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The Antiseptic

A Monthly Medical Journal

Edited by

U. Rama Rau, & U. Krishna Rau, M.B., B.S.



CONTENTS

PAGE

Osteo-Malacia and Pregnancy. By N. A. Purandare, M.D., F.R.C.O.G., F.C.P.S., Bombay. ...	337
Phrynoderma. By Dr. K. Narayanamurthi, Hony. Physician, Govt. General Hospital, Madras. ...	344
Parathyroid Deficiency. By S. Sankararaman, M.B., B.S., Cuddalore. ...	346
Deficiency Anæmia. By M. C. Rama Ayyangar, B.A., M.B., B.S., Asst. Surgeon, General Hospital, Madras. ...	351
Pellagra in Children. By A. V. S. Sarma, M.B., B.S., D.C.H., (Eng.), Hony. Asst. Physician, Stanley Hospital, Madras. ...	355
Vitamin B₁ Deficiency and Cardio-Vascular Disturbances. By P. N. Laha, M.D. (Pat.), Lecturer in Diseases of Children, Medical College, Agra. ...	358
Some Observations on Vitamin B Complex. By CH. Krishnamurthy, L.M.P., Selection Grade Sub-Assistant Surgeon, Markapur. ...	361
Diseases of Vitamin C Deficiency. By R. K. Bhattacharyya, M.B., Nabadwip... ..	363
Some Minor Disorders Due to Vitamin Deficiencies. By Capt. N. Seshadrinathan, M.B., B.S., D.T.M., King Institute, Guindy, (Madras). ...	367
Syphilotherapy in Deficiency Diseases. By K. D. Lahiri, M.B., B.S., Skin and Venereal Department, Patna Medical College Hospital. ...	370
Deficiencies in Amœbiasis. By H. P. Mukherjee, M.B., Medical School, Bankura. ...	371
Vitamins and the Eye. By S. Sanyal, M.B., H. O. R. Institute, Calcutta. ...	379
Proceedings of Associations ...	390
Announcement. ...	390

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Index to Advertisements, see page 55.

