger was selected, and both squeezing the finger after pricking and ligating before pricking were avoided, a deep prick being made in order to ensure a free supply of blood.

The same hæmocytometer and the same microscope were used for all the observations and the same observer did the counting in all cases.

For the first four cases of whom one was a healthy control, a medical student—blood pressure, pulse and respiration were also recorded. But as they were found to be unaffected by the drug, these observations were not made in the rest of the cases.

The first seven cases were started on 10 minims of Tr. Musk in an ounce of water and as the total leucocytic count was beginning to fall after some time, they were given a second dose of 10 m. in an ounce of water and further observations were made. The observations were made every half-hour till a definite increase or a tendency to return to the normal of the leucocyte count was noticed. The remaining cases were started with 15 m. of Tr. Musk in an ounce of water only one dose being given and observations made till the leucocyte count began to decline.

TABLE A.

The following cases were given two doses of 10 m. at 1 to 2 hours interval :—

Name.	Age.	Weight.	Disease.	Leucocyte count.					
				1	2	3	4	5	6
Viswanatha Menon ...	41	91 lbs.	Ch. Malaria.	2140	6250	9000	8140	8000	10150
Vareed ...	32	106 lbs.	Dis. Sclerosis	4000	3440	7180	6200	7500	6785
Damodara Nambiar ...	22	128 lbs.	Control.	8016	8200	9000	...	...	...
Iyyappa Nair ...	24	120 lbs.	Syphilis with pyrexia.	4062	5310	5936	8200	...	...
Yellappa ...	30	105 lbs.	K A.	2190	3750	3125	3125	4150	4200
Viswanathan ...	25	138 lbs.	Control	6250	7250	6500	6250	...	...
Ramakrishnan ...	30	91 lbs.	K A.	5300	5936	5300	6900	8700	...

The first counting was done just before the administration of Musk. This gives the indication of the normal leucocyte count of the individual at the time of administration.

It will be noticed from the above table that the healthy controls show very little change ; the student's count (case No. 3) going up by about 900 at the end of the hour and in the case of No. 6, a doctor, the count rose by 1000 in half an hour returning to the original figure at the end of $1\frac{1}{2}$ hours.

TABLE B.

The following cases were given one dose of 15m. in 1 ounce of water to start with :—

Name.	Age.	Weight.	Disease.	Leucocyte count.			Remarks.
				1	2	3	
Kondan ...	23	98 lbs.	K. A.	4400	5000	5120	
Arulakuppan ...	28		K. A.	4060	5500	4700	Acute case.
Perumal ...	30	130 lbs.	K. A.	4225	7500	5850	Fall in 1 hour, Acute case.
Gopal Nair ...	25	125 lbs.	Enteric	4550	7000	4875	Acute case.
S. Gopal ...	25	125 lbs.	Pyrexia	4550	6500	6175	Acute fever 4 days.
Kanniah ...	29	100 lbs.	Pleurisy	2275	4875	4225	With effusion.
Subrahmaniam ...	23	110 lbs.	Influenza	6500	7800	7800	Temperature normal at time of examination.
Krishna Nair ...		120 lbs.	Influenza	7150	9100	9425	Temperature normal.
Veeraraghaviah ...	20	90 lbs.	K. A.	1300	2600	3500	
Ramachandran ...	12		Influenza	5850	7475	5850	Given 4 hours after food.
Muthukaruppan ...	19		Splenomegaly	2925	7150	6825	3 hours after food, Spleen 6".
Ismail ...	15		Convalescent after Pneumonia	8775	10400	9090	3 hours after food.
Viraraghaviah ...	20		K. A.	2275	4550	3900	Spleen 6". 3 hours after food.
Mohiuddin ...	24		Malaria	5525	6500	4875	Enlarged spleen and liver.
Arulsami ...	18		K. A.	1300	2925	2275	Spleen 2". Liver $2\frac{1}{2}$".
Subramani ...	24		Pneumonia	10400	11050	9750	Temperature 103·8. Cyanosed.

The above tables show that the most noteworthy results are obtained in cases of leucopenia, which invariably show an increase in leucocyte count, sometimes even double the initial count.

It may be further observed that the cases with acute infections show a more steep fall in leucocyte count after the preliminary rise. (Table B, 3, 4 & 5.)

Cases with moderate or well marked leucocytosis are not responsive. In case No. 16, the initial count of 10400 became 11050 in half an hour, but in another half an hour it was only 9750.

The above results are of the total leucocyte count. No change in the relative proportion of the various leucocytes have been noticed in the preliminary and later counts.

Discussion.

The question arises as to the mode of action of musk in thus causing an increase in the total leucocytes of the blood. We have not attempted to explain the mechanism of action. Bohlandt (1899) stated that a theoretical basis for pharmacological actions on the leucocytes cannot be given. Their number in the superficial blood may be changed by the distribution of the blood; or an increase in the leucocytes may occur owing to an increased emission from the blood forming organs and by new formation. A leucocytosis may also be caused by a contraction of the smooth muscles of the spleen and lymph glands, as after pilocarpine. The increased number of

leucocytes which one observes with increased activity and hyperæmia of the gut, and which is known as a digestive leucocytosis, may depend on an effect on the lymphoid tissue. Such a leucocytosis has often been observed after numerous substances which irritate the mucosæ, such as the bitters, etc. (Pohl. 1889).

Hyperleucocytosis is also produced by radium, owing, it is presumed, to an irritation of the blood forming organs; and lastly the number of leucocytes may also be indirectly changed by various drugs, like skin irritants. In which of these various ways does musk bring about leucocytosis? If the action were similar to that of bitters, one would expect a uniform increase in the leucocytes in all cases. But musk seems to have the maximum action in cases of leucopenia and little or no action in normal cases and in individuals with leucocytosis. Has musk got a selective action on the hæmopoietic system? or does it act through the endocrines?

Conclusions.

Musk has no direct or indirect action on the heart and the circulation. But it has a well marked effect on the cellular elements of the blood. The total number of leucocytes are increased after oral administration of musk. This effect is very marked in cases of leucopenia, the total counts being doubled in some cases, and comparatively little change is noticed in normal cases and in individuals with leucocytosis. This points to the probable therapeutic usefulness of musk in those cases where an increase in the leucocytes seems desirable.

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PEPTIC ULCERS.

By B. V. N. Mangesh Rao, F.R.C.S.

(These may be divided into Acute and Chronic.)

