

MEDICO-SURGICAL SUGGESTIONS

Vol.

JUNE 1936

No. 6.

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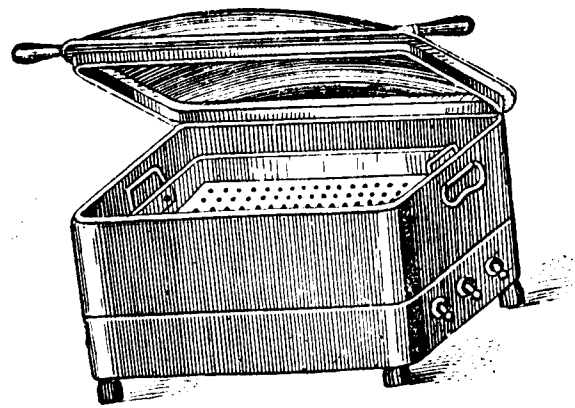
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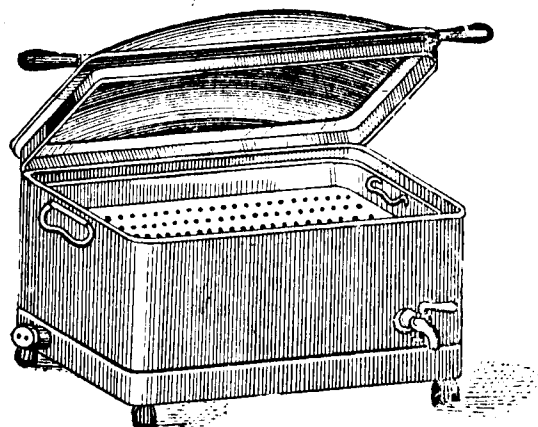
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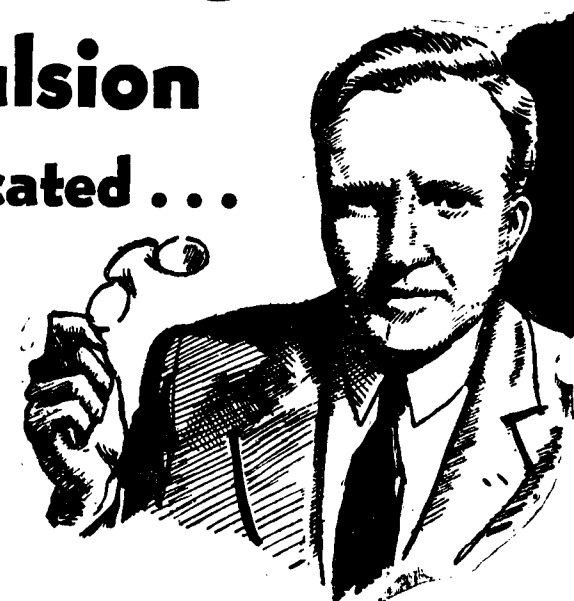
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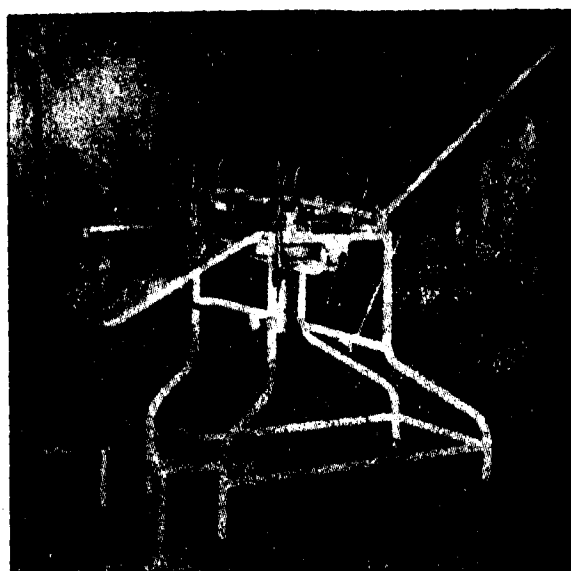


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MEDICO-SURGICAL SUGGESTIONS

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VOL. V

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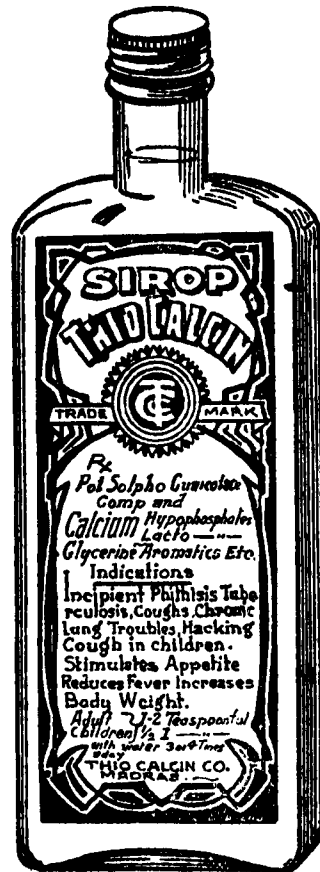
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Leading Articles

FOOD, NUTRITION AND DISEASE

BY RAO BAHADUR M. KESAVA PAI, M.D., O.B.E.,
Madras.

If there be one subject which is more important than most others in the progress of our country today it is that of nutrition. There is an erroneous impression even amongst the educated classes that different countries, races and climates need entirely different kinds of food for the maintenance of health and vigour. Whilst there is no denying the fact that a difference of climate demands a certain difference in the diet, it is idle to insist that the diets which we have been adopting in different parts of the country are the ideal ones for each of us, simply on the flimsy ground that they have been found to be so by the experience of centuries. Time was when, due to limited communications and climatic handicaps on the production of the various crops, the different parts of the country had to adjust their diets to their agricultural and other resources, but with the present day facilities of railways, steamships and fast communications it is quite possible to obtain at reasonable cost any food material from distant provinces and it is therefore mere ignorance and blind adherence to old traditions that makes us cling to our old defective and unbalanced diets.

The object of food is to nourish the body, sustain its structure and function and provide the energy for the work that has to be performed by it. Diet must naturally differ for the different age periods, being of a more easily absorbable type and of a proportionately larger volume for the infant and the growing child than for the adult who has ceased to grow.

There is no single foodstuff in nature which can supply all the wants of the body, except in the infant, who can live on mother's milk alone, being a perfect food for its sustenance. For the growing child, and growing and grown-up adult there must be a correct

combination of different foodstuffs constituting what is known as a balanced diet. The proteins, carbohydrates, fats, mineral salts, vitamins and other substances essential for the formation of special tissues must be present in the diet in proper proportions, along with pure air and pure water. A certain amount of undigestible roughage or chaff is also essential for the stimulation of intestinal movements and discharge of effete material.

The value of a balanced diet can be demonstrated by the observations that have been made amongst the different races of India as also by laboratory experiments on animals. It has been proved time and again that the dietetic habits and the physique of the various races of India have a direct bearing on each other, climate, surroundings and infection playing quite a minor part. The races of Northern India, especially the north western part of India, who are eaters of wheat along with dhal, milk, vegetables and fruits being physically much more efficient than the Bengalis and Madrasis, who are predominantly a rice eating race with a poor protein content in their dietary and have the poorest physique. The Sikhs and the Pathans of North West India are the hardiest and finest of the Indian races, whilst the Bengali and the Madrasi, though full of intellectual vigour, are physically weak and non-sustaining races. Sir Robert McCarrison proved experimentally that sets of white rats fed on the various diets of India varied in their growth, physique, health and mortality with the kind of diet on which they were fed. The Sikh and the Pathan rats were the healthiest and largest of all, the Maharatta rat of middling size and vigour and the Bengali and Madrasi rats the poorest and least healthy. Maternal and infantile mortality and disease were rare amongst the Sikh and the Pathan rats whilst stunted growth, and diseases mostly of the gastro-intestinal tract and of the lungs were common amongst the Madras rats. Ulcer of the stomach which is much more common in South India than in the Punjab and the North West Frontier was also found to prevail more amongst the Madrasi rats than amongst the Punjabi rats. A similar difference was noticeable as regards the incidence of stone in the urinary tract amongst the two races of India as well as in the two classes of rats.

Plenty of fresh air and pure water constitute scientifically important constituents of our food, though popularly we do not class them as such. An abundance of drinking water to flush the skin and the kidneys is essential for good health, and pure air and its oxygen constitute the purifiers of the blood.

Proteins or nitrogenous foodstuffs are necessary for growth and repair. Proteins are found both amongst animal and vegetable foods including meat, eggs, fish and milk, legumes, cereals, seeds, nuts and vegetables. Animal proteins are said to be more nourishing than vegetable proteins, but milk which is really an animal food

along with vegetable proteins more or less suffices in a tropical climate like ours. Wheat, ragi, dhal, soya beans, legumes, nuts, with milk and milk products can, in proper proportions, make up a perfect and well balanced diet, without the flesh of animals.

Several minerals, about 20 in number, particularly calcium, phosphorus, iron and iodine are essential for the formation of the body tissues and fluids, especially in pregnant and nursing women and growing children. Milk, nuts, eggs, cereals and greens are all rich in these minerals, whilst condiments, cloves, ginger, pepper, &c., are said to be rich in iodine.

Vitamins whose great importance in food has been proved in recent years are at least of five different kinds as known at present. Vitamin A which is essential for the prevention of infections is present in milk, cod-liver oil, animal fats like butter, ghee, eggs, fish and greens. Vitamin B prevents the disease of muscles and nerves and of the alimentary and nervous system. Vitamin B is found in egg yolk, yeast, the polishings and bran of rice and cereals, in wheat and in greens. Vitamin C when deficient gives rise to anæmia, bleeding, fragility of bones and other degenerations of tissues. This vitamin is present in vegetables, orange and lemon juices, green chillies and fruits in general. Vitamin D when deficient causes rickets, caries of teeth, etc. It is present in cod-liver oil, fish, butter, yolk of egg, etc. Vitamin E is said to nourish the productive system and is present in wheat germ, milk, vegetables and cereals. A diet which is deficient in vitamins is apt to cause the respective diseases mentioned above, particularly in children and growing adults.

