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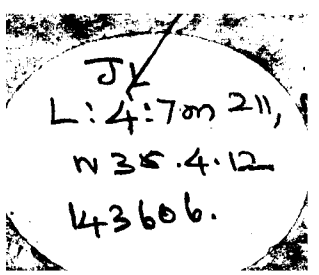
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CONTENTS

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Special Article

MODERN DIAGNOSTICS AND TREATMENT OF CICATRISED OESOPHAGEAL STENOSIS

BY R. DEMEL

A report from the Second Surgical Department, Rudolfspital, Vienna.
Director: Prof. R. Demel.

With cicatrised oesophageal stenosis we generally succeed in relief of the patients' troubles by means of sounds used for dilatation. There are, however, cases of intense or impenetrable stenosis, when we have to proceed to a complicated and dangerous ante-thoracic plastic surgery of the oesophagus. I need not enter into particulars, successful results of ante-thoracic plastic surgery of oesophagus being well known. A great many of the patients have been cured of their trouble by this operation.

Due to progress in diagnostics a considerable change in the treatment of intense cicatrised stenosis has taken place of late. The consequence is that in most of the cases we can desist from ante-thoracic plastic surgery, in removing stenosis by another and less dangerous method. This procedure being insufficiently known even in Europe—as far as I can see from literature—while, on the other hand I presuppose sufficient cases of the like available in India, I wish to call people's attention to it.

The salient point for undertaking this new direction of treatment concerns the distinction in a case of either penetrable or really impenetrable oesophageal stenosis. For a final judgment methods of investigation have been employed as follows :

The oldest method consisted in introducing a bougie, whether a cylindrical, or an English, or a filiform one, or may be in using catgut strings for the purpose of determining the seat and intensity of stenosis. Since X-rays have been applied the patients were made to drink a liquid casting a shadow by which method a thorough

explanation about the character of the narrowing was obtained. A chemical method was also applied in order to test the stage of permeability in the narrowing.

To that purpose and as suggested by LOTHEISSEN the patient had to swallow a very small quantity (a tea-spoonful) of a 2·5 per cent solution of lactate of iron. Fifteen minutes after, the gastric juice was drawn off from the gastrostomy tube into a test-tube and diluted with a solution of ferro-cyanide of potash. (With all those patients gastrostomy was done before hand on account of intense stenosis.) If on account of stenosis the slightest quantity, one drop, of lactate of iron has passed into the stomach, intensive colouration of blue will occur in the dilution of the gastric juice with the ferrocyanide of potash. (Berlin blue reaction). This is a very sensitive reaction.

Ewald has indicated another chemical method consisting in a strong solution of iodide of sodium, a small quantity of which is administered to the patient. Fifteen minutes after that a rinsing of the stomach takes place by means of the gastrostomy tube, the fluid of which is diluted with fuming nitric acid and shaken with chloroform. If on account of oesophageal stenosis the slightest trace of iodine has passed into the stomach the colour of the rinsings turns purple-red.

There is a difference in efficiency of these 3 methods. Oesophageal sounding fails pretty often, as with intense stenosis one seldom can reach into the stomach. X-ray examination works out much more accurate. Experience, however, has given evidence that it is not altogether reliable. It occurs pretty often that on the basis of an X-ray examination stenosis is mistaken for being impenetrable while, in the end it proved to be penetrable. Such errors have become scarcer since chemical tests have been applied, being far superior in sensitivity to X-ray findings. Still, in the valuation of chemical tests too, we had to relearn lately as cases were observed when stenosis found impenetrable by the help of chemical tests later on was known as being penetrable. Until some few years back the chemical test passed for being the most sensitive process for a clear distinction between either penetrable or impenetrable stenosis. There were, however, a sufficiently large number of cases of stenosis which on the basis of a chemical test were considered impenetrable. In such cases all we could do was to suggest to the patient some method of plastic surgery of oesophagus though it is technically not simple and altogether serious. The patient had to submit to several operations and was confined to the hospital for a couple of months.

For that reason it is highly interesting to learn that *in the electrolytic sound* we have met with a method by which in recent years we have successfully determined permeability in most of such cases

of oesophageal stenosis which hitherto had passed for impermeable. At this rate the number of cases of really impenetrable stenosis was lowered to a minimum.

Electrolytic procedure was first applied successfully in the year 1884 by E. BOECKEL of Strassburg, later on by J. A. FORT and STROEN. In Germany JENCKELS was the first man to use electrolysis for that purpose in 1912. In Vienna it was LOTHEISSEN who introduced that method.

Electrolysis is an outstanding method in order to attain permeability in impermeable stenosis. It requires an electrolytic sound consisting of a piece of copper-wire of about 1 meter's length (40 inches) isolated outwards, at the end of which there is a little screw-thread for screwing up different metal olives. The sound is connected to the negative pole of a constant electric current, while the positive electrode is being applied to the patient dorsally.

The site of oesophageal stenosis is observed in the oesophagoscope and the electrolytic sound is introduced as far as to the narrowing with a slight pressure at the place of the cicatrised constriction. Then a constant electric current of 2—5 milliamperes is turned on which is to operate for no longer than 5 minutes in order to prevent effects of caustics or injury through electrolysis. The desired effect of electrolysis should be merely to produce hyperaemia and moistening of the cicatrix in order to ease the sounding.

In case of a continued slight pressure against the cicatrised constriction of stenosis during the exposure one suddenly gets the impression of an obstacle giving in so much as to allow the sound to advance into the depth without any force used. If you are very fortunate you may experience this effect at the very first treatment, thus in cases of a short narrowing the sound may be advanced way down into the stomach. With more severe cases one must repeat the treatment after 5 to 7 days in order to prevent burning of the tissue.

When we have succeeded in pushing down the sound into the stomach we may get out the end of the sound by way of the aperture of gastrostomy and connect a permanent sounding. To this end a thick silk thread is fastened to the sound, which along with the thread is pulled up in a retrograde movement leaving the silk thread to pass through the oesophagus, with its ends sticking outside of the mouth and of the aperture of gastrostomy. By means of a conical, elastic bougie attached to both ends of the silk thread alternately, sounding and dilatation of stenosis takes place step by step to attain the width required of oesophagus.

With the aid of electrolytic procedure I have been treating 5 cases successfully during the last few years. I wish to briefly enter into particulars about two sample cases only, in order to emphasize the efficiency of this method.

(1) A woman, age 30, a sempstress, drank a solution of caustic soda 12 years ago. She avoided appropriate treatment, and attended the hospital only for some time later, when she revealed impenetrable stenosis even for liquids. Gastrostomy was performed. In the course of these 12 years the gastrostomy tube had to be exchanged several times, its diameter finally amounting to 5 cm. (2 inches). The patient had taken her meals by the way of chewing the solid food and expectorating it forth into the gastrostomy tube. At the end of these 12 years she went to see the clinic for the first time with a view to undergo an operation. X-ray test as well as a chemical test had revealed impenetrable oesophageal stenosis at the level of the 6th thoracic vertebra. Preliminarily to resolving on ante-thoracic plastic surgery of oesophagus the electrolytic procedure was applied for the purpose of ascertaining the impenetrability of the narrowing. When the electrolytic sound had been applied for 5 minutes with an electric current of 3 milliamperes, a sudden relaxation in the resistence of the tissue at the place of cicatrised stenosis was noticed; whereupon the sound could easily be let down by 10 centimeters (4 inches). After that the sound was extracted. As there was no certainty as to whether the sound had reached the stomach, another exposure was planned to be held a week after. It was surprising all the same when the patient, after a week from the first treatment with the electrolytic sound, admitted that for the first time in 12 years she had been able to take water and milk by mouth. She was then made to swallow a grain of shot which after having dropped into the stomach, was pulled out along with the silk thread by the way of the aperture of gastrostomy. Permanent bougie treatment was started. When four months had elapsed the patient was able to introduce bougie No. 22 herself and take food by mouth. One year afterwards gastrostomy was healed up. For all that time, that is to say, for these last 7 years, the patient has been in perfect good health and has taken any kind of food normally.

Hence it was by means of the electrolytic method only that in this case oesophageal stenosis considered impenetrable for 12 years was diagnosed as penetrable and removed for ever by a conservative way of treatment (bougie).

(2) A workwoman, age 34, drank a solution of caustic soda six weeks ago. In spite of using bougie stenosis, developed, so gastrostomy was performed. X-ray examination revealed an interruption of oesophagus at the place of bifurcation. In the attempt to make her swallow a grain of shot along with a silk thread the grain was caught in the narrowing. The chemical test also turned out in the negative. In a similar way as it was done in the first case, with the oesophagus grounded the electrolytic sound was applied preliminarily to decidedly accounting for impenetrable stenosis. After connecting a constant current of 3 milliamperes and after a

permanent exposure of only 4 minutes the sound was carried through the stricture into the stomach. With this case also a permanent bougie was connected and when 3 months had passed the patient could introduce the sound No. 24 herself and was able to take any food in a normal way. Gastrostomy likewise was healed up one year later. For these four years the patient has been entirely well and has taken food like any person in good health. In this case likewise it was the electrolytic method which revealed penetrability of stenosis and prevented ante-thoracic plastic surgery of the oesophagus.

These two cases are to be used as examples which display the great advantage of that method of examination. Observations as those two once referred to must necessarily excite suspicion as to a large part of plastic surgery of oesophagus done successfully in different countries and made publicly known. Such operations could have been avoided if people had only used beforehand all methods of examination yet most and foremost the method of the electrolytic sound. That this is a rightful presumption we may derive from a verbal communication by LOTHEISSEN according to which he had not met with any possibility of carrying out ante-thoracic plastic surgery of œsophagus in cicatriced œsophageal stenosis for many years. We made the same observation. We may consider that LOTHEISSEN does have a great deal of material available in Vienna.

We further learn from these observations that we have to be most careful in diagnosing impenetrable œsophageal stricture, realising the scarcity of real and permanent impenetrability of stenosis.

In our patients' own interest we are bound to ask for application of all methods of examination in any cases of œsophageal stenosis. Electrolytic procedure should not be left out as in most of the cases we may save our patients the trouble of the complicated and rather dangerous plastic surgery of œsophagus.

Attending to this request and with increasing knowledge and use of these methods of examination I feel thoroughly convinced the number of serious operations will still diminish which is all in our patients' favour.

