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Editor : DR. M. C. CONDILLAC, M.R.C.S.

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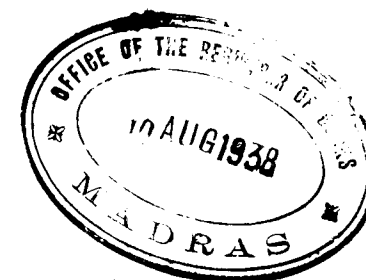
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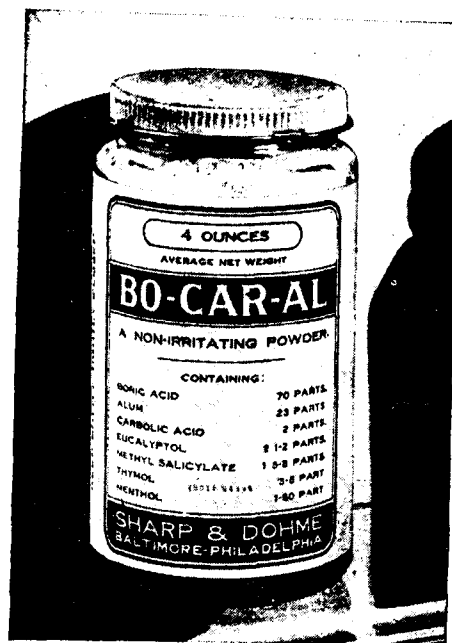
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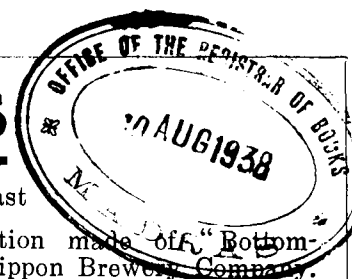
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conditions is he in a position to gauge the exact nature and degree of the lesion as in head injury, from the physical signs.

Recently I saw a case which was diagnosed as a 'simple' injury by a medical officer. The patient walked into the dispensary, was quite conscious, and more or less normal except for a lacerated wound in the head and a black eye, so the doctor did not examine the case as thoroughly as he would have done otherwise. On further examination it was revealed that he had hæmoptysis, unequal and sluggish pupils, pulse 64, and the cerebro-spinal fluid (on lumbar puncture) was mixed with blood. He obviously relied more on the symptoms, but after full examination there was no doubt that it was a case of 'grievous hurt' if not a dangerous injury.

In some cases the patient will try to belittle the seriousness of the injury for some reason or other. Generally, there is some motive behind it and unless the doctor makes a careful examination, he may be involved in serious trouble. In one case where a person was very badly beaten, the doctor did not report the matter to the police, as he did not consider the case to be serious enough. The injured person also requested him not to report it to the police. The motive of the patient is difficult to understand in such a case. It may be that he wanted to take the revenge himself and a police record would have gone against him in that case. However, the person died on the second night after the injury, and in his judgement the judge passed an injunction on the conduct of the doctor. The doctor's statement was that he did not consider the case to be serious enough. In my opinion he can only be blamed if he did not make a thorough examination, which he stated he did. So in these cases it will be wise for rural practitioners to record the negative evidence as well in the case sheet, at least for his own protection.

From these two examples it is evident that no reliance can be placed on the patient's statement or appearance. The patient in an average medico-legal case will try to hoodwink the doctor by feigning to be more seriously ill than he really is and the attending physician must rely on the actual physical signs present and must record them, not only for evidence, but also

for assessing the progress and guidance for further treatment. For this reason a record of negative findings is no less important than positive ones.

Classification of head injuries.—Injury of the head may cause the following lesions, either singly or in combination:—

1. Simple scalp wound.
2. Simple fracture of vault.
3. Fracture of the base.
4. Concussion.
5. Cerebral compression.
6. Cerebral laceration.

Investigation of a case of head injury

As soon as a case of head injury arrives, an attempt must be made to find out the exact nature and degree of damage sustained by the skull and its contents.

I. Preliminary examination

1. Obtain a careful history.

The exact nature of the fall or violence must be enquired into and the sequence of events that followed immediately subsequent to the injury should be noted. The period of unconsciousness following the injury is important; whether there was temporary disturbance of slight concussion, a brief loss of consciousness followed by rapid return to consciousness; or deep unconsciousness of a severe concussion; or yet again the consciousness which is retained for some time after the accident, but presently followed by a state of stupor. The third denotes cerebral compression.

2. Is shock present, and if so to what degree?

Shock is an inevitable accompaniment of concussion and actually there is no clear line of distinction between the two. For the moment, the occurrence of shock is not without advantage, for on account of the associated lowering of intra-cranial pressure, through reduction of general blood pressure, it affords temporarily a safeguard against bleeding immediately after the accident. So although the shock should be treated, none of the intensive stimulating methods should be employed.

3. Examination of the local part.

The wound in the scalp and the amount of damage to the skull are noted. If there is any depression, its nature and degree are also noted.

4. The whole body should then be carefully examined to find out if any other injury is present.

II. Detailed examination to assess the degree of cerebral damage

This must be recorded.

1. Is the patient unconscious?

When the patient is unconscious, begin the investigation by estimating the depth of unconsciousness. This is an index of the degree of intra-cranial damage and provides a standard by which we can contrast further improvement or deterioration. Four tests are applied in the following order:—

(i) A simple question such as 'What is your name,' spoken in the ordinary tone of voice.

(ii) An imperative question spoken loudly close to the ear as 'Raise your hand' or 'left foot.'

(iii) Induction of a painful stimulus by pressing the finger nail against the supra-orbital notch.

(iv) Testing the corneal reflex.

Response to the first indicates comparatively slight concussion, while failure to react to the last shows the deepest degree of unconsciousness compatible with life.

2. Pulse.

Quick pulse of small volume is the usual accompaniment of concussion, but in the absence of complications, it soon returns to normal.

A slowing pulse rate accompanied by increased volume is a hint that compression is developing.

A quick pulse rate maintained at high speed and yet slowing increased volume is an indication that some degree of cerebral irritation without localized compression exists.

3. Respiration.

Changes in respiration are less constant, but they too are important.

In concussion, respiration is slow and shallow. Irregularity of respiration and particularly Cheyne-Stokes' breathing indicates increased intra-cranial pressure.

A much quickened respiration points to serious cerebral laceration, generally of a diffuse character.

In advanced cerebral compression there is irregular stertorous breathing which indicates increasing bulbar compression and failure of the vital nuclei in the medulla.

4. Pupil.

As a general rule, pupils are moderately contracted and react sluggishly to light.

Inequality or asymmetry of the pupils indicates cerebral lesion and any further change in them will indicate that the lesion is still progressing. The inequality in the earlier stages is due to contraction of the pupil on the same side as the lesion (due to irritation), while on further compression, it is dilated on the same side as the lesion and contracted on the opposite side, and on still further compression both are dilated.

If one pupil is widely dilated and fails to react to light, the other presenting a normal appearance and retaining light reaction and this is noted comparatively suddenly in the progress of the case, one or other of two explanations is likely, a pontine hæmorrhage or œdema, or cerebral compression developing on the same side as the dilated pupil. The latter is of significance in the recognition of compression syndrome.

5. Temperature.

Assuming that extra-cerebral causes have been excluded, a rising temperature means that there is some degree of cerebral damage.

Uncomplicated concussion does not exhibit pyrexia.

Few individual features are more suggestive than pyrexia, and in general it may be stated that the earlier the manifesta-

tion and the more intense the pyrexia, the more serious the cerebral damage.

6. Bleeding from the ear, nose, or throat.

If blood is escaping from the ear, its character should be noted; if the consistency is more watery than normal or coagulation is delayed, the probability is that cerebro-spinal fluid is mixed with it.

7. Blood pressure.

This must be recorded at two-hourly intervals of 24 hours.

Immediately after the injury, the blood pressure falls and as recovery advances, it rises. If it does so gradually, and after rising a few points above normal readjusts itself to average figure, prognosis is good.

If after the preliminary fall, it rises to a high level and is maintained, it indicates increased intra-cranial pressure.

On the other hand, in very severe injuries, as associated with extensive fractures of the skull, the blood pressure may remain low for the entire period of the illness.

8. Paralysis, twitching and convulsion.

Any evidence of twitching is noted, the limbs are moved in various positions in order to ascertain if there is any flaccidity or spasticity.

9. Escape of cerebro-spinal fluid from the wound or ear.

10. Reflexes.

The tendon, plantar and abdominal reflexes are examined, but their evidence is not particularly trustworthy in head injury.

III. Clinical features of cerebral damage

A. Concussion.

B. Compression.

C. Laceration.

A. CONCUSSION

The clinical appearance resembles that of surgical shock with the addition of disturbance of consciousness in a varying degree. The patient may be unconscious to the first or second

degree, pulse quick and of reduced volume, respiration quick and shallow, blood pressure reduced, temperature subnormal, muscles flaccid, appreciation of pain diminished, skin pale and surface temperature lowered.

B. COMPRESSION

1. Due to cerebral œdema. It is due to the local œdema which follows on every injury of the brain. After about 24 to 48 hours, the patient passes into an exaggerated state of reaction, he lies curled up in bed, face buried under the bed clothes and resists all forms of interference, specially exposure to light, and complains of severe headache. Temperature is raised, face flushed, pulse full and slow.

2. Due to intra-cranial hæmorrhage.

Remembering that concussion almost always follows immediately after a severe head injury, it is obvious that a latent period must follow before the symptoms of intra-cranial hæmorrhage can develop. In extra-dural hæmorrhage the symptoms may appear in a few hours, but in sub-dural hæmorrhage it may take several days.

In extra-dural hæmorrhage a period of remission generally intervenes between the concussion which immediately follows the injury and the compression to which the hæmorrhage gives rise. In this period the patient may recover consciousness and there may be a lucid interval of perhaps a few hours. During this period the blood pressure is steadily rising, the patient begins to complain of increasing head-ache, then he becomes irritable and later drowsy and finally comatose with stertorous breathing, flushed face, and slow, full pulse. There may be hemiplegia on the opposite side of the body with rigidity and possibly Jacksonian convulsions in the early stages. Later on, rigidity may appear on the same side as the lesion from generalized compression and, unless its true nature is recognized, it may lead to erroneous diagnosis of the side of hæmorrhage. Pupillary changes have already been described. Still later, the medullary centres become paralysed, the pulse becomes rapid, weak and irregular, and the respirations uncertain and gasping; these indicate that the terminal stage is fast approaching.

In the more serious cases the stupor of concussion passes almost imperceptibly into unconsciousness of compression, and unless a very careful watch is kept, the opportunity of affording relief may pass before the condition is diagnosed.

The other and the most important sign of increased intra-cranial pressure is the increased tension of the cerebro-spinal fluid. So, briefly, the evidence of compression is

- (i) The effects of general increased intra-cranial pressure as evidenced by pulse, etc.
- (ii) Effects of local cortical pressure as shown by convulsions or paralysis.
- (iii) The rate at which the cerebro-spinal fluid comes out on lumbar puncture.

Possible later effects of cerebral compression.

- 1. A delayed compression oedema, generally associated with cerebral laceration.
- 2. Meningo-encephalitis—usually due to infection.

IV. *Dangers of fracture of the base of skull*

The gravity of fractures of the base in contrast to those of the vault is mainly due to the relation of the dura to the bone; in the former the dura is invariably torn; whereas in the case of the vault the dura separates with the greatest ease and may escape being torn.

Anterior fossa. There is no immediate danger, but the injury is liable to delayed sepsis owing to its communication with the nasal cavity.

Middle fossa. The dangers are both immediate and remote, because an extensive hæmorrhage very often accompanies the fracture and because the line of it may involve the sella turcica and endanger the pituitary or its stalk; remote, because the communication with the ear may induce sepsis.

Posterior fossa. Fracture of this fossa causes the most immediate concern because of its proximity of the vital area of the medulla, but on the other hand they are in a large measure free from the risks of secondary infection.

Treatment

I. *General*

Undress the patient with the minimum disturbance.

Put him to bed with the head on one side without a pillow, Ice bag or cold compress should be applied.

Concussion is tantamount to shock and the treatment should be carried out accordingly. Hot-water bottles, fluids preferably by mouth, otherwise subcutaneously or rectally. Intravenous methods are inadvisable because a sudden rise of blood pressure may initiate bleeding or may produce cerebral oedema.

Restlessness. Put the patient in a dark quiet room. Restlessness may indicate the onset of compression. Morphine should not be given. Bromides and chloral gr. 15 to 20 each should be given. If the patient is unable to swallow, paraldehyde, one dram per stone of body-weight, made up with mucilage of acacia in a proportion of two parts to one of mucilage, may be given per rectum. If this is not effective, hyoscine should be given.

II. *Local*

Fractures involving the vault.—It is better to shave the head or cut the hair as short as possible. This will enable better examination and the ice cap or cold compress will be more effective. Moreover the part will be kept ready for immediate operation if necessary.

Simple fissured fracture. This should be left alone.

Compound fissured fracture. The wound should be cleaned, and necessary treatment for the general condition adopted. Watch should be kept for symptoms of compression.

Depressed fracture. If it is pressing on the brain, it must be operated on and the pressure relieved. Otherwise it should be left alone.

Fractures involving the base.—The same line of treatment as already indicated should be undertaken. All obvious sources of infection must be avoided. Put a few drops of 5 per cent argyrol into the auditory meatus and if possible spray the nose with the same solution.

Restlessness is usual and should be treated at an early stage with judicious sedatives.

The pulse, respiration, temperature and blood pressure should be recorded every two hours and the bladder should be watched.

Treatment of Compression.—Compression due to extradural hæmorrhage may appear at any stage but if it be due to cerebral œdema it generally manifests itself after 24 hours by deepening unconsciousness and alteration of the character and the frequency of pulse, blood pressure, respiration and temperature as described above.

When symptoms of compression appear the following treatment is advised:—

Lumbar puncture. If there is moderate rise of pressure, the fluid should be drained until normal pressure is attained.

If there is excessive rise of pressure, hypertonic therapy is indicated. If the patient is able to swallow, 2 drachms of magnesium sulphate in 2 ounces of water are given and thereafter 1 drachm of the salt in 2 ounces of water is repeated every two hours until a watery motion is evacuated. If swallowing is impossible, 3 ounces of magnesium sulphate in 6 ounces of water are given as an enema. The injections may be repeated if necessary. Hypertonic solution such as 50 per cent glucose or 30 per cent saline, injected intravenously, is powerful in lowering the intra-cranial pressure. Moreover, a hypertonic solution of glucose and saline will combat the shock very well. If the symptoms reappear, treatment should be repeated. This is all that should be done in a small dispensary. In *tehsil* headquarters dispensaries further treatment as detailed below may be carried out.

If compression persists or increases in spite of the above treatment, it is not simple general cerebral œdema. It is more likely to be hæmorrhage which is slowly but insidiously progressing and one is forced to decide whether to operate or not. If operation is decided, it must be done as soon as possible and it cannot be expected that a local decompression will afford relief to a brain that is already water-logged.

Technique of operation

Local anæsthetic, 1 per cent novocaine with adrenaline, is used. The infiltration is carried out along a line which extends vertically upwards from the zygoma, one inch in front of the external auditory meatus for a distance of 3 to 4 inches.

The superficial tissues are divided, temporal fascia is incised, fibres of the temporal muscle are split and retracted and the bone of the temporal fossa is exposed. The bone is perforated with a trephine or bur at a point about two finger-breadths above the zygoma and with a pair of nibbling forceps the vault is cut away until an aperture $2\frac{1}{2}$ inches in diameter is made. If there is a clot, it should be removed and most likely the middle meningeal artery will be found ruptured and if so it should be ligatured.

Failing this, the dura should be opened in a cruciform manner, avoiding the dural vessels. If they have to be divided, they should be picked up with a fine round needle and stitched and each ligatured individually.

The dural flaps are widely separated, the split in the temporal muscle is restored a few interrupted catgut sutures, temporal fascia is united with by a continuous catgut suture and the edges are united with silkworm gut or horse hair.

If there are still signs of further compression, alimentary dehydration as already indicated should be continued. Failing that similar decompression operation may be done on the opposite side.

After-treatment

The patient should be kept in bed for 3 to 4 weeks, on a low diet with abundant fluids. Half an ounce of magnesium sulphate solution should be given every morning. Ten grains of potassium bromide should be given every day. If the patient is suffering from insomnia, 2 gr. of luminal or $7\frac{1}{2}$ gr. of medinal, or 5 gr. of soneryl may be given.

Early Diagnosis and Treatment of Heart Failure

BY

WILLIAM EVANS, M.D., F.R.C.P. (Lond.)

FEW illnesses can more readily compel a medical attendant to apply immediate therapeutic measures than heart failure.

So alarming may be the symptoms and so great the anxiety surrounding the patient that a demand for hurried action is everywhere evident. Never is the necessity greater, however, for the doctor to make haste slowly, to act promptly but not precipitately, and yet again to treat cautiously but expeditiously. The need to do something is always present, but it should be no greater than the need to know everything before doing anything; so that an immediate perception of the expected course of the illness, and the processes involved, is an essential preliminary to the competent treatment of heart failure.