Statistics of different countries have shown that intestinal and lung diseases are mostly caused by defective diets. Thus tuberculosis is said to be twice as prevalent in Madras as in the Punjab. Diseases of malnutrition like leprosy, ulcer of the stomach, rickets and anæmia are much more common in South India than in North West India. Infantile mortality is also being recognized as due to vitamin deficiency with its resulting gastro-intestinal and pulmonary disorders and is greater in Madras than in the Punjab. Under-weight, under-growth, digestive disturbances, anæmia, rickets, caries of the teeth, and tuberculosis are all the result of malnutrition and are more prevalent amongst races with a diet deficiency. Maternal and infantile mortality could be controlled by the regulation of the diet of the mother and the infant.

I shall now indicate in what way the Madras diet is defective and how it can be made a balanced diet. The rice diet of the Madrasi is far too deficient in protein and since the rice now consumed is milled and thereby deprived of the bran which is rich in vitamin B and some protein, the rice eater of to-day is much worse off than his ancestor of the last century. The amount of milk and

vegetables consumed by him is far too little and if he happens to be a vegetarian he cannot get the benefit of the protein of the meat, fish or egg which he would otherwise be having. Hence the top heavy brahmin of Southern India who easily tops the list in the Universities and competitions for the Civil Services but has no sustaining power and cannot hope to be even a C class recruit to the army. The Telugu recruit fared hopelessly in the Indian Army 50 years ago and had to be eliminated. The Tamilian recruit is necessarily from the meat eating classes and even then is unsatisfactory. If the Madrasi could shake off his conservatism and free himself from his old tradition in the matter of food, using wheat, ragi, dhall, soya beans, rice bran, and a little extra milk and vegetables, in the place of the bare rice and rasam with a pittance of milk and vegetables, he is bound to come up to the physical standard of the stronger races of the North in the course of a generation or more. It is the difficulty of making him break tradition that hampers his physical progress. Wheat and ragi are nowadays available cheaper than rice. He has only to take the trouble of studying how to eat them.

As regards infants and children the evil has crept into modern Indian society of using the patent artificial foods which have ruined the constitution of the child and the finances of the parents which are already on the debit side. Mother's milk is the best food for the infant and if the mother cares to study dietetics and feed herself better, the baby shall not want. If for any reason the mother's milk proves insufficient, cow's milk is the best substitute as it is rich in protein and vitamins and salts. The addition of fresh wheat and ragi as the child grows will again produce a higher nutritive effect at a lower cost than the patent foods. Rice is a poor food for the growing child, perhaps poorer, than to the adult. If our babies could be fed on cow's milk, fresh whole wheat meal and ragi flour with a little vegetable and fruit juices, the next generation should be a healthier and stronger one and there would be less of infantile disease and mortality.

A meat diet is not quite essential for health especially in a tropical climate like ours. Sir Robert McCarrison has demonstrated by experiments and by observation that a vegetarian diet is sufficient for the maintenance of good health and sustaining power. In fact he considers the Sikhs and the Pathans of North West India are amongst the finest races of the world. The Mahratta diet is a medium diet consisting of wheat, rice, dhall, ghee, milk and vegetables. That accounts for the health and martial spirit of the Mahrattas. The ragi eaters of the Mysore plateau and the Ceded Districts are healthier and hardier than the rice eating Madrasi. Will the Madrasi take a lesson?

In cooking food it is conducive both to economy and to nutritive value to steam-cook all articles of diet, instead of boiling and

straining them. We in Madras have yet to learn the value of this great rule, which prevents waste and adds to the nutritive value of the material ingested by us.

In the inculcation of the correct standards of food and nutrition we, medical men, have an important duty to discharge as social workers, by propaganda work amongst the public and steady and persevering methods of educating the families under our charges. A great deal of disease, physical incapacity and financial hardship can be averted by our propaganda efforts. We ought to be more of preventive men than of curing physicians and surgeons. Remembering that the high incidence of disease and mortality in India is due either to infection or to malnutrition, more to the latter than to the former, our social duty is evident *i.e.*, the education of the masses in correcting their present ill balanced diet. We are now on the threshold of constitutional changes and if we wish to put our house into thorough order and produce an A1 nation from a C3, which I am afraid we are at present, and if we intend to develop the martial spirit to guard against aggression from outside and prevent foreign domination which has been our lot for centuries, one of the major items in our constructive programme should be that of improving national nutrition.

[Read before the third annual meeting of the West Godavari District Medical Association, Ellore, on the 29th March 1936.]

AFFECTIONS OF THE LUNGS AND PLEURA

BY S. SANKARA RAMAN, M.B., B.S.,

Cuddalore.

(Continued from the May issue.)

EMPHYEMA.

Primary empyema is rare, except as a result of pneumococcal infection. In the majority of cases, empyema occurs secondarily to a preceding or co-incident infection of the underlying lung such as tuberculosis, abscess, gangrene, etc. It can also arise secondary to a septicaemia, as a lymphatic borne infection conveyed from the abdominal cavity, and as a direct spread from a septic focus in the chest wall. In neglected and untreated cases, pus may track and discharge in various ways. The commonest is rupture through the visceral pleura into the lung, and discharging through a bronchus. The pus may track through an intercostal space, generally the fifth near the nipple, producing an empyema necessitatis. It may pulsate if situated on the left side of the chest. Perforation may occur into the pericardium or into the oesophagus with the formation of a pleuro-oesophageal fistula. The diaphragm may be perforated and a sub-phrenic abscess may result.

Treatment. Ascertain the presence of pus, analyse bacteriologically the pus and take a skiagram.

Indications for aspiration: (1) If the patient is too ill to justify thoracotomy or rib-resection; (2) if the pus is thin and partly purulent; (3) if the underlying lung is the site of infection; (4) if the infecting organism is a streptococcus; (5) if the effusion is diffuse.

Indications for thoracotomy. (1) When aspirations repeated every third day fails to cure; (2) in pneumococcal infections.

The treatment of empyema has not kept pace with the advance of surgery in other directions. It is regarded even now by many that empyema is like an abscess to be opened as soon as recognised. Before beginning to treat a case of empyema it is necessary to distinguish between two classes of empyema. (1) Those in which pus is free in the pleural cavity; (2) those in which it is shut off by adhesions. In pneumococcal cases, the effusion does not become purulent until after the pneumonia has subsided and it gets walled off from the general pleural cavity by adhesions. In streptococcal cases, effusion develops early and is copious whilst the lung is still the seat of bronchopneumonic changes, and the fluid is free in the pleural cavity. Hence an attempt to drain the pleura in a streptococcal case produces an open pyo-pneumo-thorax, with all its changes of collapse of the lung which increases the respiratory movements of the opposite lung, which, in turn, transmits it to the mediastinum, mobilising it. This mobilised mediastinum disturbs the heart. The collapse and mediastinal mobilisation add an additional burden to the lung which is crippled by bronchopneumonic changes. The Empyema Commission in America showed that if aspiration is done at first, and time allowed for formation of adhesions thus localising the empyema and then operation is performed, there is a diminished mortality of 26 per cent, the death rate falling from 30 to 4.3 per cent. But early operation is desirable in pneumococcal cases to prevent them from becoming chronic empyemas.

Chronic empyema: results from (1) too early opening in streptococcal cases, (2) delayed opening where the thickened pleura prevents expansion of the lung; (3) insufficient drainage, (4) foreign body, (5) formation of a bony ring around the drainage tube; (6) pocketing, and (7) broncho-pleural fistula.

Treatment: Another incision in a more suitable place combined with irrigation with Dakin's fluid may bring about a cure in the majority of cases. Estlander's operation though successful with small cavities does not allow sufficient mobilisation of ribs in larger ones.

The Fowler-Delorme operation of decortication aims at reaching the plane between the pleura and adherent membrane and removing the latter. When the exudate cannot be removed, Ransohoff's operation of gridironing the thickened membrane by incisions down to the level of the pleura at right angles to one another and one-fourth inch apart, is to be performed. Young points out that these operations

are serious in patients already debilitated by long discharges and with diminished vital capacity and must not be undertaken lightly.

ACUTE PNEUMOTHORAX.

This may occur—

- (1) Through the visceral pleura. In the majority of cases, it is caused by rupture of a sub-pleural tubercular focus. It may also occur as a result of injury, abscess of lung, carcinoma and rupture of an emphysematous bulla;
- (2) through the chest wall, as a result of penetrating wounds or a discharging empyema necessitatis;
- (3) through the mediastinum, by ulceration of an oesophageal growth;
- (4) through the diaphragm, from some hollow viscus, as perforating gastric or duodenal ulcer.

If the opening is in the chest wall, the intra-pleural pressure is equal to that of the atmosphere. If the opening is in the lung (1) the opening may remain patent, thus keeping the pressure equal to that of the atmosphere; (2) the opening may be valvular, permitting entry of air into the pleura during inspiration and preventing its leakage during expiration thus keeping the pressure higher than that of the atmosphere; (3) the opening may become sealed when the pressure will be equal to, greater than, or less than that of the atmosphere, depending on the amount of air in the closed pneumothorax.

Symptoms: The onset is sudden with severe pain. Dyspnoea is present. Cold clammy sweats, rapid pulse and even sudden death occurs, but the symptoms usually subside after a few hours. The alae nasi are working hard, rapid shallow breathing continues, cardiac impulse is displaced away from the affected side, there is no chest movement, vocal fremitus is absent, percussion is tympanitic, and auscultation shows absence of breath sounds. Bruit de airain is a valuable sign.

Treatment: In severe cases, morphia $\frac{1}{4}$ grain immediately. Saugman's pneumothorax needle must be inserted in the fifth space in the mid-axillary line, and left as long as air can be heard whistling out. When the symptoms subside without complications, the patient is kept in bed until the collapsed lung re-expands. Afterwards, it is safe to let him return to the ordinary mode of life. When the patient remains in good health and no effusion occurs, but the lung does not re-expand, remove the air with a pneumothorax apparatus until there is negative pressure in the thorax.

If effusion occurs but is clear and sterile, treat it as one of pleural effusion.

If effusion occurs and becomes purulent, pus must be aspirated and the pleural cavity washed with 1:4000 Methylene blue solution. The dye may stain the sputum blue if the perforation in the visceral

pleura is not closed. Unless it is made to heal there is absolutely no chance of saving the patient.

If the visceral pleura is closed, the pus is aspirated and replaced by sterile air to leave a negative pressure to allow the lung to re-expand.

If aspiration and gas replacement done thrice do not expand the lung, the cavity must be washed with Dakin's solution.