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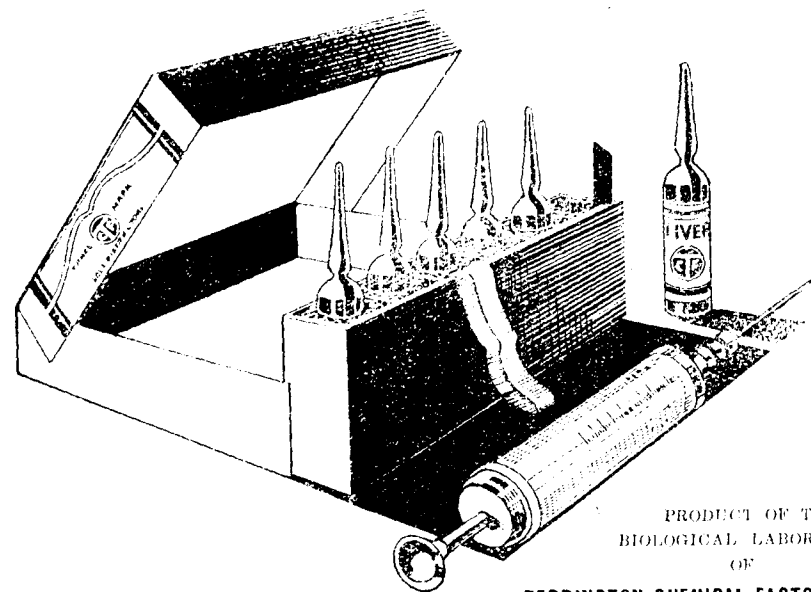
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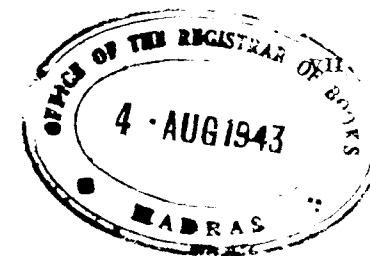
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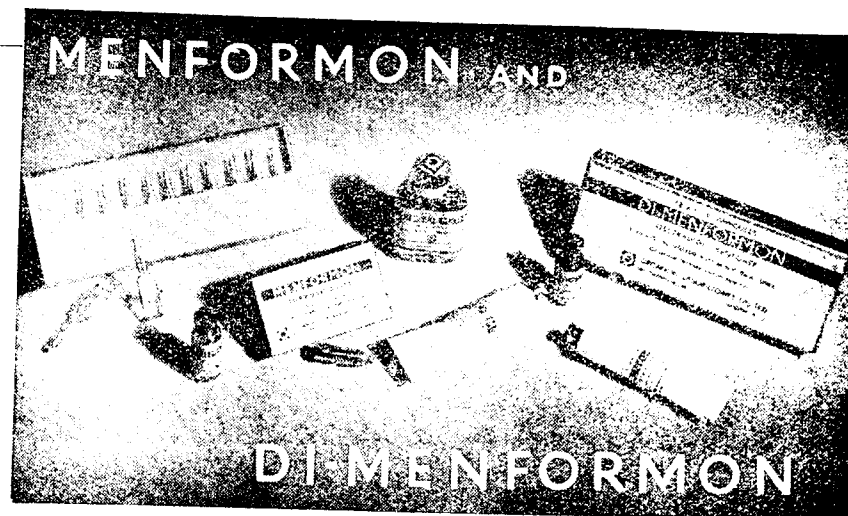
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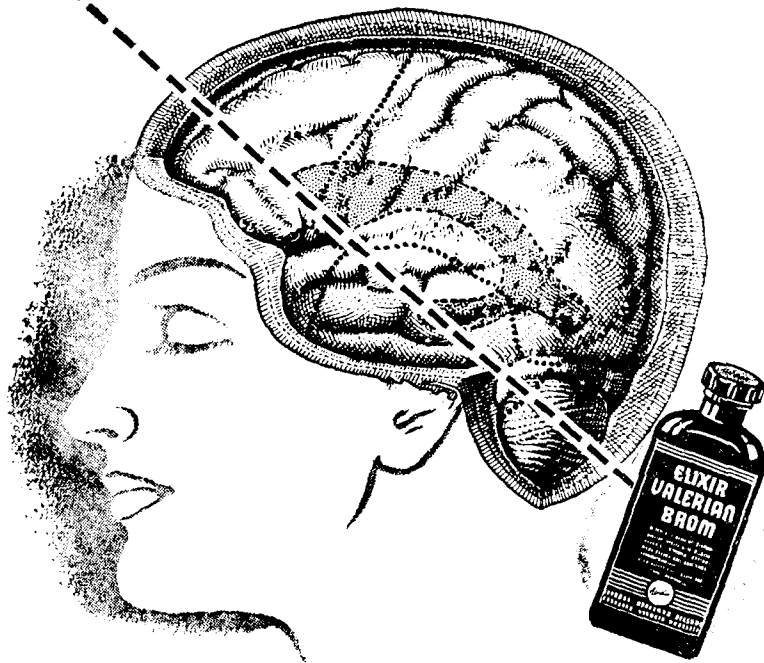