According to Hurst all peptic ulcers start as acute and the vast majority of these heal. But a few for some reason fail to do so. But some think that chronic ulcers are a definite entity—a definite specific affection no doubt due to a single factor but as yet undetermined. Faber of Copenhagen considers that acid dyspepsia, gastritis, and gastric ulcers as closely allied conditions, if not different phases of the same condition. Both acute and chronic ulcers have been found in the same stomach.

The normal mucous membrane of the stomach has an Anti-pepsin which prevents auto-digestion (Weinland). According to Hunter living cells are not vulnerable to peptic juices. When diffects due to perforation are closed by living tissue as omentum, no auto-digestion occurs. So long as living tissues, whose circulation is intact are used no digestion of such tissue occurs. But in Gastrostomy cases a troublesome eczema occurs round the opening owing to peptic juices. Mann and Williamson have experimentally produced peptic ulcers in the Ileum by eliminating bile and pancreatic juices by resection of the second part of duodenum.

Acute ulcers.—These arise in different ways and may be caused by the interplay of a number of varying factors. They occur in association with some infective focus elsewhere in the body. They develop quickly and

heal as quickly. They are often terminal lesions. They may give rise to no symptoms unless they erode a blood-vessel or perforate. Sometime they give rise to severe and even fatal hæmorrhage. Perforation is rare. They are prone to be multiple and occur quite frequently on the cardiac portion of stomach. They are cleft like or rounded in shape and vary from a pins head to two c. m. in diameter. In depth usually the submucosa is exposed but sometimes it may penetrate down to the peritoneal coat, and even perforate. Surrounding stomach is soft and pliable but may be oedematous and slightly inflamed. Bleeding is from the small arteries in the mucosa or submucosa and never from a main vessel. They heal and leave behind small scars difficult to identify in the voluminous folds of the gastric mucosa.

Chronic Peptic Ulcers.

Site of ulceration.—That part of Gastro-intestinal tract which comes into contact with acid gastric juice, as the stomach, first part of the duodenum as far as bile papilla and after a gastro-jejunostomy either the new stoma, the efferent limb of jejunum up to a distance of four inches, or rarely the afferent limb. Sometimes an ulcer has been found in a Meckels Diverticulum but always associated with presence of gastric glands. Chronic peptic ulcer is usually single but two or more may occur simultaneously. Both gastric and duodenal ulcer may be found in the same person. Duodenal ulcer is more common than gastric, 16:1 in Madras. Gastric ulcers are most common in the middle of lesser curvature and more common towards the pyloric than the cardiac end. They are more frequent on the posterior than on the anterior surface. Chronic ulcers in the cardiac portion or on the greater curvature are rare. The area

of distribution of these ulcers corresponds to the route followed by the duodenal secretion which flows back into the stomach. Similarly after a gastro-jejunostomy ulcer occurs at the meeting place of the two juices. It would seem that the interaction of the two juices, seems to render conditions more favourable for ulcer formation. The meeting place of the juices is shifted to the first part of the duodenum in cases of hyperchlorhydria and hypermotility of the stomach with the result, that the site of the ulcer is also shifted to this place, and a similar result occurs after gastro-jejunostomy.

Aetiology.—As yet no definite causative factor has been found, but certain definite facts have been noted in connection with them. Experimentally ulcers have been produced but they rapidly heal. All ulcers start as acute but the majority of ulcers heal, a few however owing to some unknown cause pass on to the chronic stage (Hurst).

Normal gastric mucosa cannot be auto-digested, but gastric juice can digest any localised patch of devitalised tissue, in the gastric mucosa, and so produce an ulcer. Therefore the two main factors in gastric ulceration are (*a*) devatalised patches in gastric mucosa, (*b*) presence of activated gastric juice *i.e.* pepsin and hydrochloric acid. Minute areas of necrosis or hæmorrhage first occur in the mucosa then the gastric juice acting on it produces the ulcer which is acute at first. Such necrosis and ulceration may occur in the rest of the intestines, but ... owing to the alkaline nature of the contents healing takes place rapidly. Focal sepsis seems to be an important factor. Solitary ulcer of the bladder-elusive ulcer, Hunner's ulcer in the ureter are examples of ulceration due to focal sepsis.

Factors concerned in the production of local necrosis.

1. *Anatomical.*—(*a*) *Vascular supply.*—Reeves has shown that the submucous artirial plexus at the lesser curvature has vessels of smaller calibre and greater length and that the anastamosis between them is poor *i.e.* the terminals are practically end arteries. When the stomach is full all the rugae disappear except two near the lesser curvature, running from the cardia to the pylorus along the anterior and the posterior wall of the stomach. Thus the spiral arteries along the lesser curvature are not straightened out, and being long and narrow the circulation to the part is poor.

The duodenal bulb is supplied by a branch of the Gastroduodenal (Kocher's). Its terminals in the submucosa are practically end arteries with narrow lumen and poor circulation. These terminal arteries in the duodenum and the stomach are fairly closely surrounded by muscle fibres, and are likely to be further affected by contraction of the muscle wall during the course of digestion. The round shape of the ulcer can be explained by supposing that a nutrient vessel becomes blocked by emboli leading to the formation of infarcts.

(*b*) *Presence of lymphoid tissue.*—Minute lymphoid follicles are present at the bases of the glandular crypts, superficial to the Muscularis Mucosæ. These are found in greater numbers along lesser curvature than at the cardia or at the greater curvature. They are more liable to be infected by ingested bacteria than by hæmatogenous infection. It is doubtful if chronic ulcers arise out of follicular ulcers (Hurst), though some think so.

2. *Infection* is the main factor in the production of devitalisation of the gastric mucosa

(*a*) *Bacterial organisms.*—Acute ulcers are found

post-mortem in deaths from acute infections as septicæmia, pyæmia, scarlet fever, etc. In some cases bacteria have been found in the edges and floor of the ulcers, and yet some ulcers have been found sterile. It is possible that in the latter cases it is some toxin that produces the primary necrosis. Primary ulcers are the result of hæmatogenous infection carried from some septic focus as pyorrhea, root-abscess, septic tonsils, nasal sinusitis. As HCl is a strong antiseptic ingested bacteria and their toxins are usually destroyed, but if heavy enough are more likely to cause a general gastritis with resultant inhibition of gastric juice. Moynihan ascribes Juxta-pyloric ulcers to an ascending infection via the lymphatics from foci chiefly in the right iliac fossa as chronic appendicitis, salpingitis and chronic cholecystitis. In cases operated for such septic conditions he has observed increased vascularity of the pylorus and hypermotility of the stomach. Braithwaite has shown the possible lymphatic path in this ascending infection. But Continental Authorities especially von Haeberer (Innsbruck) dispute this point. Rosenow of the Mayo Clinic carried out experiments to prove the "Focal Infection Theory". It was confirmed by Nakamura also of the Mayo Clinic. He was able to isolate streptococcus from both gastric and duodenal ulcers, and which seems to have a special affinity for the mucosa of these areas. He has experimentally produced these ulcers in animals by intravenous injections of cultures grown from both ulcers as well as root-abscesses, etc. He is of opinion that a special variety of streptococcus with an affinity for gastric mucosa is the causative factor.