If in spite of it pus re-forms, gelatino-thorax may be induced. 2 per cent solution of gelatine in normal saline is mixed just before use with Acriflavine to a dilution of 1: 2500.

Thoracoplasty is recommended in intractable cases, but the result of this treatment is far from satisfactory.

Selected Articles

INTESTINAL TUBERCULOSIS

BY Y. G. SHRIKHANDE, B.Sc., M.B., B.S., T.D.D. (Wales),
King Edward VII Sanatorium, Bhowali, U. P.

Infection of the alimentary tract may occur (1) by direct invasion of the lumen or (2) through the lymphatics or (3) by the blood stream.

Tuberculous ulceration of the intestine which occurs so frequently during the course of pulmonary tuberculosis is the result of direct infection from swallowed sputum containing tubercle bacilli. Infection by the blood-stream is very rare but may occur in miliary tuberculosis. Animal experimentation has shown that hæmatogenous infection is followed by the development of tubercles in the walls of the alimentary tract. Such lesions should theoretically be isolated and widely scattered throughout the alimentary tract. In practical experience, however, this is not found in intestinal tuberculosis where the lesions are invariably located in the vicinity of the ilio-coecal region. Infection through the lymphatics may take place by local retrograde extension from tuberculous mesenteric glands and is more common than is supposed. Cases are met with where with an advanced caseous glandular lesion lines of recent tuberculous lymphatics are seen extending to the gut and small recent ulcers are noticed within the latter. Stewart believes that the spread in these cases has been centrifugal and he accounts in this way for a localised group of ulcers high up in the ileum and jejunum with no evidence of lesions in the more usual sites.

Pathologically and clinically, however, there are only two important varieties of intestinal tuberculosis which may concern us:

- (1) Primary, hyperplastic or proliferative type.
- (2) Secondary or ulcerative type.

It may be mentioned here that by primary tuberculosis of the intestine is understood that the intestinal mucous membrane is the

portal of entry of the tubercle bacillus which passes through it without causing any apparent injury and no ulceration occurs at this stage. The first localisation of the bacilli takes place in the mesenteric lymph nodes where tubercles are formed causing sensitisation of the body and serving as depots from where further dissemination can take place. Ulceration, on the other hand, is a manifestation of reinfection and it is, therefore, necessary that the focus of primary infection is established either in the intestinal lymphoid tissue or elsewhere before ulceration of the intestinal mucous membrane can take place.

PRIMARY TUBERCULOSIS OF THE INTESTINE.

This is a comparatively rare type of tuberculous lesion and is essentially proliferative in character. Owing to the massive infiltration of all the coats of the intestine, a tumour is formed which may be indistinguishable clinically from any other granuloma or from malignant disease. Ulceration may or may not be present and, if present, does not form a conspicuous feature of the disease. It is often impossible to be sure of the nature of the disease even after the operation until, (a) several sections of the tumour or, (b) neighbouring lymph glands or, (c) with a negative histology, positive inoculation results are obtained.

The site of the lesion is the ileo-caecal region, the most frequent site being the caecum, but the terminal ileum and the ascending colon may also be involved. Occasionally, the appendix is also affected.

The important factors concerned in the explanation of this peculiar lesion probably are: (a) a fairly high grade immunity to tuberculous infection, (b) infection by a small number of bacilli, and (c) infection by bacilli of low virulence, in many cases bovine in origin.

Two important phenomena are associated with the condition, namely (a) a large tumour is palpable in the caecal region and (b) signs of chronic intestinal obstruction manifest themselves. The treatment consists in resection or short-circuiting operation.

The disease is said to be fairly prevalent in Europe and less so in America. It is rare in India probably because bovine tuberculosis, as has been pointed out by Lankester is not common in this country and milk which is the main source of the bovine bacillus is not taken raw. Further, judging from the universal and abominable habit of spitting anywhere and everywhere which is so prevalent in this country, the human bacillus would appear to be the commonest source of infection and intestinal tuberculosis is, therefore, found to occur as a very common complication of and secondary to the disease in the lungs.

SECONDARY INTESTINAL TUBERCULOSIS.

This is a very common complication of pulmonary tuberculosis and is the result of swallowing sputum consciously or unconsciously.

Calmette, however, believes that the constant elimination of the bacilli in the bile may also furnish the source of infection to the intestinal tract.

Secondary tuberculosis of the intestine is a manifestation of Koch's phenomenon and is therefore, frankly ulcerative. Owing to the sensitisation of the body that has already taken place by the presence of the disease in the lungs, the chief struggle for mastery has been transferred from the regional lymph nodes with which the body is naturally endowed, to the cells of the mucous membrane of the intestinal tract on which has now been conferred the specific defensive role as a result of the primary infection in the lungs. There is not much of reactive fibrosis and the muscular coat is rarely penetrated completely in this type. Intra-peritoneal perforation is uncommon owing to the formation of adhesions between the coils of the intestine. In contradistinction to the hyperplastic type, this variety rarely gives rise to any serious obstruction partly due to the destructive character of the lesion and partly because the patient usually dies before cicatrization can take place to any appreciable extent.

The most frequent *site* of affection is the ileo-caecal region namely the terminal part of the ileum, the caecum and the first part of the ascending colon. The probable reasons for this are:—

- (a) Tuberculous disease first attacks the lymphoid tissue and this in the form of solitary follicles and Peyer's patches is abundant in the terminal part of the ileum.
- (b) Absorption of the last products of digestion takes place in the terminal ileum and the infected material comes in direct contact with the mucous membrane.
- (c) The bowel contents remain for quite a long time in the ileo-caecal region thus giving an opportunity to the bacilli to cause infection in this region.

It is well to bear in mind that the lower portion of the ileum, the caecum and the ascending colon have two points in common. Each represents the part of their respective portions of the digestive tract where slowness of the passage of contents and absorptive activity are most pronounced.

Pottinger has suggested the possibility of trophic disturbances in the cells of the intestinal wall brought about by the prolonged stimulation of the sympathetic and vagus nervous systems which lower their resistance to infection.

It is interesting to mention here that tuberculous ulceration of the intestine spreads at right angles to the long axis of the bowel in contrast to the ulceration of typhoid fever. The reason is that tuberculous infection spreads along the course of the lymphatics which run around instead of lengthwise of the intestine.

The problem of secondary intestinal tuberculosis is exactly what the problem of pulmonary tuberculosis was some fifty years ago; its onset meant the rapid termination of the life of the patient. This view-point may hold good even to-day if we were to wait for its diagnosis until the classical symptoms like tenderness and pain, diarrhoea and emaciation manifest themselves. These are not early, diagnostic signs but late prognostic omens. They should be considered as late signs of intestinal ulceration, as bubbling rales and cavities are of pulmonary tuberculosis. If we are not aware of this complication until these late symptoms have been established, we are no wiser than our forefathers and the outlook must still remain hopeless. For here, as in pulmonary tuberculosis, the diagnosis to be of any value must be made early and to make an early diagnosis we must consider what may be called the borderline symptoms.

It would be fortunate if there was any pathognomonic sign of intestinal tuberculosis but unfortunately there is none. The symptom-complex is not constant; in fact, it is quite variable. One may have violent cramps, another only abdominal consciousness; one may have diarrhoea, another constipation; one may have anorexia, another good appetite.

Moreover, some of these gastro-intestinal disturbances may be due to causes other than tuberculous ulceration. (a) They may, for instance, be due to the sedentary life and generous diet employed in the treatment of pulmonary tuberculosis. Over-feeding and other errors in diet may be responsible for dyspepsia or inactive life for constipation. The unfortunate tuberculous patient must be pardoned if he suffers from ills not necessarily tuberculous in nature. Morriston Davies has shown that intestinal stasis not infrequently gives rise to gastro-intestinal disturbances and is brought about by the decomposition of food-stuffs and the production of amino-acid derivatives. (b) Diarrhoea may be due to the insufficiency of digestive glands or to the administration of certain drugs for treatment. (c) Amyloid disease of the intestine may cause digestive disturbances, particularly diarrhoea. (d) Digestive disturbances may also be due to the toxæmia of pulmonary tuberculosis or be 'reflex' in origin and produced, as shown by Pottinger, by the stimulation of the peripheral ends of the sympathetic and vagus systems.

But all said and done, intestinal ulceration remains the sinister complication of pulmonary tuberculosis and the fight is often fought and won or lost not so much in the lung as on the gastro-intestinal front. Old sayings like "stomach is the greatest friend of the consumptive" or "poor eaters do badly" or "a flux of the bowel leads to a fatal outcome" have apparently been coined to stress the importance of the alimentary tract in the treatment of pulmonary tuberculosis.

SYMPTOMS.

The earlier symptoms of intestinal tuberculosis are generally due to lesions in the small bowel, the later to lesions in the large bowel. Ulceration in the small bowel tends to retard the passage, in the large bowel to accelerate it. The earlier symptoms are gradual and slight change from the patient's normal temperament towards nervousness and irritability; from usual and normal appetite towards slight impairment and from usual bowel-habit towards constipation. Nervousness, slight impairment of appetite and constipation beyond usual, form, therefore, the early and most useful diagnostic triad. An ulcer in the small intestine produces spasm and it is to this spasm that the earlier symptoms are due. Later, when the large intestine also gets involved diarrhoea with foul-smelling stools results. Constipation is thus a special feature of ulceration in the small bowel while diarrhoea is a symptom of ulceration in the large bowel. Alternating constipation and diarrhoea indicate ulceration in both the tracts. Tenesmus occurs when the rectum is also involved.

In order to understand the symptom-complex of intestinal tuberculosis it must be borne in mind that the underlying pathology is an ulcerative entero-colitis. The physiological response to ulceration is increased irritability of the bowel. This increased irritability is manifested by hypermotility. In the small intestine, moreover, ulceration produces spasm. Hence constipation and diarrhoea become important factors. The stools are soft and unformed, often liquid and may contain mucus, blood or pus.

With ulceration of the intestines comes peritoneal irritation and this results in abdominal pain, abdominal tenderness and abdominal rigidity. The pain tends to come on shortly after eating and may be colicky in character. Tenderness is first noticed over the ileo-caecal region and later over the whole abdomen. Similarly, rigidity commences in the right iliac fossa and spreads over the entire abdomen. The mesenteric glands may be palpable in the right iliac fossa or round about the umbilicus. While it is generally true that secondary tuberculous involvement is not accompanied by the enlargement of the regional lymph nodes, this rule does not hold if the infection is sufficiently severe. For this reason the mesenteric lymph glands are sometimes found to be enlarged and even caseous.