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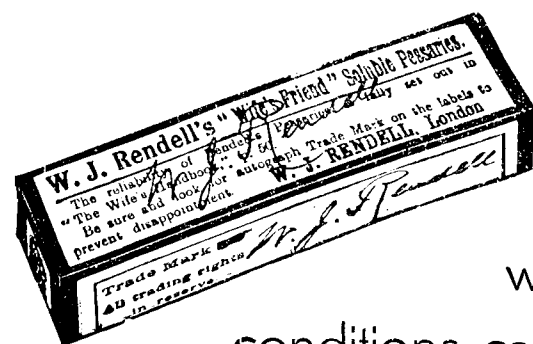
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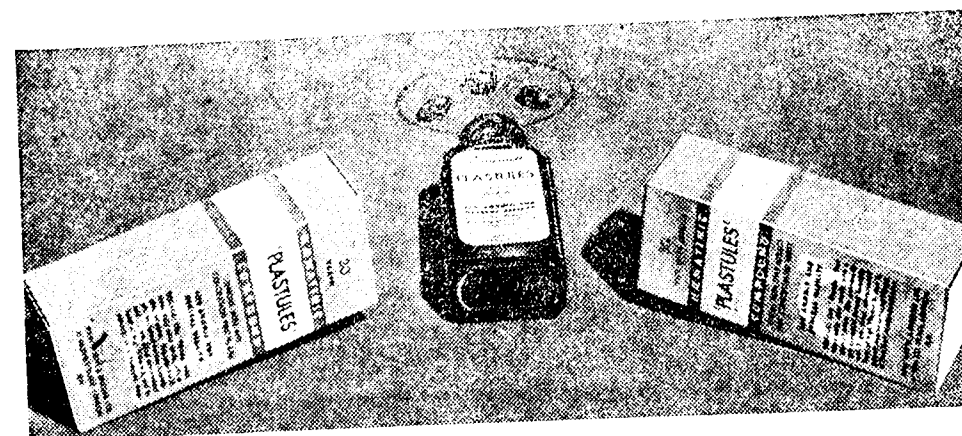
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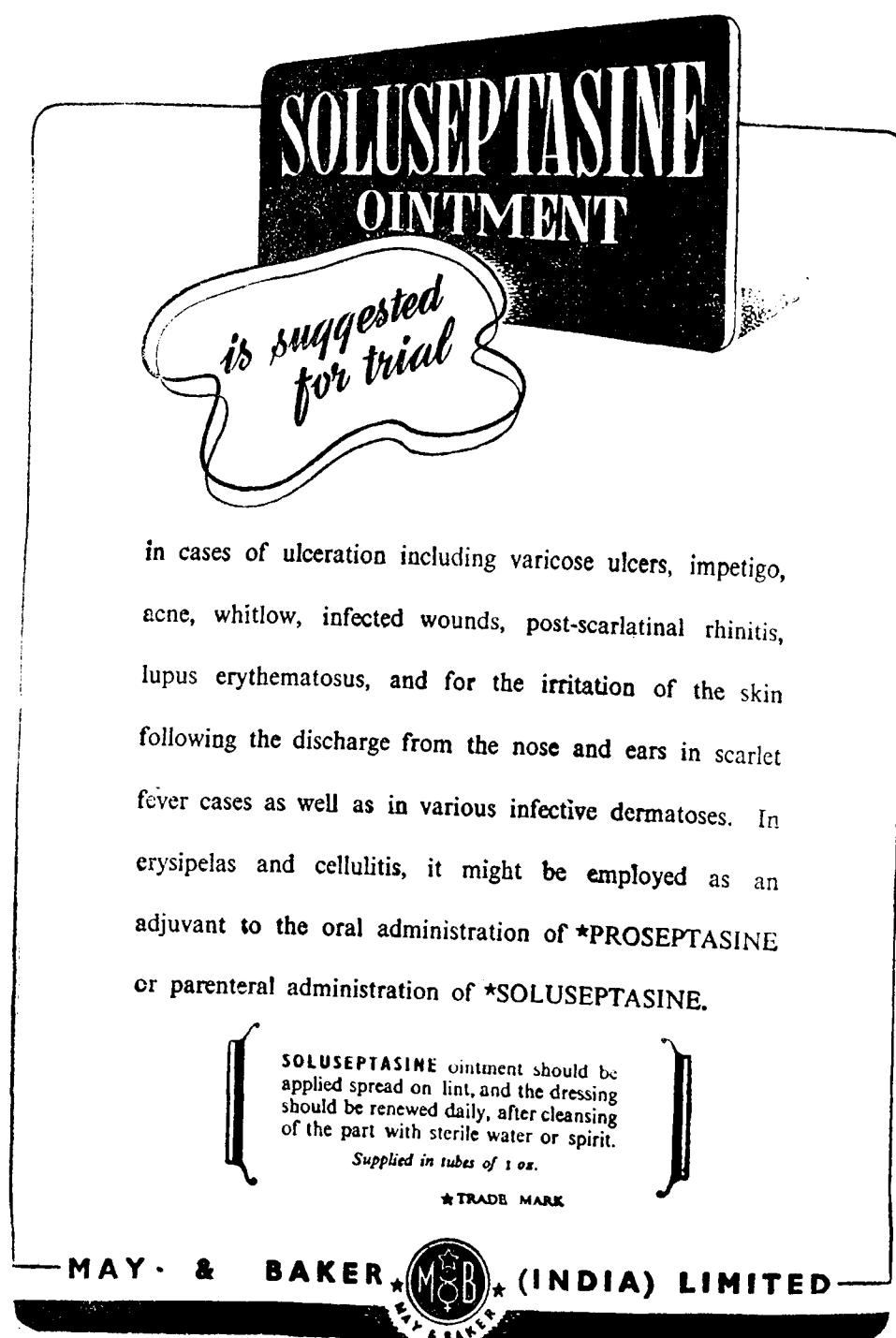
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No. 7.

