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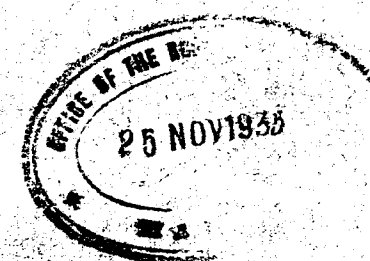
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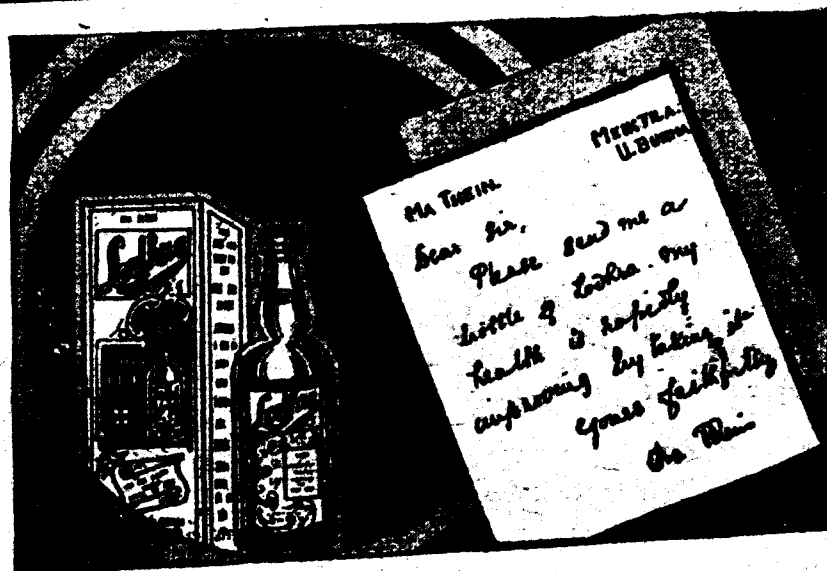


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RENAL LITHIASIS

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PART I

This disease is probably as old as the human race. Ayurveda gives descriptions of bladder stones and also mentions a supra-pubic removal. CHARAKA, SUSRUTA and VAGBHATTA describe the symptoms and treatment of vesical stones. Four types of stones are described: *large white stones* (probably phosphatic), *spiky stones* (probably oxalate stones), *bile-coloured stones* (probably urate stones), and *sperm stones*. The last type probably refers to prostatic stones. SUSRUTA, in the fifth century A. D., operated for vesical stones by lateral lithotomy without a staff.

GEOGRAPHICAL DISTRIBUTION.—Stone-formation naturally is more frequent in the Old World than in the New, and is more frequent in Asia than in Europe. India, Mesopotamia and South China, where the oldest civilizations have existed from the dawn of history and where the standards of life are still poor, show a great incidence of the disease. Western Europe, where lithiasis was not uncommon, has shown a rapid fall in the incidence of the disease, especially in the young, owing to improvement in the standards of life and sanitation and a change to a mixed diet rich in fat-soluble vitamins.

In India, stone-formation is more common in the Indus Valley and Upper Gangetic Valley. In this Province, it is more prevalent in Districts where the staple food is maize. It is comparatively rare along the Coast Districts where rice, fish, and vegetables form the staple diet.

RACE.—The Indians, the Arab, the South Chinese and Anglo-Saxons seem to be more prone to lithiasis (in order of frequency).

IN ANIMALS.—Stone is rare in wild animals, especially carnivora, but is more frequent in domesticated animals, especially the horse. Animals oxidise their uric acid into allantoin, which is freely soluble in urine at body temperature. Calcium carbonate stone predominates in animals.

AGE.—Vesical stone is not uncommon in children of the poorer classes in this country. It is also not uncommon in old age. Renal lithiasis is more common from 20 to 50 years. In children, sodium and ammonium urate stones are more common. These being smooth are passed down the ureter and per urethra. But, sometimes, they get held up in the bladder. It is owing to the introduction of X-rays, the cystoscope, and improved methods of diagnosis, that renal lithiasis is now more frequently diagnosed.

SEX.—*Vesical stones* is a disease of the male sex, and is rare in women. *Renal lithiasis* is probably equally prevalent in both sexes. Some say that in the male it is twice as common as in women. This may be due to the fact that in the male the kidneys are liable to pathological changes from back-pressure and infection from diseases of the urethra and prostate.

URINARY CHANGES AND LITHIASIS

To understand the formation of renal calculi, it is necessary to learn the physical and bio-chemical nature of urine. Urine is really a super-saturated solution of uric acid, calcium oxalate and calcium phosphate. (An adult passes 3 to 4 grammes of the salts per diem in about 1,500 c.c. of urine. It would require about 15 to 20 litres of water to dissolve this amount of the salts). This super-saturation is brought about by the presence in the urine of minute amounts of "Reversible Colloids," approximately 0.83 gm. per litre. Some of them are mucin, nucleic acid, urochrome, and a complex carbohydrate containing nitrogen compound. They are in a colloidal state of minute globules and hold the molecules of the salts by surface tension, a process known as "Adsorption," and render the solution transparent. Normal human beings can be divided into three classes, according to the amount of these *reversible colloids* present in their urine. In the *first* type, the urine contains such large amounts of these colloids that it does not form any deposit, if kept *statically*, in 24 hours. In the *second* type, these colloids are just enough to hold the salts in solution. Hence, though the urine is clear on passing, a deposit is formed on standing for a few hours. This is due to some of the colloid globules uniting to form larger globules and thus throwing some of the salts as deposit. This process of forming larger globules of colloids is

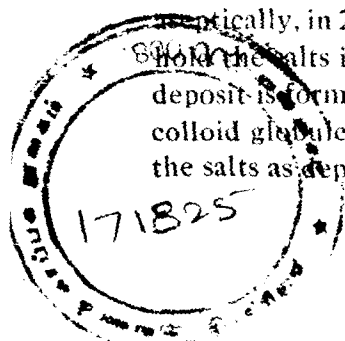
known as "ageing" and is seen in colloid solutions. The *third* type has a deficiency of these reversible colloids, and hence salts are precipitated in the urinary tract. In such individuals, the freshly passed urine shows a deposit of salts which increases in amount after standing for a few hours. The *first type* is never likely to suffer from stones, the *second type* may suffer if the kidneys become damaged by infection, and the *third type* may pass through the usual span of life without stone-formation, but in this any damage to the kidney is very liable to end in stone-formation.

Only certain salts, such as uric acid, urates, oxalates and phosphates of calcium, cystine and xanthine, show a tendency to form stones. Though crystals of these salts may be passed out, as in phosphaturia, etc., usually they show no tendency to conglomerate into calculi. In ordinary inflammations of the kidney, the non-albuminous colloids in the urine are increased, giving a protection from the formation of calculi.

The concentration of salts in the urine as compared to that in the blood is seen in the table given below.

	Blood plasma per cent	Urine per cent	Concentration No. of times
Water	99.93	95	
Proteins, fats and other colloids	7.9	nil	
Dextrose	0.1		
Urea	0.03	2	60
Uric acid	0.004	0.05	12
Na	0.3	0.35	1
K	0.02	0.15	7
Ca	0.008	0.015	2
NH ₄	0.001	0.04	40
Mg	0.0025	0.006	2
Cl	0.37	0.6	2
PO ₄	0.009	0.15	16
SO ₄	0.002	0.18	90

PHOSPHATURIA.—An adult, on a mixed diet, eliminates about 2 grammes of phosphoric acid per diem. This is chiefly derived from complex organic compounds as nucleo-proteins and phospho-proteins and the inorganic salts present in milk, eggs and vegetables (chiefly as magnesium



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salt in the last). Only small amounts of phosphorus are absorbed from a vegetarian diet, the rest being derived from sodium phosphate and casein of milk. The turbidity of the urine of vegetarians is due to its alkalinity, and in them an amorphous deposit of calcium phosphate occurs. The acidity of urine is dependent on mono-sodium phosphate. Excretion of this salt in the urine is dependent on the alkalinity of the blood. Increase in the amount of protein in the diet increases the acidity of urine by greater excretion of mono-sodium phosphate, and in consequence the urine becomes clear, though the total amount of phosphates in the urine may actually be greater. The presence of di-sodium phosphate in excess renders the urine alkaline. Calcium phosphate occurs either as long crystalline needles in acid urine, or as an amorphous deposit in alkaline urine.

Treatment. In phosphaturia, with sterile urine, an increase of proteins in the diet and administration of mono-sodium phosphate, ammonium chloride, or ammonium benzoate, by mouth, would clear the urine by rendering the urine acid.

OXALURIA.—A precipitate of calcium oxalate may occur either in acid or alkaline urine, but a reaction near the neutral point is the most favourable. The deposit is absent in highly alkaline or acid urine. It is often associated with uric acid in acid urine and amorphous phosphates in alkaline urine. This condition is entirely due to a deficiency of the specific colloids mentioned above. The crystals are clear. We eliminate about 15 to 20 mgm. of oxalate per day. It is partly derived from vegetables as spinach, rhubarb, sorrel, beans, peas, roots, tomatoes, berries, potatoes, and tea. It occurs as calcium oxalate in these, and especially in the growing points of vegetables. Normally, about 10 per cent of this is absorbed from the intestines, but this amount may be increased by giving hydrochloric acid, and decreased by giving alkalis. Intestinal fermentation increases the amount of soluble oxalates and, hence, their absorption. In hyperchlorhydria and jaundice, their absorption is increased. The endogenous source is from the metabolic processes of the body, and during complete starvation oxalates are still passed in urine.

Treatment.—Regulation of bowels to prevent intestinal fermentation is of importance. Intestinal antiseptics are also of use. Eliminate external source of oxalates by avoiding the vegetables mentioned above. If the urine is acid, alkalis such as sodium citrate with large amounts of water are to be taken. Alkaline waters of Vichy, Vittel, Evian, and Contrexeville are useful.

Alkaline diuretics limit or prevent precipitation of crystals. A mixed diet is better than a carbohydrate diet for these patients.

URATURIA.—A deposit of uric acid and urates may occur in acid urine on standing, especially in pyrexial conditions. This is due to the lowering of the temperature of the urine; the deposit redissolves on warming the urine. In uraturia, however, the precipitate occurs in the urinary tract as agglutinations of crystals into small round spheroids (microliths) either in the kidney or bladder, and if retained they form stones. They are colourless, but become yellow from deposit of urochrome. Being smooth, they may silently pass down if of small size; when fairly large, they may give rise to attacks of renal colic, but roetgenograms show no shadows, as urate microliths are transparent to X-rays. I have seen these tiny microliths in the bladder of a couple of cases. They appear like little pearls, but dull in colour. On a mixed diet, an adult passes from 0.8 to 1 gramme of uric acid per day, and a little less on a vegetarian diet. Its origin is partly endogenous and partly exogenous (from food). Its endogenous source is greater in growing youths, during absorption of exudates (as in pneumonia), when destruction of cells occurs (as in leucocythaemia). In man, 60 per cent of the purines in circulation are converted into uric acid, and a smaller amount into urea. In other mammals, 70 to 90 per cent of uric acid is converted into allantoin, which is freely soluble in water at body temperature.

Treatment.—Since most of the exogenous source is nucleo-proteins, avoidance of purines in the diet (lean meat, liver, kidneys, pancreas, beans, peas, etc.) and inhibition of large quantities of water as barley water, weak tea, alkaline waters as of Vichy, etc., are advised. Four to five pints of urine should be passed per day. Lithia and piperazin have no influence on this condition.

CYSTINURIA.—This is uncommon in India, as the majority of the people are vegetarians, by choice or necessity. It is a familial disease, but only 2 to 3 per cent develop actual cystine stones. Normally, cystine is absent in the urine, or only traces are found. In cystinuria, 0.5 to 1.5 gm. of it may be passed per day. It is a complex organic compound containing sulphur, and results from deranged intermediate metabolism of the body. A protein-free diet reduces the amount excreted. It occurs partly as a crystalline deposit and partly in solution. Being a congenital condition,

stones may form in children and young adults. It is often bilateral and liable to recur after removal.

Treatment. A strict vegetarian diet, with restriction of proteins to a minimum, and administration of large doses of sodium bicarbonate, to render the urine alkaline, reduce the amount of cystine excreted and render it soluble in urine.

FORMATION OF CALCULI

Though crystals may be passed in urine in lithiasis as mentioned above, actual stone-formation may never occur in the individual until other factors come into play.

Diet.—A monotonous diet, rich in carbohydrates and deficient in fat-soluble vitamins, predisposes to it. It is found that the incidence of stone-formation in Europe has gone down rapidly, especially in the younger generations, since the advent of a mixed diet rich in fat-soluble vitamins, rendered possible by a rapid improvement in the standard of life of the working classes, in whom formerly it was not uncommon. According to McCARRISON, deficiency of vitamin-A and some factor in the germs of cereals, present especially in wheat, but absent from rice, encourage stone-formation.