The electric sound is to be had from: H. Reiner, Wien, IX. Van Swietengasse 10, at a price of about 23 Austrian shillings.

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Selected Articles

CONGESTIVE HEART FAILURE

BY CLOUGH TURRILL BURNETT, M.D.
Denver, Colorado, U. S. A.

Of functional disorders of the heart angina pectoris and congestive heart failure are of chief importance, the former more dramatic in its manifestations, the latter more certain as to the ultimate outcome. Both are due to functional insufficiency, the former of the coronary circulation, the latter of the myocardium.

Failure on the part of the heart to maintain an adequate circulation represents a potential congestive heart failure. This may be so slight as to pass unnoticed by the patient, or may occur with the suddenness of an anginal attack. Between these two extremes lie the chronic progressive failure associated with valvular disease, hypertensive heart disease and luetic heart failure. While the causal factor of any of these cannot be removed, the early recognition of congestive failure will do much toward retarding downward progress through adequate management.

Three types of circulatory failure: I. Peripheral failure—failure of fluid to heart, shock, toxic states etc., II. failure of rhythmic mechanism—Stokes-Adams, venticular asystole, vagus effects, ventricular slowing and fainting, auricular fibrillation and auricular flutter; III. failure of heart muscle: (*a*) Congestive, (*b*) anginal, and (*c*) paroxysmal—cardiac asthma, acute pulmonary edema, etc.

Clinically acute heart failure falls into three general groups: I. Those in which the auricles are primarily involved, as in chronic rheumatic mitral disease and thyrotoxicosis. In these cases total arrhythmia is associated with dyspnoea, cyanosis and usually edema; II. the cases in which the conduction system between the auricles and the ventricles is disturbed, leading to attacks of bradycardia and syncope; these are usually found to be cardiosclerotics; III. the cases in which the failure is primarily of the ventricles. These may be cases of valvular heart disease, either rheumatic or syphilitic, but frequently are associated with hypertension or coronary disease, or both. Pain, dyspnoea—often nocturnal—frequently pallor, lowered pulse pressure and collapse are the striking features; and in contradistinction to the rheumatic valvular cases, the rhythm is usually normal. Muscle failure occurs in acute infections and is nearly always ventricular in type. Diphtheria offers an example of this type of failure. The pulse pressure drops alarmingly, due in part to ventricular failure and in part to capillary paralysis. The

effect of edema on the utilization of oxygen by the tissues and the resultant metabolic disturbances are peripheral factors of importance in certain instances.

It is the group of cases associated with circulatory failure with which I am especially concerned at this moment. To these the term decompensation has been applied but congestive heart failure is a better descriptive term, for, according to Wenckebach, under certain circumstances the cardiac output of the failing heart—the heart showing evidence of congestive changes—may be normal or even increased for a time, yet coincident with this normal or increased output congestive changes occur, evidenced by slight breathlessness to profound engorgement of the venous system.

Mechanism of congestive heart failure: In heart disease one chamber or side of the heart is involved to a greater extent than the other with rare exceptions. For example, in the earliest form of heart disease—congential heart disease—various types of developmental defects throw an unusual strain upon one side of the heart, right or left. This strain may be for a time entirely borne by one chamber, the auricle or ventricle, but usually the effect is noted both proximal and distal to the lesion. If the compensatory process is adequate the circulation is maintained in spite of the defect—a condition which occurs frequently in congenital hearts, and unless infection causes an added insult to the heart, through endocardial infections or myocardial damage, this compensatory stage may continue for many years—in fact, through the normal life span; but should there occur an excess load through any cause, as infection, strain or anything which furnishes a demand in excess of that of which the myocardium is capable, there at once appears a different picture—congestive heart failure.

The next group of cases in order of age occurrence is the large one of rheumatic valvular heart disease. Here mitral lesions occupy the place of prominence, with the stenotic factor the most important. Pure mitral stenosis is a rare pathological finding, but in the combined lesion of mitral stenosis and mitral insufficiency the former is by far of the greater importance in the production of myocardial strain and ultimate failure. For a time the left auricle carries the load, but sooner or later this overstrain is reflected to the pulmonary circulation, and we have the first manifestation of congestive heart failure. This stage may be long or short but always the right ventricle receives an added load. If the patient be spared physical strain and infection, especially pulmonary, the ventricle may undergo considerable hypertrophy so that the evidence of circulatory failure is limited to signs of pulmonary vessel stasis; but if the coronary circulation is inadequate hypertrophy fails to develop, or if it has developed it may prove inadequate to the needs of advancing stenosis and a general systemic stasis develops—the last stage in

this progressive process. I have discussed only one example of rheumatic valvulitis. Should the aortic valve be the seat of the disease the resulting insufficiency will produce its effect on the left ventricle; however, the course is the same as with initial disease. With complete compensatory increase in the size and efficiency of the affected chamber the heart carries on as though naught had occured; but, failing in this, somewhere the circulation shows a deficiency. If distal to the lesion, the signs will be those of malnourishment of tissues; if proximal, those of blood stasis. This then is in general the mechanism of congestive heart failure. It may appear in the forties with no antecedent history of heart disease. A recognized or unrecognised early syphilis may solve the riddle. At this age, likewise, the effects of hypertension become manifest in the heart; first hypertrophy and compensation, but with continued overload a final stage of dilatation and failure.

A last group we have to consider the cardiosclerotics. In these the earliest manifestations of cardiac insufficiency are usually those of coronary artery narrowing, either slow and progressive, or the spastic type so frequently associated with the anginal syndrome. In these, circulatory stasis is not a striking feature; in fact, it may not occur at all or late.

Failure of the auricles plays a limited role in congestive heart failure, although the usually associated auricular fibrillation furnishes an added source of embarassment to the already overtaxed ventricles; likewise, failure of one ventricle taxes the functional capacity of the other ventricle. In hypertensive heart disease, following the initial stage of hypertrophy of the left ventricle when the increasing load becomes too great for this chamber, dilatation occurs and, with this, relative mitral insufficiency. The left ventricle fails to pump the blood onward which comes from a still competent right ventricle, and the left auricle dilates. In time pulmonary vessel engorgement becomes so great that the right ventricle in turn dilates, and the last stage of congestive heart failure appears. Common precipitating causes of congestive heart failure are any sort of strain—at times that of strenuous effort—the changing of an automobile tire, a sudden sprint in one not accustomed, a too strenuous golf game. Sometimes it may accompany a trivial physical strain, a slight respiratory infection, emotional strain or an unwise meal. These may appear to be sole causes in certain cases, but a careful analysis of all factors will usually show a heart already seriously damaged by valvular heart disease, by long standing hypertension, or by coronary artery disease. Other common basic causes are chronic pulmonary disease and thyrotoxicosis. With the latter frequently are associated rapid heart action due to auricular fibrillation and flutter, and paroxysmal tachycardia in which fatigue is an important factor. While all of these more frequently cause the

primary failure on the left side, recent studies show the importance of pulmonary tissue changes and pulmonary sclerosis on the integrity of the right ventricle and the not infrequent failure of this chamber before there is any appreciable change in the left heart. Continental writers, and recently Coombs, have called attention to the real danger of cardiac failure in angular deformities of the spine through aortic kinking, inadequate pulmonary air spaces, and diaphragmatic restriction.

Age plays an important part in this process. Failure occurs in childhood, though infrequently, and then usually due to an extensive pan-carditis. More often even in the badly damaged hearts this is postponed until the sixth decade when coronary artery changes limit the blood supply to the overtaxed ventricles. It is questionable whether the undamaged heart may ever fail due to strain alone, but long continued functional tachycardia may so fatigue the normal muscle that it finally fails.

Pathology: White calls attention to the unfortunate trend of modern teaching to overemphasize the importance of the myocardium and to underestimate the importance of the valvular lesion. The essential factor, however, remains the diminution in the systolic power of the myocardium. There is no pathognomonic lesion in congestive heart failure. While enlargement, due to both hypertrophy and dilatation, is usually found, this is by no means the rule. According to Clawson "approximately half of the cases of myocardial failure show no anatomical changes in the heart muscle." These cases he explains as due to an exhaustion of the heart muscle. In this group fall the cases of myxedema heart and the heart in beriberi, both functional myocardial disturbances and both reversible by simple treatment; the former by thyroid extract, the latter by vitamin B. Harrison and his co-workers state that the thickness of the muscle fibers and the associated heart rate are of importance; the subjects with enlarged hearts have rates much faster than optimal and fatigue may result therefrom. While these hearts show hypertrophy and dilatation, there may be no other evidence of myocardial abnormality sequential to congestive failure. Two years prior to the reports of Blumgart these authors suggested that a subnormal heart rate would be beneficial to "cardiac fatigue" and thus suggested the basis of total thyroidectomy in congestive failure. The remote effects of congestive heart failure on other organs are those of local circulatory insufficiency—*oedema*—which if transient may cause no permanent damage, but if long continued compression changes and fibrosis may follow.

Another factor not sufficiently recognized in congestive failure is the venopressure mechanism that pushes the blood back through the veins to the heart. It is this that often fails and precipitates congestive heart failure. Y. Henderson has shown that if in a

rabbit the pulmonary artery is clamped and the vena cava opened, 50 per cent of the blood of the body will flow out, showing that there is some other pressure than the heart behind the venous return. Henderson after 25 years' search for the cause of this venous push concludes that it is the tonus of the skeletal muscles, particularly those of the abdomen and diaphragm, and that this tonus is especially influenced by the state of the respiration. If respiration is depressed, the diaphragm is relaxed, lung capacity is decreased, skeletal muscle tonus is reduced, and circulation volume is decreased. Strychnine was widely used by the past generation of doctors as a circulatory stimulant. It is probable that it increases muscle tonus and thus increases the efficiency of the venopressor mechanism.

Symptoms of Congestive Heart Failure: The earliest and most lasting symptom is dyspnea. This may develop so insidiously that the patient is not aware of it, and only after a severe break does he realize that accustomed effort has for a long time caused undue fatigue and some respiratory distress. It may occur suddenly, as in the acute cardiac asthma of advancing years, and may or may not be associated with precordial pain. As these attacks of cardiac asthma become more frequent, Cheyne-Stokes respiration appears. In extreme cases of complete failure of this type may occur the symptoms and signs of acute pulmonary edema. In all of these the cause is oxygen want in the respiratory center due to increased cerebral venous pressure which diminishes the blood flow, leading to anoxemia of the respiratory center; oxygen therapy is beneficial.