Osteo-Malacia and Pregnancy

BY

N. A. PURANDARE, M.D., F.R.C.O.G., F.C.P.S.,
Bombay.

OSTEOMALACIA (osteon—bone and malacia—softness) is an affection characterized by softness of bones. Naturally the bones are hard from deposition of calcium salts in them. It is in the organic matrix of the bone where the mineral salts are deposited. This process is called calcification. It is always occurring and is associated with another process of resorption whereby the bone is decalcified. So both the processes of calcification and decalcification are taking place at all times in the bone.

The source of the mineral salts is the food-stuff ingested. They are absorbed from the food in the gastro-intestinal tract. That they may be readily absorbed, certain factors should be operating. First, the food should be sufficient in quantity and good in quality so as to yield adequate quantity of calcium salts; secondly, it should contain enough vitamin D, which for the most part controls calcium and phosphorus metabolisms in the body economy. To support the action of vitamin D, food should also contain other vitamins in order to preserve healthy state of the body by favouring other metabolisms. The other factor which facilitates calcium and phosphorus absorption in the bowels is the acid-base value of the diet. If the diet contains organic acid, such as tartaric acid or citric acid and sodium tartarate or sodium citrate, there is greater absorption of calcium and phosphorus on account of organic acid—citric acid is more potent in its action than the tartaric acid—while there is reduction in elimination, thus inducing greater retention, whereby a positive balance is established since the output is less than the intake.

Of the mineral salts absorbed, 99 per cent. of the calcium is deposited in the bones and teeth as calcium phosphate and calcium carbonate compound, a double salt. Very small proportion is in the body fluids. Calcium in the blood is called serum calcium, and phosphorus as serum phosphorus. Serum calcium is in the form of calcium ions and calcium proteinate. The

proportion of serum calcium is 10.5 mgm. in 100 c.c. of blood; that of serum phosphorus is $3.5 \pm .5$ mgm. in 100 c.c.; thus the relation between the two is 2:1. Phosphorus in the blood is as soluble calcium phosphate. In normal condition serum calcium level is maintained; whenever the level of calcium ions gets lower than the normal, it serves as a stimulus to the parathyroids to produce their hormone. It is obvious, therefore, that the calcium and phosphorus metabolisms are under the influence of vitamin D and parathyroid hormone. Vitamin D governs absorption, assimilation, retention, utilization, elimination of calcium and phosphorus; whereas parathyroid hormone maintains the normal level of calcium ions in the blood.

From the above description it is quite apparent that the calcium and phosphorus metabolism in the bone goes on properly when there is adequate amount of vitamin D in the body; but whenever the vitamin is deficient it affects the normal processes and produces affection of the bone generally just as deficiency of vitamin A produces night-blindness and affection of the epithelium, deficient vitamin B₁ causes beri-beri, B₂ pellagra, C scurvy, so deficiency of vitamin D gives rise to rickets in children and osteomalacia in adults. Therefore osteomalacia is one of the deficiency diseases.

Aetiology.—Predisposing causes. Osteomalacia is a disease to be found especially in women arising in connection with pregnancy or lactation and getting worse and worse with each subsequent pregnancy. Sometimes girls may show signs of affection of bones at the time of puberty which are more like rickets in their character and therefore some prefer to call the affection then as late rickets. Rarely even some boys are seen suffering from late rickets.