Atypical Crystals. The crystals found in stones are of different shapes from those found normally in urine. These abnormal forms of crystals are found in lithiasis and in stones. KEYSER's experiments have shown that the nature of crystals changes when stone-formation begins.

Presence of Specific Colloids.—A stone is formed both of colloids and crystalloids. The crystals can be dissolved out by solvents, leaving a framework of colloids—a stone of colloids only. If the colloids are dissolved out by antiformin, the crystals fall to the bottom of the vessel as a deposit. These colloids are *irreversible*, and, therefore, a stone, once formed, cannot be redissolved in the urine, as we know of no drug that would convert these colloids into a reversible type. The colloid found in the nucleus of a stone is not fibrin, and very few of the people that have suffered from renal inflammation get renal lithiasis. Hence, one concludes that these colloids in a primary aseptic stone must be abnormal ones. Some consider them to be specific ones, and there may be more than one of these. They are irreversible, and once they are thrown out of their colloidal state they cannot go back into that state. As they hold some of the crystalloids by adsorption in

their colloidal state, both become precipitated together. The nature of these colloids is not yet understood.

Retention of Crystals.—This occurs most often in the lower calical branches. The lower calyx is imperfectly drained in all but the prone position, on account of the anatomical position of the kidneys. The flow of urine is about three drops a minute in a calyx, and the current is, therefore, not strong enough to wash down the crystals, which, therefore, may sink to the bottom of a calyx and remain there. This tendency is greater in bed-ridden patients (as after fracture of femur). Such cases are liable to develop bilateral calculi. These crystals have a thin covering of an irreversible colloid. A small spheroid or microlith forms by further deposit of crystals. Such a microlith may get pushed by the urine into the pelvis and travel down the ureter to the bladder and out through the urethra. So long as they are smooth and of small size they may cause no discomfort, but any roughness or increase in size may give rise to transient attacks of ureteral colic, more so in people with narrowing of the ureters. This occurs in a fair number of people of the *third type* mentioned above. These spheroids are made of irregularly arranged crystals bound together by a thin covering of irreversible colloid. I have seen these tiny spheroids, 1 to 2 mm. in size and of a dull white colour, in the bladder of people complaining of gravel. The urinary sand or gravel of these cases is made of these microliths. If the pelvi-ureteric junction is abnormal, these spheroids may be held up in the renal pelvis. Congenital narrowing at the pelvi-calycal junction may retain a spheroid in the calyx, and it may grow in size and become too large for passing through the pelvi-ureteral junction. Similarly, constrictions in the ureters may hold up a spheroid, which may grow there in size or be passed down after attacks of ureteral colic.

Growth of Stones.—Once a small stone is held up, it starts to irritate the lining membrane of the containing cavity. These spheroids have a rough surface. A reactionary inflammation results, as shown by the presence of red blood corpuscles, leucocytes and epithelial cells in the urine. Exudation of plasma and serum occurs. These are irreversible albuminous colloids, and cover the rough surface of the microlith, giving it a smoother surface. This new coat is spongy, fairly thick, and contains a smaller number of crystals and a larger amount of albuminous colloids. The surface being now smooth, irritation subsides, and with it the inflammation. The urine becomes clearer, and the patient is less troubled with pain. After some

time a new layer of crystals with a minimum amount of irreversible urinary colloids is deposited. This layer is formed slowly and is compact, i.e., rich in crystals, and, hence, the surface becomes rough. Irritation of the containing cavity gives rise to an inflammatory reaction, and a thick layer of spongy albuminous matter with minimum amount of crystals is deposited. This process of alternate irritation and quiescence gives rise to lamination of stones by spongy and compact layers alternatingly. During the irritation period pain and haematuria with pus cells are evident, and during the reactionary state the deposit is more cellular and pain becomes less, and at the end of this stage the urine becomes clearer.

Infection.—A stone may develop as a result of infection—e.g., an attack of pyelitis. Such a stone is secondary in type, and is liable to form in people of the *second type*, and more often in the *third type*, of humanity mentioned before. The nucleus in these is made of bacteria, epithelial debris, fibrin and pus cells. The foreign albuminous colloids of inflammation give rise to a thick spongy layer with small amounts of crystalloids. If the infection persists, the resulting stone is spongy throughout; if it subsides it may get a coating of a compact crystalloid layer. But more often the former occurs owing to persistence of infection. *Staphylococcus albus* is the organism often found in secondary stones; it breaks up urea into ammonium carbonate and phosphates. *Bacillus coli* is more often found with calcium oxalate stones. *Mixed infection* may also be present.

Secondary stones are chiefly made of triple phosphates of ammonium, calcium and magnesium, and calcium carbonate, as a result of alkaline decomposition of urine. Ammonium-magnesium phosphate occurs as a crystalline deposit, and calcium phosphate as an amorphous deposit. The presence of a primary stone is sooner or later followed by infection and decomposition of urine. This gives rise to a heavy deposit of earthy phosphates increasing the size of the stone rapidly.

Foreign Bodies.—These may be organisms, blood clots, etc. Usually, the inflammation set up gives rise to a spongy deposit, and if infection be also present a deposit of earthy phosphates. Rarely, a deposit of compact layers may occur round a foreign body in the absence of infection and inflammation.

The Presence of Excess of Calcium in the Urine. Hyper-calcaemia of the blood is seen in hyper-parathyroidism and after fractures, as of the

femur, entailing a long recumbency. Long residence in places with supplies of hard waters may favour the formation of stones. Recumbency in fracture adds the factor of inadequate drainage of the lower calyx. The least soluble salts in the urine are precipitated first; therefore, calcium oxalate preponderates in these cases. Calcium oxalate is held in solution by acid sodium phosphate and is in a fairly high concentration favourable for precipitation.

It is evident from what has been mentioned above that there are several factors in the causation of stones.

All calculi have an organic frame-work on which inorganic salts are built in or deposited. The organic matter in primary aseptic stone-nucleus is chiefly a urinary irreversible colloid. The spongy layer of aseptic stones is chiefly of foreign albuminous material, with a small amount of crystalloids. In the presence of infection and decomposition of urine, earthy phosphates and calcium carbonate preponderate, with foreign albuminous colloids, giving rise to secondary septic stones. These are soft and easily broken up. The aseptic ones are firmer and even hard.

TRACHOMA *

DR. L. R. FERNANDEZ.

Trichinopoly

The word TRACHOMA is derived from the Greek *trackus*, meaning rough. The disease is known as *Egyptian ophthalmia* because it was first discovered in Egypt about 3500 years ago. It is termed also *granular ophthalmia*. The famous Greek physician, HIPPOCRATES, was well acquainted with it. CELSUS, a Roman surgeon who lived in the 1st century A.D., gave a good description of the roughness of the inner membrane of the lids and recommended treatment by rubbing and scarification.

TRACHOMA is a contagious chronic inflammation of the conjunctiva, characterized by the presence of granules or papillae, accompanied by mucous secretion, and followed by cicatrization. Children are generally affected, especially during the first two years of life. Children playing with infected children, or using the same towel, soap, and basin as those infected, easily contract it. Absence of strict cleanliness is almost universal among the lower classes and facilitates spread of contagion. An attack of acute conjunctivitis frequently precedes infection with trachoma. Hence, in former times, it was thought that trachoma always began acutely. This, however, is rarely the case. The acute symptoms are usually caused by infection with well-known bacterial organisms, the more important of which are the *Diplobacillus of Morax-Axenfeld*, the *Koch-Weeks bacillus*, and the *Gonococcus*. One interesting point is that most trachoma patients are hypermetropic. Very rarely do myopes suffer from it. What bearing this has on the etiology it is not easy to say, but the correction of the longsight by glasses is very necessary in the treatment. Possibly, myopes are protected from infection by the glasses they wear.

Trachoma may begin in a fairly acute form with much secretion, and its granular nature not be recognized until the acute symptoms subside. This was responsible for the view formerly held that trachoma is sometimes the result of gonorrhoeal ophthalmia. More often, the onset is gradual, and, indeed, it is often so insidious that when it is discovered it is usually in a

fairly advanced stage, the patient not having been aware that anything was wrong. When the inmates of an infected institution are systematically examined, many cases of the disease are found presenting no symptoms. Occasionally, chronic cases present acute exacerbations. Usually, the patient has the symptoms of conjunctival catarrh, viz., lachrymation, photophobia, and secretion (watery or more often mucous) causing adhesion of the lids in the morning, and also pain and heaviness of the lids, which may be marked. The lids droop because they are swollen and really heavy and in a condition of ptosis, and this gives these patients a very characteristic sleepy expression. The conjunctiva is swollen and injected. Its appearance varies with the type of the disease. In the *granular form*, grey, translucent, round bodies are found, mainly in the retro-tarsal folds; these are more numerous in the upper fornix than in the lower. These bodies project above the surface and can be squeezed out. They present no regular arrangement, as the similar bodies in follicular conjunctivitis do. Unlike the latter, where they shrink and disappear, either in the evolution of the disease (as happens sometimes), or as the result of treatment, these granules leave behind cicatricial changes of considerable importance. In the *papillary form*, the tarsal conjunctiva, mainly of the upper lid, presents a velvety or raspberry-like papillary hypertrophy, followed later by cicatrization. In the *gelatinous form*, the thickly-set granules fuse by softening. The overlying epithelium also softens by fatty or hyaline degeneration, and the whole lid is lined with a thick, swollen, gelatinous looking material, from which granules can be squeezed out, and which stains brown with tincture of iodine, though it is probably in no way an amyloid change.

The sight is often interfered with by two corneal complications. *Pannus*, or vascularisation of the cornea, is common and may, by its interference with sight, be the means of first calling the patient's attention to the disease. The trachomatous pannus is a vascular growth of round cells lying between Bowman's membrane and the corneal epithelium, and affecting usually the upper half of the cornea, which is the part in contact with the upper lid; the rubbing of the cornea by the rough granules is not, however, the sole cause, as you may find no pannus with many granules and pannus where they are few. The process is essentially trachomatous, and the upper half of the cornea is almost always first affected, because it is in constant contact with the granules, this favouring infection. Pannus is generally accompanied by iritis. The sight is also interfered with by

*Read before the 5th Annual Gathering and Conference of the Trichinopoly District Medical Association on December 21, 1934

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ulceration of the cornea, which leaves scarring in the shape of nebulae and leukomata. These ulcers are often transparent, i. e., not accompanied by any infiltration of the cornea, and they do not stain with fluorescein.

After the trachoma has lasted some time, the mildest cases usually recover spontaneously. In other cases, further serious consequences are met with. The granules may subside leaving cicatricial contraction behind. The conjunctiva becomes smooth, pale, and thin. The transitional folds may disappear, the mucous glands atrophy, and the conjunctiva may become dry and xerotic, likewise the cornea also, leading to total blindness. In others, the tarsal plates become thickened and bent by the contraction of the conjunctiva, so that *entropion* ensues. The bending occurs along the line where the vessels perforate the tarsus. The entropion leads to *trichiasis*, i. e., turning in of the lashes, and this further increases any pannus there may be, or may cause pannus to arise. Trachoma sometimes spreads to the lachrymal sac and causes thickening of it with obstruction. The changes described are usually spread over years. Fortunately, all cases do not suffer from them. But even the mildest case may go on to them, and should hence be seriously regarded. There is no relation between the amount of irritation and the objective appearances, or between the changes in the lids and in the cornea. Relapses and exacerbations often occur unaccountably.

DIFFERENTIAL DIAGNOSIS.—Trachoma is differentiated with difficulty from certain other conditions. These are:

(1) *Follicular Conjunctivitis.* The clinical picture of follicular conjunctivitis consists in the formation of follicles on the conjunctiva of the lower lid. The diagnosis of follicular conjunctivitis can only be verified by the cure of the condition, and the disappearance of the follicles without cicatrization and without the development of pannus. The treatment of the condition is by painting with silver nitrate, 2 per cent solution, if there is a discharge. When the discharge ceases no treatment is necessary, attention to the general health only being required.

(2) *Spring Catarrh.*—Two types of spring catarrh exist: the palpebral form and the bulbar form. In a typical palpebral case, the conjunctiva of the tarsus, especially of the upper tarsus, is covered with papillae which are broad and flattened, so as to make the conjunctiva appear like a pavement

of cobble-stones. Over the whole lies a delicate bluish-white film, as if a thin layer of milk has been poured over the conjunctiva.

In a bulbar case, there is thickening of the conjunctiva at the limbus in the form of brownish, uneven, hard nodules of gelatinous appearance; these extend partly into the clear cornea, but still further in the opposite direction into the conjunctiva. The signs of spring catarrh disappear with the cure of the disease, without leaving any trace of their former existence.

(3) *Parinaud's Conjunctivitis* may easily be mistaken for trachoma with acute onset. This disease is a monocular inflammation of the conjunctiva with formation of follicles, accompanied by swelling of the pre-auricular gland, and sometimes by fever.