The Mechanism of Heart Failure

Although the true mechanism of heart failure continues to remain an unsolved problem, our knowledge of it on the one hand has helped us to conceive certain principles in treatment, and on the other hand our lack of knowledge is unlikely to have withheld anything revolutionary from present-day methods of dealing with the condition. The changes during failure are familiar to us; it is the evolution of these changes which eludes precise exposition.

The back-pressure failure theory envisages stasis of blood in the vascular areas which drain in the direction of the affected heart chamber, so that obstruction in the mitral valve is expected to produce the following sequence of changes: distension of the left auricle, engorgement of the lungs, distension of the right ventricle and of the right auricle, engorgement of the veins of the neck and cyanosis, distension of the liver, and oedema of the lower extremities. Recently it has been shown (Thompson and White, 1936) that left ventricular strain from hypertension, aortic valve disease, or infarction of

the left ventricle is the commonest cause of hypertrophy of the right ventricle, which occurs regardless of the presence of clinical evidence of left ventricular failure, although this increases the degree of hypertrophy.

The forward-pressure failure theory, supported by Sir James Mackenzie, postulates systematic failure following inadequate output of the left ventricle, while pulmonary symptoms in the same way are the outcome of failure of the right ventricle. This deficiency in the outflow of blood results in a depletion of the vascular areas and in ischaemia of the tissues situated in front of the affected heart chamber.

It is likely that both back-pressure phenomena and a diminished cardiac output are features that exist together in many if not in most instances of congestive heart failure, but the knowledge of the part which each plays in the development of failure is unlikely to contribute in a greater measure to the treatment of the condition than to indicate the need of relieving congestion and of increasing the efficiency of cardiac contraction.

Size and Position of the Heart in Failure

Cardiac enlargement is usual in failure, but it is rarely the direct result of failure, and its distribution and extent depend chiefly on the lesion which is providing the cause of the failure. Either with or without failure the progression of cardiac enlargement is very gradual, so that sudden dilatation of the heart seldom occurs. Whenever such an event has been implied from a clinical examination, and displacement of the apex beat to the left has been noticed, three influences contriving to produce this change must be kept in mind before attributing it to cardiac enlargement. They are tachycardia, distension of the liver and ascites producing elevation of the diaphragm, and a right-sided hydrothorax. Even on radiology caution must be exercised when assessing the development of cardiac enlargement, so that the "squatting" posture of the heart, assumed as a result of a raised diaphragm from any cause, must not be interpreted as enlargement. Again, the possibility of pericardial effusion enlarging the cardiac silhouette as seen on radiology should receive attention in any case where an exacerbation of

failure signs has occurred with the formation of effusion within serous cavities.

It is held by some that hydrothorax (usually right-sided) from heart failure does not cause cardiac displacement, but this is fallacious. Certainly it does not displace the heart to the same extent as does an inflammatory pleural effusion, because the quantity of fluid is not so great, and, further, whenever the effusion in heart failure has become considerable pulmonary congestion with pleural effusion occurs on the opposite side, and this opposes the initial displacement.

Evidence of Early Failure

The need for recognizing early heart failure is only equalled by the need for early treatment, and early treatment depends on early diagnosis. In order to gain this end the earliest evidence of failure must be sought in those patients known to suffer from heart disease which is expected to give rise to heart failure sooner or later. In them symptoms develop gradually and almost imperceptibly, so that our awareness of this happening will usually lead us to seek for premonitory signs and not to wait for the fully developed clinical picture of heart failure characterized by cyanosis, considerable breathlessness, conspicuous engorgement of the veins of the neck, pulmonary congestion with right-sided hydrothorax, enlargement of the liver with ascites, oedema of the lower extremities, and scanty urine with albuminuria. The recognition of early congestive heart failure depends on attention to the following essentials: undue breathlessness to customary exertion, venous engorgement, pulmonary congestion, hepatic distension, and a familiarity with the causes of heart failure.

Undue Breathlessness

Breathlessness on exertion occurs in health. With advancing age or with the onset of obesity this natural reaction to increased physical performance becomes accentuated. The breathlessness which heralds the commencement of heart failure is characterized by its occurrence during exercise which hitherto produced no breathlessness, or which is in excess of that measure which was customarily induced by a standard form of exercise.

Thus the hall-mark of pathological breathlessness is present when exercise which usually produced no breathlessness induces it, when exercise which previously initiated breathlessness causes undue breathlessness, and when the breathlessness has shown progression within a relatively short period of time.

Venous Engorgement

An increase in the venous blood pressure may be regarded technically as the first evidence of heart failure, but unfortunately its estimation cannot be carried out with the same ease as the registration of the arterial blood pressure. The direct method of recording the venous pressure by connecting a venepuncture needle to a manometer filled with citrate solution cannot for obvious reasons become common practice in clinical medicine, so that some other means denoting a change in the venous pressure is desirable. Sir Thomas Lewis (1930) directed attention to a method which permits an approximate clinical estimate of the venous pressure. He pointed out that in health all veins which lie at a higher level than the manubrium sterni remain collapsed and those below are distended. Thus in a healthy patient in the reclining posture with the head resting on a pillow the external jugular veins are distended for about one-third their course from the clavicle to the angle of the lower jaw. In a patient presenting a slightly raised venous pressure the column of venous distension reaches the centre of the sternomastoid muscle or higher. The veins in the neck are not evident in a healthy subject in the erect position, but in established heart failure they are visible as cords reaching from the clavicle to within a variable distance of the mandible. This clinical indicator of the venous pressure should gain significance not only on account of its relative accuracy, but also on account of the simplicity of the observation.

Pulmonary Congestion

When persistent crepitations are elicited over the bases of the lungs, mostly on the right side, from heart failure, the presence of a considerable degree of pulmonary congestion can be presumed. Earlier evidence of pulmonary engorgement is marked by vascular congestion in the hila of the lungs. Careful

auscultation along each side of the spine over the middle zones will often disclose, especially after coughing, isolated coarse crepitations at the end of a deep inspiration, but commonly the presence of this degree of congestion can only be made out on radiology; certainly the extent of hilar congestion can only be told by this method. The picture obtained is a characteristic one presenting wing-like shadows spreading from each hilum, and caused by an increase in the density of the pulmonary vessels, which are depicted here and there in cross-section and appear like ink blots.

The help provided by radiology in the recognition of early heart failure has not yet gained the reputation it deserves. Nor should the method be applied solely in diagnosis, for it also provides an index in the evaluation of progress during treatment, so that the extent of hilar congestion as determined at intervals by radiology determines the advance or retreat of the process of failure.

Hepatic Distension

An increase in the volume of the liver is a sensitive indicator of an increase in the venous blood pressure. Although this natural manometer does not supply accurate quantitative values, when enlargement of the liver has been made out it serves as early evidence of heart failure, so that whenever failure is suspected or expected distension of the liver should be sought. Palpation immediately below the right costal margin may elicit tenderness in a patient presenting hepatic distension, but a quest for the descended liver edge as the hand travels upwards from a low position is a more reliable method of examination.

Specific Causes of Heart Failure

Familiarity with the conditions capable of precipitating heart failure ensures timely and efficient attention to a patient in whom failure threatens. To anticipate heart failure is often to postpone it; to remain unaware of its causes and its early manifestations is to allow its grosser signs to develop. The causes of heart failure may be conveniently considered in two groups, as suggested by Parkinson and Clark-Kennedy (1926);

but here a reference to these will only include in the first place any special features which facilitate early diagnosis, and secondly those details concerning the treatment of each group which cannot be regarded as common to all cases of failure.

Treatment of Established Heart Failure

Certain general principles apply to the treatment of most of the common examples of heart failure, so that in the first place it is opportune to consider these and later to detail any particular remedy or procedure which may apply when certain specific causes are present. An outline of those therapeutic measures to be adopted in the general case of heart failure is facilitated by referring to three distinct clinical types—established heart failure, early heart failure, and abrupt heart failure.

The designation "established heart failure" is applicable when a patient presents characteristic physical signs of failure which have developed gradually and have progressed during the absence of treatment or with inadequate treatment. No rigid rules can be formulated to govern the management of all cases of established heart failure, but although every patient must be studied individually the treatment should generally provide for adequate rest, digitalization, and diuresis.

REST

In ambulatory cases a limitation of activity is of first importance in treatment, and in order to gain the fullest benefit from this curtailment of exercise the patient's usual daily habits should be ascertained and a programme modifying the day's activities should be complete in detail. A later rising time in the morning and an earlier retiring time at night, periods of rest in the reclining posture after meals, walking slowly on level ground and avoiding hurrying and climbing, are among the instructions imparted to the patient in whom established heart failure is controlled by digitalis medication. At night the provision of extra pillows promotes rest and sleep.

In non-ambulatory cases absolute rest is sought, and a patient is discouraged from indulging in any exertion, not even turning in bed. Again, the head and shoulders should be supported in the upright posture, and although devices to meet

this necessity have been improvised in the form of chairs, extension of these amenities to the construction of special beds has received but scant attention. Such a bed was designed by Sir Thomas Lewis, but the adoption of this helpful instrument in the treatment of heart failure in the wards of a hospital has proceeded very slowly.

Inseparable from any method adopted to ensure rest in a case of heart failure is the alleviation of the patient's natural apprehension and the provision of sleep. Alcohol, in that it is conducive to sleep and causes a feeling of well-being, may be dispensed with benefit, but in no sense must it be regarded as a "heart stimulant". A mixture containing 20 grains of potassium bromide and chloral hydrate often suffices as a hypnotic, or resort may be made to the barbiturates (sodium luminal 1 to 2 grains) or morphine (1/2 grain dissolved in the mouth, or 1/4 grain hypodermically). Certainly these drugs should be used with determination, as they are important agents in the treatment of heart failure.

DIGITALIS

Good wine needs no bush, and certainly the utility of digitalis in the treatment of heart failure does not need to be advertised; it has proved its worth. What does require emphasis is that it should be used in adequate doses, so that our duty in treating heart failure is not so much the giving digitalis as establishing a state of digitalization. Digitalization is only present when the patient's symptoms are lessened, when diuresis has resulted, when the pulse rate has been slowed to 70 per minute or under, when the ventricular rate has been similarly diminished and the pulse deficit annulled, and when depression of the R-T segment is a feature of the electrocardiogram.

A choice of the preparation of digitalis must necessarily be made before determining the dosage. It is most desirable to adhere to the administration of a single preparation, and acquire experience of its effects in different patients. Nothing is gained, in fact a good deal is lost, by substituting various preparations of digitalis, either in the same patient or in different patients. It is far better to acquire exact knowledge of the value of a single preparation than to nourish a doubt concerning the

comparative value of a number of preparations. The following may be regarded as representative of the digitalis group and dependable when used in the indicated daily doses: powdered leaf (4 grains), tincture (45 minims), Nativelle's granules (1/125 grain), and digoxin tablets (0.45 mg.). Although these doses are comparable and basic they are not to be regarded as absolute standards in all patients with established heart failure. The dosage should be adjusted according to the severity of the failure, weight of the patient, and particularly the length of time required to establish a state of digitalization. In most instances the suggested standard dose is the approximate one, but often it will need increasing and sometimes decreasing.

DIURESIS

To produce efficient diuresis should be a constant aim in every case of established heart failure, and there is more than one method available to attain this object.

That rest in bed combined with a diminished fluid intake can produce prominent diuresis has not gained sufficient recognition. Fig. A illustrates this effect in a female, aged 36 years,

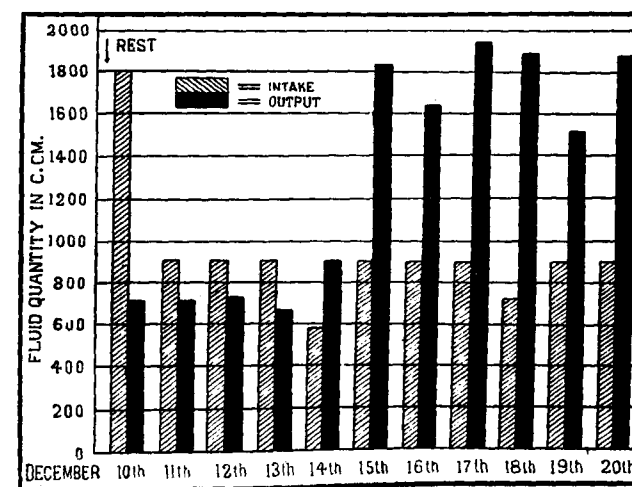


FIG. A.—Chart showing diuresis following rest in bed and a diminished fluid intake in a patient with mitral stenosis and heart failure with normal rhythm.

with mitral stenosis, in whom the features of early heart failure presented. Cognizance of this happening is necessary in the evaluation of any remedy credited with diuretic properties: in the patient instanced here unmerited reputé might have been accorded any drug professing a diuretic action if it had been introduced when the patient was voiding scanty urine on admission to

hospital. The lesson in this connexion is twofold: first, it impresses the need of prescribing rest combined with a diminished fluid intake ($1\frac{1}{2}$ pints daily) in patients presenting heart failure; and, secondly, it emphasizes the importance of adequately controlling all clinical tests conducted to assess the diuretic properties of any drug.

Foremost among the drugs which can effect diuresis in heart failure, when administered by mouth, is digitalis, and in most instances it should be preferred to other remedies in this group, such as ammonium chloride or the purine diuretics—for example, theobromine, diuretin, theophylline, or euphylline.

Intravenous and intramuscular salyrgan or novurit in 2 c.cm. doses are pre-eminently the most powerful diuretics we possess, and they form important implements now available to combat the effects of heart failure and to help restore cardiac efficiency. Parkinson and Thomson (1936) have shown that novurit gives satisfactory results when used as a suppository, although of course its diuresis is not so profuse as when it is introduced intravenously or intramuscularly; but the advantages afforded by the simplicity of this method of administration are obvious. The prescribing of ammonium chloride along with salyrgan increases diuresis, but this is not always considerable. The most recent advancement in the treatment of heart failure has been the addition of mercurial diuretics to the other effective therapeutic agents—rest, digitalis, and hypnotics.

The effect of total ablation of the thyroid gland in heart disease presenting paroxysmal nocturnal dyspnoea and heart failure has to be discussed. Impressions concerning the benefit gained from this procedure are best got from a study of the results obtained in seventy-eight cases [thirty-six by Blumgart and others (1935), and forty-two by Levine and Eppinger (1935)]. The therapeutic benefit enjoyed by some of these patients corresponded with the lowering of the basal metabolism, and when this became reduced to minus 30 per cent myxoedema frequently intervened, and this in turn responded to small doses of thyroid without materially detracting from the improvement resulting from the lowering of the basal metabolism. If heart failure was present with persistent oedema the operative risk

increased considerably, so that it is evident that total thyroidectomy is not often desirable in instances showing failure, and certainly this procedure should be undertaken only when the recognized methods of treatment have failed and when the operation can hold out a reasonable prospect of considerable improvement.

Treatment of Early Heart Failure

A patient with heart disease is commonly examined during the pre-failure period, when he will derive greater benefit from observation and instruction than from the initiation of any form of zealous treatment. Medicinal treatment, apart from a placebo, is unlikely to produce material benefit during this period. Certainly any attempt at digitalization at this stage is ill advised and without promise of improvement, so that it should be discouraged even though it might be given without harm.

In particular the patient is to be instructed on the subject of activities and diet. While it is permissible or even desirable to indulge in limited or graduated exercise, exertion is prohibited. In connexion with diet it is desirable that it should not be heavily salted, and stress is directed to two main essentials—namely, the avoiding of large or indigestible meals and, if obesity is a feature, the resorting to a fat-reducing diet.

To gain the co-operation of the patient during this pre-failure phase should be a first consideration. No amount of enthusiasm or even heroism in treatment will produce improvement if the patient is not the doctor's ally. Nor must the psychological side be neglected, so that encouragement and just optimism should be dispensed along with firm dissuasion where needed. The over-cautious should receive encouragement, and the reckless should be restrained and be made to realize the consequences of any disregard to instructions. Improved health should be promised on conditions and as a reward of attention to advice.

If the signs of early heart failure are beginning to collect and there is increased breathlessness, slight venous engorgement in the neck, and hilar congestion detected by radiology, apart

from the precautions already enumerated concerning exercise and diet, periods of controlled rest should be outlined; these will be in the mornings and evenings, after the midday meal, and at the week-ends. Digitalis should be added if auricular fibrillation has set in. The administration of mercurial diuretics combined with a much-reduced fluid intake should be conjoined with the week-end rest.

The treatment adopted for this group of patients almost merits the designation "prophylactic treatment"; at any rate, it aims at postponing the signs of established heart failure. The measure of success gained from the care devoted to these cases justifies the zeal involved in collecting cases within the scope of this group.