Osteomalacia is not such a common disease. In the west with the improved conditions of living, the disease has become very rare. In Europe some time before, cases of osteomalacia were seen in the Rhine and Danube valleys, but there too the incidence has become very low. In America and in North Canada the disease was prevalent before. In the east, cases are still seen in China, Japan, and in India. In China the binding of the feet of girls and in India the observance of *purdha* are responsible for the incidence of the disease. The sunlight is the great powerful factor in preventing the disease. It acts through its cogent ultra-violet rays, which, acting on the exposed skin both of animals as well as human beings, produces in it previtamin, 7 Dehydrocholesterol, which is ultimately stored in the fat as vitamin D. These ultra-violet rays of the sun get cut off when the light is passing through clouds, both of the rain as well as of the smoke, and through glass or clothes. The disease is observed more in cities and towns than in villages and in greater number in congested localities. But it may look strange that more women of the middle class suffer from the disease while poor labourers escape. The latter go out and work in the sun, thereby getting advantage of the ultra-violet rays; whereas the women of the middle class deem it too humble to work outside in the open even though their circumstances may be too precarious. The sun seems to be indulgent to the labourers for their industry by his beneficent ultra-violet rays which produce vitamin D in the body and favour calcium and phosphorus metabolism; while he inflicts punishment on those girls who sit idle at home in their dark ill-ventilated rooms and eat at the same time diet which their poor circumstances afford and which is invariably

not only insufficient in quantity but also deficient in quality. Vitamin D they get neither from the sun nor from their poor fare. As a result the calcium and phosphorus metabolism is very defective in them.

Besides the sun, the other sources of getting vitamin D are some articles of diet. The chief among them are milk, butter, eggs, animal-fat and fish liver oils. Not only should vitamin D be present in the food but there should also be calcium and phosphorus in the food-stuffs in adequate quantity. Unless the mineral salts are absorbed from the gastro-intestinal tract, the vitamin D is ineffective in carrying out calcium metabolism.

In order that the calcium and phosphorus metabolism be conducted in an effective manner, the diet should contain also other vitamins, as A B (complex), C and K. Particularly vitamin C exerts its influence in calcification of the bone, since its deficiency inhibits the function of the osteoblasts.

Proper diet is very necessary to preserve health and maintain an agreeable state of the body. Yet many a time it is found defective. Generally it is due to poor circumstances; sometimes it is due to ignorance and sometimes even due to faulty likes and dislikes of the individual.

With a view to knowing what is generally the diet of vegetarian and non-vegetarian families of the middle-class poor society, inquiries were instituted and the following information was obtained:

Vegetarian family.—Rice with *dhal* formed the staple food. Rice bread or rice and *bajari* bread was eaten every day mostly in the evening. Green vegetables, either fruits or leafy, formed an additional dish which was taken twice or thrice in a week. Butter-milk as *kuri* is taken with rice once or twice in a week. Pure curd is never taken, milk as such is never drunk except what little can be had in tea which is taken once or even twice a day.

Non-vegetarian family.—Rice and *dhal* and bread form their common diet, having fish *kuri* rather than butter milk. They do not have milk or even eggs regularly every day. Leafy vegetable dishes are very seldom taken. Some have flesh once in a week and even fish dishes once or twice a week. In this way vitamin D is very little in their food.

Mineral salts are also poor in quantity in their diet. Nowadays rice that is sold is generally polished containing very little calcium or vitamin B₁, the latter being removed in the germ portion and barn. In the same way, whole grain wheat flour is not used for preparing bread. Again the water that is used in boiling cereals and, legumes is either thrown away or not served to the persons eating or used by themselves; thus there is loss not only of vitamin B₁ but also of the mineral salts. These are the additional factors which make the diet yet further poor.

Exciting causes.—Pregnancy and lactation are two important factors which bring on osteomalacia especially in those women who are subjected to vitamin D deficiency and usually having food-stuffs poor in calcium and phosphorus and other vitamins. Thanks to Nature's kindness, who does her utmost to render help even in adverse conditions, the disease is not very common. Many a woman has to submit to disabilities arising from deficiency in diet and vitamin, yet the wonder is how very few develop the

disease. Only those women who have altogether disregarded early warnings and failed to take suitable steps to prevent the disease and proper remedies to cure it in time, have to suffer from it. These women may have, besides faulty calcium and phosphorus metabolism, other metabolisms going wrong; they may be anæmic; they may have soreness of mouth and looseness of bowels; they may even show signs of toxæmia of pregnancy. Such women may find the strain of pregnancy and lactation too overwhelming. Their calcium and phosphorus metabolism has been too low. The proportion of serum calcium and serum phosphorus is also too small. In spite of all this, the blood has to carry out the additional work of conveying calcium salts during pregnancy and lactation to the placenta and to the breast. This drain sets up negative calcium balance in the mother's organism and, inducing exaggerated function of the parathyroid, produces pathological changes in the bones.