(4) *Atropine Irritation.*—Formation of follicles occurs in some individuals who have an idiosyncrasy for atropine. The condition is cured by stopping the administration of the drug.

PROGNOSIS. In those cases in which treatment is commenced before any marked vascularisation of the cornea has occurred, an ultimate cure under favourable external conditions within a limited time can be promised. When gross anatomical changes have already occurred, such as vascularisation of the cornea and incurving of the lid, it is useless to expect complete restoration to the normal. This should be remembered when the cry is raised that trachoma is incurable.

Blindness may be caused by simple trachoma in one of several ways:

- (a) By the formation of thick fleshy pannus;
- (b) By giving way of the cornea under the normal ocular tension and formation of keratoconus or anterior staphyloma;
- (c) By destruction of Bowman's membrane by pannus, and subsequent opacity of the cornea.

TREATMENT.—In spite of repeated experimental and clinical research, not much advance has been made in the treatment of trachoma. General treatment is rightly considered to be of great value. We know that disease of any kind is more easily shaken off when the patient has a sufficiency of food and air and a sanitary habitation. In the local treatment of the disease we have two objects in view: (1) to reduce the inflammation

and secretion, and (2) to cause the disappearance of the granules and conjunctival hypertrophy with as little cicatrization as possible. Both objects are attained by the use of caustics in varying degree. They act by setting up a leucocytosis. When discharge is present, daily application of 1 per cent solution of silver nitrate by means of a cotton swab, and frequently washing with biniodide of mercury lotion (1 in 5,000) do good; adhesion of the lids in the morning is prevented by nightly insertion of boro-vaseline between their edges. In mixed infections, especially with the Morax-Axenfeld diplobacillus, zinc applications are useful. Massage of the lids with yellow oxide ointment is very useful. This is more useful when the secretion is reduced, and then too the lids may be everted and copper sulphate applied. Pannus does not contraindicate the use of copper. Severe photophobia with spasm of the orbicularis is best treated by canthotomy of the external canthus. This divides the orbicularis fibres, enlarges the palpebral opening, and so facilitates treatment; it gives much relief also by the free bleeding that ensues. It is often necessary owing to the contraction of the palpebral fissure that occurs.

When the granules are well-marked they should be expressed. This is done by squeezing them out with Knapp's roller forceps. The operation may require repeating as younger and small granules that escape the forceps grow later. Treatment must go on with copper also after expression. Application of $\frac{1}{2}$ per cent chaulmoogra oil and copper sulphate ointment often does good. Injections of bismuth also have been tried with benefit.

SOME RECENT ADVANCES IN THE TREATMENT OF THE ANAEMIAS *

Dr. U. D. GOPAL RAU,

Madras.

Of the many advances that have been made in Clinical Medicine in recent years, one of the most satisfactory is that in the TREATMENT OF THE ANAEMIAS. A noteworthy feature of this advance is that it has led to newer conceptions of the nature and causation of many of this group of diseases.

Till a few years ago, a good deal of confusion of thought prevailed regarding the pathogenesis of many of the Anaemias. Recent work has clarified the subject considerably, and it is now generally admitted that the old division into PRIMARY and SECONDARY anaemias is no longer appropriate; further, it has come to be recognised, and this is of great moment in regard to therapeutic endeavour, that some of the Anaemias, hitherto looked upon as of unknown origin, are essentially *deficiency diseases*. On the basis of the newer conceptions, OTTENBERG (1933) has suggested placing the Anaemias in three groups:

- (1) DUE TO SOME DEFICIENCY.—To this group belong the anaemias due to lack of the "anti-anaemic" factor (e.g., pernicious anaemia), the nutritional anaemias, and the anaemias due to haemorrhage.
- (2) DUE TO DIRECT INJURY TO THE BLOOD-FORMING ORGANS (*and consequent interference with blood-formation*).—In this group are placed aplastic anaemia (idiopathic and secondary), the anaemias due to radio-active substances, and those associated with certain bone diseases.
- (3) DUE TO HAEMOLYSIS. The anaemias secondary to infections such as malaria, streptococcal septicaemia, and so forth, the anaemia noted in acholuric jaundice, and those due to haemolytic poisons constitute this group.

* A paper read before a Clinical Meeting of the South Indian Medical Union on Thursday, April 15, 1935. Re-written and rearranged for publication.

As a working classification "appropriate for our present day knowledge of anaemias" this is probably the most suitable; but it must be admitted that cases are not infrequently met with which cannot be placed in any of these groups.

Another useful classification is that recently suggested by HADEN. It is given below:

- I. Increased blood loss
 - (a) Mechanical loss from acute haemorrhage
 1. Trauma
 2. Peptic ulcer
 3. Uterine bleeding
 4. Disturbances in blood coagulation
 - (b) Accelerated destruction
 1. Chronic haemolytic jaundice
 2. Haemolytic anaemia due to infections and poisons
 - II. Decreased blood formation
 - (a) Depression of marrow function
 1. Idiopathic aplastic anaemia
 2. Cachexia, chronic intoxication, metabolic disturbances, radio-active substances, malignancy, or infiltration of marrow by leukaemia
 - (b) Deficiency in specific substances necessary for normal marrow function
 1. Deficiency in specific anti-anaemic factor in liver — pernicious and other macrocytic anaemias
 2. Deficiency in iron and other substances necessary for haemoglobin formation: idiopathic chronic hypochromic and microcytic anaemia, diets deficient in protein and iron, chronic haemorrhage
- From the standpoint of treatment, the following classification, adapted from the one recently made by MUSSER and WRIGHT, will be convenient.
- I. Anaemias benefited by vitamin therapy
 1. Pernicious anaemia
 2. Sprue
 3. Pernicious anaemia of pregnancy
 - II. Anaemias benefited by drugs
 1. Idiopathic hypochromic (microcytic) anaemia
 2. Normocytic anaemia
 3. Simple microcytic anaemia
 4. The anaemia of pregnancy
 - III. Anaemias benefited by splenectomy
 1. Splenic anaemia
 2. Chronic haemolytic jaundice.

It is to be noted that since the new developments in treatment took place, the outlook in many of the anaemias has changed considerably. For example, pernicious anaemia, which was once the despair of therapeutics, is now regarded as a disease eminently amenable to treatment. Further, it has come to be recognized that amelioration of symptoms in many of the anaemias is a matter of judicious therapy on modern lines.

The first clinical results which compelled appreciation of the new methods of treatment were obtained in connection with PERNICIOUS ANAEMIA. This disease, of which instances had been reported as early as 1823 (COMBE), was first clearly described by ADDISON in 1855 (and for this reason is often referred to as *Addisonian Anaemia*). BIERMER did much by his writings (1871—72) to awaken interest in the malady, and he gave it the name *progressive pernicious anaemia*. W. PEPPER (1872) described the changes in the bone-marrow occurring in the disease. WILLIAM HUNTER (1888, 1901, 1903) drew attention to the oral and gastro-intestinal sepsis which he believed was the essential factor in the etiology of the condition. Further contributions to the study of the malady have been made, especially in recent years, by CABOT, MINOT and MURPHY, WHIPPLE, ROBSCHT-ROBBINS, CASTLE, and others.

Failing strength, pallor of the surfaces—most marked in the lips, gums, and conjunctivae—dyspnoea on exertion, palpitation, and a mild degree of general oedema are some of the main symptoms. A systolic murmur is often audible at the pulmonary base; and the spleen is palpable in most cases. Many of these patients are remarkably vigorous even though they are extremely anaemic. They are generally able to move about until the red cell count has fallen to nearly 1,000,000 per c. mm. The blood picture shows: (a) a reduction in the number of the red blood cells with relatively less decrease of haemoglobin so that the *colour-index* is greater than 1; (b) intense staining of the corpuscles with irregularity in shape and size—*hyperchromia*, *poikilocytosis*, *anisocytosis*; (c) *nucleated red cells*—*normoblasts*, *macroblasts*, *megaloblasts*; (d) Reduction of white corpuscles. Myelocytes are frequently present in the blood. The blood plates are usually reduced in number. Nervous symptoms such as tingling and numbness, ataxia, and muscular weakness, are present in some cases. Achylia gastrica is an almost constant feature of the disease.

The TREATMENT OF PERNICIOUS ANAEMIA was formerly in a most unsatisfactory state. It consisted in (a) such general measures as rest in bed, nourishing food, and attention to oral hygiene, (b) the administration of arsenic (introduced by BYROM BRAMWELL in 1877), and (c) the correction of infective processes in the alimentary tract (on the basis of HUNTER'S theory). These measures were of little avail, and the cases steadily progressed down-hill, with occasional intermissions, to a fatal termination. The position is very different to day. With the introduction of *Liver Therapy* by MINOT and MURPHY in 1926 the disease has been successfully brought under control. As BECKMAN says, "Since that time the mortality rate for pernicious anaemia has declined at every age-group; so uniformly successful has been the treatment, indeed, that the few failures reported are looked upon with suspicion—either, it is felt, the liver was not properly taken or else diagnosis was faulty." Further improvements and advances which took place since have made the treatment even more satisfactory. Among these may be mentioned parenteral use of liver extract, administration of dessicated stomach, and employment of diets rich in vitamin B.

Liver Therapy. The introduction of *liver* as a therapeutic substance has revolutionized the treatment of the Anaemias generally, and of Pernicious Anaemia in particular.

WHIPPLE and his co-workers (1920, 1925) found that regeneration of blood took place much more rapidly in experimental animals fed on a diet rich in liver than in those in whose diet liver was not included. This formed the basis of the work of MINOT and MURPHY which led to the discovery in 1926 that in the liver an *anti-anaemic factor*, capable of stimulating blood regeneration, is present. MINOT and MURPHY fed patients with pernicious anaemia on a diet rich in liver and red meat and found that this was followed by remarkable improvement in their clinical condition. Since their results were published in 1926, confirmatory reports rapidly accumulated, and it was generally recognized that pernicious anaemia could be effectively treated by the oral administration of liver. A point which was early appreciated was that the liver should be uncooked, if possible, or should be only lightly cooked, it having been found that heat destroyed the anti-anaemic factor.

Further work by COHN, MINOT and others led to the preparation in 1927 of a liver-extract containing the essential anti-anaemic factor, which on oral administration proved as efficacious as whole liver. This was

indeed, a great achievement: for, it made possible the large-scale manufacture of potent liver-extracts of known strengths which could be used in place of whole liver. Many such extracts are on the market; some of these are *Hepol* (Allen & Hanburys), *Livogen* (British Drug Houses), and *Hepatex* (Evans). Latterly (since 1931), extracts containing the anti-anaemic factor have also been prepared for parenteral administration. The best known of these are *Campolon* (German) and *Hepatex* (British).

The clinical studies of CASTLE and others suggest that the anti-anaemic factor is produced from the protein of food during normal gastric digestion—an *intrinsic factor* secreted by the mucosa of the stomach interacting with an *extrinsic factor* supplied in food and forming the anti-anaemic factor. This is stored in the liver, kidneys, and brain for subsequent utilization in blood formation. The nature of the anti-anaemic factor is unknown, but it is claimed to be a peptide. DAKIN, WEST and HOWE have suggested that it is a compound of beta-hydroxyglutamic acid and gamma-hydroxyproline. According to MEULENGRACHT, the intrinsic factor is produced in the "pyloric-gland region" of the stomach and duodenum, that is, in those regions of the stomach and duodenum where *pyloric glands* are present. As regards the extrinsic factor, STRAUSS and CASTLE are of opinion that it is closely related to vitamin B₂, if it is not vitamin B₂ itself.

Recent researches have elicited further information regarding the anti-anaemic factor. GOLDHAMER reports experiments with extracts of human liver which show that (1) the liver of foetuses seven months old contains the anti-anaemic factor; (2) the liver of patients with pernicious anaemia who had been adequately treated contains the factor, but not of those who had not been adequately treated; (3) damaged livers, as from cirrhosis, may not contain the anti-anaemic factor. The last finding is believed to explain the occurrence of macrocytic anaemia in cases of cirrhosis of liver. GOLDHAMER maintains that the anti-anaemic factor found in the liver is merely a storage product and not an intrinsic part of the liver substance. He says that the active anti-anaemic principle elaborated in the stomach is taken to the liver where it is altered and stored so as to be readily available for utilization by the haemopoietic tissues. The researches of WILKINSON and KLEIN show that the anti-anaemic factor is present in normal human liver and in the liver of treated cases of pernicious anaemia in active remission, but is absent from the liver of untreated cases.

Effective treatment with liver produces in patients with pernicious anaemia certain well-defined changes. These may be considered under two headings: (1) changes in the blood, and (2) other changes.

1. *Changes in the Blood.* As MINOT, MURPHY, and STETSON have shown, the response to adequate treatment with liver consists primarily in a prompt and well-marked, but temporary, increase in the number of the reticulocytes circulating in the peripheral blood. The reticulocytes are young red blood cells, and their presence in peripheral blood in large numbers indicates active blood regeneration. In normal blood they constitute 0.5 to 1 per cent of the red blood cells.