(*b*) *Toxins.*—Rosenow was able to produce similar lesions with the toxins. Toxins absorbed from primary foci as teeth, tonsil, etc., may produce local necrosis. Or

the toxins may be elaborated by bacteria in the lymphatics or in the vessels of the mucosa near the ulcer. Non-bacterial toxins may also produce the same effect—duodenal ulceration occurring in cases of burns. Bolten has shown that under certain circumstances metabolic toxins may produce auto-digestion, and he suggests a toxin of endogenous origin as the causative factor. According to Stewart toxæmias of infections account for 50% of the ulcers. Chronic toxæmias may account for some of them, *e.g.*, chronic duodenal ulcers in connection with chronic appendicitis and cholecystitis.

Bacterial emboli may produce septic infarcts in these end-arteries, or thrombosis, and the toxins may produce local necrosis. The activated gastric juice then digests dead tissue and an ulcer results.

3. *Hydrochloric Acid.*—Normal secretion of HCl free and combined lies between 0·1 to 0·2%. Chronic Peptic ulcers are found usually in parts which are constantly in contact with acid gastric juice. The fundus of the stomach, owing to the gas cap and the vertical position of human-beings, is not in contact with the acid for 15—16 hours of the day, and chronic ulcers are uncommon here. In the later stages of a large chronic gastric ulcer even complete achlorhydria may be found throughout a fractional test meal, but such a condition is never found in a duodenal or jejunal ulcer. There is usually a long history of indigestion in achlorhydria cases. The achlorhydria may be due either to the accompanying chronic gastritis, the free HCl, being neutralised by the accompanying mucus, or may be due to the onset of malignant changes. If the mucus is got rid off by proper gastric lavage before a test-meal, free HCl can always be found enough to activate Pepsin which is found in normal quantity. The presence of HCl

is essential in the formation of the ulcer. Acute ulcers have a tendency to heal, but in some cases one may show no tendency to heal. The increase in HCl, coarse food, irregular meals, presence of free acid in an empty stomach are probably some of the factors in preventing healing (Hurst). It is the persistence of acid gastric juice in the stomach rather than the increase in the acidity which is the important factor, especially in an empty stomach.

Factors which give rise to increase in HCl.

1. *Spices*, condiments naturally give rise to an increase in the gastric secretion, they also act as irritants to gastric mucosa. Alcohol and vinegar also act in a similar way. Meat diet also increases the gastric juice more than a vegetarian diet. When chemical irritants as alcohol, etc., are taken with food or immediately after, they get diluted and are not so deleterious as when taken on an empty stomach or between meals. They not only increase the gastric secretion but also act as irritants to gastric mucosa. When taken on empty stomach they pass along the lesser curvature to the duodenum and so the stress is felt along the lesser curvature. Drinking of very hot or cold drinks may have a similar effect.

2. *Smoking.*—Excessive smoking is more often associated with duodenal ulcers. This probably accounts for a higher incidence of duodenal ulcers in men than in women. The nicotine paralyses the sympathetic synapses which inhibit peristalsis and secretion. The Vagal supply becomes uncontrolled, leading to hypersecretion and hyperperistalsis (Langley). The appearance of pain in a quiescent ulcer, or a relapse may follow excessive smoking. Smoking on empty stomach (cigerettes) is the worst. Chewing of tobacco will have a similar effect.

3. *Food-mechanical.*—Proper mastication of food bring it into a non-irritating state, physical as well as chemical. The habit of bolting food, or in edentulous cases, brings coarse food to the stomach and the stress of this is felt mostly along the lesser curvature of stomach, in the Orthostatic Hour-glass or the Spasmodic Hour-glass stomachs (the result of chronic appendicitis). The lesser curvature is less mobile owing to the gastro-hepatic omentum. All food is completely softened before reaching the duodenal bulb. Irritation due to chemical food factors has been mentioned above.

4. *Nerve disturbances.*—Stimulation of the Vagus produces hyperperistalsis and hypersecretion. Stannke produced ulcers along the lesser curvature by this. Yzeren produced chronic ulcers along the lesser curvature by section of Vagus below the diaphragm. In this peristalsis was exaggerated into cramplike contractions which preceded ulcer formation. From these experiments it was assumed that the ulcer resulted from some disturbance of the Vagitative Nervous system an imbalance between the Para-sympathetics and the Splanchnic sympathetics. The fact that the removal of lesser curvature where the nerves of the stomach are chiefly aggregated does not lead to the formation of any more ulcers is brought forward to support it, and is also advocated for treatment of gastric ulcers (Shoemacker). After a sleeve resection the denervated distal part shows. no tendency to ulcer formation. This theory was chiefly supported by Von Bergman. The action of nicotine from smoking or chewing tobacco has been already mentioned.

5. *Vitamin Deficiency.*—The possibility of deficiency of some vitamin in the food as predisposing the

person to peptic ulceration has been brought forward. MacCarrison has conducted some experiments in rats and has produced ulcers but it must be remembered that in gastric ulcer cases operation wounds of stomach heal by primary intention without any immediate tendency to ulceration.

6. *Ulcer Diathesis.*—Campbell and Connybeare have shown that a large percentage of men with hyperchlorhydria have also hypertonic stomachs—Hypersthenic Gastric Diathesis *i.e.*, both hypertonus and hyperperistalsis. This condition predisposes to duodenal ulceration, and is more common in men and also runs in families. In this type undiluted and very highly acid gastric contents pass into the duodenum and the duodenal bulb is in contact with it for several hours of the day. It is possible that this diathesis is due to lessened activity of the sympathetics. But then it is difficult to suppose and explain a Gastric Diathesis. Uncomplicated gastric ulcers are rarely associated with hyperacidity and never with hypertonus. It is more often associated with hypotonic stomachs. Orthostatic Hour-glass stomach (hypotonic dropped stomach) is present in such, and in these the middle of the stomach forms a definite obstruction to the passage of food so long as the erect posture is maintained. This results in the mucosa along the lesser curvature of the stomach being subjected to friction and prolonged contact with acid gastric juice. Therefore a hypotonic dropped stomach is associated with ulcer of lesser curvature (J shaped stomach). The stag-horn stomach is a high hypertonic stomach with hyperchlorhydria and occurs particularly in men of an athletic build with broad chests. Low hypotonic stamachs (J shaped) occurs in men of less vigorous type with long norrow chests.