As a consequence of the ulcerative entero-colitis certain constitutional signs and symptoms result such as slight elevation of temperature without any increase in the pulmonary symptoms or failure to improve and gain in weight when the pulmonary lesion is quiescent or shows signs of improvement. Not infrequently, the regular curve of temperature in pulmonary tuberculosis becomes irregular and varies from day to day—becomes regularly irregular, if it may be so described. Digestive symptoms also appear such a

nausea, vomiting, flatulence, discomfort and change in the bowel-habit, *i.e.*, a patient who has been constipated shows an increased frequency of stools or a patient who has had normal bowels becomes constipated. In the opinion of Bandelier and Roepke, diarrhoea occurring at night in phthisical patients is very suggestive of intestinal tuberculosis. Owing to the increased irritability of the intestine the food taken early in the day is discharged in the night and the movements continue during the day.

DIAGNOSIS.

The methods of diagnosis of intestinal tuberculosis may be discussed under three headings—(a) clinical, (b) laboratory, and (c) radiological.

(a) *Clinical diagnosis*:—The outstanding signs and symptoms have already been mentioned. One thing must be emphasized, namely, the earliest symptoms must be anticipated. There is a wide difference between the normal digestive functions of even healthy persons. Small disturbances in these may easily, therefore, be attributed to lapses on the part of persons from their normal diets and when brought about in patients by tuberculous ulceration of the intestine are considered to be due to a lack of control of the palate. The patient, therefore, fights shy of mentioning them to his physician until they are well established. It is up to the physician and not the patient to be on the look out for them to make a diagnosis. Similarly, slight deviation from normal temperament towards nervous irritability is attributed to lapses in self-control. When a patient who is usually placid begins to get irritable, nervous and querulous the physician must think of intestinal tuberculosis as a likely cause. It is an old dictum that the disease above the diaphragm makes a patient optimistic while the disease below it makes him pessimistic.

(b) *Laboratory diagnosis*:—The laboratory diagnosis of intestinal tuberculosis is notoriously uncertain. The presence of tubercle bacilli in stools is not proof positive of intestinal ulceration in the presence of active pulmonary tuberculosis. The sputum may be swallowed consciously or unconsciously. Women are the worst culprits in this respect. They swallow the sputum deliberately either from indolence or from an aversion to the spitting flask. The presence of pus and blood in stools is presumptive evidence when the patient is suffering from pulmonary tuberculosis and gastro-intestinal symptoms are present. Occult blood, when found, is a fairly strong evidence but need not be a constant finding. The blood picture usually shows a moderate amount of leucocytosis but this may be due also to the presence of cavity in the lung. Tuberculin test may cause some increase of abdominal pain but aids little in diagnosis.

(c) *Radiological diagnosis* :—The first worker to use the radiological method in the diagnosis of intestinal tuberculosis was Stierlin who, in 1911, showed that in ulceration of the ileo-caecal region the opaque meal is hurried over this portion of the gut throwing little or no shadow on the film six or seven hours following the ingestion of the meal. Pirie, in 1917, recorded somewhat similar findings. But no serious attention was given to this method of diagnosis until, in 1919, Brown and Sampson worked it out by X-raying the alimentary tracts of all patients in the Trudeau Sanatorium.

In this method of diagnosis fluoroscopic observations are made every half-hour or so from the seventh to the tenth hour following the ingestion of the barium meal and finally at the twenty-fourth hour. The patient is instructed not to use any laxative for thirty-six hours prior to the ingestion of the barium meal in order to exclude the possibility of any hypermotility from its use and he is also advised to abstain from eating or drinking anything in the interval between the barium meal ingestion and the first fluoroscopic observation to enable to estimate the emptying time of the stomach. The barium enema may be given a day or two later to confirm the barium meal findings but is not absolutely necessary. Films are usually taken when there is most to record, namely, at the seventh, tenth and twenty-fourth hour.

It has already been shown above that pathologically the caecum and ascending colon form the chief storm-centres of ulceration, at least as far as the large bowel is concerned. A well rounded smooth-walled caecum still fairly filled after 24 hours with the tail of the meal gives a normal barium appearance to establish a negative diagnosis. On the other hand, a ragged, dwarfed, shrivelled caecum or a caecum that does not fill or shows filling defects or that empties quickly and seems intolerant of barium pretty much makes the diagnosis. Variations at the right or ascending side of the large bowel are more significant than at the left or descending side. Gastric retention suggests irritation lower down in the small intestine. Segmentation in the lower part of the ileum suggests damming due to ulceration and consequent spasm.

The findings of Brown and Sampson may be summarised as follows :—

- (a) General hypermotility with complete or nearly complete emptying of the colon in 24 hours.
- (b) Failure of the caecum or of ascending colon and hepatic flexure to retain the barium.
- (c) The presence of spasm or spastic filling defects, irregular contour and absence of haustrations in the ascending colon.
- (d) Segmentation of the coil of small intestine with or without dilatation of the coils.

(e) Ileal stasis and gastric retention.

It is important to remember that these findings indicate only the presence and not the nature of ulceration. While hypermotility may be present in other conditions also, its combination with filling defects confirms the presence of ulcerative condition of the intestine. In the presence of active pulmonary tuberculosis the tuberculous nature of these ulcers may be inferred with some degree of certainty. Moreover, other types of ulceration do not occur primarily in the ileo-caecal region. The absence of hypermotility and filling defects excludes for practical purposes the presence of tuberculous colitis unless, of course, the condition has already healed up.

Differential Diagnosis :—Since the direct diagnosis of intestinal tuberculosis is based on the evidence of hypermotility of the intestinal tract, mistakes are likely to be made and the differential diagnosis must be made from other pathological conditions which give the same results. Gauss, Singerman and Black have discussed the differential diagnosis from the following conditions :—(1) protozoal infections, (2) bacterial infection of the colon, (3) tumours of the intestine, (4) disorders in the formation, position and configuration of the colon, (5) subacute and chronic appendicitis, (6) digestive disorders like achylia gastrica or pancreatic insufficiency, (7) nutritional disorders like pernicious anaemia or pellagra, (8) blood dyscrasias like myelogenous leukaemia, (9) endocrine disorders like hyperthyroidism or Addison's disease, (10) cardio-renal disease, (11) industrial poisons like lead and arsenic, (12) surgical complications like nephrectomy or adhesions and (13) rectal disorders like proctitis, fissures etc.

A careful consideration of the clinical features together with the X-ray findings and other necessary tests will, in most cases, differentiate the above conditions from intestinal tuberculosis.

The importance of X-ray in the diagnosis of intestinal tuberculosis cannot be denied. In a proverbially poor country like India however, the radiological method of diagnosis cannot be employed as a routine measure for the simple reason that it is too expensive for the average patient. Moreover, there are still no facilities for X-ray in this country. This does not mean that X-ray should not be employed wherever it is possible to do so. But considering everything greater importance should be attached to physical signs and symptoms.

TREATMENT.

General Measures :—As soon as the diagnosis of intestinal tuberculosis is made, certain general principles must be observed. The patient should be asked to rest in bed as exercise may stimulate the peristaltic movements of the bowel. The rest should be continued as long as active bowel symptoms are present and for some time after. If the lung lesion is active a serious attempt should be made to bring

about a successful control of the pulmonary condition to obviate the possibility of re-infection of the intestinal tract from this source. The patient should be advised to use constantly an abdominal binder covering the whole of the abdomen. This keeps the abdomen warm and protects it from catching chill. Similarly, chilling of the feet should be avoided as it has also been found to act adversely.

Dietetic Treatment:—Tuberculous intestine, like any other organ affected by the disease, requires rest. It has been found that in patients who have an irritable bowel the mere act of eating produces reflex peristalsis of the terminal ileum and colon quite independently of the nature of food taken. Distension of the bowel tends to stimulate the peristaltic movements; consequently food that causes fermentation such as peas, beans, cabbages, potatoes, etc., should be excluded. Milk also causes flatulent distension and diarrhoea in some patients and has to be stopped. Experience has shown that raw eggs sometimes give rise to loose motions. Raw white of the egg is not digested easily and has been found experimentally to stimulate only partly the flow of the gastric juice. On the other hand, cooked white of the egg is easily digested and well utilised by the economy. In the opinion of Fishberg, as long as eggs are not consumed raw they make an excellent food for the tuberculous patient. Foods which leave a good deal of residue in the bowel irritate the ulcerated mucous membrane and cause diarrhoea; such foods should be stopped. Alvarez has advocated the smooth, cellulose-free diet which aims at supplying the necessary nourishment with the least amount of work and irritation to the intestine. It is small in bulk, high in caloric value, fairly varied, easily prepared, palatable and easily assimilated. With cellulose pretty much removed the starches are acted upon more freely. If bulk is needed agar can be added. It seems to lessen flatulence, discomfort and bowel irregularities. Lean meat is the basis of the diet.

In a vast country like India, the selection of diet for a consumptive is a very difficult problem indeed. Normal diets vary with different provinces and many are strictly vegetarians. It is well known that the proteins of vegetable origin are not easily assimilated and vegetable food leaves a great deal of residue behind which stimulates peristalsis.

Lassabliere has recommended a diet exclusively of sweetened condensed milk diluted with four parts of rice water of which a tea cupful is taken every two or three hours, one or two litres being taken in 24 hours. No other food, liquid or solid, has to be taken. The number of stools are said to be reduced in two or three days after which other ingredients may be added gradually to the diet.

Morriess has found bacillus-acidophilus-milk treatment, first described by Rettger and Chaplin, of great therapeutic value. He has

given one pint of milk with 50 grammes of lactose daily with satisfactory results. Sufficient care has to be taken in regulating the quantity of milk and of lactose. In India, sour milk in the form of *dahi*, butter-milk etc., has been used extensively from time immemorial and proves satisfactory where pure milk does not agree with a patient.