The response may be evident as early as the third day after commencement of treatment if the amount of liver administered is adequate; if the dose is less than the optimum, it may be delayed till even the ninth day. It usually reaches a peak in 6 to 9 days; then it falls off, more and more mature red blood cells reach the blood, and the reticulocyte count returns to the normal by about the end of the third week. The rise in the reticulocyte count is directly proportional to the reduction that had taken place in the red blood cell count; in cases in which the reduction in the red cell count is small the reticulocyte response is slight, whereas in cases in which the red cells are greatly reduced in number the reticulocyte response is well-marked.

Along with the rise in the reticulocyte count, there is also a rise in the red cell count. This rise is at first slight, but becomes well-marked with the continuance of the treatment. Eventually, in eight to ten weeks, the red cells approximate the normal in number, provided adequate treatment has been maintained. A compensatory polycythaemia has been noted in some cases. The rise in the red cell count is accompanied by a slow, but steady, rise in the haemoglobin percentage, which also returns to the normal in about the same time as the red cell count. There is, in addition, a return to the normal in size and shape of the red blood cells.

The blood platelets and the granular leucocytes also are increased in number as a result of the treatment. A well-marked leucocytosis may often be noted. An eosinophilia of 20 or 30 per cent (and occasionally even one of 70 per cent) is frequently observed 4 or 5 weeks after the commencement of treatment.

2. *Other Changes.*—Even during the first week the skin begins to acquire colour, and it rapidly becomes healthy and moist in appearance. The mucosae also share in the change. In a large proportion of cases the tongue lesions clear up, and the organ becomes normal. The haemic murmurs, oedema, and splenic enlargement disappear as the blood condition improves. The weight increases, the gastro-intestinal disturbances disappear, the basal metabolic rate becomes lowered, and an increase in strength follows. Rapid and well-marked improvement results in the mental condition. The appetite returns, dizziness and dimness of vision disappear, and headache and dyspnoea are no longer complained of. The tingling and numbness of the extremities become less marked, and may disappear entirely.

The improvement is maintained as long as the patient continues to take liver in adequate doses.

As in diabetes mellitus, so in pernicious anaemia, a *cure* is not to be expected, and the aim of treatment is to restore the condition of the patient to the normal and to maintain him in continued health and strength. In pernicious anaemia this aim can be achieved, as has been mentioned, by the continued administration of liver in adequate doses. But what constitutes adequate dosage?

The amount of liver required to restore the condition of the patient to normal, that is, the *restorative dose*, is by no means the same in every case, as patients differ widely in their requirements. Early experience suggested that an *adequate restorative dose* for an average adult would be half-a-pound per diem of whole liver. Some patients may require as much as even one pound per day. The liver may be given either raw or lightly cooked. Various recipes have come into use for preparing the liver. It is, however, important to see that in cooking the anti-anaemic factor is not destroyed. Prolonged boiling and over-cooking destroy it. The anti-anaemic factor is water-soluble, and, therefore, the watery part of liver preparations should also be taken by the patient.

Instead of whole liver, the patient may be given by mouth a liver extract, preferably a palatable one, such as *Hepatex* or *Livogen*. The dose of the extract is based on its potency in the anti-anaemic principle as compared with whole liver. An amount of the extract having the same potency as half a pound of whole liver would be an average *restorative dose*.

for an adult. The small bulk of the extract, its keeping properties, and its palatability are advantages favouring its employment. The extract is available also in powder form. It may be taken in any suitable vehicle, according to the taste of the patient, as it matters little in what form the anti-anaemic factor is taken, provided the dose is adequate.

Parenteral administration of liver extract, intramuscular and intravenous, has many advocates. It was introduced by GANSSLEN in 1931. It has certain distinct advantages. It produces a more rapid and more powerful action than oral administration. *In desperate cases, the intravenous route is the route of choice.* An intravenous injection will act at once, and show its effect immediately; by the oral route much too long a time has to elapse before the effect is obtained. Further, it must be noted that the response to treatment is of a quantitative nature, a large dose given every 6 days will produce the same effect as one-third of the same dose given every two days. Intramuscular injection is less rapid in action than the intravenous, but is more rapid and potent than oral administration.

Of the extracts available for parenteral administration, two have been specifically mentioned, viz., *Hepatex* which is available both for intramuscular and intravenous administration, and *Campolon* which is available only for intramuscular use. I would give a word of warning in regard to *Campolon*. Recently, one of my patients, who was being treated for an anaemia of the pernicious type, developed after an injection of *Campolon* severe anaphylactic shock, and within half an hour was on the verge of collapse. It is necessary, therefore, to use this preparation guardedly.

As regards these extracts in general, it is sometimes very difficult to determine in what doses they should be used as many of them are not standardized. DAMESHEK and CASTLE have pointed out recently that the descriptions of potency of the commercial extracts offered for parenteral use are often extremely vague. As they have rightly stressed, unless the dose of these preparations is clearly defined in terms of what constitutes the maximal response, it will be impossible to form an accurate estimate of their relative potency.

When the condition of the patient has improved, and restoration of the blood to the normal has taken place, the dose of liver extract is to be reduced. He should now be given a *maintenance dose*. The maintenance

dose is half-a-pound of whole liver, or its equivalent of a liver extract orally or parenterally every two days.

DAVIDSON has found that the intramuscular injections of a liver extract (*Campolon*, 5 c.c.) at fortnightly intervals more effective in maintaining the patient in good health than peroral administration of liver or liver extract. As he claims, this procedure is certainly cheaper. But, in this connection, it will not be out of place to quote the words of BECKMAN: "There can be no doubt there are circumstances in which it is of distinct value to have available an actively potent substance which may be given parenterally, such as in a moribund patient, or a "resistant" case when the resistance may be due to poor alimentary absorption, or in an individual who is absolutely unable to take the whole liver or extract by mouth, but to advocate the sweeping replacement of oral by parenteral administration, merely because the latter is at the moment cheaper, appears to me not to be in keeping with the tenets of sound therapeutics. This is of course merely the viewpoint of one who is primarily a pharmacologist, but I have been delighted to find MUSSER expressing the same feeling from the practical standpoint of a research clinician. Let us delay as long as possible before we foist upon pernicious anaemia patients that slavery to the needle which is the bane of the diabetic's existence."

Stomach Therapy. In 1929, STURGIS and ISAACS made the discovery that oral administration of desiccated and defatted hog's stomach was as effective as liver in the treatment of pernicious anaemia. This discovery, which was proved not only of great practical importance, but also of considerable theoretical interest, obtained speedy confirmation from other observers, notably SNAPPER and du PREEZ, WILKINSON, GANSSLEN, and DAVIDSON. And it is universally admitted at the present time that stomach preparations can be used in place of liver, with the proviso that the doses employed are adequate. It is also felt that the relative cheapness of these preparations, of which there are many on the market, e. g., *Ventriculin* (Parke, Davis & Co.) *Gaster Sicca* (British Drug Houses), and their freedom from bad taste might make them supersede the liver preparations intended for peroral medication.

The efficacy of stomach preparations in pernicious anaemia has been explained as due to the presence in them of the active anti-anaemic factor. It is believed that when fresh stomach tissue is ground up, the *intrinsic factor*

of CASTLE found in the *pars mucosa* acts on the *pars muscularis* (which provides the *extrinsic factor*) to form the active *anti-anaemic factor*. The anti-anaemic factor thus formed is retained in the stomach preparation. On ingestion of the stomach preparation the active anti-anaemic factor is rapidly absorbed and becomes at once available for blood regeneration. Recent work by WILKINSON and KLEIN substantiates this view. These observers have separated from the stomach an enzyme-like substance called *haemopoietin*, and they have found that when this haemopoietin is incubated with beef-muscle an active haemopoietical element is obtained which closely resembles the anti-anaemic factor in the liver. This haemopoietical element may be prepared in a form suitable for parenteral administration.

As regards the dosage of stomach preparations, STURGIS and ISAACS found 10 gm. daily for each million deficit in the red blood cells to be a satisfactory commencing dose. The maintenance dose varies somewhat with different patients; but in most cases 10 gms. daily for five or six days a week will suffice.

Liver Gastric Therapy. It has been found that by the addition of a small quantity of stomach tissue to liver extract a product could be obtained which, when given orally, is three times as effective as liver extract and five times as effective as prepared stomach. *Extralin* is a product of this kind prepared by incubating liver extract with stomach tissue. It is put up in capsules containing 0.5 gm., which is equivalent to 20 gm. of fresh liver or half that quantity of fresh stomach.

Yeast (or vitamin-B₂) Therapy. This has not proved effective in pernicious anaemia, probably because the intrinsic factor is lacking in the stomach in this disease. But WILLS has successfully used marmite (autolysed yeast containing vitamin-B₂) in the treatment of cases of tropical macrocytic anaemia. This anaemia resembles in blood picture pernicious anaemia, but differs from it in other features. It is likely that the intrinsic factor is present in the stomach in this type of anaemia, and that the interaction of marmite with the intrinsic factor produces the anti-anaemic factor.

Diet in Pernicious Anaemia. A suitable diet is of the utmost importance not only in pernicious anaemia but also in the other anaemias. There are not hard and fast rules to follow, but the dietary should include a good proportion of fresh fruit and lightly cooked or raw vegetables, red meat in finely divided form (at least 4 oz. daily), and liver (about 4 oz.

daily). Fat intake should be restricted as far as possible; cheese, bacon, and fried foods must be avoided. Sweet and starchy foods, especially potatoes, rice, milk, and bread, should be taken only in limited amounts.

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Of recent years, a form of Anaemia, to which the name SIMPLE ACHLORHYDRIC ANAEMIA, IDIOPATHIC HYPOCHROMIC ANAEMIA, and IDIOPATHIC MICROCYTIC ANAEMIA are given, has attracted a good deal of attention. As these names suggest, it is a hypochromic, microcytic anaemia, associated with hypochlorhydria. It occurs especially in middle-aged and elderly women, has an insidious onset, and is attended by dyspeptic symptoms. The usual clinical features are loss of appetite, palpitation, asthenia, glossitis, numbness of the extremities, and dysphagia. Blood examination shows reduction in the number of red blood cells and a low haemoglobin percentage. The colour index is very low, often 0.5 or 0.6. The average size of the red cells is decreased. The combination of glossitis, anaemia, and dysphagia (usually occurring in this disease) is known as the *Plummer-Vinson Syndrome*. The cause is unknown. Deficiency in iron and defective diet are suggested as probable factors. *Treatment* consists in attention to general hygiene, the ordering of a diet rich in vitamins, and the administration of iron in large doses. Dilute hydrochloric acid should be given to correct the achlorhydria. When the blood has returned to the normal, a maintenance dose of iron should be prescribed to prevent recurrence. DAMESHEK advises 15 grains of iron and ammonium citrate daily for this purpose.

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The variety of Anaemia to which the name CHRONIC SECONDARY (OR SYMPTOMATIC) ANAEMIA was formerly given, and which is referred to now as CHRONIC SECONDARY MICROCYTIC (OR HYPOCHROMIC) ANAEMIA should receive brief consideration here. It occurs with some other disease to which it is secondary, and for this reason is known as SECONDARY ANAEMIA. It is characterized by a low colour index and a reduction in size of the red blood cells. It results from various causes, e.g., *chronic loss of blood; malnutrition; prolonged lactation; such infections as typhoid fever, rheumatic fever, sepsis, and malaria; the action of such poisons as lead, mercury, and arsenic; infestation with Ankylostoma duodenale*. It is due to iron deficiency, the amount of available iron being insufficient for normal haemoglobin formation. Such an iron deficiency may be induced by (a) depletion of the iron reserve of the body through continued loss of blood; (b) insufficient iron intake (in the food); (c) improper absorption

of iron from the alimentary tract; and (d) inability to utilize the available iron.

In the TREATMENT of this form of anaemia, in addition to (1) the general regimen, which consists in rest, nourishing food, and fresh air, and (2) the correction of the causative factor at work, (3) **Iron Therapy** is of the greatest value. Reduced iron, 1.5 gm. daily (22 gr.) or iron and ammonium citrate, 6 gm. (90 gr.) daily, in three divided doses after meals, gives gratifying results. Iron by injection is of no special value. Iron and ammonium citrate, 2 gm. (30 gr.) thrice daily by mouth, increases the haemoglobin 1 per cent a day, and even more rapidly if the anaemia is very severe. The action of iron preparations is augmented in general by the simultaneous exhibition of some of the products containing vitamin-B. Extracts of liver and stomach preparations are also of value in stimulating blood regeneration.