These are the cases described by Allbutt as "ventricular defeat"—the cardiac asthmatic with hyperpiesis who suffers nocturnal attacks following exertion of the day before. Adrenalin (or ephedrine) often relieves these by dilating the coronaries—a fact not well recognized.

Acute pulmonary edema, a less frequent syndrome, is likewise usually nocturnal and always of grave prognosis; death frequently occurs in the attack. In these cases the left ventricle suddenly fails while the right continues to function with a disproportionate force, the result being that since the left heart fails to force the blood onward, there is rapidly increasing engorgement of the pulmonary circuit. While pain is not a part of the picture, excepting when this is associated with coronary thrombosis, the seizure is no less dramatic than in coronary thrombosis or angina pectoris. Allbutt, describing such an attack wrote ". . . Someone exclaimed, 'Angina pectoris.' Thoracic agony it was indeed, but dyspnea is not a character of angina pectoris."

Sudden or paroxysmal right heart failure is a rare event, one-fourth as frequent as left ventricular failure excepting as a sequel of thrombosis of the right coronary artery or of an acute pneumonia; but slow progressive failure of the right heart is a common event in rheumatic heart disease and is characterized by its chronicity and

tendency to occur in bouts from which the patient makes a partial recovery and may even live for several years. Contrast this with the failure of the left heart in syphilitic or hypertensive heart disease. Congestive heart failure with leutic aortitis rarely clears because the narrowed coronary orifices reduce permanently the blood supply to the left ventricle. In either the sudden or slow progressive type of right heart failure cyanosis and other evidence of venous engorgement dominate the picture.

More commonly both the right and left ventricles are involved in congestive heart failure, though often disproportionately. Here signs and symptoms are due to the dual involvement. Dyspnea becomes more constant and urgent, and orthopnea supervenes. To slight cyanosis is added liver engorgement and dependent edema, the former more readily noted in bed patients; in those up and about, edema of the legs occurs first. A distended liver may be easily missed by placing the palpating hand too high on the abdomen. The "lumbar pad" may be the sole evidence of dependent edema in the bed patient. In the ambulant patient there may be difficulty in the determination of the cause of the dependent edema, especially in fat women with the present style shoe; but edema due to peripheral causes, anemia or debilitating disease rarely causes pitting except after exertion in hot weather. Slight puffiness of the feet and ankles is commonly encountered in those whose occupation requires standing without the massaging effect of muscular exercise of the legs. Pleural transudates may occur with any right heart failure. These are more frequent on the right, and should there be an old obliterating pleurisy, bizarre localization may be noted, as in one case recently reported by Steele of an interlobar pleural fluid in each of four attacks of heart failure.

Abnormal pulsations in the neck veins furnish accurate information of right heart failure and this increase in venous pressure, described as the positive venous pulse, usually precedes liver enlargement or they occur simultaneously. These patients are breathless, either at rest or with very slight exertion. In the presence of extensive lung changes, as in pneumoconiosis, pulmonary fibrosis or bronchiectasis, it is often difficult to determine to what extent dyspnea may be due to associated cardiac insufficiency. Lewis has stated that "if it is established that a patient has no congestion of the venous system and is yet breathless in bed, then that breathlessness is not primarily cardiac in origin."

Vital capacity determinations may be of considerable assistance in determining the extent to which the pulmonary extensibility has been reduced by pulmonary vessel stasis. They do not add much in diagnosis after a careful physical examination, including inspection of the neck veins, has been made, but they may be of considerable value in the management of the patient and furnish some prognostic evidence.

Other signs of value may be mentioned: The protodiastolic gallop rhythm of ventricular dilatation and impending failure; pulsus alternans, the alternating strong and weak beat, recognizable oft-times by palpation of the pulse—always by the sphygmograph and always a sign of left ventricular failure—in fact, the only pathognomonic sign of pure left ventricular failure. This may not occur continuously but only for a few beats following a premature contraction; accentuation of the second sound at the pulmonary artery area, indicating an increased pulmonary vessel pressure, but not always of a failing heart; x-ray evidence of dilatation of one or all chambers of the heart; the fluoroscope offers much that is missed in the film, since the extent of systolic contraction may be estimated.

The study of murmurs plays a minor part in the determination of heart failure especially of the left heart, excepting that murmurs frequently disappear with dilatation. Lewis has stressed the fact that in failure heart signs do not change much, but peripheral signs and symptoms change frequently. Demonstrable increase in heart size within a short period means dilatation or pericardial effusion; with this the sounds become more distant and less distinct; if effusion be excluded the picture is that of failure.

Blood pressure is usually diminished but not always; in fact, it may remain elevated, while the heart is dilating.

So far no mention of the *electrocardiogram*: What can it tell us of failure? Much can be told regarding hypertrophy of the right and left heart, practically all we need to know regarding myocardial degeneration, the arrhythmias, and conduction disturbances which so frequently initiate heart failure, but what of failure itself? In the absence of coronary artery disease low voltage is probably the sole evidence of the failing heart obtained from the electrocardiogram, and even this change has been noted in the absence of clinical evidence of failure.

Treatment: Since by the time congestive heart failure appears there is usually irreparable damage to the heart muscle, little can be expected from treatment but an amelioration of the distress resulting from functional failure. The average duration of life after the onset of symptoms of congestive heart failure is brief in all but the rheumatic and thyrotoxic cases and the small group occurring in the course of an acute infection. Romberg found this about six months in a group of poor patients; in those more fortunately situated it will be more. If the failure occurs in a bed patient little can be expected from treatment.

In spite of all of the advances made in other fields of medicine rest and digitalis remain our sheet-anchor.

Rest will vary from a simple elimination of the types of strain which precipitate the mild attacks to the complete and absolute bed rest required in the failure secondary to a coronary thrombosis.

Where absolute bed rest is necessary the physician too often errs in allowing a resumption of effort at too early a date. In the patient who requires a long continued bed regime attention should be paid to back rest, or, better still, an adjustable bed of the type of the Gatch bed, or one divised by Lewis in London and recently modified by White in Boston. I have seen patients perform more work in bed than they would have in being assisted once or twice a day into an easy chair, and more work in the use of the bed-pan than in being lifted to a commode. The old-fashioned rocker is a convenient substitute for a special bed since it can be adjusted to any position; if not so adjusted it should be blocked to prevent tendency to rock in such a chair, which entails no small amount of work in the course of an hour or so. Rest should also imply rest from disturbing factors. One who has passed through a personal hospital experience knows how frequently the kindly visitor saps the energy.

Digitalis: The most frequent error is that this drug is not given in sufficient doses. In urgent failure, when the drug has not been given in the preceding week or two, the massive method of Eggleston or some modification is desirable. Eggleston gives within twenty-four hours 1.5 gr. of the dried leaf or 1.0 c.c. of the fat-free tincture to 10 pounds body weight. Most doctors have their personal likes as to preparations. Mackenzie and Wenckebach stated that life was too short to learn all they needed to know about one form of this drug. If a dependable dried leaf is available, it is probably preferable, but for most of us a dependable fat-free tincture is preferable because there is less chance of deterioration. In the presence of nausea and vomiting the intramuscular injection of fat-free tincture is advisable because all of the gastro-intestinal tract is oedematous and non-absorptive. In case of generalized oedema it may be necessary to give a water soluble digitalis intravenously, although this method carries a greater degree of risk than does the intramuscular method; in the majority of cases the oral administration of digitalis is preferable. When large doses are given or the drug is employed over long periods, the toxic effects of the drug should always be constantly looked for. In addition to the usual anorexia, nausea and vomiting at times diarrhoea, and the direct cardiac effects such as slowing irregularities due to premature contractions, one may encounter visual disturbances of equal significance sometimes preceding any of the above mentioned symptoms of cumulative effect. Withering noted that under these circumstances all objects appear green and yellow, at times red and blue, or take on a snow or hoar-frost appearance. All of these are increased in the sunlight and to a less extent by artificial light. Dim, veiled, cloudy or cottony vision may likewise occur. In none of these is the mechanism clear. Delirium may occur rarely as an unusual toxic symptom.

For the relief of cardiac oedema digitalis is ordinarily the best

diuretic. If this is inadequate, one may employ mercury and for this purpose novasurol or salyrgan (1 or 2 c.c. intramuscularly) are often followed by significant diuresis. Because it is attended by less renal irritation, salyrgan is more frequently employed and best preceded a day or two by ammonium nitrate or chloride—in fact, often these latter drugs alone suffice. Recently urea has been administered in cardiac oedema. This acts as a diuretic without impairment of the kidneys and capable of use over long periods. The purin diuretics—caffein, theobromine and theophylline—are less spectacular in their results and are more liable to cause nausea. Any of these is often more effective after tapping the pleura or abdomen. Likewise, a distended bladder in the male may seriously interfere with diuretics. Incision of the legs by multiple punctures or Southey tubes may be employed in oedema not responding to other methods. In the use of any diuretic the daily record of fluid intake and output and of weight loss is most important.

The method of venesection used by the earlier generation of dotors is too little used to-day. In the presence of urgent pulmonary congestion the removal of 300 to 600 c.c. of blood is usually followed by prompt improvement in symptoms.

Diet plays a minor part in cardiac therapy so long as a light diet is employed. One should prohibit a heavy evening meal for most cardiac patients. A light supper will often prevent the occurrence of pre existing nocturnal dyspnea. In cases where there is marked oedema the diet or Karell—800 c.c. of a milk a day for a few days—may act as an effective diuretic, to be followed later by a modification of the same by adding the usual light diet. In certain instances there appears to be a relationship between oedema and the sodium-potassium ratio in the diet. By substituting potassium for sodium, especially in non-valvular heart disease, a marked diuresis may be obtained. While the evidence is somewhat conflicting as to the role of salt in hypertension, it is probably wise in this condition, with or without oedema, to restrict the salt intake.

In any case of sudden failure associated with marked dyspnea, with or without pain, morphine will be of equal value to digitalis. The relief is prompt and permits the rest so sorely needed. Codeine, pantopan, bromides, and barbitals may be used in less urgent situations. In acute pulmonary edema atrophine may be advantageous.

In severe cases oxygen therapy by the open or by the closed (tent) method will be of great assistance. The cyanosis improves, the patient breathes without so much effort, rest is obtained and diuresis may continue one to several weeks after the withdrawal of oxygen. The effect of too sudden withdrawal will prove conclusively the advantage of the procedure.