Treatment of Abrupt Heart Failure

In some patients in this class the precipitation of heart failure may be an unforeseen contingency, and this particularly applies to cardiac infarction. It may also occur unexpectedly in hypertension, although paroxysmal nocturnal dyspnoea will often serve as a premonitory sign. Sudden exacerbation of failure symptoms may also occur in cases with established heart failure hitherto controlled by treatment, or even in some instances of early failure; in both of these groups either a period of increased activity and exertion or some intercurrent illness has caused a damaged heart to fail suddenly.

It is in this class of cases that the urge to do something is so strong and good judgement so often yields to mismanagement created by over-enthusiasm. The term "cardiac stimulant" designates the drug whose purpose it is to help the heart in this sudden crisis. Many drugs have gained a reputation in this field, but usually this has not been merited and has been gained through uncontrolled clinical impressions. Strychnine, camphor, and pituitary are in this category, and have far survived their usefulness in this connexion. A warning is overdue concerning the danger of the wholesale and indiscriminate use of proprietary preparations imputed to be of this class of "cardiac stimulants" in virtue of their action, variably claimed to be on the medulla, on the peripheral circulation,

on the coronary circulation, or as "cardiac hormones." A plea for resort to controlled clinical assay is made in order to regain a proper perspective of the value of these potions.

Strophanthin has seldom shown superiority over digitalis in cases of precipitate heart failure. Digitalis, particularly digoxin, has great value in abrupt heart failure associated with auricular fibrillation. Oral administration of a 1 mg. of digoxin can lower the ventricular rate in auricular fibrillation within a few hours. Its success when the heart rhythm is regular is not so assured. Little or no advantage is gained by intravenous administration of digitalis.

Whenever cyanosis is a prominent feature and there is conspicuous venous engorgement in the neck, venesection is good practice. If restlessness and sleeplessness present, hypnotics must be administered in adequate doses after providing support for the head and shoulders and making the patient comfortable.

If oedema and ascites are features salyrgan is indicated. In the case of a female patient with much oedema it is necessary to anticipate the considerable diuresis by tying in a urinary catheter in position so that the patient's energy is in this way conserved. Salyrgan medication has dispensed with the necessity of purging an oedematous patient for the purpose of ridding him of superfluous tissue fluids.

Heart Failure in Specific Conditions

Certain special features in the mechanism, early diagnosis, and treatment of specific conditions which may initiate failure will now be described.

HYPERPIESIA

Whenever congestive heart failure appears in a patient and the heart rhythm is regular, hyperpiesia may be regarded as the likely cause. To bear this in mind is to recognize the underlying condition when the failure has produced a fall in the blood-pressure value. Sometimes when the onset of failure has caused the systolic figure to fall to a normal level the diastolic value has continued to remain high, permitting the

diagnosis of hypertension. Often, however, a fall in the diastolic blood pressure has accompanied a similar change in the systolic blood-pressure value, and the diagnosis then rests on a record of a raised blood pressure during the pre-failure period and the finding of cardiac hypertrophy chiefly involving the left ventricle. An ominous sign has appeared when heart failure has been added in a case of hyperpiesia. Improvement may occur for a time, but longevity can hardly be mentioned in connexion with prognosis.

Digitalis is without any great reputation in the treatment of hypertensive heart failure, but it always merits a trial in individual cases, as it will usually produce some degree of improvement. Certainly it is the treatment of choice in the less common instances where auricular fibrillation is present.

The mercurial diuretics salyrgan and novurit hold first place in the treatment of hypertensive heart failure, and the administration of these should be augmented by providing organized periods of rest and a diminished intake of fluid.

Further, when failure has set in as a complication of hypertension, paroxysmal nocturnal dyspnoea (cardiac asthma) is a frequent accompaniment, and demands specific treatment in addition to the general regime adopted for the failure. The patient must be propped up in bed, and potent hypnotics such as sodium luminal, $1\frac{1}{2}$ grains, or morphine $\frac{1}{4}$ grain, administered each night until the general condition is improved from the use of diuretics. Digitalis medication should also be given a trial in these cases.

AORTIC INCOMPETENCE AND AORTIC STENOSIS

Aortic incompetence from syphilitic aortitis and endocarditis invariably precipitates heart failure with a regular cardiac rhythm, and so does rheumatic aortic incompetence if the mitral valve has remained healthy or is only slightly damaged. Similarly the cardiac action is regular in aortic stenosis with heart failure if the aortic valve is the only valve affected. As in the case of hyperpiesia the onset of failure is a serious complication, and signals a grave outlook.

The failure associated with aortic valve disease also simulates hypertensive failure in that digitalis assumes second place to salyrgan or novurit, except in the less frequent instances where failure has recurred with auricular fibrillation.

CARDIAC INFARCTION

Although arrhythmia, especially auricular fibrillation, is sometimes present in cardiac infarction, in the majority of cases normal rhythm is preserved. The signs of failure may appear abruptly in this condition, and if the area of infarction is a large one the signs are correspondingly increased; thus pulmonary congestion becomes prominent, and not infrequently crepitations are found scattered over both lungs. The liver, too, may become distended early in the illness, and slight oedema of the ankles may appear.

Treatment of the failure in this instance resolves itself into dealing with cardiac infarction, so that rest ensured by morphine is the procedure to adopt in patients in this group.

CONGENITAL CARDIOVASCULAR DISEASE

Heart failure is not a common feature of congenital cardiovascular disease, and pulmonary infection and subacute bacteriaemic endocarditis are the more likely complications to ensue. Cyanosis in these cases usually only serves to indicate the presence of a venous-arterial shunt, and is seldom an expression of the presence of heart failure. In those instances of congenital cardiovascular defects where cyanosis appears later in life the onset of heart failure may have caused cyanosis by producing a reversal of an existing arterio-venous shunt into a venous-arterial shunt. The symptoms of dyspnoea and cyanosis, therefore, in congenital heart disease are not commonly manifestations of congestive heart failure; they do not call for special care or treatment except in those few instances which show frank signs of failure.

The cardiac rhythm continues to be regular after failure has set in unless congenital heart-block is present as a rare feature of interventricular septum defect. When failure has set in it is usually progressive, although adequate rest and

treatment with mercurial diuretics and digitalis may produce temporary improvement.

EMPHYSEMA AND BRONCHITIS

Too often in the past a patient giving a history of a productive cough occurring mostly during winter months and presenting great breathlessness, cyanosis, and abundant adventitious sounds in the chest, has been regarded as a case of heart failure. More notice should be taken of emphysema as a common cause of extreme breathlessness, and less of its influence on the heart. The severe breathlessness associated with emphysema is the direct result of a diminished pulmonary ventilation, and is only rarely the sequence of heart failure. The effects of emphysema on the heart have recently received attention. Parkinson and Hoyle (1937) determined the size and shape of the heart by radiology in eighty patients suffering from a high grade of emphysema. Enlargement of the pulmonary artery stem and its branches and of the conus of the right ventricle was present in about one-half of the cases. Changes due to hypertension were also commonly met with. About one-third showed no cardiac enlargement. Heart failure was infrequent (thirteen out of eighty), and in seven of the thirteen cases hypertension was a feature. They stated that congestive failure purely from emphysema and chronic bronchitis was rare and had a grave prognosis, so that *recurrent* failure was almost unknown.

Of even greater importance in the management of a case of heart failure is the recognition of the effects of emphysema and chronic bronchitis in a patient presenting heart disease. The occurrence in these cases of attacks of acute bronchitis produces an exacerbation of symptoms and may cause alarm, for the breathlessness may be extreme and accompanied by a distressing cough with the voiding of thin sputum and often haemoptysis. In the presence of mitral stenosis and auricular fibrillation such symptoms have been attributed too readily to heart failure, and the added pulmonary signs have erroneously assigned to pulmonary congestion from failure instead of to diffuse bronchitis and emphysema. The treatment of each condition is so dissimilar as to urge the necessity of recognizing in many cases

of heart disease this prevalent "bronchitic element," and of deciding its influence in producing the symptoms before treatment is commenced. Adrenaline or ephedrine and a mixture containing potassium iodide, stramonium, and ammonium carbonate prove more beneficial than an increase in the dose of digitalis. It is likely, from experience with this type of case, that certain proprietary adrenaline-like preparations have gained an over-measure of trust in the treatment of all cases of heart failure.

NEPHRITIS

Heart failure is a common event in nephritis when hypertension is present. In chronic nephritis cardiac asthma is often an added feature. This frequent occurrence of heart failure in nephritic hypertension is not universally realized, particularly in hypertension of acute nephritis. Ellis (personal communication) found evidence of heart failure in twenty instances among 100 patients with acute nephritis, and in nine of these it was severe. The importance of a more general acceptance of the common incidence of heart failure in nephritic hypertension is found in a wider application of specific remedies in these cases to cardiac rather than to renal failure.

ADHERENT PERICARDIUM

Adhesions may form between the parietal and visceral layers of the pericardium, partially or completely obliterating the sac, and yet produce no cardiac embarrassment nor result in cardiac enlargement unless valvular disease is present. In another group calcification of the pericardium may be added, but without symptoms. Where extrapericardial adhesions have bound the heart to the surrounding structures (mediastino-pericarditis) cardiac enlargement is often present, but even here coincident valvular disease may account for the enlargement. A special feature of a fourth group is the constriction of the superior and inferior venae cavae, and to a less extent the heart, by adhesive pericarditis, giving rise to a characteristic clinical syndrome (Pick's syndrome). The effects of these changes is to produce a condition comparable to that of heart failure but without pulmonary congestion, so that the symptom

of breathlessness is never prominent in the initial stages. The means of recognizing cases in this group has been clearly detailed by Paul White (1935), who refer to the leading clues in the diagnosis in the following order: The insidious onset of dropsy and oedema of the lower extremities in a young person. Preponderant enlargement of the liver with ascites. Undue prominence of the jugular veins with increase of the venous pressure. Absence of any heart disease or nephritis in a case presenting dropsy. Small and regular pulse with a low systolic blood pressure and pulse pressure. Evidence of chronic pleurisy. Calcification of the pericardium seen on radioscopy. Low-voltage curve in the electrocardiogram with flattening or inversion of the T-wave in one or more leads. Previous history of pericarditis and polyserositis.

Pericardial resection by an experienced thoracic surgeon is the only method of treatment which holds promise of recovery from the effects of constrictive pericarditis, and the number of patients who have gained benefit from this surgical procedure is fast increasing.

ARTERIO-VENOUS ANEURYSM

When a small communication has been established between an artery and a vein there is no demonstrable change in either the anatomy or function of the heart, but if the fistulous opening is a large one then symptoms and signs of heart failure develop. It is the arterio-venous aneurysm resulting from trauma, therefore, and situated in the extremities, particularly the thigh and leg, which specially produces changes in the heart. Here breathlessness and palpitation are common symptoms, and considerable enlargement of the heart, especially the left ventricle, is demonstrable on radioscopy. The pulse is regular, a little rapid, and is collapsing in character with an increase of the pulse pressure; obliteration of the arterio-venous shunt by digital compression raises the systolic and particularly the diastolic blood pressure, and the collapsing nature of the pulse disappears; the pulse rate also falls abruptly. With the fistula open the venous blood pressure is increased and pulmonary congestion is visible on radioscopy. Extirpation of the aneurysm causes the cardiac enlargement and the

pulmonary congestion to disappear within a short period of time (see Figs. 7 and 8), thus providing a rare example of reversible cardiac enlargement.

MITRAL STENOSIS

Most instances of mitral stenosis showing evidence of heart failure present arrhythmia, usually in the form of auricular fibrillation, but occasionally as auricular flutter; in some the rhythm remains regular. It is likely that early indications of heart failure are invariably present before the onset of fibrillation, and that the adoption of efficient treatment during this pre-fibrillation period postpones the onset of fibrillation. Once auricular fibrillation has become established in a case of mitral stenosis digitalis is pre-eminently the medicine to prescribe. In the uncommon instances of mitral stenosis presenting prominent and aneurysmal dilatation of the left auricle the absence of gross heart failure symptoms may be a notable feature.

THYROTOXICOSIS

Slight cardiac enlargement occurs commonly in patients with thyrotoxicosis in the absence of auricular fibrillation and heart failure, but failure is seldom if ever present with a normal cardiac rhythm. Again, not all cases of thyrotoxic fibrillation show evidence of failure. When it has set in digitalis will produce some improvement, but subtotal thyroidectomy after adequate treatment with Lugol's iodine is unquestionably the proper course to adopt. An attempt to re-establish normal rhythm by quinidine administration during the pre-operative period is of doubtful value.

COMPLETE HEART-BLOCK

When complete heart-block is complicated by heart failure and when Adams-Stokes attacks are absent digitalis is worthy of trial because it often produces clinical improvement and the failure symptoms disappear without causing undue slowing of the pulse. In cases subject to Adams-Stokes attacks digitalis is contraindicated as it usually increases the incidence of the attacks.

Conclusion

In conclusion it may be said that in the diagnosis and prevention of heart failure it is important to seek early evidence of this in patients presenting heart disease which can ultimately precipitate failure, and that in treatment it is necessary to individualize, to treat with determination but not over-zealously, to prescribe remedies whose worth has been established by repeated and controlled clinical trial, to gain the full co-operation of the patient, and to give constant encouragement.

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Modern Treatment of Malaria

THE modern treatment of Malaria may be summed up as the return to a short course of treatment by quinine and the discontinuance of the use of the various gametocidal and antipyretic, synthetic products.

The fourth general report of the League of Nations Malaria Commission repeats the suggestion of the 1933 report which strongly recommended the short course of Quinine treatment i.e., 15 to 18 grains of Quinine for 5 to 7 days.

Whether Quinine acts directly on the parasite or indirectly increases the parasitocidal powers of the blood has not been established.

Experiments have shown that small doses of quinine during the early days of the attack are sufficient to kill the majority of parasites.

Turning to the gametocidal action of quinine, while quartan and benign tertian forms are easily killed by quinine, the malignant tertian crescents are not. In these cases one must make use of plasmochin. Here again however one can omit plasmochin because the red blood cells in which the crescents live, die in about a month, and the enclosed gametes are destroyed with them.

Gametes do not produce relapses. It is the asexual survivors that are responsible. The gametes however indirectly infect other humans through the agency of the biting mosquito.

Relapses in Malaria are frequent and the tendency to chronicity the rule.

Atebrin and plasmochin because of their toxic action and expense are gradually being relegated to the background.

Relapses with these treatments have been no less than those in treatment with quinine; besides one could never put down, for certain, that cures were definitely due to atebrin or plasmochin as these were invariably used along with quinine in separate form or compounded with plasmochin.

The treatment with quinine is simple. It is economical. Patients take a short course which does not necessitate immediate control of the doctor nor lying up for days in bed. Quinine is cheap. Its absorption can be controlled and checked.

A short course of treatment with quinine and the daily prophylactic five grains has stood the test of time.

The Trichinopoly District Medical Association
TRICHINOPOLY

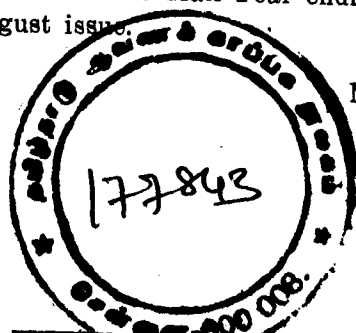
Statement of Accounts for June 1938

RECEIPT	EXPENDITURE
Rs. A. P.	Rs. A. P.
Brought Forward Cash 36 13 9	Clerk's Salary for May 18 0 0
Bank Balance ... 144 4 8	Office Rent for May ... 5 0 0
Subscription from Members ... 42 8 0	Sweeping charges do. 0 8 0
Advertisement charges 9 12 0	Stationery ... 0 4 6
	Printing charges for April & May ... 13 8 6
	Cycle Repairs and New Tyre & Tube ... 4 15 6
	Cartage ... 0 7 0
	Monthly Meeting (Baksheesh to peon) 1 0 0
	Stamps (includes postage accounts for Miscellany) ... 17 10 3
	61 5 9
	Cash Balance on hand 30-6-'38 ... 34 8 6
	Bank Balance on 30-6-'38 ... 137 8 2
233 6 5	233 6 5

Credit Balance on 30th June 1938 is Rs. 172-0-8.

Miscellany accounts for the Half-Year ending June 1938 will be published in the August issue.

30th June 1938.



M. C. CONDILLAC,
Treasurer.

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(REGISTERED)

JULY 1938

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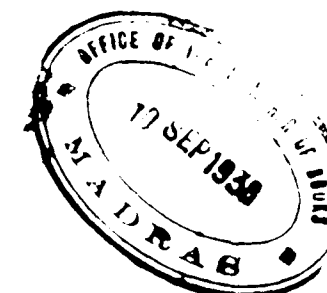
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Editor: DR. M. C. CONDILLAC, M.R.C.S.