The experimental studies of HART, STEEBECK, WADDELL, ELVEHJEM, and SHERMAN have shown that, in the presence of **copper**, iron salts are more effective in promoting haemoglobin formation than when they are given in pure form. MILLS reports cases of microcytic anaemia in which the combined use of iron and copper was effective when iron alone had failed. It can scarcely be denied that small quantities of copper are helpful in enabling the organism to utilize the iron in blood building. But, as recent investigations show, the minute quantities of copper required for this purpose are found in green vegetables and other articles of food that it is scarcely necessary to administer copper so long as well-balanced diets containing these articles are given. Further, it has been found that the ordinary commercial preparations of iron used for medicinal purposes contain as impurity sufficient copper for the needs of even the anaemic organism. Besides copper, other mineral substances, e. g., *manganese*, *germanium*, have been considered useful in stimulating blood-building. But the evidence available does not justify their employment in practical therapeutics. **Arsenic** enjoys considerable reputation as a haematinic, but some recent observations seem to throw doubt on its efficacy in this respect. It may, however, be used as an adjunct to iron in selected cases.

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Anaemias due to IRON DEFICIENCY are frequently met with in infancy and childhood. They are *nutritional anaemias* for the most part.

It is known that iron deficiency in children may predispose to repeated attacks of bronchitis, otorrhoea, nasal discharge, and cervical adenitis. In looking for the causation of this iron deficiency, it has been found from calculations of an elaborate nature that the deficiency cannot be accounted for in the average child on the basis of relative absence of iron in storage at birth and normal rapid growth, and it has become necessary to seek for other factors that reduce the amount of iron in the body. *Prematurity* is one such factor. *Infection* is a second. *Malnutrition* is a third. **Iron** is the sheet-another of *treatment* in these cases. To withhold iron is to court disaster. BASS and DENZER say, ".....what happens to anaemic children who do not receive iron? In the group of children with mild anaemia there is a strong tendency to spontaneous improvement and sometimes to cure with advancing age. Thus, many of these babies, develop into children somewhat pale and generally inferior, and the infantile anaemia responsible for their condition may be forgotten entirely. Another group of infants succumb to intercurrent infection, and the anaemia plays an important but unsuspected part in the fatal outcome. One of us has called attention to the relation between pneumonia and anaemia and the beneficial effects of transfusion. A third group of infants develop anaemia of such severity that transfusion becomes an emergency measure... When seen at this late stage of anaemia, one is apt to forget that iron therapy earlier in the disease might have prevented a catastrophe." Iron should be given in large doses; 2 to 4 gm. (30 to 60 gr.) daily are advocated even for infants. Further, it is felt, by the administration of iron as a routine at welfare centres, it would be possible to effect a considerable reduction in infant mortality, as anaemia due to iron deficiency is an important cause of death in infancy, especially among the poorer classes.

GLEANINGS FROM MEDICAL LITERATURE

7 THE DEBT OF SURGERY TO ROENTGENOLOGY

DONALD C. BALFOUR

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On December 3, 1934, The Radiological Society of North America inaugurated the Carman Lecture in honour of the memory of Russell D. Carman, one of the pioneer radiologists of the world, whose untimely death took place in 1926. On this occasion Dr. Donald Balfour of the Mayo Clinic gave a lecture from which the following is reproduced. Dr. Balfour is a world renowned surgeon and gastro-enterologist, and a summary of The Debt of Surgery to Radiology by him, dealing as it does with aspects of Gastro-enterology, will be, it is hoped, instructive.

"It has been during the past twenty-five years that the progress of our knowledge in respect to lesions of the stomach and duodenum has been most rapid. In the first place, pathologists took advantage of the opportunity afforded by the surgeon of studying lesions in their various stages of development, rather than being confined to study of those lesions which had terminated the life of the patient. For this reason a more satisfactory classification of such lesions, from a pathologic standpoint, can now easily be made.

Great advances also have taken place in our knowledge of the physiology of the stomach, both in health and disease, and particularly in its motor and secretory functions. The clinical manifestations of disease have been better understood because of this pathologic and physiologic background and the known character of lesions in their various stages and their effect on gastric function.

Strangely enough, not yet is as much known about the uninterrupted clinical course of benign lesions of the stomach and duodenum as is desirable, in the first place because many of these lesions do not give sufficient symptoms to be recognized, and secondly because the course of the benign lesion is usually so chronic in a large group of cases that a much longer period of observation is necessary to get a true estimate of the prognosis. The great stumbling block is the lack of knowledge as to the cause of benign lesions of the stomach and duodenum, and in the absence of any known cause, theories are numerous.

The greatest impetus to our knowledge, however, has been the accuracy of diagnosis, since all therapeutics must depend for its success or failure primarily on this, and it is in accurate diagnosis that the difference exists between quackery and scientific medicine. Since roentgenology has made such accuracy possible, it is difficult to overestimate the direct benefits of roentgenology to humanity. And, when it is considered that as yet no direct method, such as intragastric photography or visualization by gastroscope, has equalled in any respect what can be determined indirectly by roentgenology, one's amazement grows that any such method should attain such precision. To take advantage, therefore, of this precision, I shall attempt to

outline briefly what appear at the moment to be some of the more important lesions of the stomach and duodenum and their treatment.

The most common lesion of either the stomach or duodenum is ulcer, and there are certain important facts which have significance to the clinician, roentgenologist, and surgeon. The lesion is very frequently multiple; in some recent reports ulcers or scars of ulceration have been shown to be present in 100 per cent of cases. It is this multiplicity of lesions and the constant tendency for such lesions to heal which is responsible for the great variety of deformities of the duodenum seen at operation. The most constant of these is the shortening of the duodenum that can be shown in all cases in which a lesion has undergone any attempted healing. Perhaps of greater interest to the roentgenologist is the pouching that may occur on either the superior or inferior border, or on both, and which sometimes results in what appears to be a true diverticulum. The most important consideration from a clinical standpoint, perhaps, is that by far the greatest number of the extensive lesions are on the posterior wall, and appears that these lesions are the most difficult to heal, probably because they become easily fixed to retroperitoneal tissues, namely, in the head of the pancreas. When such fixation has occurred, healing seems almost out of the question. While inflammatory processes may occur in any situation, they are usually confined, however, to the first portion of the duodenum, and they exhibit lesions in every stage of development and healing.

One of the most interesting problems to the solution of which the roentgenologist contributes is that of duodenitis and its relationship to actual ulcer. It is, I think, commonly agreed that the inflammatory area of the duodenum may be, and probably is, the precursor of actual ulceration, so that the exact recognition of duodenitis by roentgen rays would be a very great accomplishment. The difficulty is that duodenitis is so frequently found to be associated with other lesions that management must be directed toward a multiplicity of lesions rather than toward a single inflammatory process. Just as the pathologic and physical characteristics of duodenal ulcer are variable, so the symptoms are exceedingly variable, and it is difficult in many cases to correlate these symptoms with the pathologic changes present. For the same reason treatment must be adjusted to these symptoms, so that it will vary from none at all being indicated, the lesion healing spontaneously, to removal of the inflammatory area with a substantial portion of the stomach to eliminate all known factors in the production of the lesion. Between these extremes, however, there are a number of measures designed for control. All these have the same purpose, namely, to provide as much rest as possible for the lesion and to so modify gastric function that sufficient rest will be maintained. In medical treatment this is attained by frequent feedings of a small, bland diet, with necessary alkalies, to control hyperacidity; from a surgical standpoint the same purpose is attained by various types of operations.

The roentgenologist will, with experience, find in the behaviour of the stomach of the patient with duodenal ulcer certain suggestions as to what will be the most successful operation. When the patient has marked obstruction from duodenal ulcer, impairment of motility, as shown under the fluoroscope, suggests, or at least very clearly indicates, that relief of this impairment will be all that is necessary. In this his supposition would be quite correct, because satisfactory results are obtained in cases in which there are such disturbances of function. When the stomach shows great hyperactivity, the operation of gastro-enterostomy, which is so

effective when an obstruction exists, does not bring about as satisfactory results, for it is this very hyperactivity that makes possible the development of recurrent lesions. In this type of case, therefore, local operations on the outlet of the stomach are preferable, and in rare cases, when marked haemorrhage has occurred, some method of partial gastrectomy is desirable. Of these methods of partial gastrectomy, probably restoration of gastric continuity by uniting the stomach and duodenum is preferable to uniting the stomach and jejunum. The results of treatment of duodenal ulcer show it to be a disease that can be controlled, in from 85 to 90 per cent of the cases, by the co-ordinated efforts of the roentgenologist, physician and surgeon.

Duodenal diverticulum is an example of a condition that can be detected only by the roentgenologist. It is true that only after it is found will a clinician assume that there are symptoms connected with the diverticulum, but these symptoms are so vague that I have never known a diagnosis to be made on clinical examination alone. The incidence of true diverticulum is about 2 per cent. Foreign bodies and benign tumors also can be detected only by roentgenologic examination.

Cancer of the stomach is a disease which may occur at any age, and various pathologic forms may involve any part of the stomach and vary greatly in virulence and in the symptoms produced. With all the discussion as to how more could be accomplished in attacking this serious problem, there is one fact that is more important than all others. That fact is that the hope of the patient with cancer of the stomach lies in the roentgenologist. This is for the reason that although in its early stages cancer of the stomach seldom gives rise to definite symptoms, usually there are sufficient signs and circumstances to arouse the suspicions of the discerning physician, and it is hoped that in the future the patient will be sufficiently educated to ask for the only method by which an early diagnosis can be made, namely, competent roentgenologic examination. The roentgenologist, therefore, probably has his greatest responsibility in this disease for the reason that it is in these early cases that removal is so much more likely to be followed by cure. There are certain aspects of cancer of the stomach which I think are particularly important. First, that while it is true that roentgen rays offer by far the best method of examination, it is also true that even in experienced hands a fairly advanced lesion may be overlooked. Therefore, I believe that when the clinician suspects cancer of the stomach and a single roentgenologic examination does not substantiate this opinion, a second examination or repeated examinations should by all means be made at an interval of a few days.

Again, with advanced carcinoma, the roentgenologist should co-operate with the clinician and surgeon in deciding whether exploration is worth while because of involvement of the upper part of the lesser curvature, since it is possible to remove the entire stomach and it has been shown that a patient can live in comfort with an anastomosis between the oesophagus and jejunum or the oesophagus and duodenum. I believe, therefore, that a patient with cancer of the stomach should never be dismissed as hopeless until there is a direct discussion of the whole situation with the roentgenologist. In the treatment of cancer of the stomach, curability, under the circumstances, should always be stressed, and when the disease is confined to the stomach, complete removal of the lesion at operation results in patients being alive and well at the end of three years in approximately 50 per cent of the cases. There are a great number of

procedures for dealing with gastric cancer, all being based on general principles, namely, thorough removal of the growth and restoration of continuity so that the anastomosis will function well.

Gastric ulcer is much less common than gastric cancer. Its well known predilection for the lesser curve at the angle is fortunate because this is a situation in which the lesion is readily accessible to surgical attack. It has been shown that those lesions on the greater curve are more likely to be malignant than benign, and for that reason they should perhaps be distinguished from those on the lesser curve. Lesions high on the lesser curve are perhaps of a less serious nature than those between the incisura and pylorus. There is still a good deal of confusion about the pathology of ulcerated lesions of the stomach, and I think this is largely unnecessary because as yet there is no way of determining by any gross methods of examination what the microscopic picture of a lesion is; however, more important contributory evidence has been brought by roentgen rays to differentiate malignant and benign lesions. Nevertheless I think one of the most important points in this respect was made by Carman when he adopted the practice of frequently referring to ulcers of the stomach as "lesions" rather than as "ulcers."

Caution should also be used in drawing deductions while observing the course of gastric ulcer by means of roentgenologic examination. It is well-known now that the malignant ulcer may respond to intensive medical management not only in relief of symptoms, but also in decrease in size of the lesion, yet we have seen a number of cases in which the result of medical management has cost the patient his life, in that it seemed to prove the lesion was benign. The treatment of gastric ulcer should also take into consideration these possibilities, and in those cases in which surgery seems indicated the most important fact is that if the competent roentgenologist has identified the lesion in the stomach, however small, the surgeon is probably wrong if he fails to find it. I have been amazed on a number of occasions to find, after great difficulty, of course, a lesion awkwardly situated on the posterior wall and of such small size that it could be entirely over-looked had not the roentgenologist stated that it had been seen.

Gastritis is probably the most common of all conditions of the stomach, since it always accompanies in some degree any lesion whether malignant or benign. As yet in this country there has been no tendency to direct any surgical measures toward gastritis *per se*, but I believe one should keep an open mind as to the relation between gastritis and chronic lesions of the stomach, and we will probably again be indebted to roentgenology for establishing more accurate methods of determining the presence and degree of gastritis.

Benign tumors of the stomach are either symptomless or associated with secondary anaemia, the source of which would entirely be unsuspected were it not for the visualization by roentgen rays. Since these tumors are often the cause of very serious injury to the blood, and since they can be readily removed by operation, with complete cure of the patient, roentgen rays can again be given the entire credit for the successful management of the condition. Again, with foreign bodies, and with gastric syphilis, the establishment of the diagnosis depends to a large extent on roentgenologic examination.