Experimental evidence shows that glucose furnishes a temporary increase in the supply of carbohydrate, especially to the respiratory

center. Feeding appears to be preferable to the intravenous route because in the latter the increase is of short duration. It may act as a cardiac stimulant, but Smith and Luten claim that it is not effective in restoring and maintaining compensation. Yet there appears to be sufficient clinical evidence to justify a continuation of it in threatened or actual failure.

Coramine, a synthetic drug, pyridine beta-carbonic acid-diethylamide, has recently been introduced as a substitute for digitalis. It is claimed that there is no cumulative effect, no nausea in full doses, and that it is especially effective in the control of cardiac asthma. My personal experience is that the same results may usually be obtained with the proper use of digitalis.

Quinidine is not used in the presence of congestive heart failure because of the danger of embolism, but it is of distinct advantage in the prevention of failure since the re-establishment of a normal rhythm before failure sets in will remove an important factor (auricular fibrillation) which contributes to the onset of failure in these cases.

Thyroidectomy: In the past years several reports have appeared from Boston. Bulmgart and his co-workers have presented a group of 40 cases of congestive heart failure which had not responded to the usual methods of management. In about one-half of these there has been "economic rehabitation" over a period of three to sixteen months following the total removal of the thyroid gland, in consequence of which there was a marked reduction in the basal metabolic rate. Time will not permit an extensive discussion of this new procedure. It is certainly radical!but there is both theoretical and clinical evidence in support of the contentions of these workers.

There remains one more drug to mention, epinephrin, of chief value in vasomotor colapse, likewise in acute pulmonary edema, although acute pulmonary edema has been known to follow its use. It is of great value in certain cases of cardiac asthma, acting as a coronary dilator and cannot be considered as a means of differentiation between cardiac and bronchial asthma. Two recent personal experiences show that a similar effect may follow the use of ephedrine. Epinephrine is the only means for the relief of cardiac standstill, in which case it must be injected into the heart muscle. Because of its pressor effect it is to be avoided in angina pectoris, coro..ary thrombosis and aneurism.

The picture is not alluring; save in the rheumatic cases the progress is downward following the first failure; we have barely scraped the surface in demonstrating what may be done in the preventive management of our cardiac patients. The real future of cardiology lies in that direction.

—*Southwestern Medicine*, April 1935.

RETINAL HAEMORRHAGES

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To the general practitioner a recognition of the types and manifestations of retinal haemorrhages can be of practical value, in diagnosis, etiology and therapeusis.

Usually in books and articles retinal haemorrhages are relegated to a secondary role in the descriptions of the general underlying diseases. Nephritis, diabetes, arterio-sclerosis are described and the retinal manifestations are added as accompanying symptoms. To reverse the process appears illogical and yet the discovery of retinal haemorrhages before a general diagnosis has been made, is not an uncommon occurrence. A knowledge of these types and their more or less pathognomonic relationship to the general pathologies becomes of great value in furthering correct study and analysis.

Haemorrhages of the retina fall into three great classes that depend for their appearence and behavior upon the portion of the retina involved.

1. Haemorrhages beneath the hyaloid membrane and in front of the retinal structures.
2. Haemorrhages in the nerve fibre layer.
3. Haemorrhages in the granular layer.

Necessarily these types frequently are intermingled. There are many occasions, however, when the manifestation is of one type alone and here its recognition is of very practical advantage, being at times almost pathognomonic of the form of the general disease.

I. PRE-RETINAL HAEMORRHAGE.

This haemorrhage is very distinctive in appearence. It occurs as a rule about the posterior pole, between the retina and vitreous (Subhyaloid) and does not seriously involve the retinal structure so that complete absorption which frequently happens allows the return of visual acuity, the same or nearly the same as existed before the extravasation.

It is disc-like in shape, large as a rule, with a straight sharp superior edge of demarcation above a lower sac-like projection, dark red from the settling downward of the erythrocytes.

The blood is derived from the retinal vessels and breaks through the internal limiting membrane into the pre-retinal space, particularly in the macula region where the connection between the retina and the vitreous is least intimate. From the cul de sac the blood not infrequently ruptures into the vitreous.

Such a haemorrhage may be traumatic, from a foreign body or a contusion. If it is a manifestation of a general pathology such as nephritis it is accompanied by haemorrhages of other types in the retinal tissues themselves. It is not therefore of such pathognomonic importance as are the other two types.

II. STRIATE HAEMORRHAGES.

In the fibre layer of the retina the extravasations of blood take the from necessitated by pressure. They are flame-like, large or small, depending upon the quantity of blood exuded. As a rule, they are close to the retinal arteries and therefore in the anterior retinal layers.

III. DOT-LIKE HAEMORRHAGES.

These occur in the deep granular layers of the retina and are as easily recognized as the striate type. They are usually small and discrete and take their form from the anatomic arrangement of the retinal structure.

Frequently, of course, these types of haemorrhages occur together and at times are so conglomerate that they are differentiated with difficulty, especially in cases of thrombosis and profound retinal changes of mixed types of arterio-sclerosis and nephritis.

Fortunately, there are many cases in which one type appears, and the recognition of it is a guide to a diagnosis and therapy. It is of these cases that this paper will particularly deal.

The causes of retinal haemorrhages are numerous and fall into two rough groups, traumatic and toxic.

Under the traumatic are classed those from external injury, those in high myopia and the retinal haemorrhages of the newborn.

The toxic group is vague, for much must still be learned before a final reason is formed why extravasation takes place. Vessels break from over-distension as in thrombosis; from brittleness as in arterio-sclerosis. But there are haemorrhages that occur in many conditions such as pernicious anemia, tuberculosis, poisoning, albuminuria, diabetes, profound burns, etc., where our only present explanation is an altered condition of the blood, probably toxic.

The visual prognosis in these retinal haemorrhages depends upon the extensiveness and the position of the extravasations. The blood absorbs very slowly, often requiring weeks and months. If of any size the haemorrhages produce scotomata,—blind spots somewhere in the visual fields. At the macula region they may cause permanent loss; in other parts, as the periphery, little or no disability. After absorption, their presence is usually discovered by remnants of connective tissue.

Particularly interesting to the internist are these two types of striate and dot-like haemorrhages. A more general use of the ophthalmoscope by the internist will discover them frequently before there appears the necessity of an ophthalmologist.

To the internist, diabetic and albuminuric retinitis are of constant interest. Retinal haemorrhages from other causes, trauma, choked disc, optic neuritis etc., are infrequent in his daily practice and of more intimate interest to the specialist. It is not always easy to differentiate diabetic haemorrhages from those of nephritis. Broadly speaking, however, the haemorrhages of diabetes are small; exudations infrequent, and when present chiefly found about the

macula, not however presenting the stellate arrangement of the so-called albumunuric retinitis; nor is the presence of diabetic haemorrhages of the serious import of nephritic haemorrhages. Many cases of diabetes attended by haemorrhages carry on for many years, more particularly so in advanced age.

The development of retinal complications in nephritic conditions is tragic and a sign-post to early death, especially in chronic nephritis.

Frequently in acute nephritis of toxic pregnancy the retinitis will disappear with the terminating of the pregnancy. Nephritic haemorrhages are largely of two types,—those accompanying chronic nephritis with exudation, and those accompanying nephritis without exudation. The former are found with general oedema and dropsy and are almost invariably accompanied by retinal exudates. The exudates do not necessarily accumulate about the macula as a stellate mass, in fact this is a rather rare occurrence. They are usually scattered here and there with no general order and the haemorrhages are of the striate variety. Hence if the picture is one of striate haemorrhages, exudations, stellate or otherwise, irregular and inclined to rather large masses, we may presume a nephritis, acute or chronic; and if chronic, without necessarily accompanying arterio-sclerosis, for in these retinas it is not often that we find the signs of hardening of the arteries.

Another picture may arise,—few or no exudates, haemorrhages few or many dot-like in form, in the granular layer of the retina. The mind leaps at once to arterio-sclerosis and we find increased light streaks upon the retinal arteries, cork-screw appearance of the terminals, and ampulla enlargements of the veins where they underly the hard arteries.

Such a picture is that of high blood pressure, arterio-sclerosis, with or without an accompanying nephritis—the condition classified by Delafield as nephritis without exudation.

It is in these types of cases, that recognition of the variety of haemorrhages is of very practical value.

In the first type of case, so called albuminuric retinitis, the condition remains practically unchanged until death. In the latter, the haemorrhages appear and disappear with the progress of the toxaemia. With improvement of blood pressure and toxicity, the haemorrhages tend to vanish, with an increase of toxaemia they reappear. They are undoubtedly due to a toxaemia and certainly they are an index to prognosis and therapy.

No claim is made in these few random paragraphs to completeness of description or elucidation,—a practical point is however stressed. A recognition of retinal haemorrhages, particularly in nephritis and arterio-sclerosis, is a valuable index in diagnosis, prognosis, and therapy.

—*American Medicine*, May 1935.

Extracts

LEUKAEMIA: ITS DIAGNOSIS AND TREATMENT.

(N. ROSENTHAL AND W. HARRIS: *Journal of A. M. A.* 1935. civ. 702.)

The important characteristic alteration in leukaemia is the presence of a persistent relative or absolute increase in the number of mature or premature white blood cells. The type of premature cell usually varies with the duration of the disease. Acute leukaemia is characterized by the presence of the more premature types of cells, particularly the myeloblasts, lymphoblasts and monoblasts. Chronic varieties have a tendency to show more mature types such as polymorphonuclear neutrophiles and myelocytes in the myeloid leukaemias and lymphocytes in the lymphoid leukaemias. In acute leukaemias the haemoglobin and the number of red blood cells are usually reduced. In chronic leukaemias anaemia may be absent. The blood platelets present marked variations, being usually greatly reduced in the acute types and in exacerbations of the chronic types.

Weakness and fatigue are common and persistent. Abdominal distress due to enlarged viscera or lymph nodes is usual. Pain and ulceration in the upper part of the respiratory tract may occur. Purpuric manifestations or uncontrollable bleeding such as bleeding of the gums or persistent haemorrhages following a surgical procedure may be the first evidence of a leukaemic state.