7. *Deficiency of Internal Secretion.*—It is possible that some derangement of internal secretion as yet undiscovered may be the cause. The inhibitory activity of the sympathetic system is lowered by the less production of adrenalin resulting in a Vagotonic condition. It is possibly the cause of more marked symptoms at night in duodenal ulcers as the adrenal secretion is low in the night. Compare this with the appearance of attacks of colic and asthma at night. Mental and physical fatigue produce suprarenal exhaustion ending in gastric-vagal activity which often produces relapses and recurrences in the symptoms of peptic ulcers.

8. *Bile and Pancreatic Secretions.*—Mann and Williams were able to produce ulcers in the ileum by resecting duodenum with the bile-papilla, *i.e.*, abstracting bile and pancreatic juices. Morton produced in 100% cases experimentally in animals gastro-jejunal ulcers by duodenal drainage. But why the alkaline contents of duodenum fail to regurgitate into the stomach in these cases of peptic ulcers and what the cause for this is, has to be yet found. But at the same time it must remembered that the site of the ulcer is the meeting place of the two juices, as mentioned before.

After all the actual causative factor in this fairly widely prevalent disease may be a single one as yet undiscovered, and all causes mentioned above may be only aiding factors. I suggest that it may be some interference with an internal secretion as yet unknown, that predisposes the individual to peptic ulceration. On this one can explain ulcer diathesis personal or familial.

Age.—Highest incident is between the ages of 20—50, *i.e.*, the active period of life, but it has been found in

younger people. The youngest I operated on was 13 years old with a history of 2 to 3 years duration. It was a case of a callous ulcer with duodenal stenosis and dilated stomach.

Sex.—It is more common in men than in women, in Madras it being 26 : 1. This may be explained by the fact that smoking, use of alcohol, irregular meals, strenuous life, etc., are more common in men than women.

Pathology of peptic ulcer.

HCl percentage.	Free HCl	Chlorides.	Total.
Nomal stomach.	0·15 (0·1 free. 0·05 protein.)	0·1	0·25%
Peptic ulcers.	0·24	0·1	0·34%
Dyspepsia	0·18	0·1	0·28%
Cancer of stomach	0·05	0·1	0·25%

HCl acts as a strong bactericide and is an activator of pepsin for protein digestion. By itself it has no digestive powers but it may act as an irritant to an existing ulcer. Normally gastric juice is secreted when the appetite for food is roused during the act of eating and for a little while after. According to MacLean as the secretion of free HCl disminishes there is more and more secretion of neutral chlorides. In health a small meal is completely out of stomach in two hours and a large one in 5 to 6 hours.

The earliest stage of chronic ulcers is not definitely known. It probably starts as a small acute ulcer and from some unknown cause persists and takes on the charac-

teristics of a fully developed chronic ulcer. It takes one of two forms, either a flat oval ulcer, or a funnel-shaped deep cavity, with overhanging margins. The surroundings of the ulcers are greatly thickened and indurated probably from fibrosis the result of inflammatory changes. The most probable explanation is that a local necrosis or a septic infarct gives rise to ulcer from auto-digestion, but the tissue reaction is not characteristic of inflammation. There is an absence of leucocytic infiltration but the organism may be a specific one the reaction of tissues to which is characteristic.

Askanazy says that the margin of the ulcer is overhanging on the oral side and flatter on the aboral side. The following layers were seen by him. (1) A layer of surface exudate of fibrin, leucocytes, and a few erythrocytes. (2) A zone of necrosis, the dead tissue being infiltrated with fibrin. (3) A zone of granulation tissue. (4) Cicatricial zone extends widely into the surrounding parts the muscle fibres being destroyed. Arteritis Obliterans is very common in the surrounding blood-vessels and affects even the large arteries. According to Perman there are some ulcers in which granulation tissue is wanting and in perforated ulcers this is always the case, its absence being a sign of rapid spread of the ulcer. The general mucosa may show a pronounced gastritis or duodenitis in some of the cases though not commonly. These ulcers are liable to attacks of acute inflammation with periods of quiescence when the ulcers may show a tendency to heal. Acute ulcers are superficial involving the mucosa and exposing the muscularis sometimes they rapidly penetrate to and through the peritoneum. Hæmorrhage is from the smaller vessels in the mucosa and submucosa. Perforation in acute ulcer is rare. Acute

ulcers are multiple and heal quickly. Hæmorrhage and perforation are themselves acute, but they are usually with a few exceptions an evidence of chronic ulcers, which have assumed fresh activity.

Gastric Ulcer.—The process of fibrosis round the ulcer extends into lesser omentum, which becomes thickened, infiltrated with fat, contracted and distorted. The ulcer may thus be dragged back to the posterior abdominal wall. The ulcer and its surroundings form a hard callous mass, sometimes palpable through anterior abdominal wall, if the ulcer is on the anterior aspect. As the ulcer deepens it forms adhesions to neighbouring structures. The ulcer on the posterior surface often penetrates into the pancreas, which later on forms the base of the ulcer. At first the superficial layers of the pancreas get fibrosed and adherent to the ulcer, later excavation into it occurs the egg-cup excavation, in shape and depth. Anterior ulcers of the lesser curvature may penetrate into the liver in a similar way, the excavation here resembles that of a soup-spoon, shallow and oval.

Perigastritis may obliterate parts of the lesser sac, the mesocolon being dragged up to the site of the ulcer behind the stomach and sometimes so contracted that a posterior gastro-jejunostomy is an impossibility. Rarely an ulcer may become adherent to the anterior abdominal wall. The spread of the disease may be very extensive especially when multiple ulcers exist and the stomach may get fixed by perigastric adhesions to liver, pancreas, and posterior abdominal wall, making any operation an impossibility. A callous ulcer over a certain size and if infiltrating shows little tendency to

heal and requires operative interference. When healing takes place in such ulcers, cicatrisation leads to deformity, from wide spread infiltration of the gastric walls. The fibrosis at the site of the ulcer contracts as soon as the active process ceases. This causes at the pylorus, stenosis ; a saddle shaped ulcer on the lesser curvature owing to cicatricial contraction produces a ring like contraction pulling the greater curvature towards the ulcer ending in an hour-glass stomach. The pouches are often of unequal sizes and the isthmus may occasionally be of very norraw calibre. This condition is rare but is more common in women and is often combined with pyloric stenosis.