It has recently been shown that herniation of the stomach is a much more common condition than previously was thought, and while the symptoms of herniation through the

oesophageal opening have been studied by Harrington and certain significant points have been established, again the only method of determining whether such herniation exists is by means of the fluoroscope. To emphasize the contribution in this field one might say that during the past year 41 cases of herniation were identified as against approximately five cases twelve years ago.

The pylorus offers some interesting problems to roentgenology. By far the more secondary effect of intragastric and extragastric disease is pylorospasm, and there has been much speculation not only as to its significance as a reflex condition, but, and probably more important, as to its relationship to the development of intragastric disease, particularly peptic ulcer. I believe it is generally agreed that persistent and recurrent pylorospasm in some way disturbs gastric function, to an extent that inflammatory processes are not only more prone to develop, but are less likely to heal when marked pylorospasm is present. The detection, therefore, of pylorospasm, and as of the various degrees of pylorospasm, would be an important contribution to our knowledge of the etiologic factors concerning peptic ulcer.

Another condition which has given rise to a good deal of interest and which is of much clinical importance, is hypertrophy of the pyloric muscle. We have seen some extraordinary examples of this condition among adults, in which hypertrophy was demonstrated by roentgenologic examination and was present beyond any question at operation, and from which the patients were completely relieved of symptoms (usually somewhat suggestive of ulcer) by permanent ablation of the muscle. The pylorus, and its effect on gastric function and its relationship to chronic disease, is, I believe, worthy of considerable further study in which the roentgenologist will again play a prominent part.

The most difficult problems which present themselves to the clinician, roentgenologist, and surgeon are involved in those cases in which patients have not been relieved by operation of symptoms due to benign lesions, or in which secondary ulceration has developed. The clinician and surgeon are confused largely because the rule is that good results are obtained when operation is clearly indicated and properly performed. In cases of such secondary ulceration one is constantly dealing, therefore, with the exception rather than the rule, and to the problem of the lesion itself is added the necessity of evaluating the physiologic results of the operation. In a similar way the roentgenologist finds that in examinations of the stomach in which there is secondary ulceration interpretation is difficult. This is to a large extent the result of the fact that the correlation between the roentgenologic findings and symptoms in cases in which various types of operations have been performed is not as definite as in cases in which patients were not operated on, and one could very properly emphasize the danger of unwarranted deductions being made from what appeared to be deviations from normal in the roentgenologic findings. I have repeatedly seen patients, who had attained perfect results from operation and who had normal digestion, become disturbed because the roentgenologic report showed dilated intestinal loops or some deviation from normal in the emptying of the stomach.

It is just as true in the stomach and intestines as in the other parts of the body that, if normal function is present, form, position, and physical signs are not of much significance. Yet with all these difficulties of interpretation it is still true that roentgen rays offer the greatest help in these cases of secondary ulceration, and it is also true that the greatest co-operation

between clinician and roentgenologist is necessary in such cases. The determination of whether the primary lesion has been reactivated or whether a new lesion has developed is of the utmost importance in the management of the patient. In those cases in which a short-circuiting operation has been done for an inflammatory process in the duodenum, and in which there is recurrence of symptoms, the surgeon is particularly interested in how the pylorus is functioning. It may be assumed in such cases that the inflammatory process in the duodenum, if the gastro-enteric stoma has functioned well, has healed, but since healing is always associated with some deformity, unwarranted deductions must not be made because such deformity is demonstrated by roentgen rays, and it is known that even considerable deformity in the duodenum may be entirely consistent with good digestion.

The value of roentgen rays in this field is such that the experienced roentgenologist frequently is able to suggest to the surgeon what type of operation would probably be best in the case in which further surgery seems advisable. This group of cases, I believe, is the high mark in illustrating the place roentgen rays have in the diagnosis of lesions of the stomach and duodenum."

8

FOOT PAIN

JOHN FRASER

The Practitioner, Vol. CXXXIII, September, 1934, p. 304

Evidence exists that foot complaints are on the increase. This is attributable probably to two factors: (1) failure to observe physiological principles in the matter of foot-wear; (2) much walking on the unyielding surfaces of streets and pavements.

PAIN IN THE HEEL.—In examining for pain in the heel the point of maximum pain should be first located, and whether the pain is induced by pressure or is a subjective symptom should be ascertained. Pain is usually felt in the heel at one of three points—(1) the posterior aspect; (2) the under surface; or (3) as a more central manifestation—in this case it is usually referred to the medial or lateral surface.

Pain in the posterior aspect of the heel may be due to: (1) A *calcaneal apophysitis* resulting from an irritation of the growth centre of the calcaneal metaphyses, owing to severe muscular strain applied through the tendo-calcaneus upon the apophysis of the heel bone, such as may occur in a young school boy who has indulged in long cross-country runs. Pain is felt on dorsiflexion and on pressure on the posterior surface of the calcaneus. *Treatment* consists in rest, avoidance of pressure over the tender spot, and counter-irritation or diathermy. (2) An *inflammation of the bursa* between the anterior surface of the tendo-calcaneus and the posterior surface of the heel base may occur in those subject to long hours of standing and walking on uncarpeted floors and corridors, probably in tight-fitting shoes.—*Treatment.*—Relief of undue pressure on the sensitive parts, of congestion by means of heat, and counter-irritants; of failing these excision of the bursa. (3) A *teno-synovitis of the calcaneal tendon* such as is commonly met with in army recruits condemned to long hours of standing and marching in the army boots which are specially reinforced at the heel. The line of the sinew is tender to the

touch, and movements of dorsi- and plantar-flexion are extremely painful. *Treatment*.—Relief of pressure over part by a pad and counter-irritation.

In every one of the above, a search for and eradication of any toxic focus should form part of the treatment. Pain on the under surface usually originates in the fibrous attachments of the muscles and fasciae. Usually a calcaneal spur visible by X-ray exists and it is particularly liable to affect those subject to rheumatism, to osteoarthritis, and those who have had urethritis in the past. *Treatment*: Operation is usually a failure and most relief is obtained by a rubber or sponge heel with depressions cut over the painful area.

Pain referred to the side of the heel bone usually signifies a central disturbance as tuberculosis or chronic osteomyelitis, and treatment is operation or fixation in plaster or splint.

TARSAL PAIN.—Pain referred particularly to the region of the midtarsal joint may be due to: (1) *Tarsal scaphoiditis* (Kohler's disease) to be treated by prohibiting weight-bearing, and encasing part in splint for three months. (2) *Chronic infective osteomyelitis*, tuberculous or pyogenic, to be treated by conservative methods, immobilization, aided by local exercise, if need be. (3) *Plantar flat-foot*, relieved by rest, massage, bathing the feet in hot water, and supporting the fallen arch. (4) *Chronic arthritis of the intertarsal joints*, to be treated by rest during the acute stage, and later by manipulation under an anaesthetic, and massage and diathermy to restore the fullest measure of mobility in the affected joint.

METATARSAL PAIN.—Pain in the metatarsal area is usually found in the distal ends of bones, especially the second and third metatarsals, and may be due to: (1) *Static causes* as metatarsal or anterior flat-foot—to be treated by correcting the displacement by circular metatarsal bands placed immediately behind the heads or by a metatarsal bar. (2) *Traumatic causes* as in the condition known as *March fracture* to be treated by fixation. (3) *Defective causes*: osteochondritis of the metatarsal heads (Kohler's disease of the metatarsals) to be treated by fixation in plaster, with a supporting pad of felt placed immediately behind the heads.

Other conditions causing foot pain are usually such obvious conditions as *hallux valgus* and *rigidus* and *hammer-toe*. Pain localised beneath the head of the first metatarsal may be due to a fracture of one of the sesamoids and pain beneath the nail of the great toe to a subungual exostosis. It must not be forgotten that while foot pain of a general character, not localised to any particular point, with pain on movement or weight-bearing, may be met in rheumatoid arthritis or flat foot, or is frequently the result of a protracted local urethritis—the so-called gonorrhoeal rheumatism.

M. K.

THE CENTENARY OF THE MADRAS MEDICAL COLLEGE

A century is an achievement to be proud of, not in cricket alone, but in the life of any institution, especially so of an institution which has been founded with the sole aim of alleviating human suffering and ultimate prevention of human diseases. The MADRAS MEDICAL COLLEGE has already lived a hundred years, and steps are being concerted to mark the event with appropriate celebrations. We are happy to note that the first meeting that was held in this connection was very well attended by the Old Students of the College. The celebration of the *centenary* of the college is primarily the function of its *alumni*, past and present. But through the Medical Profession (including its staff), the Medical College comes very intimately in contact with the vast general public, rich and poor, humble and exalted. It is thus as much a duty of the public as of the Profession to celebrate the occasion befittingly. It was thus that the prominent citizens of the City were invited to the public meeting that was held to organize measures for the celebration. The response of the Public has been very good as seen by the large number of prominent people that were present at the public meeting. We are, therefore, confident that with the enthusiasm of the Public and the energy of the *alumni*, the event would be well celebrated. Those who are engaged in this task have been very thoughtful. They are aware that the celebrations should not lack dignity and grandeur. But what form the memorial should take has been exercising the minds of many. There are many suggestions. As usual, each of these is meant to supply some urgent want, and none is without some defect. It is, however, the common wish that, whatever form the memorial should take, it would not end in blocks of brick and mortar. Further, there is an increasing desire among medical men here that the occasion should be availed of to bring into existence an organization that would help the members of the Profession to extend their knowledge either by research or intensive courses in the wards and laboratories. Whatever form the memorial might take, fairly large funds would be required. We hope that the students of this College who are in office or practice in the various parts of the Presidency and the adjoining States would exert themselves seriously to speak to their friends and clients and help in the collection of funds. The success of the memorial depends entirely on their exertions.

MEDICAL PERIODICALS

THE INDIAN MEDICAL GAZETTE

VOL. LXX NO. 7, JULY, 1935

Arterial versus Venous Blood Sugar—Arterio-Venous Sugar Difference as a Criterion of the Severity of Diabetes, J. P. Bose, p. 361.

"In normal healthy subjects—(a) the arterio-venous sugar difference is negligible in the fasting stage, the average being 3 mgm. per 100 c. cm. (b) after the glucose meal, the arterio-venous difference rises, the maximum being obtained at the end of one half hour's time; (c) the average arterio-venous difference obtained in one hour period was 35 mgm. the variations ranging from a minimum of 26 mgm. to a maximum of 45 mgm. per 100 c. cm. of blood. (d) the arterio-venous difference rapidly decreases one hour after the glucose meal, coming almost to the post-absorption level at the end of two and a half hours.

In mild cases of diabetes—(a) the fasting arterio-venous difference did not differ materially from the normal cases; (b) after the glucose meal, the arterio-venous difference was much less marked than in the normal case, the greatest difference (13 to 20 mgm. per 100 c. cm. instead of 26 to 45 mgm. as in normal cases) occurring at the end of one hour after the meal; (c) the fall in the sugar content of both the arterial and the venous blood began at the end of one and a half hours (instead of one hour as in normal cases); (d) the arterio-venous difference regained the original level at the end of two and a half hours.

In severe cases—(a) the fasting arterio-venous difference was almost always on the negative side; (b) after the glucose meal there were at least five cases in which the rise in the sugar content of the arterial blood exceeded that of the venous blood, thus converting the initial negative arterio-venous difference into a positive one, though of a small degree. The other cases retained the negative difference throughout the test."

Tebetron in Indian Strains of Malaria, R. N. Chopra, B. Sen, and S. K. Ganguli, p. 362.

"Tebetron resembles quinine and atebrin in its action on Indian strains of malaria. It destroys the asexual and sexual forms of *P. vivax* and *P. malariae*, but only the asexual forms of *P. falciparum*. Its action on *P. malariae* is comparatively slower and less potent. Like atebrin it is not unpleasant to take, but the drug has to be given in much larger doses than atebrin. As compared with cinchona alkaloids the drug is much more expensive and appears to have no particular advantage over them."

The Treatment of Obstruction of Lacrimal Duct and Chronic Dacryocystitis, Jadavji Hansraj, p. 366.**The Rate of Development of Hookworm Eggs, P. A. Maplestone, p. 368.****Ewing's Sarcoma—A Clinical and Pathological Study of a Case, Amar Nath Goyle, A. Vasudevan, and K. G. Krishnaswamy, p. 371.**

"A case of Ewing's sarcoma arising from the femur of the left side in a man aged 35 years is described. Though the task of tracing the derivation of the neoplastic cells is difficult it is suggested that they arise from the reticular cells on the basis of their morphological and structural characters. If this view be accepted, reticulum cell sarcoma would undoubtedly be a more suitable designation for tumours of this nature from the standpoint of histogenesis than endothelial myeloma. The question of the exact site of origin of the tumour is also discussed with the conclusion that it may arise in the medullary as well as the cortical part of the bone. A clinical study of the case including X-ray picture before and after irradiation is also presented."