Heliotherapy:—Heliotherapy has been used successfully for several years by Rollier in the treatment of surgical tuberculosis. Its value in the treatment of intestinal tuberculosis could not be established as long as no definite method of diagnosis was known in order to decide when the disease in the bowel began or whether or not it was at all present. The X-ray method of diagnosis has, however, made it possible to employ heliotherapy in the treatment of intestinal tuberculosis. Heliotherapy may be natural or artificial. The latter is preferable because it contains pure ultra-violet rays while with the former are associated the luminous rays also. For artificial heliotherapy carbon-arc lamp or mercury-vapour lamp may be employed. The mercury vapour lamp is, however, simpler to use and can be controlled by the patient himself. Advantage must of course, be taken of sun's rays in the absence of ultraviolet-ray lamp.

Limited areas of skin are first exposed to the ultra-violet rays for short periods and then the time of exposures and the extent of the skin exposed are gradually increased in order to avoid severe local or general reactions. It is said that patients whose skin becomes pigmented easily have a better prognosis. The results of treatment by heliotherapy have been reported to be quite satisfactory. Pain, nausea and vomiting are quickly relieved but diarrhoea and digestive disturbances take some time. Better results are said to be obtained when the treatment is commenced soon after the symptoms manifest themselves.

When the desired results are not obtained by heliotherapy, X-ray treatment of the intestinal tract has been suggested. Great care has, however, to be taken in the use of X-ray as it has been shown experimentally that the intestinal mucous membrane is very susceptible to hard and short rays.

Tomato juice and Cod-liver oil treatment:—During his trip to America in 1930, the writer was much impressed to find that tomato juice and cod-liver oil was being employed on a very large scale. This method of treatment is the result of experiments carried out by McConkey who discovered that it was necessary to have a deficiency of vitamin C in diet in order to produce intestinal tuberculosis in experimental animals. McConkey thought that since heliotherapy and cod-liver oil are equally efficacious in the treatment of rickets and since intestinal tuberculosis can be treated successfully by heliotherapy, it was not unreasonable to expect equally good results from the use of cod-liver oil. Cod-liver oil alone gave unpleasant taste and upset many patients but the combination of tomato or

orange juice with the oil gave better results. McConkey believes that the treatment effects some change in the calcium metabolism which brings about immediate relief of symptoms and the healing of ulcers. The treatment is very simple—half an ounce of cod-liver oil in three ounces of fresh tomato juice is taken three times a day immediately after the principal meals. The juice of an average sized orange may be substituted for tomato juice. According to McConkey, this treatment is more economical than artificial heliotherapy and may also prove as a means of preventing intestinal tuberculosis in pulmonary cases. As we know, cod-liver oil contains vitamins A and D and tomato or orange juice contains vitamin C. The treatment is, therefore, a simple high vitamin diet treatment. Our experience with this method of treatment has, however, not proved very encouraging perhaps because the patients treated with it were already on a diet usually rich in fresh vegetable produce and fatty material.

Medicinal treatment:—Drugs of almost every kind have been used in the treatment of intestinal tuberculosis, such as antiseptics, astringents, sedatives, opiates etc., but they have been used more for the relief of symptoms than for the cure of the disease. The list of these drugs is very long and ever increasing but the fact that we have a thousand and one remedies speaks more loudly than words for their failure as a "cure."

Jacquelin has described certain simple procedures such as reaction to litmus paper, time taken for food to pass through the alimentary tract, microscopical examination of stools etc., in order to determine the type of enteritis present and to treat the condition accordingly. He has classified diarrhoea into four groups requiring different methods of treatment:—

(1) *Simple enteritis*.—Accompanied by fever and coated tongue. Treated by dieting, gentle saline purgation and intestinal antiseptics especially lactic ferments. Treatment not to be kept longer than is absolutely necessary.

(2) *Diarrhoea associated with hyper-functioning of stomach, liver and pancreas*.—Best treated by pepsin, pancreatin and soda-bicarb at meal times and small doses of hydrochloric acid after meals. At other times, powdered charcoal, prepared chalk and powdered nuxvomica may be taken.

(3) *Putrefactive or fermentative diarrhoea*.—Treated by lessening the intake of carbohydrates and of meat and eggs.

(4) *Ulcerative Enteritis*.—Characterised by abdominal pain and the presence of blood and albumin in stools and by its intractable character. This is the tuberculous type of enteritis and is treated with antiseptics, astringents, sedatives, kaolin, calcium chloride injections, opium etc.

TREATMENT OF IMPORTANT SYMPTOMS.

(1) *Diarrhoea*.—(a) Calcium chloride has been used extensively and is best given by the intravenous route. The usual dose is 5 c. c. of 5 per-cent solution given once or twice a week but it may be used more frequently and in greater concentration according to the necessity of the case. It is believed that calcium chloride acts on the vegetative nervous system and decreases the peristaltic movements by reducing the irritability of the smooth muscle of the intestinal wall and thus gives rest to the affected part. It also gives relief to pain and bleeding.

(b) *Castor oil*.—Yields good results in some cases. Morriston Davies has spoken of the 'charm' of castor oil in the treatment of intestinal stasis. Sodium Ricinoleate has been used in place of castor oil with better results probably because it is free from disagreeable and nauseating taste and is, therefore, capable of being administered for as long a time as desired. It maintains the normal intestinal gradient, reduces toxæmia and relieves symptoms without producing unnecessary purgation.

(c) *Bismuth and astringents and their combinations*.—Have also been used; substances containing tannic acid usually affect the stomach slightly but with the alkaline reaction of the intestine, they precipitate the protein and so protect the mucous membrane which is thus less stimulated and peristalsis is lessened. The mucus may be reduced and fermentation hindered.

(d) *Certain inert powders* like animal charcoal and kaolin are prescribed for coating and protecting the mucous membrane and for the absorption of gases.

(e) *Antiseptics* like creosote, iodoform, dimol, benzonaphthol, methylene blue etc., have been used from time immemorial.

(f) *Opiates* are resorted to when other remedies fail in checking diarrhoea.

(g) *Enemata* either of saline alone or combined with tincture of opium or one of the silver preparations have been found to give relief in some cases. Starch and opium enema may have to be tried. It must not be forgotten that many patients feel better when the bowels are loose.

(2) *Constipation*.—Liquid paraffin, castor oil and other mild laxatives are used. Several patent medicines like paragol, petromol, petrol-agar etc., are available in the market. Kaylene-Ol, a combination of kaolin and liquid paraffin, has been found to be very useful in the writer's experience.

(3) *Pain*.—Calcium chloride injections are supposed to give relief. If it persists, hot fomentations, hot wet compresses, turpentine stupes, antiphlogistine poultice etc., may be applied externally over the abdomen. Internally, benzyl-benzoate, atropine, or opium may be given. Ispaghula has been found to give relief to intestinal

symptoms and is used extensively in this country. Spogel seeds contain, as we know, much mucilage and are given in the form of cool demulcent drink for pain and diarrhoea.

Surgical treatment:—Surgery has a very limited field in the treatment of secondary tuberculosis of the intestine. For the operation to be of any value the ulceration must be confined to a limited area so that the diseased portion of the bowel may be excised with some hope of success. Short-circuiting operation has been tried but not with any great success.

The injection of oxygen in to the peritoneal cavity has been advocated and satisfactory results reported. The technique is practically the same as employed for artificial pneumothorax. McBurney's point is selected for the injection and 300 to 500 c. c. of oxygen are injected after anaesthetising the needle tract including the peritoneum with novocain solution. The intervals between the injections vary between one to three weeks depending upon the condition of the patient.

PROGNOSIS.

The occurrence of persistent diarrhoea in a phthisical patient has from time immemorial been regarded as of grave significance. The old Hippocratic saying "diarrhoea attacking a person with phthisis is a mortal symptom" has been held valid until very recently. Autopsy figures show that at death intestinal tuberculosis occurs in from 50 to 80 per cent or even more of all cases of pulmonary tuberculosis. It has been estimated by Drolet that about 18,000,000 patients died from pulmonary tuberculosis throughout the world during forty-years. Of this number, from 9,000,000 to 14,000,000 had intestinal tuberculosis. It is doubtful if these figures include the mortality figures from India where no reliable and accurate statistics are available and the mortality from pulmonary and intestinal tuberculosis must necessarily be very great. Intestinal ulceration would appear to be more common and more severe in Indian patients because the people of this country have not yet developed that amount of racial immunity which people in the West have done. The prognosis is, therefore, more serious in Indian patients.

The prognosis of intestinal tuberculosis is determined by the character of the disease in the lungs; in other words, the amount of tuberculous enteritis is directly proportional to the degree of activity in the lungs. It has been found that healing in the intestine is much more frequent in the productive type of pulmonary tuberculosis than is the case in the exudative form. The explanation for this probably lies in the fact that the intestine reacts either slightly or vigorously in proportion to the number of tubercle bacilli swallowed with the sputum and this factor is largely governed by the character of the pulmonary lesion. It will thus appear that factors like the number of bacilli swallowed with the sputum, the condition of the

patient and his vitamin balance in the diet are controlling factors in the production of intestinal tuberculosis.

If the diagnosis of intestinal tuberculosis is made in good time, if the general condition of the patient is good and if the pulmonary condition is not in an advanced stage and if the patient is placed under proper and prolonged treatment at this time, the prognosis would be brighter. Tuberculous lungs heal; tuberculous lesions of the intestine tend also to heal and can be shown by barium meal to have healed. At the same time the difficulty which attends the healing of severe wide-spread infection in the intestinal wall, particularly when frank ulceration has taken place, must be quite evident. The surface of the ulcers is constantly bathed in irritating mixtures of digestive juices and food in various stages of digestion which produce more or less maceration of the tissues and retard healing. When, therefore, the ulceration is wide-spread and the pulmonary condition is far advanced and the general condition of the patient is poor, little can be hoped for except amelioration of the symptoms. In such cases the intestinal lesion only hastens the inevitable end. The prognosis of intestinal tuberculosis, therefore, depends, as in pulmonary tuberculosis, upon early diagnosis and proper treatment. We are still making the diagnosis at a late stage. By methods we now have at our command we can make it much earlier and if we do this there seems no reason why intestinal tuberculosis should not yield to treatment just as pulmonary lesions do.

PREVENTION.