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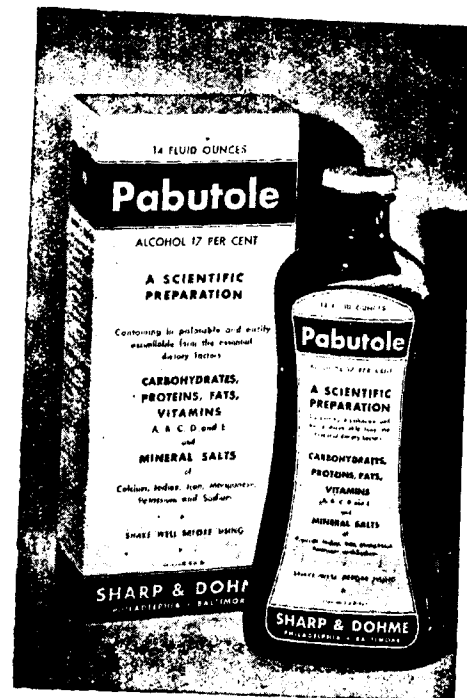
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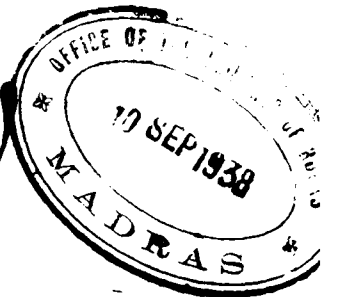
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AUGUST 1938

[No. 8

Indications for Mastoid Operation

BY

K. G. MENON, M.B., B.S.,

Ear, Nose and Throat Specialist,

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A Mastoid infection is diagnosed by most general practitioners only when a tender post-aural swelling develops in the course of an acute or a chronic Otorrhoea, but this sign develops only in fairly advanced mastoid infection. A mastoid infection has to be diagnosed much earlier than this because the results of operation are far more satisfactory when the condition is diagnosed early, and operated on than when operated at this late stage. Further, it is only in a few percentage of cases that this post-aural swelling manifests itself. In a large number of cases this sign does not make its appearance; and consequently a definite mastoid infection is treated as a simple otorrhoea with the result that grave complications set in and endanger even the life of the patient. Tenderness over the mastoid is not, as many imagine, in itself an indication for operative treatment because even a catarrhal inflammation of the mastoid antrum and air cells produces a temporary tenderness over the mastoid, which is reflex in origin and which passes off when the inflammation subsides. If on the other hand the tenderness persists in spite of adequate drainage, then there is a definite indication for operative interference. The points of

tenderness to be looked for in a suspected mastoiditis are (1) Over the Mastoid antrum, (2) Over the tip of the Mastoid, and (3) Along the posterior border of the mastoid.

It is not possible in a short article like this to go into the signs and symptoms of Mastoiditis in detail, so I shall confine myself to a few of the important signs and symptoms which would enable one to diagnose Mastoid infection in its early stages.

- (1) Profuse, thick, muco-purulent discharge lasting for more than 2 weeks in spite of adequate drainage.
- (2) Pulsating pus in the perforation in the drum, which fills up the meatus soon after the meatus has been cleaned.
- (3) Tenderness over the mastoid lasting for more than a week in spite of adequate drainage and conservative treatment.
- (4) Throbbing pain in spite of good drainage, rigor and headache.
- (5) A persistence of a high pulse rate with or without a certain amount of rise in temperature.
- (6) A sagging down of the postero-superior quadrant of the external auditory canal at its junction with the tympanic membrane.
- (7) Facial paralysis in a discharging ear.
- (8) Signs and symptoms indicating inflammation of the memirges or the labyrinth.
- (9) Post-aural swelling with tenderness over mastoid.

Before concluding I wish to draw the attention of medical practitioners to the fact that it is very very important to distinguish between a post-aural swelling due to mastoid infection and one complicating an external otitis. The differential diagnosis is of the utmost importance from the treatment point of view for, upon a correct diagnosis depends, whether the patient is to be subjected to a serious operation or not. I shall not go into the details of the differential diagnosis in this paper but I would

like to say before I conclude that many eminent surgeons have mistaken a post-aural swelling complicating external otis for a mastoid abscess and opened up the mastoid bone to find a perfectly healthy mastoid antrum and air cells. I have committed this mistake myself and hence my justification in emphasising this point so much to my brother practitioners, who may not be constantly seeing these conditions.

About the usefulness of Röntgenography in diagnosing an early mastoid infection, I am rather pessimistic about its utility. A skiagram will certainly help in the cellular type of mastoid but in the a-cellular or dense mastoids I do not attach much value to an X-ray picture because in these cases it is very difficult if not impossible to make out evidences of early inflammation of the antrum and air cells.

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The Early Diagnosis of Pulmonary Tuberculosis

BY

ANDREW MORLAND, M.D., M.R.C.P.

Methods of Detection

WHEN the patient presents himself for examination in this early stage his chest must be carefully examined, differences in the percussion note between the two sides, especially the apices, duly noted, and in the stethoscopic examination local differences in air entry looked for in addition to the search for râles, which are most easily picked up during the sharp intake of breath which follows a cough at the end of expiration. A small proportion of early lesions, superficially situated, produce physical signs which may be elicited in this way, but the great majority do not give rise to any abnormal physical signs. The stethoscope rightly occupies a place of honour in clinical medicine but its uses do not include the elimination of pulmonary tuberculosis, and if applied to this purpose it becomes a public danger, as most cases are no longer early by the time physical signs appear.

The detection of tubercle bacilli in the sputum is, of course, the only absolutely certain method of diagnosis and at least three sputum examinations should be made whenever there is the slightest suspicion that this disease is present. Should there be no sputum an expectorant mixture containing potassium iodide may be successful in producing a specimen for examination. Although most early cases have little or no cough and no sputum an appreciable number pass through a stage early in the course of the disease during which a positive sputum may be obtained. In the early tuberculous infiltrations described by Assmann the typical history is that of a febrile attack, of only a few days' duration, towards the end of which there is a cough and frequently a little positive sputum; the symptoms usually disappear quickly, but within a few months the patient develops signs of phthisis which is often of a severe type. The initial attack is usually taken for influenza, although the

absence of any other cases in the neighbourhood may arouse suspicions. The belief that tuberculosis frequently follows influenza has been proved to be erroneous by the absence of any increase in notifications following widespread epidemics of the former disease.

Radiology has made a revolutionary change in the diagnosis of pulmonary disease, and controversy has arisen concerning the relative merits of film and stethoscope. Defenders of Laennec's tube have dwelt at length on the difficulties of radiological interpretation. Difficulties there undoubtedly are, although these are becoming less with improving knowledge and technique; but the subject is not one for controversy of this kind, early diagnosis being so difficult that every method must be made full use of and given due weight in the final decision.

There is reason to believe that in the majority of cases radiological signs precede symptoms and the logical conclusion from this has been drawn by D'Arcy Hart in his recent Milroy Lecture in which he maintained that the mass examination by X-rays of groups of apparently healthy people was the only way of effectively increasing the proportion of cases diagnosed in the early stage of the disease.

Suggestive Symptoms

Pulmonary tuberculosis is not a difficult disease to diagnose when once suspected; it is true that in rare cases the disease may exist (as shown by a positive sputum) without radiological signs, but in the great majority of cases a normal film properly taken and interpreted will be sufficient to exclude the disease with only a small margin of error. A doubtful infiltration, coupled with suggestive symptoms, should lead to a presumptive diagnosis and suitable treatment, but not necessarily notification.

Among the symptoms for which X-ray examination is essential the most obvious is *hæmoptysis*; no chest has been thoroughly examined that has not been X-rayed and this symptom is so strongly suggestive of tuberculosis that it is inexcusable to dismiss the patient on the strength of a negative physical examination and sputum test even if he has a sore

throat from which the blood might have come. *Pleurisy* is also presumptive evidence of tuberculosis and requires radiological examination as soon as the patient is fit enough; even if this shows the lung parenchyma clear on both sides the patient remains a suspect and, after a period of treatment on sanatorium lines, not necessarily in a sanatorium, he should be kept under observation for several years, the weight carefully controlled and the slightest departure from normal health should be the excuse for further X-ray examination. Cases of *anal fistula* should be treated in much the same way.

Cough is usual in smokers and common in others; it is often absent in early disease; when it persists after a febrile attack, usually referred to as "influenza" it should excite suspicion. Chronic bronchitis with cough and sputum is typical of fibroid phthisis in elderly people; such persons are dangerous carriers of the disease although repeated sputum examinations may be negative; a timely X-ray may save the lives of children and others living in close contact with such cases.

A slight evening rise in *temperature* is often absent in early cases but when present it is important. Small temperature variations can only be established by careful technique; as gentle exercise habitually causes an appreciable rise in the temperature of normal subjects, it is important that the suspect of tuberculosis should have been at rest for at least half an hour before temperature taking. Axillary temperatures are useless, and mouth readings, although less accurate than rectal, will be the usual method used; the best times for temperature taking are (1) on awakening, (2) 6 P.M., (3) 9-30 P.M. The end of the thermometer must be placed well under the tongue and the mouth kept closed for at least five minutes. A rest temperature of 99° F. taken in this way is definitely above normal.

Loss of weight, important when it does occur, is often absent in early cases. The association of consumption with wasting has caused the disease to be overlooked, with disastrous results, in many well-nourished people. The fattest man I have ever seen had advanced tuberculosis, and it is extremely important to be on the look-out for phthisis in the obese.

The *phthisical chest* occurs in normal as well as in tuberculosis subjects, and most patients with early phthisis have chests of normal shape.

Amenorrhœa is unusual but may occur, especially in cases in which the onset of the disease has been preceded by a long period of overwork and worry.

The most constant and typical symptom of early pulmonary tuberculosis is *lassitude* which is often associated with anorexia and vague digestive disturbances. Radiological examination is certainly not frequent enough, and those who fear to waste their patients' or public's money may be reminded that one positive early case in a hundred means a great saving of money as well as the probable saving of a life and further dissemination of the disease.

Summary

- (1) Early pulmonary tuberculosis has no physical signs and often no symptoms.
 - (2) When suggestive symptoms are present or when the history gives rise to suspicion X-ray examination of the chest is essential.
 - (3) Physical signs in the chest and the detection of tubercle bacilli in the sputum have great positive value in the diagnosis of pulmonary tuberculosis, but should never be relied on in eliminating the presence of the disease.
-

The Treatment of an Early Case of Pulmonary Tuberculosis

BY

GEOFFREY S. TODD, M.B., Ch.M., M.R.C.P.

IN view of the modern methods of diagnosis, it is considered at the present time that it is best to judge a case of pulmonary tuberculosis as *early* not so much from the extent of lung involved as from the time of onset of the first definite symptoms. It is then possible to deal with the treatment of these patients (excepting, of course, rapid cases—the old “galloping consumption”) admitted to sanatorium within two or three months of diagnosis. The aim, however, of all medical practitioners must be to begin treatment as early as possible; and when the diagnosis has been established steps should be taken at once. On admission to an institution, the case should be submitted to a thorough clinical examination, followed by X-ray and blood sedimentation-rate examinations. On the result of these the line of therapy must be decided.

The Early Case with Fever

It is proposed first to describe in detail the treatment of an early active case, with disease limited radiologically to the upper half of one lung, with a temperature around 100° F. nightly and a high sedimentation-rate. The patient must be kept strictly in bed and the temperature and pulse recorded in the morning and late afternoon. If at the end of one month of this regime no striking improvement has taken place, artificial pneumothorax should be attempted. Before its induction, however, it must be ascertained that there are no contra-indications, such as super-added bronchitis, heart disease or extreme debility. The question of age (with the exception of extremes) need not be taken into account in view of the present modern technique.

On the day of the induction, it is advisable to give the patient an injection of morphine approximately quarter of an hour before beginning. The induction completed, the patient is warned to cough as little as possible and, if it is necessary, is

instructed to place the hand over the site of puncture (which is covered by a small pad of gauze) and thus minimize as far as possible the danger of surgical emphysema. This complication following induction is not in itself serious but is tiresome for the patient. Absolute rest is prescribed, and the patient is not allowed to wash or feed himself. Opinions differ as to the side on which it is best for him to lie but, as the question is probably not important, the patient may choose for himself.

The first refill is given twenty-four hours after the induction, when a similar procedure is adopted; but morphine is not given and a second needle is used. Both at the induction and first refill 300 c.cm. of air are given in the normal way. The second refill is given forty-eight hours after the first, and then the patient is refilled twice weekly for three weeks. For the first week of the treatment the patient is kept on absolute rest and for the next two on “Bed I”—by which it is understood that he is not allowed to wash himself but may read and write letters and feed himself. It was found in a long series of cases that one week’s “absolute” is quite sufficient and well tolerated by most patients. Longer periods cause depression and rather tend to militate against the success of the treatment. At the end of the third week the patient is carried down to the X-ray department, and the amount of collapse of the lung is noted. On the degree depends the subsequent interval between refills. I am quite convinced that the period between refills and the amount of air given are better regulated by screen appearances than by final pressure readings. In most instances, after three weeks the interval between refills is increased one day each time until one week is reached. The patient is kept on weekly refills until the collapse of the lung is well established. Here again I believe that small refills at this interval are much better than large ones at infrequent intervals.

During the period up to and including the weekly refills the question of what the patient is doing as regards rest may be asked. At the end of the fourth week he is allowed up to tea in his room; and if no reaction, such as rise of temperature or attacks of dyspnoea, takes place, he is allowed downstairs for tea on the third day after he gets out of bed. If all goes well,

he is allowed up to lunch and tea after a week of "tea only" downstairs; and one week later is allowed to stay up for dinner as well, returning to bed at 8-30 P.M. Ten days later he gets up to breakfast, thus being up from 8 A.M. until 8-30 P.M. This is termed being "on rest," as after meals (except between tea and 6 P.M.) he rests prone on his bed. After two weeks on rest he is given an exercise test of half-a-mile. He is instructed to take twelve minutes for the walk and to do it on as level ground as possible. On his return the following procedure is adopted: pulse and temperature are recorded as soon as the patient lies down, again after five minutes, thirty minutes and one hour. Rectal temperatures are taken for these tests. A satisfactory result is achieved when the temperature has become normal at the end of half-an-hour and the pulse has dropped to between 70 and 80. The patient is now kept on "half-a-mile in the morning" and "rest" in the afternoon for one week, after which he proceeds to half-a-mile in the morning and the same distance in the afternoon. After a further week a one-mile test is given and the time taken must be twenty-four minutes. The same tests are carried out on his return. Further tests are given at increasing intervals, until the patient is able to walk about six to eight miles a day. He is then, if all other tests are normal, allowed to return home and have his refills continued by his own practitioner. While he is carrying out the exercise described he is screened once a week, and the interval between refills is regulated according to the degree of collapse noted. Individual cases vary greatly but, if the patient's condition is satisfactory and he is walking six miles on discharge, the interval between refills is either two or three weeks.

The Early Case Without Fever

The treatment of the early case with a lesion confined to one apex, with a normal temperature but a positive sputum and a slightly raised blood sedimentation-rate will next be considered. The most important point is to rest the patient in bed for at least one month after admission and then take a further X-ray photograph. If improvement has taken place—as it should—he is allowed up on the same principles as the artificial pneumothorax case and proceeds slowly to the various grades

of exercise. It may happen that although he is able to withstand exercise and pass all the tests, the sputum remains positive. In such a case a course of gold treatment is considered at the end of the month's rest in bed. It is essential to be quite sure that the patient's urine is normal, and after beginning the injections this should be tested the day before and the day after each injection. The initial dose must be small—0.05 gm. being given intravenously. Any reaction of the patient must be carefully noted during the next few days, and if a severe reaction takes place the treatment must be suspended. The second dose should be given one week after the first and should be the same. If no untoward reaction occurs, 0.1 gm. may be given the following week. This dose is continued weekly for four weeks, and if all is well 0.2 gm. is then given weekly until the conclusion of the course. I am fully convinced that larger doses than 0.2 gm. are fraught with danger and certainly do not give better results. The reactions for which to look and which may necessitate early termination of the treatment, are albuminuria, abdominal pain and diarrhoea, dermatitis, stomatitis, and severe pain in the joints. Here a word of warning must be added. However small the reaction, the course should never be continued even with a reduced dose, as I have quite often seen very grave results follow a disregard of this principle. The full course suggested—and in use at Midhurst—is 5 gm. for a man and 4 gm. for a woman. During the course the ordinary exercise and rest treatment is carried on as previously described.

Other Measures

Other forms of therapy are available for the early case. Thus, though the induction of artificial pneumothorax in a patient, who has a small thin-walled cavity at one apex with very little infiltration surrounding it is too drastic a measure, "phrenic crush" should be tried and serial X-rays taken at monthly intervals during the ordinary routine treatment. If the cavity becomes progressively smaller and finally disappears, the phrenic paralysis should be made permanent. If no effect on the cavity is apparent, then artificial pneumothorax or other forms of therapy will have to be considered. I have quite often seen small apical thin-walled cavities closed by "phrenic crush"

alone, and it is always worth a trial before embarking on major procedures.