Calcinosis Cutis, R. N. Chopra, and J. P. Bose, p. 375.

"(1) Two typical cases of the metabolic type of calcinosis cutis (calcinosis circumscripta) have been studied and described. The histological changes indicate that calcification took place in the collagenous fibres of the corium and in the subcutaneous tissues. (2) A possible lowered function of the *pars anterior* of the pituitary gland as one of the aetiological factors is suggested and treatment of this condition by extract of the anterior pituitary lobe is recommended. (3) It is suggested that investigation on the lines recommended may throw more light on the aetiology of the disease."

Evipan-Sodium, P. N. Banerjee, p. 380.**A Note on the Relative Vitamin C-Values of Milk and Curd, A. R. Ghosh and B. C. Guha, p. 382.**

There is a gradual but slow loss of ascorbic acid in cow's milk stored at 0°C. (2) The formation of curd stabilizes the ascorbic acid of cow's milk completely even if the curdling is carried out for 24 hours at 35°C. The curd retains the entire amount of the ascorbic acid of fresh milk. (3) As there is no increase in the ascorbic acid content during the curdling process, the lactic acid bacillus apparently cannot affect the synthesis of this vitamin under the stated conditions."

A Note on the Vitamin B₁-B₂ and C Values of Country Liquor prepared from the Date, H. G. Biswas and B. C. Guha, p. 382.**A Report on Plague in Peermade (Travancore State), S. Krishnan Tambi, p. 383.**

JOURNAL OF THE INDIAN MEDICAL ASSOCIATION

VOL. IV, NO. 11, JULY, 1935.

Some Recent Nutritional Researches with Reference to Indian Food-Stuffs and Dietaries, B. C. Guha. p. 485.

"Poverty is by far the most important cause of malnutrition in India. Apart from poverty, however, ignorance is a very important contributory cause. This ignorance is two fold. Firstly, there is a lack of detailed knowledge about the nutritive values of the vast numbers of our own food-stuffs and the consequent inability to construct model dietaries. Secondly, there is the ignorance of the public about the little knowledge which has been gathered in this respect. It is the duty of the scientific and medical men and publicists to make this 'knowledge available to the public.'"

During pregnancy and lactation, one might empirically suggest for Bengali women in particular (1) increased consumption of iron by means of the iron-containing food-stuffs as well as by occasional administration of iron in the form of ferric chloride or ferric ammonium citrate. (2) increased consumption of calcium and phosphorus by means of certain vegetables, if not by means of milk, which is the best, and (3) increased consumption of vitamin A, B Complex and of C by means of green vegetables (raw or gently cooked, and not fried), eggs, potato, *begun* (egg-fruit), *kanchamung*, wheat (which is much richer in vitamin B1 than milled rice) and fresh fruit juices (like mango, orange, pine-apple, etc.)."

Treatment of Smallpox with Liver, V. Govindan Nair. p. 488.

- "1. A series of twenty-eight cases of smallpox treated with liver extract is reported.
2. The liver treatment modifies the course of the disease, shortens the duration, aborts the eruption and avoids the consequent disfiguration from deep scarring and pitting. The systemic disturbances disappear soon and the patient goes through the attack with comparative comfort.
4. None of the cases reported showed itching at the time of incrustation, nor developed boils and subcutaneous abscesses, secondary septic fever, toxic rash or phlebitis and thrombosis, the usual complications of untreated smallpox cases.
5. Two brands of liver extract were tried.—(1) Campolon of Messrs. Bayer-Meister-Lucius, Germany, and (2) Liver extract Injectable of Messrs. Bengal Chemical and Pharmaceutical Works, Ltd., Calcutta. Both gave equally good results."

Cholera kidney, a clinical, biochemical and functional study, Dharendra Nath Banerjee and Sunil Krisna Datta. p. 492.**Nutritional Research in India, Cedric Dover. p. 499.****Welfare Work in America, Ruth Young. p. 501.**

THE INDIAN MEDICAL JOURNAL

VOL. XXIX, NO. VII, JULY, 1935

Treatment of Leprosy, R. C. Acharya. p. 339.

"(a) Eliminate the predisposing diseases and debilitating factors which prepare the body soil to facilitate the growth of the bacilli. (b) Increase the body resistance against the infection by improving the general health and maintain the proper nutrition of the patient by suitable and balanced diet. (c) Prevent the superinfection by proper attention to hygienic measure as described. (d) Take special care of the patient during reaction and control it efficiently. (e) Treat the patient by Hydriocarpus preparation and locally apply trichloroacetic acid solution as described; treat the complications when necessary."

Some Recent Advances in the Treatment of Anaemias, U. D. Gopal Rao. p. 349.**Dysenteries in Bengal, Nripendra Nath Mallick. p. 350.**

THE CALCUTTA MEDICAL JOURNAL

VOL. XXIV, No. 12, JUNE, 1935.

Anatomical Basis of Hilum Shadow and Lung Markings in Chest Roentgenography, Dharendra Nath Banerjee, P. 653.

"*Roentgen anatomy of the root of the lung.*—The chief structures forming the roots of the lungs by means of which the lungs are attached to the heart and great vessels are the bronchi, pulmonary arteries, pulmonary veins, bronchial arteries and veins, pulmonary plexus of nerves, lymphatic vessels, bronchial lymph glands and areolar tissue all of which are surrounded by the pleura. The roots of the lungs lie nearer the posterior than the anterior part of the lung and nearer the apex than the base. They lie opposite the bodies of the 6th and 7th dorsal vertebrae.

The trachea divides into the bronchi at the level of the 2nd costal cartilage anteriorly, while posteriorly at the level of the upper border of the 5th dorsal vertebra and lies behind the lower border of the arch of aorta. The right branch, the more direct division, is about 2.5 c.m long and enters the right lung opposite the 5th dorsal vertebra. The right pulmonary artery lies at first below and then in front of it. The left bronchus is about 5 c.m. long and enters the lung opposite the 6th dorsal vertebra. The left pulmonary artery lies at first above, then in front of it.

The bronchial lymph glands include glands in association with the wall of trachea, main bronchi and their branches. The pulmonary glands lie in the lung tissue and drain into the bronchopulmonary glands.

The anatomical basis of hilum shadow and lung markings.—"In summarising the above findings we come to the conclusion that the main factors producing the hilum shadow and lung markings are the blood vessels, rather than the pulmonary arteries.

They produce a branched system of solid cylindrical shadows when running at right angles to the path of the rays and solid circular ones when running in the plane of the path of the rays. They gradually become finer and finer and ultimately disappear altogether in the peripheral zone. The wall of the finer bronchi do not cast any shadow. The main branches of the bronchi appear as double fine linear shadows with a clear area — the lumen in the centre. They run intermingled with the shadows of the pulmonary vessels, giving the composite picture a mottled appearance, but generally their shadows are masked by the shadows of the vessels. The main branches of the bronchi running in the line of the rays appear as annular shadows with clear centre. It is on this account, and more specially, as still some misunderstandings exist it is also safe not to use the term "bronchial" and the term "peribronchial markings" to denote the hilum shadow or lung markings.

From these considerations it is quite evident that any change of the vascular and bronchial condition as found normally during the different phases of respiration, especially forced respiration, must influence the hilum shadow and lung markings."

Studies on Cerebrospinal Fluids in Meningococcus Meningitis, S. G. Seal, p. 663.

"1. Seventy five samples of cerebrospinal fluid were collected from fifty seven suspected cerebrospinal meningitis cases, four of which were later on diagnosed otherwise, thus leaving fifty-three cases only. 2. Complete analysis of all the samples were not possible mainly for want of sufficient fluid. The result of examination of altogether sixty-eight samples have been reported. Of these sixty-three were examined for reaction, twenty-seven for specific gravity, fifty-four for globulin, twenty-six for total protein, twenty for chloride, thirty-two for sugar and only nine for urea. 3. A great increase of total cell count and of specific gravity have been specially noted. Also, in the cytological changes besides polymorphonuclear leucocytes, the appearance of endothelial cells in the fluids seems to be an additional characteristic feature in meningococcus meningitis. 4. Culture results were highly satisfactory, the percentage of successful cultures being about eighty-five. Meningococcus has been isolated from 80 per cent of the cases. In the primary isolations glucose broth and Lowenthal media have given the best results. 5. A bacteriophage-like phenomenon has been observed in several primary cultures. Further investigations regarding this observation are necessary. 6. Varying number of fluids have been tested for different chemical constituents and methods employed have also been mentioned. Most of the fluids have been more or less acid to phenol red; globulin and total protein are both highly increased, while the sugar content is greatly reduced. Chlorides are usually slightly diminished and the urea is not much affected."

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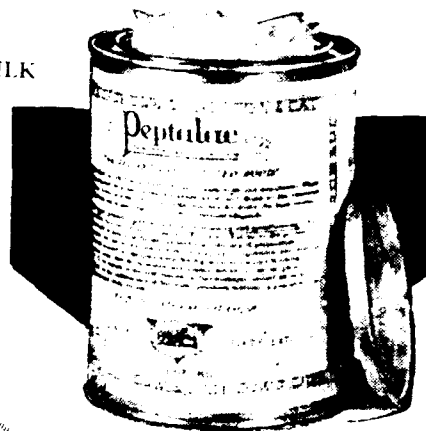
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Dicalcium Phosphate.....	2.00 gr.
Ferri et Ammon. Citras....	1 gr.
Vitamin D.	150 units.

Clinical trials which have been made with this preparation have satisfied us that it is a dependable product which may be employed in conditions where calcium therapy is indicated. As far as we can judge, the calcium in it is rapidly absorbed from the alimentary canal and readily utilized by the tissues. Its Iron and Vitamin content makes it particularly valuable in such conditions as *Tuberculous glands* and *Rickets*. We employed it in young children during the period of dentition and the results obtained were uniformly satisfactory. We have but one suggestion to make, namely, that it may be made even more palatable than what it is. But it should be said many of the patients who took it, including children, found it palatable enough.

P.

ASSOCIATION NOTES

CHETTINAD MEDICAL ASSOCIATION.

The usual monthly meeting of the above Association was held on 28-7-'35 at the S. M. Hospital, Tirupthur, under the presidency of Dr. Mangalam Pillai, as the President was absent. About 20 members were present. After tea, Dr. T. S. Shetty, the Secretary, read the minutes of the association.

Dr. T. S. Shetty read three interesting case reports, two of Diphtheria cases, one in a child of 3 years, another in an adult of 35 years, both Indians, one coming from Madras and the other from Ramnad District. He wanted to know if any doctors had any case in their practice. Dr. F. Ysander cited two cases from his experience.

There was a discussion on the advisability of doing Tracheotomy in advanced cases with breathing trouble. Dr. F. Ysander suggested that intubation is a better method and easily done, but Hospital stay was essential lest the tube be coughed out. Dr. Shetty spoke about a very interesting case of cerebellar abscess operated on in his Hospital in a case of neglected mastoid infection. Dr. Victor reported two orbital tumours he had operated on at the S. M. Hospital. Dr. F. Ysander demonstrated some interesting x-ray films of the gastro-intestinal tract. After this an interesting lecture on "Record-keeping" was delivered by Dr. D. O. Sendel, M. B., M.R.C.S. He suggested some novel and very easily manageable record keeping for out-patients and in-patients in both private and public institutions.

After the President's concluding remarks, and the usual vote of thanks, the meeting terminated at 7.30 p.m.

ALL-INDIA MEDICAL LICENTIATES ASSOCIATION, MADRAS.

"Dental Profession as it is practised in India" was the subject of keen discussion that arose among the Medical men of Madras, that gathered at a Clinical meeting held under the auspices of the above Association on Wednesday the 10th July '35, at No. 10, Thambu Chetty Street, Madras.

Dr. S. R. Bhat, M.B., B.S., L.D.S.C., was the lecturer. Major G. R. McRobert, I. M. S., presided.