Leukaemia must be differentiated from pernicious anaemia, purpura haemorrhagica, agranulocytosis, subacute endocarditis, splenic anaemia, and various other conditions. Its diagnosis should be based on the characteristic blood changes. These depend not so much on the number of white blood cells as on the presence and persistence of specific types of cells such as myelocytes, myeloblasts, and a relative and absolute lymphocytosis. A confirmatory diagnosis of the more obscure varieties of leukaemia may be made by biopsy on the sternal bone marrow or a lymph node.

Because of the limitation of our knowledge concerning the etiology of leukaemia, no specific remedy is known and treatment is therefore essentially symptomatic. The aims of treatment should be to improve the general condition, to render the patient comfortable by rest, regulation of the diet, and the administration of sedatives, and to increase his strength and efficiency by blood transfusions and roentgenotherapy. The cases in which treatment is most successful are those of chronic myelogenous and lymphatic leukaemia of the leucocythaemic variety. Radium, in the form of surface application or packs, is used only in the event that roentgen therapy is not available, or in patients who cannot be moved from their homes. The aim of treatment should be to reduce the number

of white blood cells and increase the haemoglobin and red blood cells. Reduction of the number of white blood cells may be accomplished by chemical, biological, or physical methods. The performance of splenectomy in leukaemia should be discouraged since, as leukaemia is a disease of the entire haematopoietic system, it is unreasonable to expect any constant alteration in the general condition from the removal of only one of the organs affected. Arsenic, transfusions, and particularly roentgen irradiations are the chief means of inducing symptomatic improvement and remissions, and possibly prolonging life.

Irradiation therapy is of value in both accute and chronic leukaemia, both myeloid and lymphoid. The marked radiosensitivity of leukaemic tissue and the liability of the blood count necessitate extreme caution. In general, marked reactions should be avoided, and rapid regressions of leukaemic deposits should not be sought. In the authors' series the areas treated were the spleen in myeloid cases, and palpable lymph nodes and spleen in the lymphoid cases. Occasionally the long bones or the flat bones were irradiated, but the response appeared to be somewhat slower. The effect of roentgen treatment is measured by the degree of symptomatic improvement. In favourable cases there is a rise in the haemoglobin and an increase in the red blood cells, a response that occurs more rapidly in myeloid cases. In lymphoid leukaemias the improvement is not so marked. After the first series of roentgen treatments the blood picture may return to normal, particularly in chronic forms, and it may remain so for a variable period—from a few weeks to nine months. The symptomatic improvement, however, may be of longer duration. Additional treatment in their cases proved less successful. In time, patients become more resistant to the treatment and, as the disease progresses, do not tolerate it well.

THE SIGNIFICANCE AND TREATMENT OF COUGH.

(J. B. ALEXANDER: *Practitioner*. 1935. cxxxiv. 83.)

With the exception of "pain," cough is probably the most common symptom about which a patient consults his practitioner. The practitioner must be wide awake to the extreme diversity of the causes of cough; it may denote anything from mere nervousness up to such grave conditions as pulmonary new growth or aneurysmal swellings. Every medical man must recall many occasions on which the slight and unimportant cough (according to the patient's own description) denoted serious pulmonary disease, hitherto undetected, and equally many occasions on which, in spite of the relatives' alarming description of the cough, nothing more serious than a slight laryngeal irritation from excessive smoking has been the cause. The information imparted by the patient regarding the type and character of his cough should be carefully considered.

Local causes: The most common causes are: (1) Tonsils and adenoids, most particularly in children. (2) Long uvula. (3) Pharyngitis, often associated with dyspeptic conditions. (4) Laryngitis. (5) Tracheitis.

Conditions affecting the lungs and pleurae: Under this may be included the cough of acute and chronic bronchitis, acute pleurisy, pulmonary emphysema, bronchiectasis, pulmonary tuberculosis, fibrosis of the lungs and pulmonary neoplasms. The cough which is brought about by effort on the part of the patient, if not of a painful nature as in pleurisy, is usually an indication of cardiac dilatation, and may be present before any marked degree of shortness of breath occurs.

Conditions with pressure on some part of the respiratory tract: These are chiefly: (1) Enlarged mediastinal glands. (2) Mediastinal new growths. (3) Aneurysm. The brassy cough occurs when the main bronchus or the trachea is pressed by a large mass of glands or by a mediastinal new growth or by an aneurysm, and is almost always diagnostic of some severe degree of pressure.

Reflex causes: The most important of reflex coughs are those seen in children due to some local irritation in the ears or teeth; the most common cause in adults is usually supposed to be due to dyspepsia and co-existent with pharyngitis, and is usually a morning cough. There are instances of cough in which no satisfactory explanation of the cough is forthcoming and cause much worry and great anxiety. In many instances there are examples of *habit cough* and are actually reflexes—the result of an irritant condition at some previous date.

The nervous cough is usually of a barking nature and is more distressing to others than to the patient and is non-productive. A still more harassing form is the hysterical cough, which may be very alarming, and is often actually a symptom produced through a desire for sympathy. In this type of cough no less than in other forms the true cause of its nature is often shown by the results of treatment.

The treatment of cough: naturally depends upon a correct diagnosis having been made. The indiscriminate use of cough mixtures is often harmful as it may tend to mask the true underlying cause.

It is obvious that a cough due to enlarged tonsils and adenoids must be treated surgically. The long uvula may be cut, if other causes have been carefully excluded. For the pharyngitis the dyspeptic condition should be corrected, with advice regarding diet.

In *laryngitis* and *tracheitis* relief can be usually obtained by a mixture to lessen congestion of the inflamed mucous membrane, promote secretion, and thus lessen the painful character of the cough.

Sod. Bicarb. gr. xv
Tinct. Ipecac. m viiss

Syrup. tolu. } aa. m xxx
Syrup. scill. }
Spirit. chloroform. m x
Infus. senegae ad oz. i.

One oz. every 4 hours until sputum has become free.

Inhalations are also useful, such as, Tinct. benzoin. co. m 60 to the pint of water. Or to this may be added 3 minims each of menthol and oil of eucalyptus. Another remedy for relieving the cough of laryngitis and tracheitis is the application round the neck of anti-phlogistine or warm olive oil.

In the *early stages of branchitis*, when the cough is troublesome, painful, and non-productive, the same mixture as for laryngitis may be used.

The following mixture is also useful.

Vin. antimonialis m xv
Ammon. chlorid. gr. x
Spirit. chloroform. m x
Syrupi aurantii m lx
Aquam camphor. ad oz. i

This should be discontinued, probably after 2 or 3 days, when the sputum becomes free.

In the *later stages of bronchitis* when expectoration is copious, the cough calls for stimulating expectorant drugs, as in the following mixture :—

Ammon. carb. gr. v
Tinct. nuc. vom. m viii
Tinct. scillae m xv
Aquam ad oz. i

or alternatively—

Ammon. carb. gr. v
Tinct. scillae m xv
Tinct. opii camph. m xx
Infus. senegae ad oz. i

A very useful prescription for children suffering from a cough which is just becoming loose, is the following :—

Tinct. ipecac. m ii—v
Tinct. opii camph. m v—x
Potass. nitratis gr. ii
Oxymelis m xxx—lx
Aquam ad m cxx

In the cough of *pulmonary tuberculosis* the treatment will depend upon whether it be of a useful or useless type. In the useless type the patient can often be trained to suppress the act and to prescribe some sedative drug, especially when it disturbs sleep, as in the following :—

Heroin gr. 1/12
Syrup. virg. prun. } aa. m xxx
Syrup. codein }
Aquam ad oz. ss

The following prescription is even better : –

Heroin gr. 1/12
Codeinae gr. ½
Acid sulph. dil. m ii
Glycerini m x
Syrup. tolu ad m lx

For this type of cough dry inhalations are often of use.

Menthol m l x
Creosotum m l x
Spirit. vin. rect. oz. ss

A few drops sprinkled on a mask and inhaled for a few minutes has often a soothing effect.

When the cough is useful, every endeavour must be made to loosen the expectoration, and as in early bronchitis, ammonium chloride and antimony are invaluable.

In fibrosis of the lung the cough is usually associated with marked dyspnoea often caused by failure of the right ventricle of the heart, and relief will be better obtained by cardiac tonics and sedative cough mixtures as in the following :—

Tinct. digitalis m v
Ammon. carb. gr. v
Tinct. nuc. vom. m v
Spirit. chloroform. m x
Aquam ad oz i

In some severe cases Nativelle's granule 1/600 two or three times a day diminishes the cough.

Belladonna is of definite use in the treatment of certain types of cough in which an anti-spasmodic effect is desired :

Tinct. sumbul. m x v
Tinct. stramonii m x
Tinct. belladonnae m v
Spirit. chloroform. m x v
Aquam ad oz. ss.

The successful treatment of cough will depend upon a correct diagnosis of its cause having been made.

THE TREATMENT OF DEEP INFECTIONS OF THE HAND.

(P. H. MITCHINER: *Medical Press and Circular*. 1935. cxc. 33).

In the treatment of thecal whitlow or deep infections of the hand, indiscriminate hot bathing cannot be condemned too strongly, even after incision, for it not only causes great pain, but by increasing the local congestion in many cases leads to colliquative necrosis,

and causes sloughing and spread of infection. Warm hypertonic saline baths (10 to 15 per cent) may be given with advantage twice daily when incisions have been made, and the patient encouraged to perform active movements gently at this time, which should not exceed half an hour in all. Between whiles the best dressing to apply is hypertonic saline, though in the absence of this, weak antiseptics as acriflavine may be used. This dressing is continued for 3 or 4 days, being renewed after each bath, i.e., twice daily ; but as soon as active infection subsides, dry sterile dressings must be substituted or the area will become sodden and tend to desquamate. As in all inflammatory lesions, rest and elevation are essential in the acute stages, and between the baths the arm should be supported in a sling, with the forearm about a right angle so as to elevate the hand. For the first week the fingers and hand should be fixed to a splint applied to the dorsum of the limb ; this should be discarded as soon as acute inflammation has subsided, the patient urged to move the fingers gently, so as to prevent stiffness and formation of bands of adhesions. In palmar cases the patient should be confined to bed. The usual general measures should be taken to combat sepsis ; the bowels opened regularly but not violently ; a good nourishing diet given, alcohol in the form of stout or draught ale or port, is valuable; and narcotics may be needed for pain for a few days. In the convalescent stage cod liver oil or halibut oil is valuable and iron and arsenic tonics may be administered.