There will, of course, be a great number of early cases which do not need any treatment other than general sanatorium routine. Although nothing active is being done, the fact of being under sanatorium discipline and having something to occupy each hour of the day seems to have an extremely good effect on many patients, and they naturally do better in an institution than at home. I am quite sure that however early his case the patient should have his primary treatment and education in a sanatorium. During each of the courses of treatment described a good liberal diet must be given; and tonics, expectorants and other forms of medicines prescribed judiciously.

Treatment of Pleural Effusion

Most experts at the present time consider primary pleural effusion to be tuberculous in nature, so treatment of this condition becomes of paramount importance. As soon as it is diagnosed, the patient should be put to bed and a specimen of fluid removed for cytological examination. If this proves sterile to culture—as it should—the treatment now will consist of rest in bed until the temperature has dropped to normal and has remained so for two weeks. Aspiration should not be done at all during the early stages, unless the patient's respirations are greatly embarrassed and the heart is displaced markedly to the opposite side. If this is the case, only enough to relieve the dyspnoea should be removed. Time and time again the early removal of a pleural effusion causes a quick re-forming, with marked temperature elevation and an exacerbation of all symptoms. When the temperature has been normal for at least two weeks, the patient may be allowed out of bed for an hour or so each day. If no rise of temperature occurs, this time may be increased to three hours at the end of four days; and after a further three days to five hours, at which limit the patient should remain for a week. If all is still going well, five hours become eight hours, and after a week the patient may be allowed up all day, on rest.

The further treatment is carried out on the routine sanatorium lines already detailed. During the routine described,

serial X-rays must be taken at three-weekly intervals, to make sure the fluid is absorbing at a normal rate. Everything may be regarded as satisfactory if the effusion absorbs easily until it finally disappears at the end of three months.

Naturally a considerable number of patients will deviate from this rule. Those whose effusion absorbs sooner will cause no difficulty, but effusions which are obstinate and remain massive after two to three months present a problem which must be dealt with if the underlying lung tissue is to be left in a normal state. They should be aspirated after the conclusion of the second month and air run in to fill the space (gas-replacement). It will be necessary in this procedure to make an air level, which is done in the following manner. The aspirating needle is placed in the lowest clear space containing fluid; and a second needle attached to the artificial pneumothorax apparatus is placed in the second highest fluid-containing space, with the tubing carried over the patient's shoulder, in order to prevent fluid running into it. The aspiration is then commenced, air being bubbled in through the top needle at the same time. Very soon an air space will be obtained surrounding the top needle; and then all is straightforward, air being run in at sufficient rate to replace the fluid aspirated by the lower needle. Having obtained the air-replacement, the amount of air may in future be lessened each time and the lung slowly allowed to re-expand. Should the fluid re-form after the first gas-replacement, as often happens, it should be removed in the ordinary way of gas-replacement, as often as is necessary, the whole procedure adopted depending on screen control of the case at intervals of not less than a week.

When by screening it is seen that the lung is about three-quarters out, an X-ray should be taken, to ascertain whether there is infection with tubercle bacilli. If not, the lung may be allowed to re-expand completely. If tuberculosis is present, then an artificial pneumothorax should be kept up.

An ordinary case of straightforward pleural effusion should have at least four to five months in a sanatorium, in order to make sure of a good recovery. The habit of allowing those

cases first of all to be treated in the home and then sent for a holiday to the seaside cannot be too strongly deprecated.

Treatment while Waiting for Sanatorium

Nowadays most sanatoria have long waiting lists, and, in consequence, the general practitioner is called upon to treat cases of pulmonary tuberculosis whilst they are awaiting admission. How should he proceed? This will naturally vary according to the type of case and length of wait, but as a general rule all patients would benefit greatly by being kept at rest most of the day and not allowed any exercise. All activities should be left to the discretion of the sanatorium authorities, who have far more means of assessing a case at their disposal than the busy general practitioner. If it is remembered that however mild a case appears—even if there is no cough or sputum and there is a normal temperature—a great deal of benefit will result to the patient by keeping him at rest whilst waiting, more cases will have their period of treatment in an institution shortened and be restored to health more rapidly. Quite often a favourable case is seen in which the disease has been allowed to spread by allowing too much activity before admission to a sanatorium or other institution. A very good doctrine in the early stages at home when not sure what treatment to prescribe is—keep the patient in bed.

Conclusion

In conclusion, a warning must be mentioned. The fact that a patient's temperature is normal does not necessarily mean that disease is quiescent and that he may be allowed out of bed. Frequently patients give a history of having been allowed to get up as soon as their temperature became normal, irrespective of whether other tests were satisfactory or not. The early case, if energetically treated, has a good prognosis in most instances, and this leads to the consideration of a plan for invariable early diagnosis. Patients who complain of cough persisting for two weeks should be X-rayed if no physical signs are found as the early case is often free from all stethoscopic signs in the chest.

The Early Diagnosis and Treatment of Abdominal Tuberculosis

BY

REGINALD LIGHTWOOD, M.D., F.R.C.P.,

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INCLUDED under the term abdominal tuberculosis are the conditions of tuberculous ulceration of the intestine, tuberculosis of the abdominal lymphatic glands (tabes mesenterica) and tuberculous peritonitis, but these states so overlap that only for ease of description should they be considered separately.

Causation

The usual cause is a primary infection of the bowel but the disease may also be secondary to tuberculosis elsewhere in the body and particularly in the lungs. Thus, patients with advanced phthisis, who have not been careful to avoid swallowing their sputum, may develop symptoms of abdominal tuberculosis (auto-infection). It is also an interesting and well-known pathological fact that many young patients dying of miliary tuberculosis show at post-mortem large numbers of small and recent ulcers in the intestine. As a rule these multiple secondary ulcers do not produce intestinal symptoms and the clinical picture is likely to be that of the underlying miliary tuberculosis or tuberculous meningitis. Abdominal tuberculosis may also arise from the direct extension of a tuberculous process into the peritoneum or by lymphatic spread from neighbouring lymphatic glands, but neither of these occurrences is at all common.

When the causes of primary abdominal peritonitis are considered certain facts may be quoted which throw light on them. The maximum age incidence of primary abdominal tuberculosis is in the second year of life. Milk is undoubtedly the medium which usually bears the infection and it is just at this age that large amounts of milk are given and also when it is first used without sterilization. In 1931, in Great Britain, it was estimated that raw milk as distributed for human consumption

showed on an average the presence of living tubercle bacilli in some six per cent or seven per cent of the specimens examined. But it must not be thought that all cases of primary abdominal tuberculosis are bovine in their origin. Bovine strains are recovered bacteriologically in more than three-quarters of abdominal cases, and human types of tubercle bacilli are the cause in the remainder. In these a family history of tuberculosis is often found and it is believed that the organisms reach the intestine through the swallowing of particles of infected sputum which adhere to the fingers or contaminate articles of food. Thus contact with open tuberculosis is a cause of the abdominal tuberculosis of human origin only if carelessness and bad hygiene are allowed.

In order to understand the development of primary abdominal tuberculosis a clear idea of its essential underlying pathology is required. The changes which occur in primary tuberculosis are the same wherever in the body the primary lesion occurs. First a small primary focus of infection develops. In the case of the intestine the primary focus takes the form of a small ulcer which rapidly heals. This ulcer corresponds pathologically with the primary lung focus (Ghon's focus) which originates all primary infection of the lungs. Unfortunately, healing of the primary intestinal ulcer does not mean that the disease is at an end because infection is likely to spread in a centripetal direction along the lymphatics towards the mesenteric glands. These glands enlarge, caseate and ultimately heal by fibrosis and calcification, though in a small proportion of cases extension into other glands or into the peritoneum takes place. Every grade of severity from the most benign process to a fatal abdominal tuberculosis may be seen. Fortunately, a large majority of the infections are benign and often almost symptomless. Infections with human type bacilli are usually more severe than those due to bovine strains.

Clinical Forms

Beyond question abdominal tuberculosis, even more than other forms of tuberculosis, is declining, and this is the result of the increasing pasteurization of milk. The symptoms of abdominal tuberculosis are indeed becoming quite uncommon,

though paradoxically caseous or calcified mesenteric glands are still often found at operation and autopsy. Evidently there is much symptomless abdominal tuberculosis. In spite of the comparative rarity of clinically manifest abdominal tuberculosis the disease is often suspected and nearly as often wrongly diagnosed on insufficient data. If abdominal symptoms arouse suspicion, a tuberculin reaction should be performed. The intradermal test (Mantoux) using 0.1 c.cm. of a 1 in 1,000 dilution of old tuberculin may be employed. A negative reaction for all practical purposes excludes the diagnosis. A positive reaction should lead to a search for other confirmatory evidence.

Tuberculous enteritis.—If a tuberculous ulcer forms the onset of symptoms is likely to be insidious. The symptoms may be pain, loss of weight and diarrhoea. Occasionally the stools may contain a little pus or blood but this is seldom manifest. Confirmation of the diagnosis may be obtained by having the stools examined for tubercle bacilli. Fever may be present together with other general signs of infection. Examination of the abdomen shows it to be tumid, somewhat resistant and diffusely tender.

Tuberculous adenitis.—This must be a common condition; yet most cases pass unnoticed, so inconspicuous are its symptoms and signs. The outstanding sign is the detection by palpation of enlarged glands in a child with a positive tuberculin reaction. Their differential diagnosis requires the exclusion of the possibility that the masses may be faecal. This possibility can be settled by watching the effect of warm olive-oil enemas. Among the general symptoms of *tabes mesenterica* are abdominal pain (which may give rise to suspicion of appendicitis), slight fever, stationary weight and irregularity of the bowels.

Tuberculous peritonitis.—This is becoming more and more rare. The two chief varieties, the ascetic and the plastic, are so well described in all text-books that they do not require description here. Ascites in a child whose tuberculin reaction is positive is most likely to be due to tuberculosis. In the plastic form of peritonitis an omental tumour often forms across the upper part of the abdomen and masses may be felt in other situations. Sometimes a tuberculous abdominal mass may be

confused with a chronic intussusception. When this difficulty arises Still's sign may be invaluable: the tumour which forms in intussusception shows peristaltic contractions, tuberculous masses do not. An opaque enema may also be of the greatest diagnostic value.

Radiological assistance in the diagnosis of tuberculosis of abdominal glands can only give help in a healing stage, i.e. when calcification has begun. If a child with abdominal glands and a positive tuberculin reaction is X-rayed and calcification visible a good prognosis may be given, because the fact that the glands are visible indicate that healing has been effected or is taking place.

Treatment

Estimation of the sedimentation rate are chiefly of value in following the course of a patient under treatment. Complete rest will be required until some weeks after all the signs of activity have passed away. The signs of activity are failure to gain in weight, fever, sweating, abdominal enlargement (and ascites) and a raised sedimentation rate.

The essentials of treatment are complete rest, proper feeding and open air. The severity of the condition determines to what extent rest must be enforced. In the more acute and severe cases the legs as well as the abdomen must be immobilized and a plaster bed is useful for doing this. Other less severe cases can be in bed without special apparatus. Feeding should be sufficient to enable the child to put on weight but it is not wise to recommend "stuffing." Anything which increases peristaltic activity of the bowel must be excluded. Hence the parents are told to exclude brown bread, porridge unless sieved (and any other of the fancy breakfast cereals), raw fruit and stewed fruit unless sieved, and all green vegetables unless sieved. All jams with pips and skins must be omitted and other articles of dietary such as salad which tend to leave a residue. The diet should mainly consist of dairy produce (milk, eggs, butter, cream, cheese) for its calcium content. Well-cooked milk puddings and good home-made soups are also excellent. There is a theoretical objection to these high-fat diets on the ground that the lacteals may be blocked, but in practice, unless the stools

do become fatty there is no need to reduce the fat content of the diet. Except for vitamins drug treatment plays a minor part. Preparations containing vitamins A, D and C are especially indicated. Salts of calcium may be used in the healing stages to ensure that there is sufficient of this mineral to effect calcification of the glandular lesions. Useful preparations are:—dicalcium phosphate 20-30 grains t.d.s., calcivitan (Richter) one tablespoon daily and calcydic (A. and H.) one or two teaspoonfuls. These preparations should be taken before meals. Liquid paraffin should be given to regulate the bowels and in obstinate cases warm olive oil enemas be necessary.

The question of surgical treatment must be briefly discussed. The resemblance of appendicitis to inflamed mesenteric glands is often close and some hold the view that the appendix should be removed in all cases. This is probably too radical, but if the pain is severe, causing vomiting and associated with loss of weight, then laparotomy may be called for, with removal of the appendix and any other obvious focus, such as a tuberculous Fallopian tube. In the ascitic form of tuberculous peritonitis a laparotomy can be employed to evacuate a fluid of low bactericidal power in the hope that it will be replaced temporarily by a serous effusion of higher bactericidal power. It is undoubtedly true that many children with free fluid show improvement from the time of the laparotomy, but on the other hand it is just in the early ascitic type of the disease that general hygienic measures secure the best results. One advantage of laparotomy is that it does enable the diagnosis to be established beyond all shadow of doubt and it also confirms an opportunity for removal of the appendix which some authorities regard as the essential primary focus in almost all forms of abdominal tuberculosis. In any case surgical intervention must be followed by rest under good hygienic conditions, a generous diet and if available, sunlight. It is possible that the after-treatment is more responsible for good results than the surgical episode.

Extraordinary General Body Meeting

June 1938

AN Extraordinary General Body Meeting of the above Association was held at 5 P.M. on Saturday the 18th June 1938, in the Head Quarters Hospital, Trichy, when about 30 doctors were present. Dr. C. E. R. Norman, the President of the Association, presided. Dr. K. Raman Menon, A.D.M.O., Head Quarters Hospital, Trichy, was At Home to the Members.

After the President's preliminary remarks, the Secretary read the minutes of the previous meeting.

The following subjects that were brought forward by the Managing Committee at its meeting dated 2-6-38, were discussed:—

(1) Re-Postponement of the Association becoming a branch of the Indian Medical Association, for reconsideration at its next Annual General Body Meeting, in view of the financial position of the Association.

In this connection the Treasurer pointed out that there is arrears of subscription to the extent of Rs. 700 and odd.

Proposed by Dr. Kalamegham, seconded by Dr. Bhut and was unanimously passed.

(2) The committee's recommendations to the General Body that any committee member who is absent for three consecutive meetings, ceases to be a member of the committee.

Proposed by Dr. Iravatham, seconded by Dr. N. C. Subramanyam and unanimously passed.

(3) The committee's recommendation to amend Rule 31 of the Association Bye-Laws to read as follows:—For a quorum there shall be 3 committee members present, of which the President shall be one.

After elaborate discussion it was unanimously passed.

The following cases were shown:—

- | | | |
|--|--------|----------------------------|
| I. Case of Monoplegia | ... | By Dr. Iravatham. |
| II. Case of Compound Collis, Dislocation of Head of Radius, Dermeid of the scalp recurring | | By Dr. Kalamegham. |
| III. Case of Urachal Tumour, Ilio-caecal Tuberculosis, a typical Appendix, a typical Appendix with vague dyspepsia | | By Dr. Raman Menon. |
| IV. Case of Extotitis, Nasal Polyp, Mastoid | | By Dr. K. G. Menon. |
| V. Case of Dilated heart | | By Dr. N. C. Subramanyam. |
| VI. Cases of cirrhosis of liver with Ascitis treated on strict milk diet | | By Dr. M. A. Krishna Iyer. |

After the president's concluding remarks and request to have the next meeting at the Central Hospital, Golden Rock, the meeting terminated at a late hour with a vote of thanks to the chair and the lecturers.

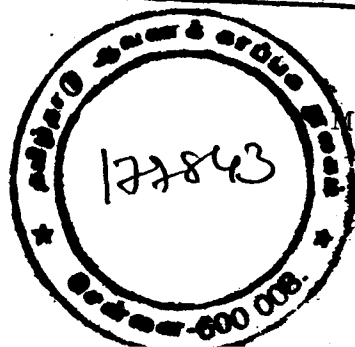
Secretary.

The Trichinopoly District Medical Association
TRICHINOPOLY

Statement of Accounts for July 1938

RECEIPT	PAYMENT
Rs. A. P.	Rs. A. P.
Cash balance on 1-7-38 34 8 6	Clerk's pay for June '38 18 8 0
Bank balance on .. 137 8 2	Office rent for June '38 5 0 0
Excess refund by clerk 0 8 0	Stationery ... 0 13 6
Subscription from Members 48 0 0	Stamp expense for Advertisers etc. ... 16 4 0
Do. Advertisers (less bank commission 0-6-0) ... 76 2 0	Stamp expense (Secretary) ... 7 0 6
	Purchase of old copies of B.M.J. and I.M.G. 0 10 0
	48 4 0
	Bank balance at the end of the month ... 206 2 2
	Cash balance ... 34 5 0
	Stamp balance (Secretary) 7 5 0
	.. (Treasurer) 0 10 6
TOTAL ... 296 10 8	296 10 8

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31st July 1938.

M. C. CONDILLAC,
Treasurer.

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(REGISTERED)

AUGUST 1938

The Editor requests contribution on Medical, Topical and News Subjects from Members of the Association and other medical men. Contributions should be sent typed in duplicate and signed by the contributor. In case where it is desired to write under a nom-de-plume the signature must be included for the information of the Editor as a token of good faith.