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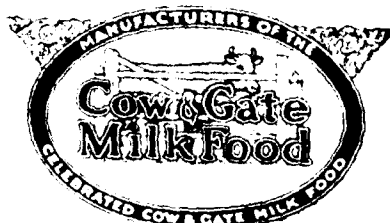
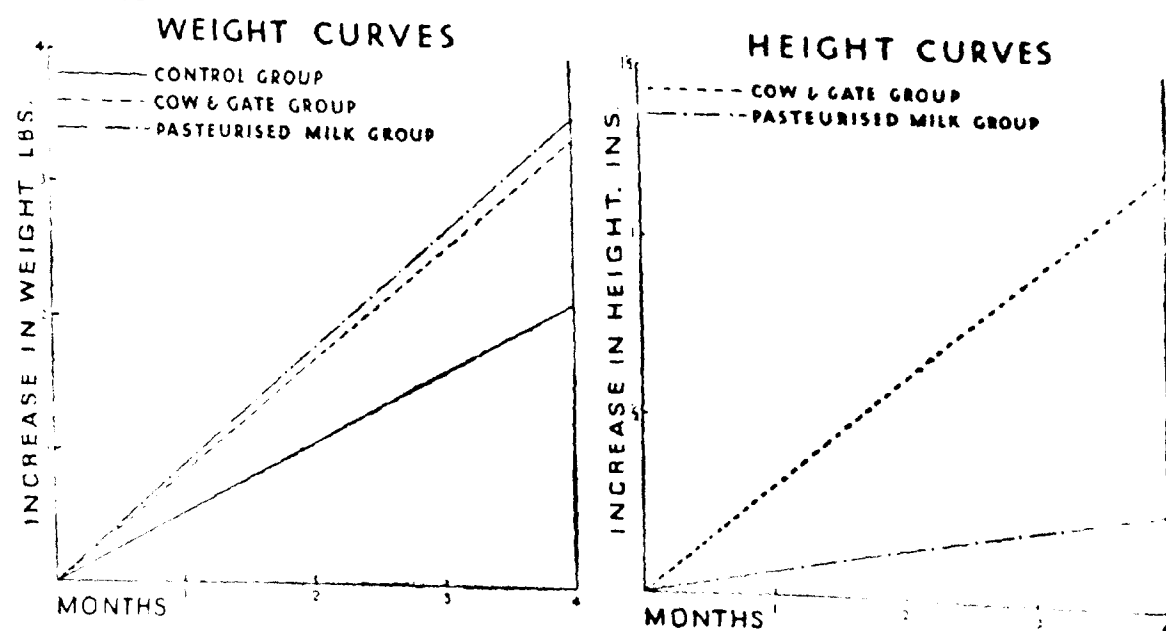
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ASSOCIATION NOTES AND NOTICES

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The Lecturer in the course of his speech pointed out that out of 5,000 men practising dentistry in India, 90 per cent are unqualified and the consequent sufferings of the public due to improper handling of dental cases are untold and unimaginable. The so-called dentists from China and Japan and others without any preliminary qualification whatsoever are treating dental cases in Madras in places open to all sorts of infection unmindful of the principle of aseptic surgery. While tracing the history, the progress and the high standard of efficiency of dental surgery in America and the Continent, the learned Lecturer stressed on the necessity of Legislation to prevent unqualified hands practising dentistry in India. He also pointed out the comments in the British Medical Journal which brought to the notice of the Government of India the need for well-organised dental institutions to train qualified medical dentists in order to eradicate the evils of quack surgery in India.

The President while concurring with the views expressed by several of the speakers, such as only qualified medical men with special qualification should practise dentistry and that they should do propaganda among the ignorant masses against the dangers of quack surgery, laid stress on the necessity of substituting well qualified dentists to meet the demand. He opined from his experience in Burma where also there are very few qualified dental surgeons, that medical organisations in India would do the needful to combat this great problem of dentistry in India.

The meeting terminated with a vote of thanks to the Chair, the Lecturer and those present.

THE TRICHINOPOLY DISTRICT MEDICAL ASSOCIATION. (Registered.)

A monthly meeting of the above association was held at 5 p.m. on Saturday, the 20th July '35, at the E. R. High School, Teppakulam, Trichinopoly, when about 45 members were present. Dr. C. E. R. Norman, F. R. F. P. & S., D. P. H., L. A. H., C. H., Chief Medical Officer, S. I. R. y. and president of the Association presided and Dr. N. C. Subramanyam, B. A., M. B. B. S., was 'At Home' to the members.

The secretary moved the following resolution which was carried unanimously:-

This Association requests the District Medical Officer, Trichinopoly and President, District Board, Trichy, to issue a general

circular to their medical sub-ordinates permitting them to attend meetings of the District Medical Association, which will be held on Sundays and penultimate Saturdays or on Holidays, so as to obviate the necessity of applying for permission each month.

Dr. B. S. Viswanathan, L. M. S., of Coimbatore delivered an interesting lecture on 'MODERN TREATMENT OF TUBERCULOSIS' and discussed the different methods of treating the disease at various stages. He also demonstrated an 'Artificial-Pneumothorax' apparatus and explained its technique. He showed a number of skiagrams of 'Tubercular lung' at various stages of treatment.

Dr. Rajan demonstrated a case of acute Pulmonary Tubercular lung which showed remarkable improvement under 'Pneumothorax treatment'. Then an interesting discussion followed in which many members took part.

Dr. Raghavan demonstrated a 'Colossal Transfusion Ampoule' and explained it was ready and simple to use.

With the chairman's concluding remarks and a vote of thanks to the host, the Lecture and the manager of the premises the meeting terminated at a late hour.

THE TINNEVELLY DISTRICT MEDICAL ASSOCIATION, TINNEVELLY.

The Tinnevelly District Medical Association held its monthly meeting on Sunday the 28th July 1935 at Courtallam (Jammal Palace) one of the most healthy spas of Southern India, noted for fine waterfalls and excellent mountainous scenery. Members numbering 57 including some lady doctors gathered from all parts of the District. Some of the officials and non-officials graced the occasion. All the members had an early bath in the excellent waterfalls which were in full swing. There were fine drizzlings and the members enjoyed the season well. M. R. Ry, I. C. Iswaran Pillai Avl., M.L.C. the President, District Board, Tinnevelly, was present for the best part of the function.

After the dinner, the meeting began at 2-30 p. m. under the Presidency of Lieut. Col. T. S. Shastry, I.M.S., the President of the Association.



INTERESTING CASES

1. A lady aged about 25 was suffering from scabies of the right nipple off and on for the last three years. Although it was ameliorated by treatment, it never healed completely. She had delivered about two years back and during lactation the scabies had disappeared but there was severe relapse after the lactation period was over. At this time the original infection was complicated by eczematous inflammation. Application of Sarcoprol in concentrated form was advised. This was done after by friction with towel. Sarcoprol was rubbed in for some time until it was thoroughly absorbed. Five days of this routine, which was repeated twice a day, brought about complete healing, leaving only brown pigmentation. The first day crust formation ceased and the secretion decreased. Itching entirely stopped after two days. The ulcers became dried on the third day and the whole lesion completely healed up after five days.

2. A female aged about 43 came to me for treatment of her obesity. She weighed 203 pounds and her height was 5' 3½". She was of sedentary habits. Her pulse was 78 and blood-pressure, 142 systolic and 104 diastolic. There was nothing abnormal about the urine. She was given two opocins of Iodobesin daily for 10 days and her diet was approximately reduced to 1800 calories per day. After 10 days of this treatment the condition was almost the same. During the next 10 days the dosage was increased, she was having 4 opocins daily. After this period her weight was found to be 196 and her blood-pressure was 135 systolic and 100 diastolic, the pulse remaining the same. She was given complete rest for the next 10 days. Then the treatment was again continued with 4 opocins daily. Her weight was brought down appreciably after 20 days, the weight being only 182. Thus the treatment was continued with 4 opocins per day for every three weeks, at the end of which she was having rest for one week, during which period she took no treatment but adhered to the diet. When this routine was continued for four months her weight was reduced by about 30 pounds. The dietetic restriction was quite moderate. There was no change in the pulse rate and the blood-pressure was brought almost to normal. The patient reported of an increased feeling of well-being. Her dullness and lethargy gave place to regular and systematic work. She is still under treatment and she weighs at present only 168 pounds.

From the Diary of a Medical Practitioner.

ADVT.

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NOTICE

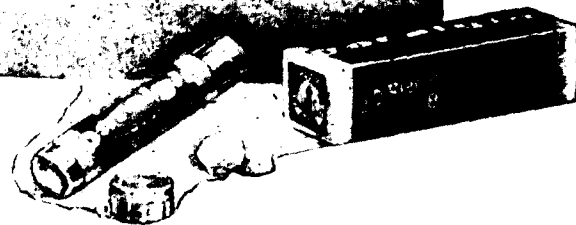
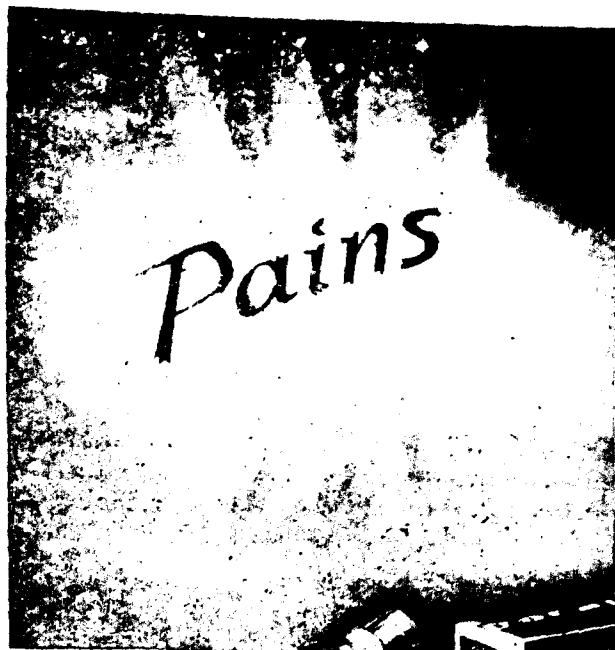
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ASSOCIATION NOTES AND NOTICES

xi

The minutes of the last meeting were read and passed. Dr. K. Kalyanaraman, L.M.P., read very interesting case notes on Pyloric Stenosis and Tuberculous Empyema of the kidney. This Kidney patient who is now in very good health was shown at the meeting. Rare and interesting specimens of (1) Epithelioma of Eyeball, (2) Cancer Breast, (3) Tumour Testis and (4) Tuberculous Kidney, successfully operated by Lieut. Col. T. S. Shastry, I.M.S. were demonstrated.

Then the discussion on 'Dyspepsia' was resumed from Papanasam meeting by Dr. A. L. Anantanarayana Iyer, M.B., B.S., of the Government Hospital, Tenkasi. There was a lively discussion on all operated cases referred to above.

After the discussion, Lieut. Col. T. S. Shastry, I.M.S., summed up the salient and important features in the correct and proper diagnosis of Dyspepsia, reviewing the most successful methods adopted in the treatment. After a hearty vote of thanks to the President, the meeting dispersed.

After tea, the party reached Tenkasi where a Health Exhibition was arranged at the Elementary Weaver's School. School boys and girls of Tenkasi and its neighbourhood assembled. The girls of the school gave an entertainment on "Kolattam" singing health songs (songs about the mischief of flies and some other health songs.) Lieut. Col. T. S. Shastry, I.M.S., welcomed the guests and requested M. R. Ry Iswaran Pillai Avl., the President, District Board, Tinnevely, to open the Exhibition. The exhibition was opened by him at 6-30 p. m. He expressed in a short speech that such exhibitions are very useful and beneficial to the masses and promised hearty co-operation by equipping schools and dispensaries with posters on infectious diseases and directing all the teachers under his control to learn from the Local Medical Officers the significance of these posters and then to teach their pupils once a week these lessons of health. Dr. G. Parthasarathi, M.B., B.S., of Tenkasi gave an interesting lecture in Tamil on the Prevention and causation of Tuberculosis, Leprosy and Cholera explaining the posters on the subject which were exhibited on the walls in easy Tamil for the benefit of little children. The exhibition was thrown open to the public till late in the evening. The exhibits consisted of posters relating to Cholera, Tuberculosis, Malaria, Small-pox, Leprosy, Fly danger, etc. This was very much appreciated by the public.

Lieut. Col. T. S. Shastry, I.M.S., offered cordial thanks to Dr. S. Kanthimathinathan, L.M. & S., and his co-workers for their kind and generous hospitality, and for organising the meeting, to Dr. S. Kalappa Pillai L.M. & S., the District Health Officer, Tinnevely, for arranging the Exhibition so well, and to M. R. Ry. I. C. Iswaran Pillai Avl., M. L. C., the President, District Board, for the great encouragement he had given.

NOTICE

The Vaccination Certificate Form to be used by District Medical Officers, Civil Surgeons, and Registered Medical Practitioners for issue of vaccination certificates to intending passengers to foreign countries from India (cited letter from the Personal Assistant to the Surgeon General, Madras. R. No 1680 A, dated 9th August, 1935) is reproduced below: -

VACCINATION CERTIFICATE

Station

Dated,

I, (name in block letters), Registered Medical Practitioner, hereby certify that Mr. Mrs. Miss.

Identification marks 1. etc.
2.

aged has been vaccinated on date
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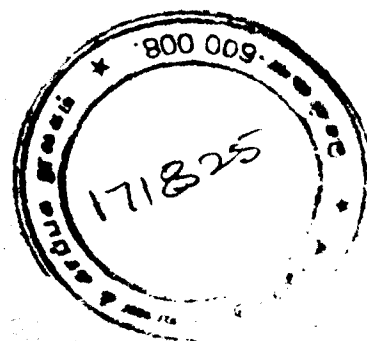
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Calf No.

Date of receipt of lymph

Date of Vaccination



Signature
(Registered Medical Practitioner)

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