Pathology.—First the exact way in which the disease was produced was not known. After the discovery of the internal secretion of the various ductless glands, the disease was ascribed to excessive activity of the ovaries producing internal secretion in great amount. As a cure of the disease the ovaries of many a girl were ablated. The removal of the ovaries stopped further pregnancy and controlled further advance of the disease only to bring on troubles of the premature menopause. Such unfortunate women had to eat the tablets of ovarian substance throughout their remaining life.

Now it is regarded as a fault of calcium and phosphorus metabolism affecting normal processes in the bone.

In normal conditions in the bone are proceeding simultaneously the processes of calcification and decalcification. Calcification means laying of calcium salts in the trabeculae of the cancellous part of the bone. The factors concerned in this process are osteoblasts or osteogenic cells, and double compound of calcium (dahllite,) calcium phosphorus and calcium carbonate, bone-matrix and phosphatase.

Osteoblasts.—Osteoblasts are also called osteogenic cells. Their main function is to form the organic bone matrix. In the matrix is the tissue-fluid which contains mineral salts brought by the bone blood vessels. Calcium is in the form of calcium ions, which forms a small proportion, and calcium proteinate, forming a greater proportion. Phosphorus is as soluble amount as soluble organic phosphorus compounds. In the little tissue-fluid, the calcium and phosphorus salts are in concentration. Again phosphatase, an enzyme, splits the organic phosphorus compounds into inorganic phosphates. Furthermore CO_2 has been added to the tissue-fluid owing to metabolic activity of the osteoblasts. Now by mass-interaction the double-salt calcium phosphate and calcium carbonate ($\text{Ca}_3(\text{PO}_4)_2 + \text{CaCO}_3$) is produced. This salt with physico-chemical affinity is laid in the organic matrix of the bone.

Decalcification means removal of calcium salts from bones. The factors concerned in this process are osteoclasts and parathyroid hormone.

Osteoclasts.—(Osteon=bone and klastos, klao, I break) are multi-nuclear giant cells. HAM regards that osteoclasts are derived from osteoblasts by fusion. They possess phagocytic property, phagocytizing bone cells and

other material. HOFMIESTER maintains that any metabolically active cell in contact with bone will liberate CO_2 and cause the calcium salts to go into solution, thus facilitating mobilization of calcium salts from the bone.

Parathyroid hormone.—As soon as calcium ions level in the blood gets lowered below the normal, a stimulus goes to the parathyroids to produce more hormone. It brings calcium to the blood not by causing more absorption in the bowels but by drawing upon calcium depots in the body. The chief reservoirs of lime salts are the bones and the teeth. But of these, bones are the chief structures which are depleted. Here the osteoclasts are excited, excessive osteoclasts takes place and calcium salts are mobilized from the bone.

In osteomalacia the process of calcification is almost at a stand-still while that of decalcification goes on with great intensity. It is because the intake is little while the output is much, owing to deficiency of vitamin D_1 and mineral salts. To this is added the loss of these salts through the placenta and the breast. Thus a pronounced negative balance is established, the serum calcium and serum phosphorus getting at a very low level. Parathyroid glands become very active, elaborating the hormone in very great quantity. The latter wakes up the osteoclasts to exaggerated activity inducing extensive decalcification to mobilize the earthy salts. But all these salts do not gain the placenta or the breasts and therefore do not satisfy the demand of the child; because increased parathyroid hormone produces hypercalciuria and hyperphosphaturia and hypophosphatæmia, thereby excreting calcium phosphates in the urine by the kidneys. Some of the phosphates are even re-excreted by the bowels. The result is the serum calcium and phosphorus level always remains low. In the bone no sooner are calcium and phosphorus ions liberated, they are carried away to the placenta or the breast, the loss at the breast being invariably greater. When the bones are deprived of the mineral salts, they are painful, soft and tender. Though the salts are removed the organic matrix is present and even more, the osteoblasts as well as phosphatase being increased in rickets and in osteomalacia. The osteoblasts lay down the matrix but are unable to deposit calcium in it. Besides, if there be marked deficiency of vitamin C, it exercises inhibitory influence on the osteoblasts and the organic matrix is poorly formed. Phosphatase may separate calcium phosphate but it is soon turned into soluble acid-phosphate and presently carried away from the bone. In this way bones, deprived of earthy salts, are made very soft and deformed.