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THE MISCELLANY

OF



THE TRICHINOPOLY DISTRICT MEDICAL ASSOCIATION

(REGISTERED)

Editor: DR. M. C. CONDILLAC, M.R.C.S.

SEPTEMBER

1938

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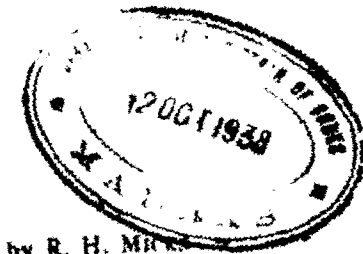
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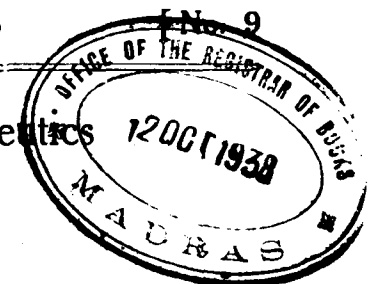
Vol. V]

SEPTEMBER 1938

The History of Therapeutics

BY

R. H. MICKS



THE term "therapeutics" means to me more than the mere knowledge of drugs, for it suggests four things: a drug, a doctor, a hospital and a patient. Let us begin by talking about patients. But a doctor has no right to tell stories about his patients, at least until they are dead; and mine are not all dead yet. So I will content myself by telling you one thing about patients, a thing that you will find out for yourselves sooner or later, that they are nearly all more frightened than they have any need to be. So you can nearly always do one thing for them, relieve them to some extent of their fears; and you can nearly always give them one medicine, hope.

I do not intend to discuss doctors either. Everybody discusses us, and let us hope that we get more credit than we deserve. So let us pass on to the hospitals and the drugs, and consider their histories.

The earliest hospitals mentioned in literature are those of India, for among the other achievements of early Hindu civilisation was the organisation of hospitals for town and villages. The objects of this hospital service were both to care for the sick and to prevent the spread of disease, and thus the Hindus were socially a step ahead of the Egyptians, the Greeks and

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the Romans, who recognised the importance of isolation and public hygiene, but made no provision for nursing as a communal service. You may be interested to hear a description of the ideal doctor from Hindu literature of about the fourth century before Christ:

"He should be cleanly in his habits and well shaved, and should not allow his nails to grow. He should wear white garments, put on a pair of shoes, and carry a stick and umbrella in his hands, and walk about with a mild benignant look, as a friend of all created beings. . . . A physician should abjure the company of women, nor should he speak in private to them nor joke with them."

Our modern hospitals have their origin in the history of early Christianity. Christianity was the first religion in which the spirit of doing good to others was an essential part of the teaching; to this day men who are at variance in points of doctrine find themselves at one in the practice of the parable of the Good Samaritan. The first Christian hospital in Rome, that is to say, the first hospital in Rome was built probably in the fourth century after Christ, and from the sixth century on hospitals were to be found in all Christian communities. The rise of the great monastic orders in Europe naturally exercised a profound influence on the development of hospitals, and from the time of St. Benedict, in other words, the early part of the sixth century, historians can trace a gradual transference of the duty of caring for the "pensioners of the Church," the sick and infirm, the old and needy, from the Bishop to the religious communities. In St. Benedict's Rule it is indeed distinctly stated that *infirmorum cura ante omnia adhibenda est*. It is not quite correct to refer to the "hospitals" of the early Church; the term "hostel," a somewhat free translation of the Greek word, conveys the meaning better, for sick, needy, crippled and aged were housed together, and prior to the eleventh century there is no evidence of the existence of any such thing as an order in which monks or nuns did the actual nursing. Probably it was done by lay attendants.

In the early twelfth century we find the first account of a hospital, one that was in connection with a monastery in Con-

stantinople. It had fifty beds, ten for surgical cases, eight for acute and eye cases, twelve for women, twenty for lighter cases and five for emergencies. There were as well five beds for fractures, and these beds were sectioned and had a "mattress divided in the middle." The staff of doctors served in two relays. There were two head doctors, three officially appointed assistants, two supernumeraries and two servants. In the women's wards there were as well, besides the physicians, a woman doctor, four official female physicians, two supernumeraries, two female servants and also two night-watchers, a woman and a man. There was a bath-house, which was used twice a week by the patients, or oftener if they needed it. We have no early record of the organisation of other ancient foundations, though the Santo Spirito in Rome, St. Peter's Hospital in York and the Hôtel Dieu in Paris were already in existence.

From the twelfth century onwards we can mark the separation of the sick from the poor, and with that the rise of the regular nursing orders. The oldest was that of the Augustinian Sisters at the Hôtel Dieu, and their influence on the development of modern nursing is incalculable. They lived a life of the hardest and most self-sacrificing routine, spending their lives in the wards, which they left only to do visiting nursing. From such orders we have received the tradition that the nurse's life is a life of self-sacrifice and monotony, a vocation almost, rather than a career for the woman with normal social instincts. We find their terminology and uniform still copied, for they had their "probationers" (*filles en approbation*), those who could wear the uniform of the white robe (*filles blanches*), and those who received the hood or *chaperon*.

In England the Reformation affected the hospitals very profoundly, for the disestablishment of the monasteries disestablished also the only hospitals then in existence. We find the City of London petitioning for permission to take over the four large hospitals of St. Thomas, St. Bartholomew, the Bridewell and Bethlehem. The same fate befell some of the famous French hospitals; the Hôtel Dieu, for example, in the early sixteenth century yielded its control to a body of lay rectors. From this time on the level of nursing in lay hospitals was very low

indeed and remained low till about one hundred years ago. The brightest spot in the nursing history of the period is the foundation of the Sisters of Charity—the “French” Sisters of Charity as we commonly call them—by St. Vincent de Paul. His “daughters of charity” took vows only for a year and did not, he insisted, form a religious “order.” With the terms of his instructions to them you are probably familiar and, if not, you should be, for they represent an ideal which modern scientific nursing has tended to ignore but has of late revived in the form of the Lady Almoner. “The Daughters of Charity shall have for monastery the houses of the sick, for chapel the parish church, for cell a hired room, for cloisters obedience, for grill the fear of God, and for veil holy modesty.”

The early part of the nineteenth century was the glorious period for Irish medicine, but it was also the glorious period of Irish nursing, for it witnessed the beginning of the reform of hospital nursing, which was up till then everywhere in the hands of old women and sluts, absolutely untrained and often grossly immoral and disorderly. It is only just over a hundred years since Ireland showed the way to other countries in the foundation of the two great nursing-orders, the Irish Sisters of Charity and the Sisters of Mercy. Florence Nightingale, to whom the organisation of nursing as a career for the lay woman is largely due, was very nearly becoming closely connected with Dublin, for we find her in 1852 writing to Cardinal Manning commending the training given by the nursing-orders, and asking him to find out “whether they would take me in at the Hospital of St. Stephen on the Green in Dublin for three months as I am. I want to learn their trade.” But she went to the nuns in Paris instead, and shortly after that the Crimean War broke out, from which she returned to stimulate the formation of training schools for nurses, at first in St. Thomas's Hospital, from which the movement spread to all teaching hospitals.

In the last fifty years our hospitals have developed more than in any previous period of their history, so that, from being places of refuge in which the needy sick were tended, they are becoming the workshop of the specialist. The hospital patient of to-day has little to complain of; he is well fed, well nursed,

and if he is not well doctored, he is, at any rate, as well doctored as anyone can be. Poverty, far from making it difficult for the poor man to obtain skilled attention, actually makes it easier for him.

Bearing all this in mind it may seem absurd to suggest that our hospitals show any lack of consideration to their patients, but nevertheless, in one way they are still barbaric. They make no attempt to segregate from the general run of patients, that is to say, the chronic cases, the convalescent and the ambulant cases, those acutely ill patients who must have peace and quiet. They make no attempt to separate into single-bed wards those patients whose presence in a communal ward is a disgrace to our humanity, the raving and restless, because they disturb and often terrify all others, and the dying. The death-bed scene is an inevitable incident in hospital life, but it is one that no sick man or boy, woman or girl should be forced to attend against his will. We must recognise that so long as we refrain from making provision for nursing a small proportion of patients by themselves, namely, the acutely ill, the restless and the dying, hospital treatment may fairly be stigmatised in this respect as inhumane, and will assuredly be regarded as such by future generations, who will wonder at our callousness; they may even call it cruelty.

If nursing had to wait till the Middle Ages to make it start, medicine had to wait still longer, for the history of medicine begins at about the period of the Renaissance, up till which time it had remained where Hippocrates had left it two thousand years before. Three hundred years intervened between Andreas Vesalius, the first accurate anatomist, who died in 1564 and Lister, who did his first antiseptic operation in 1865; these were years in which medicine was advancing along the path of knowledge, but we cannot claim that the science of therapeutics began as early as the Renaissance. Not till the middle of the last century had the knowledge of the human body and of the causes of disease in it become sufficiently profound to permit of the discovery of rational methods of treatment, for successful treatment is next to impossible without an accurate diagnosis. Robert Hutchison was right when he wrote that

there were three things in treatment. The first was diagnosis, the second was diagnosis, and the third was diagnosis.

Yet, even in the sixteenth century there was at least one efficacious new drug discovered, viz., mercury. It is mentioned as far back as 1530 as a specific for syphilis in that poem which gave the disease its name. In the poem a nymph leads the shepherd who is infected with the disease to a river: "a sacred river, whose metallic waves carry quicksilver, which will furnish thee the remedy that is good for thy disease.... When the sacred stream shall have passed over thy body three times, thou shalt be delivered of thy disease and its impure poison."

The dissections of Vesalius and the discovery of the circulation of the blood by William Harvey in the early part of the seventeenth century are historical landmarks; they remind us that physicians of the sixteenth and seventeenth centuries had at length begun to study the human body instead of theorising about it. In the eighteenth century they went a step farther, and began to study disease; it was as if they had only just begun to understand that illness could be looked on as a phenomenon of nature, something that could be studied. Many of the names of common diseases, such as Pott's caries, keep alive the memory of famous physicians and surgeons of this period in which diseases were for the first time accurately described. But the descriptions were those of a naturalist rather than of a scientist, for an enquiry into the nature of disease was impossible for men who had no knowledge of the cellular structure of the tissues or of their unicellular foes, the bacteria. Yet, the eighteenth century saw the birth of that science to which above all others medicine owes its successes in therapeutics, the science of chemistry, and we would not be far wrong if we were to select as the greatest name in eighteenth century medicine, not that of a physician, but that of the chemist Lavoisier, who died beneath the guillotine in 1794.

In the meanwhile a few more useful drugs had been discovered. In 1638 quinine was introduced into European medical practice by members of the Society of Jesus returned from South America, and hence it received the additional name of Jesuit's Bark. We who are accustomed to think of malaria as

a disease only of the tropics find it hard to imagine how great was the benefit to Europeans as a result of this. In southern Europe there is still a good deal of malaria, and three hundred years ago the disease seems to have been common in marshy districts of England.

The discovery of the effect of fruit-juice in scurvy is often attributed to the explorer Captain Cook, who made his voyages in the eighteenth century, but in fact its value was known at a far earlier date. Sir John Hawkins in 1593 wrote: "that which I have seen most fruitful for this sickness is sower oranges and lemons."

The only other therapeutic discovery in the realm of medicine was that of digitalis. William Withering published in 1785 his experiences with this drug, and his *Account* remains a monument to his powers of clinical observation and reasoning. He tells in it how he heard of an old woman in Shropshire who had brewed a tea, made of many herbs, which had great fame in cases of dropsy. He decided, when he obtained the recipe for it, that the active ingredient was foxglove. He was still thinking about whether he would try it on his patients (or, as he put it, "introduce it into the more regular mode of prescription"), when a circumstance happened which accelerated that event. My truly valuable and respectable friend, Dr. Ash, informed me that Dr. Cawley, then Principal of Brazen Nose College, Oxford, had been cured of a hydrops pectoris by an empirical exhibition of the root of the foxglove, after some of the first physicians of the age had declared they could do no more for him. Withering then determined to give it to all of his own patients who had a dropsy, thinking to himself no doubt that a University Don had at last justified his existence by acting as the *corpus vile* in a scientific experiment.

Yet the standard treatment of disease at this period was ignorant and barbarous. Not only was the condition of the hospitals and lunatic asylums incredibly bad, but a superstitious use of traditional "remedies" prevailed. Many of them were useless, but the physicians of the day seemed unable to see this; many others did nothing but harm, for example the ritual of bleeding and purgation. The practice of bleeding, which

encouraged the surgeon to drain away in a few days almost every available drop of blood in the patient's body, was so obviously bad that it disappeared completely from medical practice, but the ritual of routine purgation was much slower in disappearing, and indeed it may still be said to linger on, if we may judge from the attitude of the ward-sisters of our hospitals, who demand that every patient shall make at least a daily offering to Cloacina. The pharmacopœias of the period reflect the ignorance and credulity of the age. Towards the end of the seventeenth century the London Pharmacopœia contained "cock's comb, bee-glue, fur, feathers, hair, human perspiration, human placenta and wood-lice." Its redeeming feature is that it included one drug still held in the highest honour—Irish whiskey.

The first half of the nineteenth century was the period of the rise of many schools of clinical medicine, each with a noble list of names of famous physicians and surgeons, but not, you must note, of famous physiologists, pathologists or bacteriologists. As yet there was no science of medicine, there was no knowledge of the cellular structure of the human body either in health or in sickness, no knowledge of bacteria. But from these great schools of medicine, inspired by these great teachers came the men who were the first pathologists, the first bacteriologists, the men on whose work the whole of modern medical knowledge is based. The first and greatest of these schools was the French School, and the teacher in it most worthy of your attention to-night was Pierre-Charles-Alexandre Louis (1787-1872). Revere him, not as the man whose studies, begun at the bedside and carried on to the post-mortem room, produced the first accurate description of the course of phthisis and differentiated typhoid from typhus fever, but as the man who first insisted that the effect of drugs must be studied by a statistical method. At a time when bleeding and purgation were still the approved treatment for pneumonia, he dared to assert that bleeding killed more patients than the disease, and to teach his pupils to judge the value of a treatment not by its correspondence to tradition or the rules of an academic system, but by the superiority of its results over those of other treatments.

Philippe Ricord, too, deserves mention for his work in distinguishing syphilis and gonorrhœa, for it, like Louis's differentiation of typhoid from typhus, shows us very clearly how ignorant physicians of the earlier ages were, and how well the great observers of the early nineteenth century prepared the ground for their successors. Oliver Wendell Holmes describes him as "the Voltaire of pelvic literature—a sceptic as to the morality of the race in general, who would have submitted Diana to treatment with his mineral specifics, and ordered a course of blue pills for the vestal virgins."

For a short while two distinguished pupils of the French School, Robert James Graves and William Stokes, raised the Dublin School of medicine to pre-eminence among all other schools, and founded a system of clinical teaching which is still respected and copied. Before I tell you anything else about these two great men I will ask you never to forget one thing about them; for them the poor in their hospitals were not subjects for teaching and for study, but patients; they worked first and foremost not for fame and honour, but for the poor of their country. Both shared the healthy scepticism of the great Louis as to the efficacy of traditional methods of treatment, but neither made any great contribution in a constructive way towards the drug treatment of disease. Graves, however, made one notable advance in dietetics.

It is difficult for the modern student to realise to what extent the time of the physician of a hundred years ago was occupied with the treatment of the two chief "continued fevers" typhus and typhoid, the former of which he never sees, the latter but rarely. Their prevalence and the ease with which they were communicated made the medical student's and the doctor's life of that time one of real danger, so that every year many hospital students caught one or the other fever, and not a few died. Graves was the first teacher to point out that in a fever which lasted always at least 14 days the prevalent custom of starvation might in itself be fatal. Stokes records how one day Graves was going round the hospital when, on entering the convalescent ward, he began to expatiate on the healthy appearance of some who had recovered from severe typhus.

"This is all the effect of our good feeding," he exclaimed; "and lest, when I am gone, you may be at a loss for an epitaph for me, let me give you one in three words—'He fed fevers'."

From the middle of the last century onwards discoveries came thick and fast, each new discovery bringing with it a new possibility of treatment. So far I have not mentioned German Schools, not because there were not many great teachers in them, but I will now tell you the name of one man to whom, as the founder of the study of morbid histology, special reverence is due, Rudolf Virchow. His studies began about 1840 and were above all others the key which unlocked the door of knowledge through which his successors entered. In the same decade general anæsthesia was first used in operations, and a little over ten years later Lister showed his fellow-surgeons how to operate without infection. Read his life before you are much older, but read first of all the life of a greater man than Lister, a simple-minded little French Professor of Chemistry, Louis Pasteur. I recommended it to you first of all because it is delightful reading, but also that you may see for yourselves the history of modern therapeutics as it were unfolding itself before you as you read.