PAST AND PRESENT IN GENITO-URINARY CARCINOMA.

(B. S. BARRINGER: *Am. J. Surg.* 1934. xxiii. 438)

In former years a positive cure in *carcinoma of the bladder* was practically unheard of. Of 75 such cases in his experience, the author mentions 29 in which the patients are well from five to ten years, nine from ten to fifteen years, and one from fifteen to twenty years. This improvement is due not only to better methods of fulguration and irradiation but to earlier diagnosis as a result of cystoscopy, biopsy, and the recognition of haematuria as a warning signal. Large carcinomas of the bladder are now seldom seen.

In *prostatic carcinoma* the prognosis is less encouraging. Diagnosis, although materially aided by the method of punch biopsy, is still difficult in the early stages. Radical removal of the affected gland is a logical procedure only in early cases, and even then only in the hands of the most expert surgeons is it justified. The most satisfactory mode of treatment appears to be the implantation of radon seeds through a suprapubic incision. By this means and by other types of irradiation, from 10 to 15 per cent of these growths have been controlled for periods longer than five years.

The treatment of *teratoma of the testis* has become infinitely more encouraging with the advent of irradiation. Probably no individual tumour type reacts more dramatically to this form of treatment than the anaplastic, embryonal type of testicular tumour. Since there is a wide variation in the sensitivity of malignant lesions of the testis, it is of great importance to determine the exact type of morphology. This is most satisfactorily accomplished by removal of the primary growth, a procedure which is also efficacious in obviating over-radiation of the normal testis and the chances of local recurrence. With the recent development of the Ascheim-Zondek test, it has become possible not only to diagnose a malignant lesion of the testis but to determine fairly accurately the grade of malignancy and the individual type.

The end-results of treatment of carcinoma of the penis have been definitely improved by proper regard for anatomical extensions and the application of suitable surgical procedures. Thus a lesion limited to the foreskin may be treated satisfactorily by circumcision. With more extensive growths it is necessary to amputate the penis. Fortunately metastasis to the inguinal glands is relatively rare (40 per cent), and even when present, rarely, requires radical inguinal dissection, in view of the success of open implantation of radon seeds. Of 66 patients treated for this disease, 36 are alive and apparently well.

LABOUR IN PRIMIPARAE.

(J. N. NATHANSON: *Am. J. Obst. & Gyn.* August 1935).

The author has made a parallel study of labour in young and old primiparae based upon a critical analysis of 372 cases below 20 and above 35 years of age. He states that within the past two decades, late marriage and low birth rate have become important aspects of a world-wide economic and social upheaval. Many women are purposely avoiding maternity on the ground that they are too old to bear children without great hazard. The writer's conclusions are as follows :

1. It is suggested for the sake of uniformity in future studies that 35 years be chosen as the lower age limit for elderly primiparae.

2. The funnel pelvis was more frequently encountered among elderly primiparae, and the justo-minor pelvis among their younger sisters in the series. Dystocia of bony origin, therefore, may be the more frequent complication at the inlet of the young patient, and at the outlet of the old primipara.

3. Persistent occipito-posterior positions and breech presentations occurred in 21·94 per cent and 6·09 per cent of cases respectively, in old primiparae, in whom the incidence of these abnormal findings was twice as high as in the young patients.

4. There was a difference of only 11 per cent in the incidence of premature rupture of the membranes in elderly primiparae as compared to that of younger patients.

5. Labour was of definitely longer duration in the old primiparae, the greatest difference occurring in the first stage of labour,

and this is undoubtedly accounted for by the greater incidence of abnormal presentations and inertia uteri in the older patients, and by the greater elasticity of the soft tissues in the young primiparae.

6. Caesarean section was performed in 10·75 per cent of the cases among the older patients and not at all in the younger group. It is of utmost importance to note that the age of the patient was not the major indication for the operation in most cases, such potent contributing factors as pelvic deformity, non-yielding cervix, progressive toxaemia, etc., being present in the great majority of cases of the elderly primiparae in whom Caesarean section was performed.

7. Inertia uteri, both primary and secondary, was nine times as frequent in the old as in the young patients, and is a potential factor to be contended with when delivering an old primipara.

8. The still birth incidence was three times as high among the children born to the older patients as compared with those of mothers in the younger group.

9. The maternal mortality in the elderly primiparas was 1·6 per cent while none of the young mothers died as a result of pregnancy or labour.

10. The age of the primipara has little or no influence upon the sex, weight, or length of her children.

11. Toxaemia occured 1½ times as often in the old patient as in her younger sister, while complications of the third stage of labour were twice as frequent in the former as in the latter.

12. Irregularities in menstruation, and particularly the late establishment of the function, seemed to influence the type and duration of labour.

13. On the basis of this study, no inflexible rules can be laid down for the routine conduct of labour in elderly primiparae. Individualisation of each case with concomitant demands, guided by the experience, judgment and ability of the attending obstetrician, would appear to be the only ideal approach to the solution of the problem.

Announcement

INTERNATIONAL COURSES IN MALARIOLOGY (1936), LEAGUE OF NATIONS EASTERN BUREAU, SINGAPORE.

The Health Committee of the League of Notions is arranging for a Third Course of Instruction in Malariology which will commence at the King Edward VII College of Medicine at Singapore, on April 27th, 1936.

It will be preceded by a preliminary revision course lasting four days for candidates with a limited experience of the subject. The theoretical and laboratory studies will continue until the end of May, after which the candidates will proceed in groups either to Malaya, Java or French Indo-China for a further period of practical

field work extending over three weeks. The object of the course is to complete the training of medical practitioners who are engaged in the work of malaria control in their own countries. The courses will thus be of interest not only to Governments and Municipal authorities but also to all medical men practising in Eastern countries, particularly those engaged in estate work.

The administrative duties will be carried out by the Eastern Bureau.

There will be three distinct stages to each course:

(1) A preliminary revision course;
(2) Theoretical and laboratory studies with practical demonstrations;
(3) Practical field studies.

The *Preliminary revision course* is designed for students who have a limited practical experience of malaria, and those who may wish to revise the principles of haematology, protozoology and entomology, and the clinical aspects of malaria. It was introduced to avoid the inclusion of the more elementary aspects of these subjects into the main course, which were not required by the more experienced students. The preliminary course occupies four days, beginning at 8-30 a.m. on Wednesday, April 22nd, 1936.

The *theoretical and laboratory* studies of the main course will commence at the King Edward VII College of Medicine at 8-30 a.m. on Monday, April 27th. 1936, and will be completed on the 30th May, 1936.

The *practical field studies* will commence at the beginning of June, and for this the candidates will be divided into groups, one of which will study in Malaya, one in French Indo-China and probably another in Java. They will last approximately 21 days, during which the student will have the opportunity of becoming familiar with the routine of a malariologist and the actual application of anti-larval and other anti-malarial measures to field conditions.

The League of Nations is making available a limited number of partial fellowships to candidates who are nominated by their Governments, on condition that these Governments bear half the cost entailed by their nominees.

Conditions of Admission: The subscription for the theoretical and laboratory course will be 75 Straits dollars, and will be received by the Eastern Bureau of the League of Nations at Singapore.

Candidates not attending the preliminary course will be expected to possess a working knowledge of malaria and of the fundamental principles of the contributory subjects, such as haematology, protozoology and entomology.

Any further information desired will be supplied by the Director of the Eastern Bureau of the League of Nations, 336, River Valley Road, Singapore, to whom applications for admission to the course

should be addressed. These should reach Singapore not later than the 29th February, 1936, and as only thirty candidates can be admitted, early application is desirable.

SYLLABUS.

Preliminary Revision Course.

1. MICROSCOPY, 1 HOUR.

Special characteristics of the microscopes issued for the Course. Care of microscopes in the tropics. The importance of correct illumination and the relation between substage condenser and objective for optimum resolution.

2. HAEMATOLOGY & PROTOZOOLOGY, 9 HOURS.

The blood-picture in malaria ; enumeration of bloodcells, estimation of hæmoglobin in malaria practice. Theory of Romanowsky staining : special staining methods for malaria parasites. Difficulties in the examination of blood.

The human malaria parasites ; detailed study of *Plasmodium vivax*, *P. malariae* and *P. falciparum;* mixed infections.

3. CLINICAL, 8 HOURS.

Aetiology ; types of malarial fevers ; mechanism of the febrile attack ; onset, course and termination of ordinary cases, Analysis of different types ; after-fever and pseudo-recidive malaria. Malaria in pregnancy. Pernicious and chronic malaria. Diagnosis. Malaria in childhood. Treatment in general ; treatment of severe and fulminating cases and of special symptoms ; after-treatment and results. Demonstration of selected cases and discussion.

4. ENTOMOLOGY, 6 HOURS.

The general morphology, life-history and importance of insects ; their classification and a synopsis of the more important orders. The Diptera Nematocera ; the family Culicidæ, its sub-families, tribes and important genera.

Theoretical, Clinical and Laboratory Studies with Practical Demonstrations.

1. CLINICAL, 10 HOURS.

Demonstration of selected cases and discussion. Relapses and resistance to malaria ; provocation of attaks. Induced malaria ; delayed manifestations ; complications and sequelæ. Toxicology in relation to the use of quinine and other drugs. Determination of what constitutes a cure. Black-water fever.

2. PATHOLOGY, 6 HOURS.

Pathology (gross and microscopic) of acute malarial fever. The spleen in acute malaria. Pigmentation. Pathology of the metabolic changes in malaria ; changes in the blood, urine and special organs. Congenital malaria. Pathology of black-water fever.

3. PROTOZOOLOGY, 14 HOURS.

Comparison of staining methods ; the enumeration of malaria parasites ; cultivation ; observation of living parasites ; seasonal

prevalence of parasites ; *Plasmodium ovale ;* other indeterminate and doubtful species and varieties in man. Malaria in birds, monkeys and other animals. The action of therapeutic agents on the parasites. Obscure points in the life-history of the parasites, especially in chronic cases of malaria.

4. ENTOMOLOGY, 58 HOURS.

The detailed morphology of anopheline imagines, pupæ, larvæ and ova. The œcology of imagines, larvæ and ova ; physical and biotic autœcology, synœcology. The genus *Anopheles*, its classification and affinities ; the species and categories below the species ; geographical and seasonal distribution ; the characteristics of the principal carriers of the World. Susceptibility ; influences determining the efficiency of carriers. The determination of species in all stages ; the use of keys and original descriptions in a study of the Eastern fauna. The technique of laboratory and field investigations.