Deodenal Ulcer is more common than gastric ulcer. The most common situation is along the pancreatic-wall of duodenum an inch to an inch and a half distal to pylorus. These ulcers are rounded or triangular in shape and are small in size, rarely if ever they attain the size which some of the gastric ulcers may attain. But the surrounding fibrosis is well marked. The ulcer may penetrate the lesser omentum distorting the anatomy of its right border and dragging the duodenum towards the liver. Or it may penetrate towards the pancreas obscuring the line of separation between the gland and duodenum. Owing to the surrounding fibrosis and its contraction the common bile duct is brought down close to the duodenum into a position of great danger during duodenectomy. Commonly there is one on the anterior wall of the first part and one on the posterior wall of the junction between the first and second parts. The duodenum becomes distorted partly by contraction of the fibrous tissue in its own wall and partly from adhesions to neighbouring structures such as gall bladder

lesser or greater omentum pancreas, hepatic flexure or liver. It may become embeded in these adhesions. Owing to the shortening from fibrosis the bilepapilla comes to lie nearer the pylorus. The ulceration may extend towards the pylorus and give rise to pyloric obstruction (Moynihan) but others consider this as a primary ulcer of pylorus. Moynihan says that pyloric ulcer is very rare only 3 % or less occurring at the pylorus or within an inch or inch and a half of it. Contraction in the wall of duodenum causes duodenal stenosis and obstruction. It is very difficult to make out the exact position of pylorus in such cases but one can identify the pyloric vein which though not constant in position is a fair landmark for judging whether an ulcer is duodenal or gastric in type. Some duodenal ulcers may show no fibrosis and are difficult to detect by palpation. Very often the peritoneal coat over the site of such ulcers is whitish in colour with radiating streaks, and the area becomes stippled with red dots especially after a little handling. Lymphatic glands are always a little enlarged in cases of duodenal ulcer at the lower border of duodenum and along the greater curvature of the stomach. Duodenal ulcers heal and leave small scars, but scars are seen in the stomach in a slightly greater proportion than in the duodenum. Ulcers here have a great tendency to heal but callous ulcers especially those along the pancreatic wall are frought with danger of hæmorrhage. Symptoms of biliary obstruction are rare though the common bile duct may be surrounded by fibrosis. Ascending cholengitis is rare, but a slight jaundice may occur in cases of duodenal ulcers from accompanying duodenitis. The ulcer seldom extends as far as the Ampulla of Vater and there is always a healthy portion of wall between the

two. Acute duodenitis and gastritis may sometimes complicate a chornic duodenal ulcer (Judd).

Symptoms.—Acute gastric ulcer occasionally gives rise to sever bleeding and less often to perforation.

Chronic gastric ulcer may remain latent until a sudden perforation or hæmorrhage discloses its presence. Usually a characteristic group of symptoms and signs are present. Chronic gastric ulcer is more common in men than in women and usually occurs between the ages of 30 to 50. Over 50% of the ulcers occur along the lesser curvature and less than 3% at the pylorus or within 1½" of it, but this portion is the one commonly affected by cancer. There is a characteristic periodicity in the attacks of pain. The syndrome comes on due either to some error in diet, an indiscretion, or over exertion, or may follow some infectious disease. It lasts for a few weeks and there is respite for months or even years followed again by an attack. The exacerbations probably coincide with infection in the wall of the ulcer (Moynihan). When the ulcer is not active or shows some healing there are no symptoms but when it takes a new activity and its edge is spreading, there is infection all round the ulcer and a local spasm is excited. There are seasonal variations and a cyclic character in the occurrence of the symptoms. The ulcer may remain as a callus ulcer without any signs of healing but give rise to no symptoms. As the ulcer gets larger and becomes adherent or penertrates into neighbouring structures the intervals of freedom become less and less and finally they become continuous and unremitting.

Pain—this soon exhausts the patient's vitality and reserve. He looks drawn and haggard during the exacerbations to recover considerably during the intermissions. The pain comes on after a definite interval after taking food, a quarter of an hour to two hours but has no relation to the site of the ulcer. Moynihan has called attention to the quadruple rythm of gastric ulcers—Food, Comfort, Pain. Comfort till the next meal is taken when the cycle is repeated. Pain is felt in the left epigastrium but when the ulcer becomes adherent to the pancreas with perigastritis the pain becomes more constant, severe and sometimes colicky in nature. Penetration into the pancreas may cause pain across the back. Sometimes the attacks may be severe enough to require morphia. Some ascribe the pain to the irritation of the nerve endings by the highly acid juice. But the acme of pain does not correspond with period of greatest acidity during digestion. Others ascribe it to pyloric cramps. According to Hurst it is due to increased intra-gastric tension stretching the muscular wall. But Reynolds and McClure prove to the contrary. They maintain that motor or sphincteric disturbances often accompany the pain of ulcer but are not necessarily the cause of it. Spasmodic pain in gastric and duodenal ulcers may be due to stretching of certain nerve endings. Heartburn is associated with peristaltic contractions in the esophagus and a rise of pressure within it (Payne and Poulton). Nausea is also related to tension of specific esophageal nerve endings. "Sinking Feeling" is due to stretching of sensory never endings in the lower part of stomach. Pain in the upper abdomen is commonly associated with movements of pyloric portion of stomach, duodenum, or jejunum. Where postural tone is low pain may accompany relaxation of muscular wall of viscera (Strong Heaney). Spasmodic pain of ulcers may be due to tension but alkalies relieve the

pain without inhibiting peristalsis. This is probably due to relaxation of pyloric sphincter, whose spasm is apparently relieved by alkalies. Hunger pains are referred by majority of patients to lower mid-sternal region. But they were shown by Cannon and Washburn to originate from contractions of the stomach and also sometimes from the lower end of the esophagus occurring in conjunction with increased tonus. But these occur only in subjects of peptic ulcers, a fact which supports the view that pain may be due to postural tone of the affected organ than to active muscular contraction (Strong Heaney). It is possible that some chemical constituent of food or gastric juice irritates the nerve endings exposed in the ulcer and sets up a neuritis in them. Vomiting relieves the pain characteristically. If vomiting fails eructation of gas is prominent and heart-burn due to escape of a little gastric juice into lower end of esophagus.