The last 50 years have been years of unceasing discovery, and it would be impossible to name each discoverer and his work without wearying you. Let me remind you that there are men in this room who were students before the serum-treatment of diphtheria had been discovered, before radium or X-rays were known, far less used in diagnosis or treatment; before any hormone had been identified, when the existence of ultra-microscopic viruses was not even suspected. Many members of my audience remember the introduction of Ehrlich's Salvarsan, they remember the high hopes which they had that it was to be followed by many other brilliant discoveries in the realm of chemotherapy, their disappointment when none came, till recently the chemical laboratory gave us what promises to be one of the most potent weapons in our whole armoury against bacterial infection.

I will not speculate about the future. There must be a rich harvest of discovery awaiting us. It may be many years de-

layed, for the modern chemist is dealing with far harder problems than his fathers ever tackled. The day of studying the action of drugs solely by their effect on the living organism is passing away, and pharmacology is becoming a science which studies molecules. The attempt to express normal and abnormal tissue-reactions in terms of interaction between certain comparatively simple reagents, let us call them drugs, and the extremely complex molecules which cover the surface or occupy the interior of single tissue-cells or unicellular organisms is a titanic task, but it must in the end, perhaps generations hence, lead us to an understanding of disease and therapeutics beside which our present-day knowledge will seem the darkest ignorance.

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Indigestion

BY

DR. D. SRINIVASAN

WHAT I am going to write about Indigestion is about those forms of functional dyspepsias which are not organic in nature i.e., dyspepsias not associated with Cancer, with Ulcer, with hepatic or renal cirrhosis but purely a clinical form which is often met with and is not discussed in detail in books. It is common to meet patients complaining of indigestion without any obvious cause. Those are generally from the higher class, robust and strong; and who are idle and would not like the proper advice. They go from one doctor to the other and become ultimately pessimistic in medicines and lose faith in the doctor. Such cases are a nuisance to other practitioners even though they are good pay masters. The success of treatment lies in understanding the physiology and pathology of the normal Gastric digestion and thus correct the error, with the co-operation of the patient.

Indigestion is due to some fault in the secretion of Gastric Juice or the mobility of the stomach. The text-books describe the varieties as atonic, gouty, renal, irritative, flatulent and excess of acid. These names are really misleading and take us nowhere in the treatment. It is simple and clear to group the condition into two. 1. "Sthenic". 2. "Asthenic". These two present distinct and definite clinical types with different pathology, different symptomatology and very different treatment. So it is necessary to understand the digestive process.

The masticated and salivated food passes through oesophagus to the stomach and there it is acted upon by HCl and Pepsin. Formerly it was thought that the presence of Pepsin played an important role in the digestion but now we are drifting to the idea that HCl is the more important partner of the combination because the presence of the acid is most essential to the activity of the ferment. If the HCl is not in sufficient quantity, the gastric digestion comes to a stand-

still. So food though a normal inhabitant becomes a foreign body and excites the secretion of alkaline mucus from the stomach which surrounds the mass. Further process is not possible as pancreatic secretion is dependent on HCl acting on prosecretin in the alimentary canal. From these facts it is evident that HCl is the vital factor in digestion of all food products.

Conversely, if the acid is secreted excessively, it mixes with the food and the mass becomes too acid to be allowed into the duodenum. The Pyloric sphincter closes, and the passage of the food is blocked until the pancreatic juice and bile neutralise the excess of acid. Thus the food is locked up in the stomach longer. Irritates the nerve endings. Any slight upset of the mechanism of the stomach will produce dyspepsia; or in other words defective secretion of acid either in quality or quantity will lead to disorder.

It is clear in both the varieties of Indigestion the retention of food in the stomach longer than is necessary causes direct symptoms directly of pain, the outpouring of mucus and flabulence; and indirectly, to languor, sleeplessness and irritability of temper. So the general symptoms do not help us in classification, but there are certain marked differences both in the type of patients and in the nature of the symptoms. The sthenic form occurs in people who seem otherwise robust in health, who are for the most part strong, active and energetic and who may not be teetotallers. The asthenic form appears in weakly nervous convalescent or overworked persons who may be teetotallers but invariably will be tea or coffee drinkers.

	Sthenic	Asthenic
Tongue	... Generally furred	... flabby and generally clean and glazed.
Pulse	... Slow and full	... quick and feeble.
Appetite	... Voracious and ever present	... Weak and often absent.
Pain	... It is only fullness not pain	... Pain in the epigastrium spreading between the scapulas.

Administra-
tion of

full meal ... Relieves the pain ... Pain aggravated.

Mental

state ... Irritability ... depression.

Appearance... Towards the end of ... discomfort always pre-
of the digestion sent becomes acute
symptoms soon after meals.

Generally heartburn occurs only in sthenic form but it is not constant. With the above points in mind we can arrive tolerably at correct diagnosis if the patients are intelligent. Sometimes the patients do exaggerate their symptoms and put us in a dilemma. But we can arrive at it by the result of the treatment as one condition is diametrically opposite to the other. If for example we give HCl to the patient of sthenic type and thus increase the offending material, the patient may feel the pain more and this will set us in the right path.

Diet in Pulmonary Tuberculosis

BY

S. VERE PEARSON, M.D., M.R.C.P.,

AND

GEORGE DAY, M.D.,

Physicians, Mundesley Sanatorium, Norfolk.

THE science of dieting in tuberculosis is mostly empirical. Only in exceptional (i.e. experimental) circumstances can there be any controls. The one witness to the body-building power of food, namely the weighing-machine, is apt to prove deceptive. In some weeks a great number of the inmates of a sanatorium will gain weight prodigiously. In other weeks a majority will drop and the most careful analysis of the diet supplied during these periods does not reveal any causal factor. A patient undergoing pulmonary collapse therapy may lose weight continuously—although doing well in every other way—until he settles at a weight which will be his normal for some time to come. Then again, a synthetic diet, derived from the study of calorific values and the balance between proteins, carbohydrates, and fats is apt to lead to the conclusion that the ideal diet is one composed of a handful of lentils, two buttered dates and a raw carrot three times a day, a diet acceptable in theory to the laboratory worker, but not in practice to the patient.

Historical

Hippocrates (*circa* 400 B.C.) decided once and for all (apparently) that milk was beneficial "when there is no high fever." For the more acute cases he prescribed white wines, barley water and honey. Johannes de Mirfeld (*circa* 1400 A.D.) advanced the claims of river crab and sugar of roses. Both of these are sadly neglected nowadays. In fact, milk is the only relic of mediævalism extant. It appears to be an admirably balanced food for growing infants in a dilution suited to their relatively large stomachs. Bedridden adults requiring the minimum of 3,000 calories would have to accommodate $7\frac{1}{2}$ pints a day and

ambulant patients probably double that amount. Even with this bulk it is unlikely that they would increase their body-weight. Custom dies hard and it is still given more in cowardice than in hope as an "extra." Perhaps it has some catalytic action on other food-stuffs ingested, as patients who are tending to become too gross certainly lose this tendency when their daily pint of milk is cut off.

The first experiment on a large scale was made by the late Otto Walther of Nordrach who maintained that however ill a patient was, if he could eat and sleep, he would be able to get well. "Stuffing" was the order of that era. He taught his patients not to be fastidious about their food, but to eat whatever was put before them and to finish their meals however nauseating they were and however poor their appetites. In almost all cases he gave ordinary solid food and no choice of dishes. Milk was given nearly always with meals and nothing was allowed between meals; there were just three square—nay cubic meals a day. On occasion, if patients vomited, they had to start all over again. The principle was—stuff the food down and it will be digested all right. Such treatment was drastic. Its acceptance meant heavy going for the patients, particularly for those who were fastidious eaters or who suffered from indigestion. But certainly many patients gravely ill, even those with indigestion, were cured both of their indigestion and of their lung trouble. Nowadays this teaching has been modified almost out of recognition. On the other hand, the practice of resting before meals which also emanated originally from Walther has been followed in all the British sanatoria (but not so insistently on the continent); and it undoubtedly aids the tackling and assimilation of food. The body seems all the better able to receive the food and digest it, if a thorough rest has been taken before the meal.

Rationale of Modern Practice

Food, if it is to fulfil its various functions must be pleasant, varied and satisfying. It must also be adequate to maintain body-weight or to allow lost body-weight to be regained. Experience goes to show that ordinary full diet suits the average patient and various modifications of full diet are made to

suit the individual requirements of each patient. By full diet is meant:—

BREAKFAST (English variety) 8-30 A.M., i.e. fruit, cereal, fish and/or egg and bacon dish, coffee or tea, toast and marmalade or honey.

LUNCH 1-15 P.M.—A meat dish (with a reasonable choice), and two vegetables, sweet and cheese.

DINNER 7-0 P.M.—Soup, fish, meat, sweet, cheese and fresh fruit or savoury.

It is important that these dishes should consist of good food, attractively presented and with as much variety as possible. It must always be borne in mind that meals are the three important events in a bed-patient's day and that as he has done nothing whatever to deserve them and as he eats with a great attention to detail, they must be worthy of intense scrutiny. The old school tradition of—Sunday, sausages; Monday, fish cakes; Tuesday, eggs—would speedily bring trouble!

It goes without saying that the times of the meals should roughly coincide with those customary to the class of patient under treatment.

Afternoon tea at 3-45 P.M. is accompanied by the minimal amount of bread and butter or cake so as not to "spoil the dinner." It cannot be eliminated entirely as it is the one meal which patients can take with relatives or friends during afternoon visiting hours.

Common Mistakes

Indications for departure from full diet are rare. In regulating the diet in tuberculosis the three commonest mistakes are:—

(1) *Too great and prolonged restriction* (a) in cases with high fever, and (b) in cases of recent hæmoptysis. (a) With fever the appetite is rarely blunted and the digestion only slightly weakened, and when there is an abundance of cold, fresh air about the sick room—as there should be in the case of pulmonary tuberculosis—appetite as well as digestion may be improved considerably. There must be a sufficient amount of food to compensate for a rise of 7 per cent. in the basal metabolic rate with every increase of 1° F. in the temperature

of the body. Moreover, lost body-weight can be regained even while the patient is feverish. (b) It is generally unnecessary to curtail the diet appreciably in the case of hæmoptysis and it is quite wrong to keep a patient on short commons for days and days. Twenty-four hours on milk and ice-cream are usually sufficient.

(2) *Allowing the patient to overfeed.*—In these days when the importance of rest has been well emphasized and usually carried out properly, it is essential that when the patient is able to begin to get about he should not be too much above his normal body-weight. After he begins to exert himself and gently resumes his more normal activity he generally increases his body-weight further. If at first he is not only flabby but fat, then he is more liable to be short of breath, have a fast pulse and find it all the more difficult to get on to his feet. As Dr. Burton-Fanning pointed out years ago one cause of an unduly rapid pulse in a case of pulmonary tuberculosis, which is apparently getting on very well, is an undue prolongation of the administration of milk.

(3) *Neglect to see that the patient is taking enough fluid, especially when he is feverish.*—A close watch must be kept on the stomach. If a patient becomes bilious and has a furred tongue with offensive breath, lack of appetite, and perhaps a little diarrhoea it is often a good plan to use a day or two's starvation and increase the fluid intake, giving nothing but milk or ice-cream. In any case, any symptoms of this sort are best met in advance by seeing whether or not the patient is taking too much food of one sort or another.

Special Complaints

Dyspepsia is quite a common complaint among the tuberculous. An almost normal sequence of events is that a patient leaves an active existence to lie in bed when he comes to a sanatorium. In spite of the advice of his physicians, he has an almost ineradicable belief that it is his duty to stuff himself. He overeats and develops flatulent dyspepsia—with the fear that "the disease has spread to his stomach." He has to learn what to eat (i.e. what is necessary for his bodily require-

ment and no more) and how to eat it. Flatulent dyspepsia may be prevented by instructing him first and foremost to rest well before his meals, and secondly to eat dry—to drink freely between meals but not *at* meals. Heartburn at night is another most distressing symptom and may be avoided by progressively diminishing the amount of fluid taken later than the midday meal.

Extreme cachexia.—An emaciated patient will generally regain his body-weight at a reasonable speed (i.e. about $1\frac{1}{2}$ -4 lb. a month according to his size) if he is put on full diet. Occasionally there is such severe toxæmia that the whole alimentary system is sluggish. There may be achlorhydria or a catarrhal gastritis caused or enhanced by swallowed sputum. This is most often present in the early morning when 30 grains of bicarbonate of sodium in a tumbler of hot water or the Brompton Hospital "Hot Water Mixture" will often clear the way for a good breakfast.

Extras

Fats.—The full diet contains all the fats and fat-soluble vitamins necessary. Those who are unable to eat fat meat should have supplementary butter, milk, cod-liver oil, or cream. Cream should never be taken at the beginning of a meal with porridge or cereals as it tends to inhibit gastric secretion. It is better at the end of a meal.

Proteins are said to increase the respiratory activity more than fats or carbohydrates do. This seems to be an argument against excessive meat-eating, though there seems to be little doubt that purines are definitely beneficial for digestion, and that to begin a meal with a small helping of clear fragrant soup is the most elegant gastonomic prescription.

Carbohydrates in excess definitely cause an increased rate of body-building and with this increased rate the patient develops an increased resistance to his disease as shown by the sedimentation rate. The high caloric diet combined with insulin is the best method of increasing the carbohydrate intake. There is nothing like hyperinsulinism to endow the victim with the real famished feeling. Insulin injected in the correct

dosage and the injection timed to a nicety make the patient watch his door impatiently as the food-trolley rattles down the corridor. The practice at Mundesley is to give 60 units in the twenty-four hours in six injections of 10 units. Three of these are given between twenty and thirty minutes before the three main meals are served—twenty minutes is too short an interval for some patients and yet others in half an hour will suffer some of the profound disturbances from the induced hypoglycaemia. Three other injections are given at 11 A.M., 1-45 P.M., and 8-15 P.M., followed each time in half an hour by 6 oz. of fruit drink made of orange and lemon juice with sugar; ($\frac{1}{2}$ pint orange juice, lemon juice to taste, $\frac{3}{4}$ lb. granulated sugar, water up to $2\frac{1}{4}$ pints). The full diet is necessarily modified in respect of carbohydrates and the resulting diet is the ordinary high calorific diet based upon the researches of H. Himsworth of University College Hospital.

In the *high calorific diet* :—

Breakfast is the normal English breakfast, *lunch* must include stewed dried fruit or banana with sugar and a cereal made with milk. At *afternoon tea* the patient is given an extra allowance of bread and butter and jam. At *supper* the soup is thickened with flour or milk and extra bread is given. The calorific value of this diet is about 4,000.

The results of this regime are astonishing. Appended are two weight-cum-sedimentation rate charts showing how the process of regaining weight which had been progressing very slowly was accelerated by the insulin regime.

Vitamins

The full diet has adequate vitamins for the normal healthy man but some doubt is expressed as to whether the tuberculous patient does not require more vitamin C. The researches of Abbasy, Harris and Ellman (1937) show that the patient with active tuberculosis requires to take much more anti-scorbutic vitamins before it can be detected in his urine; and Radford de Savitsch and Sweeney of America (1937) showed that patients saturated with the ascorbic acid make more rapid progress in every way for at least the first six months of treat-

ment than other controls. It is true that in this series the controls caught up the subjects in nine months, but the fact that the rate of progress was accelerated for the earlier months suggests that it is worth while to give patients with active disease the juice of two oranges a day or its equivalent of ascorbic acid.