Symptoms.—These can be known better from the history that can be elicited from the patient. It runs nearly as follows:

A multipara first begins to experience pain in the back over the spine and shooting pains in the muscles of the thighs due to deficiency of calcium in the blood and the bones. She feels weakness in the muscles of the back and finds difficulty in getting up in bed. At first these symptoms may commence in the middle of pregnancy and even subside some time after delivery. In the following pregnancy the symptoms are aggravated, the bones of the pelvis, spine and ribs may particularly become painful and tender, the psoas and iliac muscles of the back may look as if paralysed owing to extreme difficulty in sitting up in bed, and contractures may occur in the abductor muscles of the thighs. She may also give history of attacks of tetany. The bones of the pelvis owing to softening may develop

pelvic deformity, leading to difficulty in labour, the degree of contraction being slight in the beginning and getting more and more pronounced as time passes. This raises obstruction in labour, the extent of which depending on the degree of contraction already produced. After delivery, instead of recovering, she continues to suffer more for some months; when she begins to move about, she cannot walk with ease and has developed wadling gait. Her skeleton is affected and her stature has grown shorter. In the subsequent pregnancy the symptoms may return early and have increased in turning severity. The patient may be finding difficulty even in her sides in bed. The bones of the pelvis may be so much affected as to cause great deformity and thus creating even impossibility of delivery by natural passages, entailing Cæsarean section.

As the softening of pelvic bones proceeds, along with it pelvic deformity goes on and on. First the promontory is pushed downwards and forwards, when the pelvis is widening transversely thus it becomes flat. Afterwards owing to further softening the heads of the femurs commence to push the lateral walls inwards, whereby the pelvis assumes triradiate shape. The pelvis as a whole grows less deep. The pubic bones become prominent and beaked. The pubic arch grows narrower and narrower until it is so narrowed as to render it difficult to insinuate even a finger through it for examination or to raise difficulty in performing the act of coitus.

Diagnosis.—The diagnosis is seldom difficult. The characteristic history, the peculiar wadling gait, way of getting up in bed, tenderness of the lower ribs and pelvic bones, beaked pubis and extreme narrowing of the pubic arch are peculiar to osteomalacia. The pain in the spine is mistaken for T. B. affection of the vertebræ and even plaster of paris is put on.

Treatment.—General condition of the patient should be attended to; she should pass one hour and a half in the open in order to take advantage of the sunlight which can be indulged in freely without any cost. Here is a service for charitable organizations which can favour such a patient with a supply of milk or eggs as she desires. Milk she should be made to drink on the spot and not permitted to take it home. Her diet should be improved if circumstances could allow it. By way of treatment, she should have cod-liver or halibut-liver oil in sufficient quantity, along with calcium salts. The effect of the fish-liver oil may be enhanced by giving in addition some concentrated preparation such as Calciferol. One will have at times to depend on the latter in case the patient does not tolerate fish-oil. It has been found out now that efficacy does not lie in the concentration of the units but their natural dispersion in the oil.

In a good family it would be possible to improve the diet. It should consist of rice cooked with just little water which could be retained in the rice or the patient may be made to drink it. Leafy vegetable dish she should have at least once a day along with other fresh vegetables. Other things are *dhal*, butter-milk, curds, whole gram, wheat flour or *bajari* bread, lemon or lemon-squash.

Eggs, fish and flesh should form part of the diet in non-vegetarians.

With pronounced pelvic deformity produced by osteomalacia, Cæsarean section with sterilization may be called for.

Vitamin D with calcium salts she should have for a long time.