5. IMMUNITY UNDER EXPERIMENTAL CONDITIONS, 5 HOURS.

6. EPIDEMIOLOGY, 14 HOURS.

Methods and interpretation of results ; measurement of malaria (spleen rate, parasite rate, sporozoite rate, etc.) and the estimation of its prevalence and intensity ; statistical analysis of data. Spleen-theory. Endemicity, epidemicity and pandemicity, their characteristics and the factors involved. The prediction of epidemics. Topographical, climatic and social factors. Anophelism *sine* malaria.

7. SYNTHETIC DRUGS IN THE TREATMENT AND PROPHYLAXIS OF MALARIA, 3 HOURS.

Plasmoquine, Fourneau 710, Atebrin, arsenicals and other preparations. Pharmacology and toxicology ; dosage and administration. Therapeutic effects ; effects on the various stages of the parasites ; effects on relapses. The present status of synthetic drugs in comparison with quinine and its derivatives.

8. CONTROL, 56 HOURS.

Surveys, urban and rural ; field methods for the measurement of malaria ; evaluation of loss from malaria ; temporary and permanent measures, their efficiency, cost and financial benefit. Protection from bites ; measures against adults; measures against larvae. Biological control ; choice and design of measures in special circumstances ; species control ; organisation of antimalaria work ; Propaganda.

Local procedure in conducting mosquito surveys ; the history of malaria control in Singapore and the organization of malaria control services in the Straits Settlements. Local anti-malaria legislation ; demonstrations and discussions on schemes for the control of malaria in Singapore.

Surveying and levelling in anti-malaria work. Major and minor engineering works in relation to the cause and prevention of malaria. The construction of sub-soil pipes and the manufacture of concrete inverts and slabs.

9. CONTROL DEMONSTRATIONS, 26 HOURS.

Beginning at 4-30 p. m. on certain days after the completion of the normal work of the Course.

Demonstrations and discussions on surveys, methods and results of anti-malaria work ; the manufacture of sub-soil pipes, concrete inverts and slabs and the mixing of concrete. For those particularly interested in field work.

10. PHYSICAL PROPERTIES OF OILS USED AS "LARVICIDES", 2 HOURS.
11. BIOLOGICAL AND OTHER NATURALISTIC MEASURES FOR MOSQUITO CONTROL, 2 HOURS.
12. SPECIAL PROBLEMS, 12 HOURS.
13. THE WORK OF THE LEAGUE OF NATIONS' MALARIA COMMISSION, 2 HOURS.
14. DISCUSSION OF GRADUATES' OWN PROBLEMS, 4 HOURS.

Medical News and Notes.

TINNEVELLY DISTRICT MEDICAL ASSOCIATION. The monthly meeting of the above association was held on the 23rd November at Srivaikuntam, with Lt. Col. T. S. Shastri, I.M.S., District Medical Officer, in the chair. A Health Exhibition was organised by the District Health Officer. After lunch, Dr. S. Kanthimathinathan, L.M.S., of the Municipal Hospital, Tinnevelly, delivered a lecture on "dyspnœa." Dr. S. Venkatachalam, L.M.P. of Srivaikuntam, read a paper on "spelenomegaly in the tropics." An account of a case of Caesarian section for placenta praevia centralis which was operated on by Lt. Col. Shastri was read by Dr. Mrs. A. Brown of the Women and Children's Hospital, Vannarpet. The President thanked Dr. Govinda Menon, the Sub-Assistant Surgeon of the Local Fund Hospital, Srivaikuntam, for his hospitality, and conveyed the Association's best wishes on his impending retirement from service. The meeting ended after tea and exhibiton of feats of physical strength.

The same association met again on the 17th December at Koilpatti. In the morning Lt Col. Shastri requested the Raja of Ettaiyapuram to open a health exhibition.The Health Inspector made a speech in Tamil on the causation and prevention of tuberculosis, flies, mosquitoes, and cholera. In the evening resolutions were passed supporting the affiliation of the Association with the Indian Medical Association. The annual meeting of the Association was announced to be held at Palamcottah on the 25th January. After exhibition of some clinical cases, the function terminated with dinner.

SOUTH ARCOT DISTRICT MEDICAL ASSOCIATION. This association met at the Headquarters Hospital, Cuddalore on the 23rd November. After tea, Dr. S. Padmanabha Sarma, M.B., C.M., District Medical Officer, delivered a lecture on "Appendicitis". He advised the

medical men to cultivate the habit of studying the case of every patient carefully, to diagnose it in the most scientific manner and then give the treatment. Several clinical cases were also demonstrated.

GUNTUR DISTRICT MEDICAL ASSOCIATION. A meeting of the above association was held at the Headquarters Hospital on the 25th November, with Major A. Krishnamurthi, B.A., M.B., C.M., A.I.R.O., District Medical Officer, in the chair. Dr. V. R. Sarma read a paper on "facial paralysis" and demonstrated some cases. Dr. N. Padmanabha Rao, Secretary of the association, read notes on "abdominal operations."

SALEM DISTRICT MEDICAL ASSOCIATION. A meeting of this Association was held on the 1st December at the Headquarters Hospital with Lt. Col. N. K. Bal, I.M.S., District Medical Officer, in the chair. Dr. A. P. K. Nair was "at home" to the Members. Dr. G. S. Sankara Aiyer of Namakal spoke on "some surgical experiences".

CUDDAPAH DISTRICT MEDICAL ASSOCIATION. This association met on the 8th December when Dr. G. Srinivasa Rao, the R.M.O., of the Headquarters Hospital read a paper on "some important diseases of the eye". Dr. Miss J. Muthiah was "at home" to the members. Rao Bahadur K. Raghavendra Rao, M.B., C.M., D.P.H., District Medical Officer, presided over the meeting.

COIMBATORE DISTRICT MEDICAL ASSOCIATION. The tenth annual meeting of the above association was held on the 11th December at the Moses Gnanabaranam Eye Hospital, with Dr. Anantharanga Rao in the chair. After group photo, reading of congratulatory messages and of the annual report, the following office bearers were elected for the coming year:—Patron: Major A.I. Cox; President: Dr. Anantharanga Rao; Vice-President: Dr. Mrs. Abraham; Secretary: Dr. N. Krishnaswami; Assistant Secretary: Dr. L. Seshadrinathan; Committee members: Drs. K. Pratap, S. Gurubatham, Velappan, C. S. Ramaswami Iyer, and Mrs. T. K. Sundaram. The gathering dispersed after music and dinner at night.

PUDUKOTTAH MEDICAL ASSOCIATION. This association was formed recently with the following office-bearers:—President: Dr. M. G. Ramachandra Rao (Chief Medical Officer of the State); Vice-President: Dr. V. Sadasiva Aiyar; Joint Secretaries: Drs. R. Narayana Rao and R. Raghunatha Aiyar; Treasurer: Dr. R. Satagopa Naidu.

COLONEL BHOLANATH'S efforts in seeking to organise and unify the ranks of the medical profession in India under the banner of the Indian Medical Association are highly laudable and there has been a fair amount of response to his appeal in the course of his South Indian tour: Medical men in South India do not have any cohesive force of an outstanding character and the Indian Medical Association

should serve to supply this need. We hope and trust this Association will gain in strength by the support of all medical men and women.

THE CENTENARY OF THE MADRAS MEDICAL COLLEGE was celebrated on the 4th November at the College premises, which were elaborately decorated, with Lord Erskine, Governor of Madras presiding. Sir Mahomed Usman, President of the General Committee of the Centenary Celebrations, welcomed the Governor and said that the Madras Medical College which was originally established as a school and later developed into a college, has earned a high reputation. Lt. Col. C. Newcomb, I. M. S., Principal, gave an account of the history of the College, which was founded on February 13, 1835 in order to "afford better means of instruction in Medicine and Surgery to Indo-British and Indian youths entering the medical branch of the service in this Presidency." On August 1, 1850, the school was raised to the status of a college. In 1852 the college started granting diplomas to successful students till 1863, when the University of Madras instituted the University Degree. Junior departments for the training of apothecaries and hospital assistants continued to be held till 1902 when they were transferred to the Royapuram Medical School, now called the Stanly Medical School. In 1857 women were admitted to the college for the first time in the world. The original staff of 4 has now become 90, and the original 4 rooms is now a huge pile of buildings. The period of 100 years is really a century of sustained effort and marked progress and of the advancement of medical knowledge in South India generally. The Governor addressed the gathering, and publicly announced the generous gift of fifty thousand rupees from Mrs. Kamala Rangachari for the institution of scholarships to the college students. Lt. Col. R. E. Wright, I. M. S. proposed the vote of thanks and presented the Governor with a copy of the centenary volume. The gathering then adjourned to tea. On the 5th Nov. Lt. Col. Newcomb unveiled the portraits of some of the past members of the college staff, and opened the centenary exhibition (showing the progress of medicine), which was open for 5 days. There was the College prize distribution in the evening, and dinner for the old boys of the College on the night of the 6th November, and the College sports on the 7th. The centenary is a historic landmark in the annals of the Madras Medical College.

THE FIRST ALL-INDIA OBSTETRIC AND GNAECOLOGICAL CONGRESS. The detailed programme of this Congress is as follows:

THURSDAY, 2ND JANUARY 1936.

10-15 A.M. Opening of the Congress and the Scientific Exhibition by Lord Erskine, Governor of Madras, at the Museum Theatre.

Welcome Address by Dr. A. Lakshmanaswami Mudaliar, Chairman of the Reception Committee.

Election of Dr. Miss Ida S. Scudder, M.D., D.Sc., as the President.

Presidential Address.

Vote of Thanks.

2—4-30 P.M. *Obstetric Section.* Reading of papers.

1. Dr. S. M. Trasi: Atonic post-partum haemorrhage.
2. Dr. N. A. Purandare: Manual removal of placenta—a study of cases from the Wadia Hospital, Bombay.
3. Dr. T. Bhaskara Menon: Placental infarcts.
4. Dr. M. B. Prabhu: Retained placenta—an analysis of 5-year statistics at the Women and Children's Hospital, Madras.
5. Dr. D. M. Satur: Causes and treatment of retained placenta.

5-15 P.M. Tea to the delegates at the Madras Medical College grounds by Dr. A. L. Mudaliar.