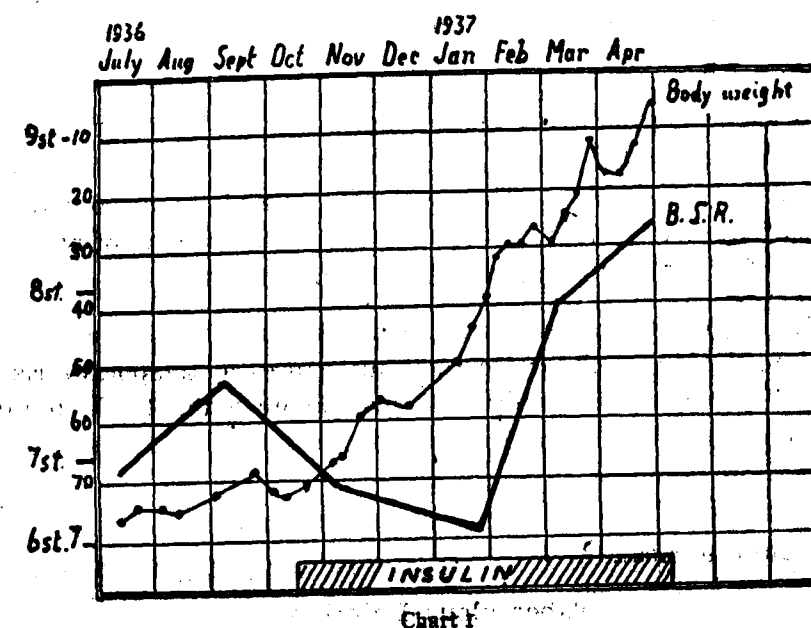


Chart 1

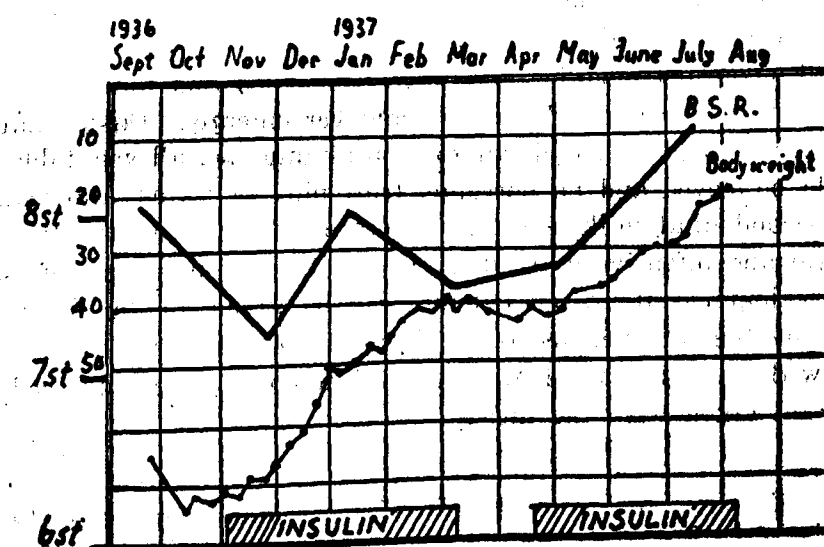


Chart 2

Stimulants

There are various methods of stimulating patients to enjoy their ordinary diet: i.e. light ales or stout served with the meal or occasionally, as "elevenses" and sometimes a lemonade made with acid. nitro-hydrochloric. dil. with lemon or orange flavour in a tumbler of water. Certain insipid egg dishes can be made savoury and appetizing by the addition of bovril or marmite.

Alcohol.—As indicated above, light wines, beers or stouts may occasionally be useful, but heavy wines or spirits should be completely forbidden. The euphoria caused in a convalescent patient invites him to depend upon them instead of accepting the feeling of tiredness and resting.

Smoking after meals is said to assist digestion and is therefore a good thing. Cigarette smoking causes a temporary rise in blood sugar and if indulged in just before a meal will blunt the appetite—a fact used some years ago in the advertisements of a certain brand of cigarette when the *slimming craze* was at its height. Therefore there should be no smoking for an hour before a meal.

Tuberculous, Enteritis

There is much controversy about the correct diet to be given when tuberculous enteritis is diagnosed—and no wonder—for all suggested modifications have been tried and found more or less disappointing. Two facts however emerge: First, that roughage should be cut down to a minimum (i.e. all vegetables should be sieved and no porridge, fruit pips or skins allowed), and second, that milk is rarely tolerated, being apt to cause painful and offensive diarrhoea. It is often given because of its high calcium content, but calcium can be given in far larger doses by the intravenous route. The diet is best kept full and dry with the addition of an ounce of tomato juice and cod-liver oil taken with the meals.

Editorial

Tuberculosis—Today's problem

EVERYWHERE supporters and organisers are doing everything to swell the funds of the Anti-Tuberculosis campaign. Not all the millions realized will help to solve our present-day problem. For we need thousands of hospitals and sanatoria, heavily staffed with specialists and specially trained workers. The cost of proper dieting will be many times heavier than that in the ordinary 'general' hospital.

So far, of all the armamentaria at our disposal, general measures to improve diet and immunity, calcium therapy and artificial pneumothorax are the only constantly successful ones.

Present-day economic problems do not permit of these lines being adopted, except in a very few cases. Limited incomes; long absence from work without pay, or the fear of losing work, result in many thousands 'carrying on' till they are prostrated and in the ravages of the disease.

Among the other few who can be treated, many have to be turned away for the want of accommodation in clinics which are pathetically few, in India. The outcome of this is that the infected thousands are allowed to mix freely and intimately with their fellows whom they soon infect.

The problem is essentially an economic one.

Better wages; better leave facilities to those infected and provision of special clinics and sanatoria, are the only solution. The solution is difficult and in our generation we can but expect a minimum realization. However, work along these lines must and will solve the greater part of this problem. The prevention of tuberculosis is intimately linked up with mal-nutrition; and when one counts up the many different clinical manifestations of the condition, one is led to label it a 'deficiency' disease. Its insidiousness, its long course, its varying signs and symptoms all point to a slow weakening of the host, instead of an increasing virulence of the parasite.

Only a correctly nourished organism can withstand the attack of the tubercle bacillus.

Today we are faced with two problems. The prevention of tuberculosis; the prevention of the spread of tuberculosis. Of these the greater problem to tackle is the prevention of the spread of tuberculosis. Its answer is segregation of those infected, and their treatment. The prevention of tuberculosis is the quest of the research worker in nutrition. The final answer to this latter is still a long way. But today we can snap the cycle of malignant spread of the disease. It calls for an awakened interest on the part of the doctor, the patient and the state. All three must realize the menace of allowing the infected person to go about free and so, easily infect others. Employers should realize how necessary it is that facilities for treatment and reasonable absence from work, should be provided; and further appreciate that tuberculosis is also the outcome of occupation and the conditions of living in industrialized areas.

And doctors must be prepared to concentrate on improving the general health of the patient and not be easily led by numerous therapeutic agents that are the vogue. It must be realized that the only satisfactory places to treat such cases are in isolated special Tuberculosis Sanatoria and finally like in all our present-day movements education of the public on these lines is the most important factor.

The Trichinopoly District Medical Association

Minutes of the Meeting of the General Body of the above Association, held at 5 p. m. on Saturday the 23rd July 1938, at the Central Hospital, Golden Rock.

A Meeting of the General Body of the above Association was held at 5 P.M. on Saturday the 23rd July 1938 at the Central Hospital, Golden Rock, when about 30 doctors were present. Dr. C. E. R. Norman, Chief Medical Officer, S. I. Ry., and President of the Association, presided and was At Home to the Members.

After the President's preliminary remarks the Secretary read the minutes of the previous meeting.

The following resolutions that were brought forward by Dr. T. S. Balasubramaniam, M.B., B.S., Trichy, were considered.—

I. This Association while generally approving of the principles of the scheme of Honorary Medical Service in the State Hospitals recently introduced by the present Ministry of Public Health, Madras, makes the following suggestions:—

- (a) The rule limiting the period of the Honorary Medical Officers to five years, should be enforced, so that the Juniors in the profession may have opportunities for those appointments.
- (b) In the Specialists' Departments a junior should also be appointed as junior specialist to work along with the Senior Honorary, their periods not being conterminous, so that the Junior may be well trained in the particular department of the particular Hospital, and step into the place of the Senior Specialist as soon as the latter vacates the office after his five years and thus keep up the efficiency and continuity in that work in that hospital.

II. This Association strongly disapproves of the scale of remuneration for the Honorary Medical Officers as being too low and highly derogatory to the dignity of our profession, and urges upon the Ministry to increase the minimum to Rs. 50/- or delete the provision of remuneration altogether.

After elaborate discussion it was proposed by the President to appoint a sub-committee for discussing the same; and it was seconded by Dr. T. S. Balasubramania Iyer himself, and was unanimously approved by all the members present.

For the discussion of the above resolutions the following were elected as sub-committee Members:—

1. Dr. C. E. R. Norman was proposed and unanimously seconded to be the Ex-Officio President.
2. Dr. T. N. Subramaniam was proposed by the President.
3. Dr. K. G. Menon proposed by Dr. Ramaswamy Nadar.
4. Dr. T. S. Balasubramania Iyer.

The aforesaid sub-committee was framed for discussing the resolutions of Dr. T. S. Balasubramaniam and was unanimously accepted by the members present that the above resolutions may be brought out for the next General Body Meeting.

The following Clinical Cases were demonstrated:—

- I. Neuritis ... by the President.
- II. Ulcerative Stomatitis .. by Dr. C. S. Sundaram.
- III. Transverse Myelitis ... by Dr. R. Kalameghum.
- IV. Acute Appendicitis ... by Dr. Ananthanarayana Iyer in which Drs. T. N. Subramaniam, M. A. Krishna Iyer, K. G. Menon and others also participated.

After the President's concluding remarks the meeting terminated at a late hour, with a vote of thanks to the Chair and the Lecturers.

O. R. BALU,
Secretary.

The Trichinopoly District Medical Association TRICHINOPOLY

Statement of Accounts for August 1938

RECEIPTS			PAYMENTS		
	Rs.	A. P.		Rs.	A. P.
Cash Balance ...	42	4 6	Clerk's pay up to 15th August '38 ...	20	0 0
Bank Balance ...	208	2 2	Extra clerk employed during absence of old clerk ..	5	0 0
Subscriptions from Members ...	63	0 0	Hire of Office for July '38 ...	5	0 0
Subscriptions from Advertisements ...	170	2 0	Miscellany printing in part payment ...	200	0 0
Security deposit by Clerk ...	50	0 0	Stationery ...	2	6 0
Adjustment deposit by Treasurer ...	7	4 0	Printing notices ...	10	4 0
			Typewriter repairs ...	3	4 0
			Cycle repairs ...	1	5 6
			Postage ...	10	15 9
			Baksheesh to peon for G. B. Meeting ...	1	0 0
			Paid to Treasurer in adjustment ...	7	4 0
			Bank balance at the end of the month (including Rs. 50 security deposit) ...	210	4 2
			Cash balance ...	63	1 3
TOTAL ...	540	12 8		540	12 8

M. C. CONDILLAC,
Hon. Treasurer.

31st August 1938.

Excerpts

The treatment of Post-operative retention of Urine

THESE are various causes of bladder dysfunction following operation among which nervous factors, disturbance of the bladder musculature, alteration of the position of the bladder itself and psychic factors must be considered. There are many arguments against the use of catheterisation and for this reason other measures in the form of hot baths and local applications and various injections have been employed. H. S. Barber discusses the different drugs available for the treatment of post-operative retention. Urotropine had a considerable vogue as a remedy for retention and usually a single injection produces a satisfactory result; but unfortunately unpleasant secondary reactions in the form of abdominal pain, headache, vomiting and tinnitus. For this reason a modified preparation called cytotropin, a combination of Urotropine, Salicylates and caffeine has been found more certain in its results and this preparation can be injected intramuscularly if necessary. Another useful diuretic compound is sold under the name Esmodil and has a direct action on the vesicular musculature. 1 c.c. of a 0.3% solution is given intramuscularly and is followed by increased movement of the alimentary canal, Salivation, perspiration and desire to micturate. The tonic contraction of the bladder lasts for several minutes and there is a complete absence of complications. Should spontaneous micturation not occur in 30 minutes, a second injection of half of the original amount is given. Intravenous administration causes pain and is not recommended. A diuretic drug with a powerful stimulative action is mecholyl given subcutaneously in doses of 5 miligrams of a water-soluble compound closely resembling acetylcholine when given by intramuscular or subcutaneous route usually produces the result in 15 minutes. Some secondary reactions however in the form of nausea, faintness, fall in the blood pressure and vomiting may occur.

From the Irish Journal of Medical Science, February 1938.

Newer Drugs for the treatment of Tape Worms

Successful results have been obtained by the use of Carbon-tetrachloride for the expulsion of *T. Solium* and *T. Saginata* and its action as well as those of tetrachloride Ethylene and Hexyl Resorcinol has been tested by J. S. Sandground. Patients suffering from infestation with *T. Saginata* and *T. Lata* were given doses of Carbon tetrachloride.....4 c.c. in well-nourished adults, 3 c.c. in depleted patients and 2 c.c. in children. The drug was given either in water or in a little milk and was well tolerated although some slight dizziness and drowsiness occurs during the first hour. The first thing in the morning is the best time for administration, in 12 of the 16 cases treated effective elimination of parasite was procured. Some warning should be given concerning dosages as deaths have been reported occurring after too severe purgation with this drug. Also patients suffering from kidney disease, chronic or acute cyanosis are contra indicated. When round worms are also present the drug should be mixed with oil of chenopodium and the blood calcium reserve and the carbohydrate medication should be increased for a few days before the administration to avoid any possible injuries to the liver. Tetrachlor Ethylene in doses of 4 c.c. given in the same way as Carbon-tetrachloride has the lower toxicity. But the results are not so constant. Hexyl Resorcinol administered in the form of 1 grain sugar-coated pills in the quantities ranging from 1 to 6 in divided doses proved still less satisfactory. The main portion of tape worm stropilla was expelled but the scole may remain viable although no signs may be visible in the faeces for several months owing to their less toxic action; however the two later drugs may be prescribed for patients in a debilitated condition or those for whom carbon tetrachloride is contra indicated.

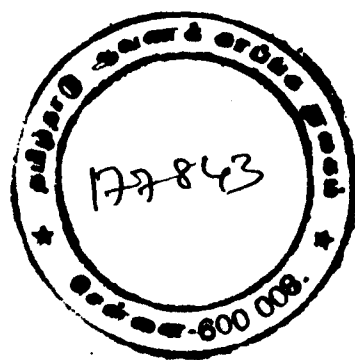
From New English Journal of Medicine, February 17, '38.

Sulphanilamide for Eczema

J. Hirshnamn writes in the Practitioner, May '38, as follows:—

I have come to believe that Eczemas are complicated by streptococcal infection when they are seen by the practitioner

or in the hospital. Whatever the primary cause the streptococcal infection is of such virulence that it swamps the first cause and leaves an almost pure streptococcal infection to be dealt with. Some time ago I began a series of experiments with streptococcal sera of various kinds. The results were disappointing and I turned towards sulphanilamide. The result in these are truly pleasing and I felt that here at last is a specific treatment for Eczema. After the first injection the irritation and weeping disappeared and the healing and local treatment were greatly helped. I tried various brands of sulphanilamide but I came to prefer red prontosil due to what appeared to be the psychological effect of this. As urine was red when voided after the treatment the patient felt or rather saw the "the poison" passing from the system, Varicose ulcers also healed quicker but here the great boon of the treatment is the cessation of the irritation. The legs can, as a result, be bandaged for longer periods and treatment shortened.



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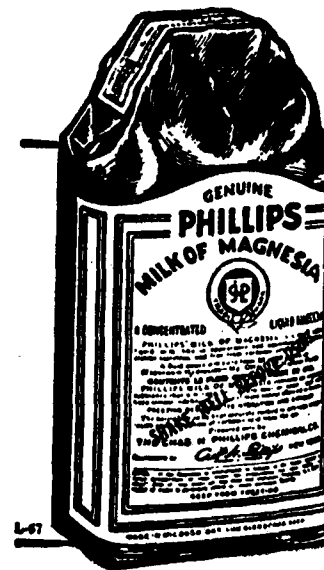
TO COUNTERACT *Acidity*

PHYSICIANS have of late years come to recognize more and more that many ailments such as headaches, dizziness, biliousness, indigestion, are caused by an acid condition in the stomach.

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Tablets bottles of 50 × 5 grs. each As. 10.

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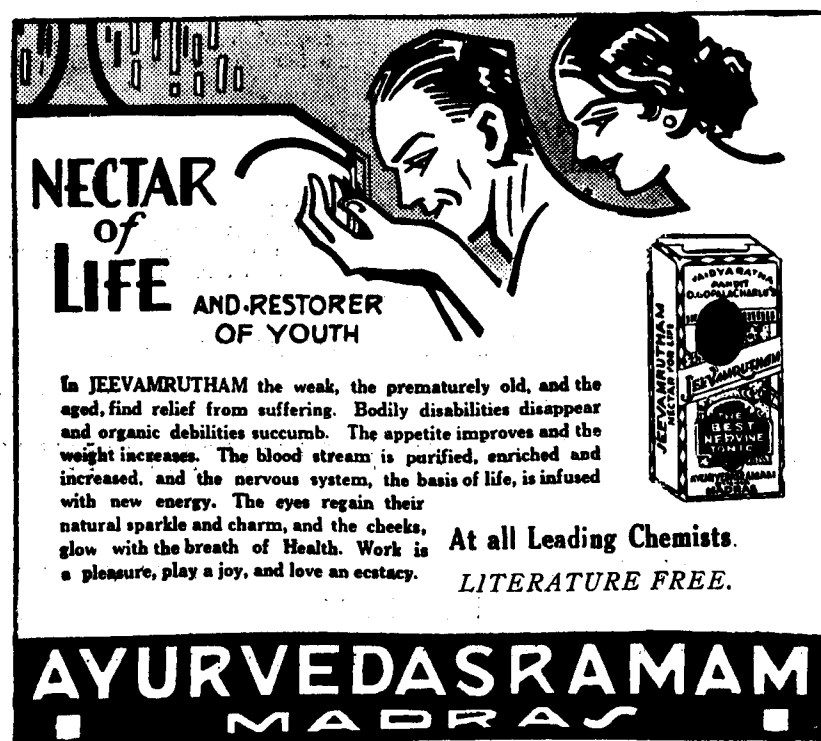
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