Pain may be mild or severe enough to be agonising and render the patient unfit. Pain may be of a stabbing, gnawing, sharp, burning, grinding or a sinking feeling type as described by patients. Sometimes patients complain of radiating pain into the shoulder especially left, round the abdomen to the back. But it always has a definite relation to food, and there is a periodicity in it. When pain has become constant, a long history can be elicited. Pain in the back and when constant indicate perigastric adhesions and penetration into pancreas. Pain in the shoulder is suggestive of penetration into the liver. Rosenthal says that 50% of lesser curvature ulcer cases complain of hunger pain.

Vomiting.—80 to 90% of gastric cases and 10 to 20% of duodenal cases give this symptom. Vomiting

usually takes place at the crisis of pain which is thereby relieved. In the early stages it is often induced in gastric cases. Later it is due to dilatation of stomach owing to pyloric or duodenal stenosis.

Periodicity of attacks with seasonal variations, in both gastric and duodenal ulcers there is a periodicity in the attacks which depends either on some indiscretion or season-cold and damp weather. At first short attacks of ill-health are separated by long periods of well being, but give place gradually to long spells of ill-healh and short periods of well being ; and ultimately the patient is more or less always ill.

Hæmorrhage, may be small in amount or severe. It is more common in woman 66%, and 35% in men. It usually occurs as hæmatemesis or malaena. Malaena occurs in about 10% of the cases of gastric ulcers. Bleeding in some form occurs in about 30% of the cases (Borster) according to Walton in 20%, and Balfour 25% of the cases. With the use of microscope and meat free diet 60% of the cases may show erythrocytes in motions.

Appetite, is good but the patient is afraid to eat, patient wants his food and can enjoy it but for the fear of pain. Appetite may be lost in the later stages owing to extensive fibrosis and hypochlorhydria or to occurrence of cancerous change in the ulcer. Nutrition fails in gastric cases as patient is either afraid to eat or has to relieve himself by vomiting. He looks therefore poorly fed in contrast to duodenal ulcer cases who is usually a well fed man unless he has pyloric or duodenal stenosis.

Signs, there may be none. Frequently there is some tenderness in the epigastrium just below the

ensiform cartilege either in the mid-line or a little to the left *i.e.*, a point corresponding to the lesser curvature of the stomach. An increased tone in the upper part of the rectus especially the right is sometimes noticeable. Rarely atumour mass may be palpable in thin subjects—a callous mass surrounding the ulcer. In neglected cases when pyloric obstruction has set in, visible peristalsis and characteristic vomiting occur.

Test meals.—Ewald's Test Breakfast consists of a slice of dry toast and a pint of weak tea without milk. An hour after it is given a sample from the stomach is taken and tested for free and total HCl. The secretory response is judged from this. In some people there may be delay in the secretion and this test breakfast is unappetising and so the stomach response may be poor. It would be better to give 300 ccs of 5 % alcohol in the form of brandy and withdraw it after half an hour. (Pennet).

Rehfuss Fractional Test.—Meal is composed of thin oatmeal gruel, samples being withdrawn every 15 minutes. By this delayed secretory response and the time when the stomach empties can be noted. Gastric function may be disturbed by the presence of the Ryle Tube. Ryle says that hyperchlorhydria is not common in gastric ulcers. Moynihan says that about 20 % of gastric cases exhibit hyperacidity, and 13% may actually show achlorhydria.

1. *X-ray examination.*—Absolute proof of an ulcer depends on the demonstration of its crater filled with barium. The crater of ulcers on the lesser curvature can best be seen in profile in the supine position as a projecting niche. For ulcers on the posterior wall of the

vertical portion of the stomach lateral, oblique or Trendelenburg position has to be used. Direct visualisation of pyloric ulcers is difficult.

2. The opaque material is apt to remain in the niche of the ulcer after the meal has left the stomach. The persistence of a fleck is characteristic. Posterior ulcers penetrating into the pancreas are best seen after the stomach empties or at the upper border of the lesser curvature in the lateral view. Shallow ulcers may be inferred from an alteration in the pattern of mucosal rugae, which are seen in a partly filled stomach at the beginning of a meal as roughly parallel lines following the curvature of the stomach. In the presence of an ulcer, they tend to radiate from the lesion in astellate manner.

3. *Disturbances of motility.*—On the screen the peristaltic indentation passing along the lesser curvature is lost owing to the fibrosis at the site of the ulcer. Gastric spasm from reflex causes disappears after full doses of atropine. In pyloric obstruction there is delay in the emptying of the stomach. Similarly there is delay from loss of propelling power of the stomach in cases of callous ulcers on the lesser curvature of the stomach. With bread and milk meal or Umbrose (barium meal) stomach normally is empty in four hours. If a six-hour residue is left it is a clear indication of some obstruction due to ulcer, perigastritis or pyloric stenosis.

4. Often there is a spasmodic indrawing of the greater curvature opposite an ulcer on the lesser curvature. Such a spasm is suggestive of an ulcer, if reflex spasm is excluded.

5. The appearance of an hour-glass stomach is typical but reflex spasmodic hour-glass should be excluded by giving full doses of atropine.

Visual examination of ulcers is now possible by the Gastroscope.

Duodenal Ulcer.

It is more common in men than in women and its incidence is at an earlier age than in the case of gastric ulcers. Usually between 20 to 60. The seasonal incidence of the exacerbations is more pronounced than in gastric ulcers. Most patients are robust—the Hypersthenic Gastric Diathesis of Hurst, and have both hyperchlorhydria and hypermotility of the stomach. But some may be thin and drawn. Hunger pains are characteristic. The triple rythm of Moynihan "Food, Comfort, Pain, till relieved by food" is noted in all. Appetite is very good and they usually feed well. The type of person is broad chested with a wide epigastric angle and a stage-horn type of stomach. Pain is felt in the epigastric angle rather to the right side, one or two hours or more after a meal and continues till the next-meal. It often wakes up the patient in the early hours of the morning and is relieved by taking a biscuit and milk or some food or alkalies. As the disease progresses the pain lasts longer, and when the ulcer has penetrated into the pancreas it may become continuous. Sometimes the ulcer may remain latent till a perforation or hæmorrhage occurs. Others may have symptoms simulating a nervous dyspepsia and such cases are very difficult to diagnose. Hyperchlorhydria, hunger pains the periodicity and retention of appetite may occur in ulcer of lesser curvature. This may occur in cholelithiasis, pericholecystitis, or chronic appendicitis

as well. It is possible that it is due to an associated duodenitis. Enlarged glands may be present on the greater curvature as an expression of both duodenitis or gastritis. Tenderness is usually to the right of the mid-line. Rarely a mass may be felt in thin subjects. Vomiting in the early stages is not common, but in some it is induced. Later with onset stenosis or extension of ulceration to pylorus (Moynihan) and spasm, vomiting is more frequent. It is present in about 50 % of the cases.