FRIDAY, 3RD JANUARY, 1936.

7-30 to 9 A. M. Business meeting.

10 to 12-30 A. M. *Obstetric Section.* Discussion on pelvic disproportion.

1. Dr. N. A. Purandare: Some principles in the management of contracted pelvis.
2. Dr. A. B. S. Smith: Pubiotomy in the treatment of pelvic disproportion.
3. Dr. Miss Telang: Review of cases of pelvic disproportion at the Cama Hospital, Bombay.
4. Dr. E. Achyuta Menon: Management of labour in contracted pelvis.
5. Dr. P. Venkatagiri: Management of labour in contracted pelvis.
6. Dr. I. S. Scudder: Infected Caesarean section.
7. Dr. Venkataswami: Lower segment Caesarean section.

2 to 4-30 P. M. *Obstetric Section* (continued.)

1. Dr. A. S. Mannadi Nayar: Blood chemistry of eclampsia.
2. Dr. Chakravarthi: Clinical aspects of placenta praevia and its management.
3. Dr. Thampan: Some aspects of accidental haemorrhage, with an analysis of 200 cases.

Gynaecology Section.

1. Dr. Subodh Mitra: Positive 5-year cure cases of carcinoma cervix uteri.
2. Col. V. B. Green-Armytage: Implantation of the tubes in the uterus for sterility.
3. Dr. Miss Dadhabhai: Functional uterine haemorrhage—a study of 500 cases.
4. Dr. A. L. Mudaliar: The use of radium in gynaecological practice.

8 P. M. Dinner at the Madras Medical College. This will be a subscription dinner to the delegates (Rs. 3 and Rs. 5 for vegetarian and non-vegetarian menu respectively). Delegates wishing to join

the dinner are requested to communicate with and get the tickets from the secretaries, Drs. M. B. Prabhu and T. Lazaro.

SATURDAY, 4TH JANUARY 1936.

10 to 12-30 A. M. *Gynaecology Section.* Discussion on displacements of the uterus.

1. Dr. H. Lazarus: Displacements of the uterus—treatment.
2. Dr. I.S. Scudder: Pleating fixation operation for retroversion.

2 to 4-30 P.M. *Maternity and Child Welfare Section.*

1. Dr. Rosenthal: Antenatal care.
2. Dr. Chinnappa: Organisation and conduct of child welfare schemes.
3. Dr. Miss A. David: The place of vitamin therapy in antenatal work.
4. Dr. M.B Prabhu: Infant mortality—its causes and prevention.
5. Dr. A. L. Mudaliar: The value of post-natal care.

Visits to the following institutions in the city are arranged during the morning hours: Government Hospital for Women and Children, General Hospital, Barnard Institute of Radiology, Government Ophthalmic Hospital, Medical College. Excursions to places of interest such as Mahabalipuram will be arranged on Sunday the 5th January 1936.

CANCER HOSPITAL FOR BOMBAY. The Trustees of the Sir Dorabji Tata Trust have decided to erect a modern cancer hospital in the Parel Hospital area, making use of the latest methods employed in cancer treatment. While radium will of necessity play an important part in the treatment, the new hospital will not simply be a Radium Institute. Surgery, X-rays and all other known methods of effective treatment will be employed. The hospital will be an independent institution specialising in the treatment of cancer and allied diseases. Provision is being made for the treatment of both free and paying patients; an out-patients' department will also be maintained. The Trustees have appointed Dr. John W. Spies, A.B., M.Sc., M.D. (Harvard) as the first Director of the Hospital. He has had extensive experience in pathology, surgery and radiology. In addition, Dr. Spies has devoted much time to research and has written numerous articles. The new Director was recently in charge of the Tumour Clinic attached to the Department of Surgery of the Peiping Union Medical College, Peiping, China. Previous to that he was associated with the Memorial Hospital of New York City, America's foremost cancer hospital, and was a Member of the Faculty of the Yale University Medical School. Dr. Spies has travelled extensively in order to acquaint himself with the leading American, European and Japanese medical centres—especially those renowned for cancer work. Dr. Spies is working on the provisional plans for the new Hospital, and it is expected that the centre will begin work some time in 1937.

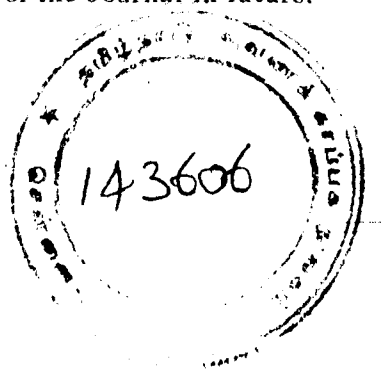
SPLANCHNIC SECTION FOR HYPERTENSION. The term "essential hypertension" implies that so far as our knowledge yet goes, the disorder is not secondary to renal disease or other focal organic lesion. Its treatment must therefore be tackled on the assumption that there is no need for the high blood pressure, and since a persistently raised pressure must eventually bring about degenerative changes in the vessels, it is right and proper to reduce it if we can, says the *Lancet*, March 16, 1935. Seeing that essential hypertension seems often to run in families, and that constitutional characteristics are held to play a part in its actiology, local changes in the vasomotor centre have been postulated as its underlying cause. And since the vasomotor centre acts principally through the splanchnic reservoir, many surgeons have tried to lower the blood pressure by section of the splanchnic nerves. Craig and Brown say that although there was an immediate fall in blood pressure after cutting the major and minor splanchnic nerves on both sides this improvement was not maintained. Adson has carried the procedure a step father by cutting the anterior spinal nerve roots from Th. 6 to L. 2 on both sides, thus paralysing the abdominal wall as well; he found a definite fall of blood pressure after the operation and that it fell still lower when the patient stood upright. It is natural to look for excess of pressor substances or deficiency of depressor substances in the blood stream and not to disease in the blood vessels themselves. Though hypertension could result from depressed activity of the carotid sinus or from deficiency of a depressor substance normally produced in the pancreas, more attention has been given to the possibility that a pressor substance is formed in excess by the suprarenal glands. Those who have investigated the blood of patients with hypertension are unable to agree that these people show it in abnormally large, quantities; and despite the observation that tumours of the suprarenal medulla may be associated with high pressure, Crile has found that denervation of the suprarenal glands is valueless except in so far as it may prevent the violent exacerbations produced in these patients by emotional stimuli. A more encouraging report comes from De Courcy and his colleagues who noted improvement after removal of ¾ of both suprarenal glands. They claim to have observed hyperplastic changes in the medulla of these excised suprarenal glands and if their therapeutic success and their pathologic findings are confirmed, a real advance may be made in our understanding of this baffling disorder.

TRANSPLACENTAL FOETAL INFECTION WITH MALARIA. Summarising his observations on this subject in the *Journal of the Ceylon Branch of the B.M.A.*, July 1935, G. A W. Wickramasuriya says that transplantal passage of malarial parasites from the maternal to the foetal circulation does occur, and depends on three factors: (*a*) Type of infection, transplacental infection occurring chiefly, if not entirely,

in malignant tertian infections. (*b*) Infection and disease of the placenta; the more severe the placental infection and the longer it has persisted, the greater is the probability of transmission of parasites. The placental barrier is broken through as a result of pathological changes induced as a result of massive infection of the placenta with parasites. (*c*) The efficacy of the treatment adopted; transplacental infection is most likely to occur in neglected and untreated malignant tertian infection. Moreover, post-mortem examination of still-born foetus born of mothers suffering from acute malignant malaria often shows enlargement and pigmentation of spleen indirect evidence of malaria of the foetus. In positive cases it is at times very easy to demonstrate parasites in the fresh foetal brain squash without any staining, and living and dead parasites are easily seen in the brain capillaries.

THE MADRAS MEDICAL COUNCIL'S NOTICE. The Registrar writes to us as follows:—It has come to the notice of the Madras Medical Council that proceedings of meetings of some District Medical Associations in the mofussil are published in the lay press. The question whether the publication of such reports in non-professional journals is permissible has been considered by the Council and it has been decided that such publication is, in the opinion of the Council, contrary to the Code of Medical Ethics, excepting in a few cases specially relating to Health Propaganda. The Madras Medical Council therefore desires the Secretaries of all Associations to be informed that they should label discussions and lectures in their Agenda as not for lay press publication whenever this course is considered desirable from an ethical point of view. (We do not think this notification is necessary.—*Editor*).

THE EDITOR'S THANKS.—The Editor conveys the season's greetings to all the subscribers, contributors, advertisers, and other readers of this Journal, thanking them for their co-operation hitherto and praying for their continued interest and assistance in the conduct of the Journal in future.



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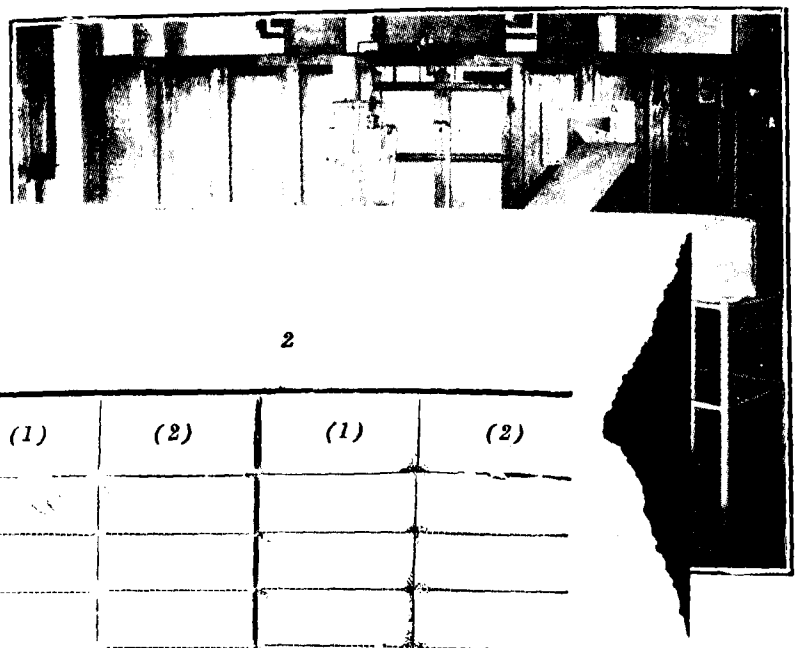

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