"Prevention is better than cure" is an old adage which is perhaps no where so true as in intestinal tuberculosis. The prevention of intestinal tuberculosis depends upon the early diagnosis and treatment of pulmonary tuberculosis. Patients should be warned against swallowing sputum either from ignorance or indolence or from an aversion to the spitting flask, as this is the chief source of infection of the intestinal tract. When one remembers that no matter how great the care taken, patients suffering from open lesions in the lung swallow bacillus-bearing sputum daily and often in large quantities, the relatively greater danger to the patient from his own sputum as compared to the danger from bacilli coming from without must be quite clear and evident. In suitable cases of pulmonary tuberculosis artificial pneumothorax should be induced to make the sputum free from tubercle bacilli. Constipation should be avoided and any catarrhal condition of the bowel should be immediately treated. Patients suffering from deficiency of hydrochloric acid in the stomach should be given this acid regularly since it is believed that in such cases the ulceration takes place higher up in the bowel. The general resistance of the patient to infection should be built up by suitable diet and particularly by a proper supply of necessary vitamins. The importance of cod-liver oil and tomato juice in the

prevention of the disease has already been mentioned. Here is some work for the King George Thanks-giving (Anti-tuberculosis) Fund to do. While educating the public against the bad habit of spitting in public places, it must also point out the danger from swallowing the infected sputum. The degree of danger to the intestine to which the patient suffering from open pulmonary tuberculosis is constantly exposed as compared to others, is not generally appreciated and must be brought home to the public by the Fund.

It is believed that tubercle bacilli are more easily absorbed through the intestinal mucous membrane when ulceration is present in it. In such cases, reinfection of the lung through blood may take place. The marked improvement noticed in the lungs of some patients who are recovering from intestinal tuberculosis, lends support to the view that ulceration of the intestine is a frequent cause of reinfection of other parts of the body including the lungs. Hence the additional danger of what may be called "enterophthisis".

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TREATMENT OF EPILEPSY

Conventionally, epilepsy has been divided into symptomatic and idiopathic according as the cause is known or not. It is claimed that this difference is only a superficial one and a hypothesis has been suggested saying that both these conditions have a common fault which is of the nature of a metabolic dyscrasia. This hypothesis, in spite of many facts supporting it, has not yet been universally accepted. According to it the case turns out to be one of idiopathic or symptomatic epilepsy according to the degree of this fault. When the dyscrasia is of a mild degree then it needs a supplementary factor in the form either of a lesion of the central nervous system or an intoxication or a peripheral irritation to set up the train of epileptic phenomena. This case would then be one of symptomatic epilepsy because of the apparent brain lesion or intoxication or peripheral irritation on which the outburst partly depends. The metabolic dyscrasia may be of such a high degree that it does not need any supplementary factor at all: this would then be a case of idiopathic epilepsy. There is also a third group where the metabolic dyscrasia is minimal or nil and in these the presence of any lesion would not give rise to epileptic phenomena.

The next interesting point about epilepsy is the present conception that it is a loss and not an exaggeration of function. Convulsions, hallucinations are considered to be release phenomena. This later concept has been responsible for the change in the life of the epileptic.

Arresting of the attack is possible only in very rare cases. When the aura starts definitely at the extremities, it has been possible to prevent its further progress by great pressure on that extremity. In some cases inhalation of amyl nitrite or inhalation of strong smelling salts is able to arrest the attack. In several cases, strong mental or physical effort is needed. However it has been found that whenever the progress of an attack has been thus arrested, the succeeding attack has been one of unusual severity.

The treatment *during the attack* is guided by the fact above mentioned that in the very large majority of cases it is not possible to arrest the attack. Further, the mortality during the attack is practically nil. Hence treatment resolves itself into seeing that the epileptic does not hurt himself in any way. When the fit comes on the patient must be placed in a lying position, safe from furniture or walls against which he may hurt himself. His clothes round the neck and the chest must be loosened. Care should be taken to prevent biting of the tongue by introducing a piece of cork or a piece of wood or any hard substance that is handy between the teeth or by imprisoning the tongue or the floor of the mouth by a handkerchief passed tight across the teeth of the lower jaw. If there is a tendency to vomit immediately after the convulsive state, the face of the patient must be turned to one side to prevent aspiration of vomited material. There should be no hurry in trying to rouse the patient before consciousness returns.

In the post-epileptic stage there may be headache which may be very severe. This can be treated by Aspirin or Phenactin in 5 grain doses. Those patients that are inclined to be hysterical may pass on from the epileptic to the hysterical stage and will have to be handled in the usual manner.

Besides individual attacks, which as mentioned above nearly always are not fatal, there is the condition *Status Epilepticus* where the fits are very frequent and the patient might be in a continuous state of unconsciousness. Here the fatal issue is to be feared in case satisfactory treatment is not instituted. The first step in the treatment should be the administration of a soap and water enema. This should be done irrespective of the state of the bowels, though these patients are usually constipated. Then, paraldehyde is given, per rectum, in a dose of 4 drams with 8 drams of olive oil. The introduction of paraldehyde treatment has reduced the mortality of *Status Epilepticus* considerably. Another drug that has been very successful in the hands of Tyler Fox is chloral hydrate, which is given by him in doses of 30 to 40 grains by mouth. If this drug is to be given per rectum, his advice is not to increase the dose. Next to chloral hydrate, is injection of Somnifaine of which one or two 2 cc. ampoules may be injected intramuscularly. Other drugs that are recommended are luminal-sodium 4 to 6 grains hypodermically,

hyosine hydrobromide 1/100th to 1/50th grain subcutaneously. When the convulsions are practically continuous chloroform should be brought into use. Here it should be remembered that deep anaesthesia is needed. Lumbar puncture is valuable in those cases where there is suspicion of kidney complications and when there is albumen in the urine; otherwise its results are not so striking. When there is a deficiency of calcium, 10 cc. of 10 per cent calcium gluconate intravenously works remarkably. Besides attending to the convulsions, it is just as important to keep a watch on the heart and circulatory system for any evidences of cardiac failure which may supervene with or without any warning. In the treatment of cardiac failure, strophanthin should be the sheet anchor and this can be supported by the usual cardiac tonics. So long as consciousness has not returned, one should always be on the look out for cardiac failure. Adequate nourishment and plentiful supply of fluids should be maintained. When swallowing is not possible, nourishment must be introduced by nasal tube. Fluids should be given in large quantities. Hyperprexia, pneumonia and transitory mental confusion may always occur in these conditions and should be treated in the usual way.

The main treatment of epilepsy is one *between the attacks*, and the purpose of this treatment is to anticipate and avert the attacks, to keep the patient in such a state as to be able to tide over those critical moments when he might have a fit. In this, the general treatment of the patient is just as important as the medical treatment. When the history gives a suspicion of epilepsy, a complete and careful examination and questioning of the patient should be undertaken to find out if there be any correlation between the epileptic attacks and particular events in the patient's life. If such correlating factors can be found, they should be dealt with immediately.

The *drugs* that are most in vogue in the treatment of epilepsy are Luminal, Sodium Luminal, Prominal and Bromides. Luminal is the first choice. It can be used for long periods without any deleterious effects on the patient. The only bad effect is when the drug is stopped suddenly as then fits recur with greater intensity. Prominal is supposed to be better than Luminal because it is less hypnotic in effect and is more efficacious in averting epileptic attacks. Whereas Luminal is not soluble, Luminal Sodium is soluble and therefore can be given in a mixture form as well as subcutaneously for which a preparation is available on the market containing 3 grains to one cc. of the solution. Bromides have been given for very long periods and they have been found also quite effective in the treatment of epilepsy. The only drawback in the use of bromides is the complication of acne which can be minimised by the addition of Liquor Arsenicalis. But, unfortunately, Arsenic has a tendency to produce pigmentation of the skin and gastro-enteritis. In few cases bromides may produce

gastric and respiratory catarrh, excessive salivation, slurring speech, sleepiness and drowsiness. The salts of sodium, potassium, and ammonium are the ones that are commonly used and these are given either singly or in combination. Some use Strontium Bromide because it is believed to be less inclined to produce acne. But this property has not yet been conclusively proved.

Other drugs that are used are Borax in 5 grain doses three times a day. It is particularly successful in cases of Petit Mal. Belladonna is given in 10 m. doses thrice a day, and arsenic is also used.

The dose and the time of administration of these drugs is to be controlled by the history of the fits. If the fits are strictly nocturnal, then a single large dose should be given on going to bed. If the fits are strictly diurnal then one large dose should be given in the morning on waking and if this be not sufficient to control the fits it should be repeated again in a smaller dose in the afternoon. When the fits are both nocturnal and diurnal then the dose should be divided into three doses given during the day and a larger dose at night. When the fits come on very soon after waking, then a large dose should be given as late at night as possible.

In cases of those people who have only one fit and who have an occasional fit once in two or three months, small doses should be given every day and this dose should be increased or decreased so that the spacing between fits becomes greater and greater and ultimately the fits do not appear at all.

In deciding upon the dosage, it is advisable to vary the dose which should be increased or decreased according to the success in controlling the number of fits. However a single maximum dose of Luminal should not be more than 1½ grains and of bromides not more than 15 grains and the total dosage a day should not be more than 3 grains of Luminal and 30 grains of bromide for an adult. Patients should be warned that when this sedative treatment is instituted there is a chance of the fits increasing in number at the beginning. In those cases where emotional states are present and are liable to produce fits or when the patient is in the presence of a particular occasion when greater nervous strain is put upon him, at the moment a fit is expected, then he should be fortified by a larger dose given a short time before this occasion.

If it is found that one of these drugs alone is unsuitable to the patient because of idiosyncrasy or because of its incapacity to control the fits, then it is advisable to supplement it with another; and both bromides and Luminal may be given. As mentioned above, Prominal is suggested to be a better drug in the treatment of epilepsy than Luminal. There are some cases that do not respond either to Luminal or Bromides or both. In such cases Belladonna and Borax should be tried. In a good few of these the fits will be controlled by either

of these. Zinc Oxide in capsules of 3 to 5 grains twice daily is another drug to be tried.

Once the fits have been controlled, these drugs must be continued with very same dosage for at least two years and after that the reduction of the dosage should be very gradual by about a third of the dose every year.