Hæmatemesis is less common but malæna is more frequent than in gastric ulcer. Occult blood is demonstrable in 70 % of the cases. Slight transient jaundice may occur rarely, due probably to the accompanying duodenitis.

Test-meal—Fractional test meal.—There is an abundant, rather highly acid, fasting juice containing little or no mucus. After an initial fall in the acidity there is a steady rise to a high level followed by a steep drop with a rapid emptying of the stomach, or a sustained acidity with a rapid or delayed emptying, and a continuous after secretion.

X-ray examination—Food is held for a few seconds in the first part of duodenum and passes rapidly through the second part, which is therefore rarely seen. With abarium meal in the duodenum the duodenal bulb appears like a Cardinal's hat, but in a few seconds it is expressed out and passes rapidly into jejenum. Photographic exposure at this moment will show no duodenal cap. Sometimes the shadow of the stomach hides it, when the stomach lies in front of it. The shape may also be changed when the duodenum runs directly

backwards. Therefore both oblique and lateral views should be taken in addition to the A. P. views. Alterations of the duodenal cap should be present in more than one view to be of value. The more liquid meals are the best. Deformity may be cicatrical or spasmodic. The actual creater is rarely seen. A niche may be seen on the greater of lesser curvature of duodenum after the bulb has emptied. A spasmodic indrawing of the border of the duodenum opposite the ulcer may be visible. Sometimes a most irregular shape may be seen owing to several areas of spasm. Infiltration of the duodenal wall leads to its rigidity and loss of distensibility, so that a flattening of the lesser curve of the bulb may occur. Asymetrical situation of the pyloric canal on the flat basal border of the cap-shadow is indicative of adhesions or distortion from stenosis or fibrosis. If the bulb is filled by manual pressure and several observations are made, it is possible to distinguish true deformity from that caused by inflammation in neighbouring organs.

Indirect signs of duodenal ulcer are seen in the stomach. When there is no obstruction hypertonus and exaggerated peristalsis are seen. Tumultuous action of the stomach, hyperactivity of pyloric antrum is shewn by the fact that there are more than two peristaltic waves visible simultaneously. When organic stenosis is present the stomach is found dilated, hyperperistalsis and reversed peristalsis may be present and inspite of hyperperistalsis there is a six hour residue of barium meal.

Bowels are either normal and regular or patient is constipated.

Complications and sequelæ of Peptic ulcers.—*Haemorrhage*—ortality from this is 8·3% (Bulmer). Patient may have a single massive hæmorrhage. More often he rallies but there may be recurrent hæmorrhages. Bleeding from an ulcer is of the secondary type and recurrence may sap patient's strength. A pulse rate of over 100 means patient has lost a great deal of blood and with only 50% hæmoglobin will not stand an operation. A blood tranfusion is necessary in such cases before operating.

Perforation.—Though an acute condition is more common in chronic ulcers than in acute ones. Moynihan and Stewart found in 61 cases of perforation 60 were of the chronic type of ulcers; and in 14 cases of death from hæmorrhage, 13 were from chronic ulcers of the stomach. In 117 cases of perforation of duodenal ulcers 109 were of the chronic type.

Late cicatrised stomach.—When the ulcer is on the lesser curvature and of the saddle-shape type it is very liable to give rise to hour-glass contraction. It is more common in women. It is often associated with pyloric stenosis. The proximal sac is usually the larger.

Perigastric and periduodenal adhesions have already been mentioned.

Linitis Plastica. — Extensive fibrosis following on gastric ulcer giving rise to the so-called leather bottle stomach is really due to scirrhus type of carcinoma supervening on ulcer.

Carcinoma of Stomach—According to Mayo clinic 70% of chronic gastric ulcers left untreated end in malignant transformation. McCarty (Mayo Clinic, 1928)

reiterates that nearly all ulcers of a diameter of 1" and over are malignant and that 12 % of apparently benign ulcers a cytoplasea of cancerous nature is recognizable by special Histological methods. Moynihan and Sherren supported this. 18·5 % of excised ulcers showed malignant changes. Till 1920 this view prevailed. Paterson, Spilsbury, Morley and Stewart have strongly criticised this view. The mistake was probably due to the fact that the distortion produced by fibrosis leading to an apparent downgrowth of epithelial cells was mistaken for malignant changes (Ewing and Spilsbury). Morley thinks that malignant change in gastric ulcer is rare. Paterson records that only 2 % of the cases of ulcers treated by gastro-enterostomy become malignant subsequently. Stewart points out that 73% of the ulcers occur along the lesser curvature, but 67% of gastric concers occur at the pylorus, or the prepyloric inch. Gastric ulcers are uncommon at the pylorus, only 3% occurring there (Moynihan), or within an inch and half of the pylorus. But this is the commonest situation for gastric carcinoma Finisterer puts it at 26·6% for all gastric ulcers. Any ulcer with a diameter of more than an inch should be considered as malignant for purposes of treatment. The present view in England and America is that malignant change in gastric ulcers is very low.

X-ray examination in carcinoma.—Presence of tumour causes a "filling defect" and absence of any movement over the affected segment. Malignant ulcers may have a deep crater but are surrounded by a prominent ridge of tumour tissue. In profile the crater rarely projects outside the plane of stomach mucosa, *i.e.*, a true niche is rarely present. If on the posterior surface the ridge of tumour is seen as atranlucent ring round a central

opacity. Professor Duval has introduced Pneumogastrogram by which the extent of cancer can be judged.

Diagnosis.—The periodicity in the symptoms, seasonal occurrence of the attacks, and the characteristic symptoms in these cases should help in the diagnosis. X-ray examination and fractional test meals will aid in the diagnosis. In a long standing case it is always possible to get this history of periods of complete freedom from pain alternating with attacks characteristic of ulcer.

Differential Diagnosis.—In some cases both X-ray pictures and clinical symptoms may suggest an ulcer but none may exist and the condition may be due to Gastroptosis. Chronic appendicitis is another disease which may simulate a duodenal ulcer. Similarly chronic cholecystitis may give rise to symptoms suggestive of gastric ulcer. Chronic pancreatitis and pancreatic lithiasis have been mistaken for peptic ulcers. In this country tuberculosis of intestines and lungs should be excluded as they in the early stages are liable to mislead. Cirrhosis of liver in the early stages may resemble gastric ulcer both in the symptoms and may give rise to hæmatemesis. It has been observed in several cases of peptic ulcer of long standing that there is an accompanying cirrhosis of the liver which may add to the difficulties of the operation, in that these patients stand the chloroform anæsthesia very badly. There are also cases of nervous dyspepsia which may resemble an ulcer case so closely that an unnecessary operation may be done with worse results. These pitfalls should be avoided, but sometimes it may be necessary to do an exploration to be certain.