Recently, for the last ten years, *ketogenic diet* has been tried as treatment for epilepsy. The diet is worked out to contain small quantities of carbohydrates with the necessary minimal quantity of protein, and fats are added to make up the rest of the calories so that ketosis may be produced. Mandelic acid has been recently introduced with the same object of producing ketosis. The experience of different workers that have been published so far concern ketogenic diet; but it is too early to say anything about Mandelic acid. The experience of the last ten years shows that ketogenic diet has a beneficial effect in converting *grand mal* type of epilepsy to *petit mal* type and it is particularly favourable in cases of epilepsy in children. The results in adults are not as satisfactory. One has still to wait some more years before one can decide upon this method of treatment of epilepsy. Based on the conception of Hippocrates and other observations, Lyon and Dunlop have suggested a dehydration treatment for epilepsy. The treatment consists in the restriction of the carbohydrate and the fluid intake to a minimum; the production of watery stools by purgatives; diuresis by urea, and acidosis by administration of ammonium chloride. In the few cases they tried, the effects were beneficial in the beginning but they wore off later. Subsequent reports were not as favourable.

Because of the chronic nature of this condition and because of weakness of human nature many other remedies have been tried but have not held their ground for a long time. Recently two lines are being further investigated. One is the use of snake venom. This is based on the well known finding in America and Africa that epileptics who have survived a snake bite have shown remarkable improvement in their fits subsequently. The second one is the encouraging response to encephalography. Both these have been tried in cases that have not responded to luminal and bromides and some satisfactory results are reported.

The *surgical treatment* of epilepsy has to be considered in those cases where there is a traumatic lesion. But it has been found that removal of the focal lesion does not always mean a cure. In some cases there is an improvement in the number of fits, but this may be only temporary. When the surgical interference has been long delayed, it seems that the central cortex develops the epileptic habit and continues as before.

Once a diagnosis of epilepsy is made the patient should be carefully examined with a view to find any correlation between the fits

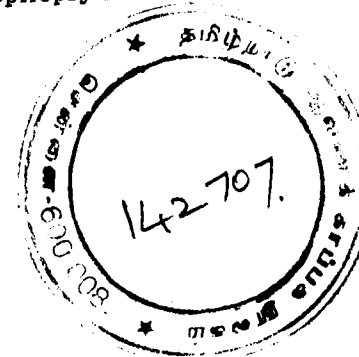
and the patient's life. In this way the possible exciting causes may be found. All sources of peripheral irritation must be attended to. The bowels need especial attention and the party must not be allowed to be constipated. There is no need to restrict the diet in any way though it can be adjusted in the light of the value of the ketogenic diet and dehydration. Carbohydrates may therefore be proportionately restricted and fats increased and fluids minimised as much as possible. Alcohol is best avoided while tobacco may be allowed in moderation. It is not advisable to restrict the daily routine in any way barring, of course, those items that may be dangerous because of the situation—like swimming, rowing, cycling, etc. On the contrary the daily life should be full and active. Such as are old and are employed should be permitted to carry on so long as their occupation is such that there is no danger of hurting themselves in a fit because of the nature of the job. Outdoor exercise should be encouraged.

Epileptic children need especial attention when the fits are very frequent. Then it is advisable to keep them away from school till such time as the fits are controlled. Such children are mentally backward and whenever possible should be sent to special schools. Quite a number of them are bad tempered, taciturn and lazy. It is not desirable that they should be sent to a normal school. Should their education be mishandled in the early stages, they grow into life with an extra handicap.

In the west there are special institutions for these epileptics. In these places the usual restrictions can be imposed with greater ease. What is of higher advantage is that the epileptic does not feel these restrictions as much as when he is outside. Here it is also possible to train him up in any handiwork suited to his abilities and temperament.

The problem of marriage of these epileptics has been recently in the forefront. Eugenically-minded States are inclined towards sterilisation of these epileptics. But still the controversy has not settled down. Sterilisation does not deny them the comforts of marriage. In Brain's series, 28 per cent gave a family history of epilepsy but to what extent sterilisation will reduce the incidence of epilepsy is a matter for the future to decide.

—Medical Bulletin, May 16, 1936.



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Extracts

EPITUBERCULOSIS.

(*British Medical Journal*. 1936. i. 220.)

The term "epituberculosis" was first used in a paper by Eliasberg and Neuland in 1920, and since then no satisfactory pathological and experimental evidence has been adduced to clear up the confusion that exists on this subject. Grancher's conception of "spleno-pneumonie" as a special type of tuberculous reaction in the lung has been ignored by most writers.

Put briefly, epituberculosis is in the main a clinico-radiological conception: there is a benign, febrile, pneumonia-like reaction in the lung, and x-ray examination reveals a slowly-resolving shadow. It is always difficult and often impossible in the early stages to differentiate by x-ray examination between the benign condition and tuberculous pneumonia of a fatal type.

The main theories to account for the x-ray and other findings in epituberculosis are that: (1) they may be caused by collapse from pressure of enlarged tuberculous mediastinal glands; (2) they may result from a chronic non-tuberculous pneumonia in a lung made more susceptible by the presence of a tuberculosis infection; (3) epituberculosis may be a non-inflammatory reaction (oedema or increase of connective tissue); and (4) epituberculosis may be a retrogressive tuberculous pneumonia.

Oppenheimer, of the Johns Hopkins Hospital, on the basis of Rich's suggestions that there were similarities between epituberculosis and caseous pneumonia, recently carried out a series of experiments on rabbits, using animals which were hypersensitive to tuberculin as well as non-allergic controls. He found that a x-ray lesion quite like that of the so-called epituberculosis followed the experimental introduction of dead tubercle bacilli into the bronchus of hypersensitive rabbits. The clinical course was similar to that of human epituberculosis. In non-allergic animals similar changes could not be obtained, and in immunized hypersensitive animals injected intratracheally with living tubercle bacilli a similar x-ray shadow was observed, which, however, spread progressively, and the animals died. From anatomical studies of the experimentally produced epituberculosis it appears that the lesion is essentially a tuberculous pneumonia, clearing by resolution and organization. From these and other considerations, Oppenheimer puts forward a working theory for the occurrence of epituberculosis in human subjects. He suggests that it is the result of erosion of a bronchus by a caseous lymphatic gland containing relatively few viable bacilli

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The characteristic x-ray shadow is caused by a lesion which is essentially a tuberculous pneumonia, brought about by the discharge of caseous material impregnated with tuberculo-protein and dead bacilli (although containing some living bacilli) into the lung of a patient, already sensitized and immunized by the previous infection. If a similar accident happens with a gland teeming with living bacilli a similar x-ray shadow will be observed at first, but the process will extend progressively. Between these two extremes are the various and clinically familiar shades which are neither so benign as the epituberculous lesion nor so acute as the fatal caseous pneumonia, the outcome depending upon the balance between the number of living bacilli in the lymphatic gland and the degree of acquired resistance and hypersensitiveness of the body. If this view is accepted the term "epituberculosis" might well be dropped. The lesion it represents would then be recognized as the most benign and retrogressive type of tuberculous pneumonia.

BILATERAL URINARY CALCULI.

(A. R. STEVENS: *Journal of A. M. A.* 1935. civ. 1289.)

The therapeutic problems peculiar to bilateral urinary calculi are not solved by the application of stated formulas. In clinical work one must secure data as complete as possible concerning the patient in general and his urinary tract in particular. The size and location of all calculi should be known, whether or not they obstruct, as well as the total renal function, the relative function of the two kidneys, and the presence or absence and degree of infection of both kidneys. There is no dependable method of dissolving calculi, but stones may pass spontaneously through the renal channels or be removed by operation.

Large, non-infected, non-obstructing, symptomless stag-horn calculi are usually left alone. Also, there is questionable advantage in trying to remove a large mass of non-obstructing symptomless small stones occupying an entire kidney. There is no hurry in operating for small symptomless stones in a kidney even in the presence of low grade infection, whereas one does not dare wait long for the spontaneous passage of ureteral calculi associated with urinary infection.

Relief of obstruction by a ureteral catheter, if possible, is usually a relatively harmless procedure; it may give temporary relief from symptoms and danger, may be followed by spontaneous passage of a calculus. Ureterotomy is one of the least dangerous major operations. Pyelotomy entails an added risk, and nephrotomy has been followed by a decided, though perhaps transient, retention of non-protein nitrogen in the blood within a week or ten days. Secondary haemorrhages and acute kidney infections, when they do occur after operation, are usually encountered after nephrotomy incisions and not after pyelotomy. The more nearly complete urinary obstruction is, the more rapidly will kidney injury occur. The greater this injury, the less the return to function is to be anticipated following operation. One should seek improvement of renal function primarily, and eventually control of infection and alleviation of symptoms.

Medical News and Notes

THE MADRAS MEDICAL COUNCIL'S NOTICE. The Registrar writes to us as follows under date 1st May, 1936: The Madras Medical Council at its meeting held on the 3rd March 1936 resolved that the following notice be published in all possible Medical Journals:—"It is a grave breach of professional propriety for a medical practitioner to take any part in the manufacture, or sale, of a proprietary food or medicine or to have his name in any way associated with it. Medical men should also be cautious how they lend themselves to the puffing of these articles. (Extract from the Code of Medical Ethics, Section 22.)"

ALL-INDIA MEDICAL LICENTIATES' ASSOCIATION. Dr. U. B. Narayana Rao, General Secretary, writes to us under date Bombay, 9th June 1936:—Special concession to Licentiates joining the A. I. M. L. Association: During the Dr. P. S. Ramachandra Iyer's Memorial Month (July 1st to 31st 1936) Licentiates will be enrolled as members of the Association on payment of Rs. 4 only, without any entrance fees. Membership will entitle them to get the *Indian Medical Journal* (the monthly organ of the Association) free for one year. The amount of the subscription may be sent to Dr. A. N. Khosla, Treasurer, A. I. M. L. Association, Ripon Hospital, Simla.

CHETTINAD MEDICAL ASSOCIATION. The sixth Anniversary of the above Association was held on the 9th April at the S. M. S. High School, Karaikudi, with Dr. B. V. Varadachari, B. A., M. B., C. M., District Medical Officer of Ramnad, in the chair. The Conference of the Tamilnad Medical Associations was also held at the same time. Dr. T. S. Shetty, Secretary, read the annual report. Among the papers read on the occasion were those on "the relationship of obstetrics and ophthalmology" by Dr. G. Joseph Gnanadikam of Trichinopoly; "non-sanatorium treatment of pulmonary tuberculosis" by Dr. W. E. Wilder, M.D., of Madura; "x-rays in general practice" by Dr. P. Rama Rau, D.M.R. (Vienna) of Madras; "a few salient points in the treatment of syphilis" by Dr. A. Narayana Menon, M.B., B.S. of Madura. The function ended after a cinema show and dinner.

CHITTOOR DISTRICT MEDICAL ASSOCIATION. A meeting of this association was held on the 23rd May at Kuppam with Dr. D. Krishnayya, District Medical Officer, in the chair. After tea, Dr. C. Doraiswamy Mudaliar read a paper on "some common diseases." Dr. Mudaliar later entertained the members at dinner.

TINNEVELLY DISTRICT MEDICAL ASSOCIATION. This Association met on the 23rd May at the Centenary Hall, Palamcottah. Lt. Col. T. S. Shastri, I.M.S., District Medical Officer, presided. Dr. N. Isaac read a paper on "tobacco and its medical aspects". Dr. K. R. Venkoba Rao spoke on "laboratory work in relation to medical diagnosis."

TUTICORIN MEDICAL UNION. A meeting of the above association was held on the 29th May under the presidency of Dr. K. S. Iyengar, L.M.S., Civil Surgeon. The following office-bearers were elected for the ensuing year: Dr. K. S. Iyengar, President, Dr. Seshadri, Secretary, Dr. P. R. Achar, Treasurer, Drs. Manuel and Miss Verghese, committee members.

EAST GODAVARI DISTRICT MEDICAL ASSOCIATION. A meeting of the above association was held on the 30th May at Amalapuram, under the presidency of Captain J. S. McMillan, I.M.S., District Medical Officer. After tea, Dr. A. Umapathi Mudaliar, M.B., B.S., of Amalapuram showed a case of pulmonary stenosis in a boy of 12, a case of congenital syphilis in a child of 2, and a case of malignant growth of the neck with pushing forward of the posterior wall of the pharynx and protrusion of one eyeball. Dr. N. S. Reddi of Amalapuram read a paper on "typhoid" and Dr. A. Perisastry on "tetanus". There was a dinner after which the guests dispersed.

TRICHINOPOLY DISTRICT MEDICAL ASSOCIATION. A monthly meeting of the above association was held on the 31st May in the E. R. High School, Teppakulam. Dr. R. Kalamegham, B.A., M.B., B.S. was "at home" to the members and Dr. V. Iravatham of Thirukattupally presided. Dr. R. Sambasivan, M.B., B.S., delivered a lecture on "pitfalls in practice." He said that with the advancement of therapeutics pitfalls were getting fewer. He instanced cases of sloughing after quinine injections, which were now entirely replaced by the safer and shorter course of atabrin treatment. Risky intravenous injections of rectified tincture of iodine have been replaced by the comparatively innocuous colloidal iodine, and the dangerous arsenicals have been replaced by safer intramuscular arsenical and bismuth preparations. He explained the risks of giving pure undiluted glycerine for enema and of hypnotics in children. He also spoke a few words as to how practitioners should behave with patients and co-practitioners.

ANANTAPUR DISTRICT MEDICAL ASSOCIATION. This association was formed on the 7th June at the Headquarters Hospital with Dr. V. Krishnamurthy, District Medical Officer, in the chair. Dr. Krishnamurthy was elected president, Dr. A. B. Siva Rao, secretary, and Dr. (Miss) Chakko, treasurer.

PUDUKOTTAH MEDICAL ASSOCIATION. The monthly meeting of this association was held on the 29th May at the General Hospital with Dr. M. G. Ramachandra Rao, M.B., C.M., Chief Medical and Sanitary Officer, presiding. Dr. V. R. Chander delivered a lecture on "filariasis".

THE INDIAN MEDICAL COUNCIL announces that a telegram has been received from the Registrar of the General Medical Council of Great Britain, to the effect that medical qualifications of the Universities of Bombay, Lucknow and Madras have been recognised with

effect from February 25, 1930 and that the Degree of M.B., B.S., of the University of Patna has been recognised, when granted on or after May 11, 1935. Qualifications of the three older Universities were recognised previously up to February 24, 1930.

OBITUARY. The death of Dr. M. A. Ansari, M.D., M.S. (Edin.) on the 10th May 1936 from heart failure is a great loss to this country. He was born on December 25, 1880 at Yusufpur, Ghazipur District, United Provinces. He was educated in the Muktab and in the Queen's Collegiate School of Benares. He matriculated in 1895 from the Government High School, Allahabad. He later entered the Muir College of Allahabad and the Nizam College, Hyderabad (Deccan) and graduated in science from the latter college in 1900. With the Nizam's scholarship he proceeded to Edinburgh and obtained the degree of M.B., CH. B. in 1905. He joined the Charing Cross Hospital London, as a demonstrator and house surgeon, and obtained the Conjoint Board diploma as well as the M.D. degree of the Edinburgh University. In 1908 he was the R.M.O. of the London Lock Hospital and obtained the M.S. degree of the Edinburgh University. After a short period of house surgeonship at the Lock Hospital for Women and Children, he returned to India and opened consulting practice at Delhi in 1910. In 1912 and 1913 he organised and directed the All-India Medical Mission for service of the wounded in the Turko-Balkan War and was the recipient of high honours from the Sultan of Turkey. After 1916 he took an active part in Indian political life and was elected President of the Indian National Congress in 1927, the greatest honour in the gift of his grateful countrymen. He helped to establish a College of Unani Medicine at Delhi. He presided over the ninth All-India Medical Conference at Bombay in 1932 and was Chairman of the Reception Committee of the 11th All-India Medical Conference in 1934 which met at Delhi under the presidency of Colonel Bhola Nath. He was having a very lucrative medical practice throughout almost the whole of Northern India and his medical advice was in very great demand by prince and peasant. A non-medical journalist sums up his life and career in these words: "A great doctor by calling, he was a great healer by instinct. Puissant princes came to him with their ailments; perplexed politicians sought his advice to set right communal or other tangles; struggling students and suffering poor made incessant calls on his generosity. To one and all, to rich and poor, to all castes and communities and sections, his advice and help was a healing balm. In all her many ailments, India had in Dr. Ansari the trustiest physician. And in his death, which occurred recently, India, still ailing, has lost a consultant, the need for whom will be felt even more in the coming difficult years than in the past."

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By Prof. NOGUER MORE.

Chief of the Dermatologic Service.

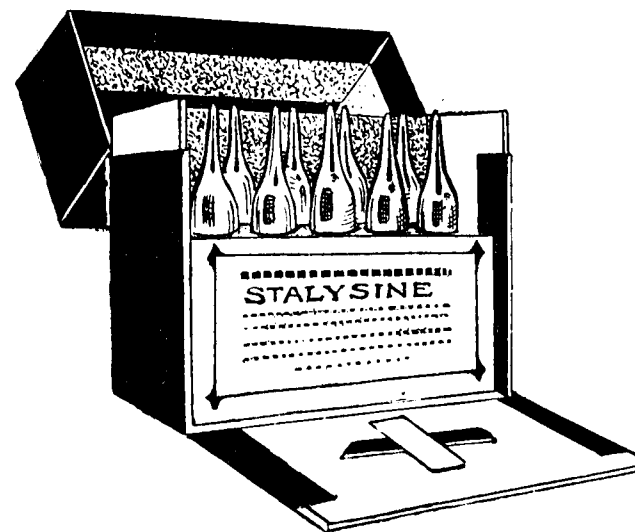
Hospital de la Santa Cruz.
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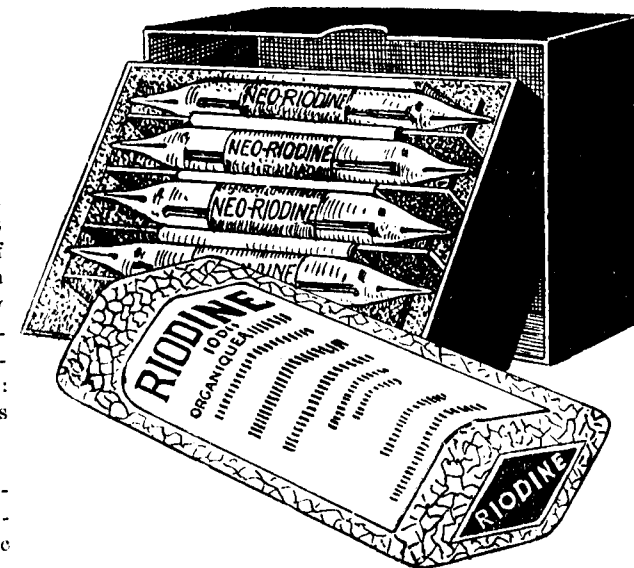
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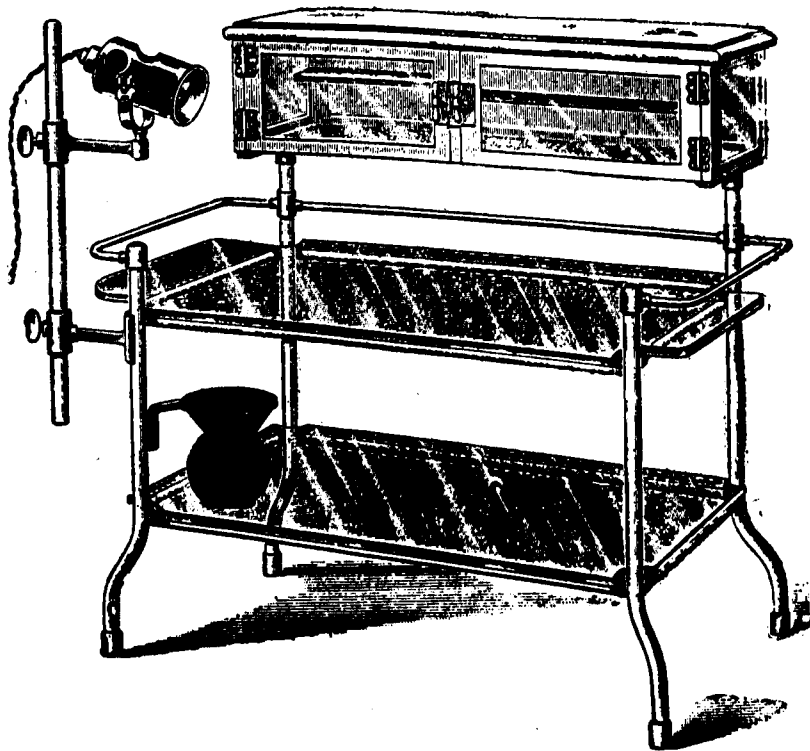
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