ASSOCIATION NOTES.

The following communication is published for the information of those interested in the question to which it relates:—

No. Z—6178/1/DMSI (A.D. 2).
Regr. No. 12299—A.D. 2. GOVERNMENT OF INDIA,
ARMY DEPARTMENT,
New Delhi, the 21st November 1928.

To

THE HONORARY SECRETARY,
INDIAN OFFICERS' ASSOCIATION (MEDICAL SECTION),
Latice Bridge Road, Adyar, Madras.

Method in which temporary Officers of the I.M.S. who have been permitted to retain their rank on demobilization should describe themselves.

SIR,

With reference to your letter No. 8/27, dated the 8th February 1928, to the Director, Medical Services in India, I am directed to say that under existing orders an ex-temporary Officer of the Indian Medical Service who has been permitted to retain his rank can describe himself as Lieutenant or Captain (as the case may be)—*i.e.*.....(name), late I.M.S. (T.C.); but if he remains in Government service, he will not be described officially by his military rank except in gazette notifications.

I am, Sir,
Your most obedient servant,
(Sd.) J. C. DAS GUPTA,
Offg. Asst. Secretary to the Govt. of India.

PROCEEDINGS OF THE BOMBAY MEDICAL COUNCIL.

The following summary of the proceedings of the meeting of the Bombay Medical Council held on the 18th February 1929 is published in the Medical Press for information.

1. The Council considered the case of Mr. Naraindas Dharamdas Lalvani, a registered medical practitioner of Hyderabad (Sind), who had been summoned to appear before the Council on the following charges:—

(1) That he being a registered medical practitioner under the Bombay Medical Act, 1912, on or about the 3rd June 1927 issued a certificate over his signature that a boy by name Gada Hussain Ghulam Hussain had been under his treatment from the 5th January 1927 to 5th March 1927 for Pneumonia following Diphteria and was advised to keep away from school on account of the highly infectious nature of the disease;

(2) That the registers of the Government High School showed that the said Gada Hussain attended the school upon all the working days throughout the months of January, February and March except one day in February when he was given leave;

(3) That it thereby appears that he did not in fact treat the said Gada Hussain during the period mentioned as stated in the said certificate and that his said certificate was in fact untrue;

(4) That in his answer dated 28th June 1928 to the Head Master, Government High School, Hyderabad, Mr. Naraindas stated that the dates of the period of illness were supplied to him by the father of the boy; and

(5) That he has committed a breach of Rule 2 of the Code of Medical Ethics by the issue of the said untrue certificate.

As Mr. Naraindas did not submit a satisfactory explanation of his conduct or express regret for it, the Executive Committee considered that the case was one in which an enquiry ought to be held and directed the Registrar to take steps for the institution of an enquiry under the rules. Subsequently, however, Mr. Naraindas having, before the date of the meeting, submitted a letter in which he admitted his negligence in not verifying the dates entered in the certificate which he granted, and expressed his regret for not having done so, the Council resolved as under :—

> "That in view of the appeal by Mr. Naraindas D. Lalvani to forgive, and his admission of having granted an incorrect certificate—which he might have done earlier with grace—the Council do not propose to proceed further with the case and warn Mr. Naraindas to be careful in future in granting such certificates."

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2. The Council resolved to accept for registration as an additional qualification the Diploma in Medical Rodiology and Electrology (D. M. R. E.) of the Cambridge University.

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3. The Council considered a letter from Mr. Dr.D. Sathaye, F.R.F.P.S. (Glasg.), requesting to be informed what procedure he should adopt if he wished to get the letters F.C.O. (Fellow of the College of Ophthalmology, Bombay) registered as an additional qualification, under the Bombay Medical Act, and resolved to inform him that the Council were unable to agree to the proposal as their policy is to recognise for registration only such qualifications as are granted by recognised Universities or other regular Corporate Examining Bodies.

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4. The Council considered an application from Mr. A. B. Yardi for permission to be registered under section 7 (3) of the Bombay Medical Act and resolved to inform Government that

in opinion of the Council the permission applied for may be granted.

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5. The Council considered an application from Mr. R. L. Mungrey for permission to be registered under section 7 (3) of the Bombay Medical Act and resolved to inform Government that as the applicant was not able to produce satisfactory evidence that he was practising medicine in the Bombay Presidency before the 25th June 1912, he could not be given the benefit of section 7 (3) of the Act.

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6. The Council resolved that the meetings of the Council be thrown open to the Press except on occasions when for special reasons cases are heard *in camera*, and that in penal cases full notes of the proceedings be recorded.

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7. The Council considered a reference from the Bombay Government communicating, for remarks, a recommendation of the Surgeon General with the Government of Bombay that Miss Holdforth, a trained midwife and nurse, who has been in charge of the Baker Hospital, Larkana, for the past 18 years and who has been doing the work in the most competent manner, may be granted permission to be registered under section 7 (3) of the Bombay Medical Act, and resolved to inform Government that the recommendation of the Surgeon General should not be accepted.

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8. The Council considered a proposal for inter-provincial registration on a reciprocal basis and resolved to inform Government that in order to give effect to the proposal the following should be added as clause (*a*) to section 7 (1) of the Bombay Medical Act :—

> "Every person who is already registered with any of the Provincial Medical Councils in India with which

reciprocity of registration has been mutually arranged, shall on the production of his registration certificate, be entitled to have registered under this Act, which are entered in the said certificate."

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9. The Council resolved on consideration of certain further circumstances brought to their notice to drop the proposal to prosecute Mr. R. K. Rawal under the Indian Medical Degrees Act.

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10. The Council considered an application from Mr. M. K. Parikh, a medical practitioner of Godhra, for the removal of his name from the Bombay Medical Register and resolved not to accede to his application.

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11. The following 6 members were elected by ballot as members of the Execute Committee.

Khan Bahadur Dr. N. H. Choksy, C.I.E.; Dr. Dinshah M. Gagrat; Dr. Y. G. Nadgir; Sir Temulji B. Nairman, Kt.; Lt. Col. R. Row, O.B.E. and Lt. Col. W. M. Houston, I.M.S.

NOTICES.

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