Even those patients who were unable to get out of bed have improved with cod-liver or halibut-liver oil and calcium treatment carried out for sufficiently long time, improvement effected being so much as to enable them to walk about freely and to attend to all their work with ease. Only defect that remains in them is that their height is permanently shortened and that their back is bent forwards. To corroborate the above statement, a case under direct observation is cited below:

A patient, now above 40, a Hindu (Brahmin) developed osteomalacia in the second pregnancy. The disease took a rapid course. The delivery occurred with much protraction of labour. She did not recover completely but within a year she conceived again. The disease assumed yet graver condition. The labour was obstructed and delivery effected by craniotomy. She was afterwards confined to bed for nearly two years. Her first two children showed signs of rickets. Her second child when about 2 years old, playing near her bedstead met with an accident. Its mother, then bed-ridden, turned over on her side to look at the child rather suddenly, with the result that one of the femurs fractured. From that time she was placed on proper cod-liver oil and calcium treatment. She went on improving and within six months she left her bed, in which she was laid up for almost three years, and began to walk about and to attend to her household duties. She enjoyed subsequently good health, with the exception of some reduction in her height, for nearly five years, when unfortunately she became pregnant again. She was this time on the appropriate treatment and did not show signs of osteomalacia. Nevertheless her labour was protracted but delivery took place spontaneously, with a living child. Within two years, she conceived again and got a son with less difficulty in labour. The son is now 10 years old.

This case has peculiarities of its own. The disease progressed, because cod-liver oil and calcium treatment she won't have until she broke her thigh bone and the surgeon insisted on the right treatment. She recovered and continued the treatment long as she was convinced of the efficacy and beneficial results of it. Some years after, pregnancy followed and the improvement was so much as to have a living child though after protracted labour, a happy termination in view of the fact that delivery had to be effected with craniotomy some years ago when the disease was running an acute course. The last labour was less difficult. Both the last two children did not show signs of rickets. All the same they were themselves having cod-liver oil.

Though the bones had become brittle owing to decalcification bone matrix was present where, with the proper calcium and phosphorus metabolism induced by the help of vitamin D and calcium salts given by mouth subsequently, proper calcification occurred and the patient recovered.

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4, Chowpathy Sea Face,
Bombay, No. 7.

Phrynoderma

BY

DR. K. NARAYANAMURTHI,

Hony. Physician, Govt. General Hospital, Madras.

DEFICIENCY of vitamin A in human beings not only causes xerophthalmia, keratomalacia but also causes a peculiar dry skin with spinous papules at the sides of the hair follicles mostly at the extensor surfaces of the extremities, shoulders and lower part of the abdomen.

The first clinical description of the changes in the skin was given by PILLAT (1929) and latter on FRAZIER and HU (1930, '31) reported the occurrence of papular lesions in the skin in a group of Chinese soldiers. Histological and pathological process was one of hyperplasia and hyperkeratinisation of the epidermis and hair follicles with associated metaplasia of the epithelium of the sweat ducts to the keratinising type of degeneration of the glandular structures of the skin. Further clinical and pathological observations made in India by DR. RADHAKRISHNA RAO and others confirm that the specific nature of follicular hyperkeratosis is due to the deficiency of vitamin A. LOEWENTHAL (1933) described a similar dermatosis associated with night-blindness among a group of prisoners in Uganda Central Prisons in East Africa. The condition could be cured with cod-liver oil and without modification of diet given for a period of nine weeks. It is also pointed out that the histological changes in the papules were precisely the same as those occurring in pityriasis rubra pilaris although the anatomical distribution of the two conditions were different. NICHOLLAS (1933) made similar observations among poorly fed labourers in East Africa, and also among convicts in a jail in Ceylon. He gave the condition the name of 'Phrynoderma,' meaning (toad-skin 1934, '35, '36). He attributed it primarily to vitamin A deficiency but considers, that there may be other food factors responsible.

GOODWIN (1934) in London, PEIPING in China in (1935) and WRIGHT (1936) in Madras, described papular skin lesions similar to those of pityriasis rubra pilaris occurring on elbows and knees both in adults and children suffering from keratomalacia. AYKROYD and RAJAGOPAL described the occurrence of phrynoderma in school children in South India (1936). In their paper the clinical manifestations and histo-pathological features of the condition were described and ætiology was discussed.

Of recent date the author has found among apparently healthy children of school-going age the same clinical condition of dermatosis without ocular signs and most of them belong to the poorer classes with deficiency of protective foods such as milk, milk products, eggs, pulses, vegetables, etc. A chubby girl is found with no other signs of ill-health except from the typical cutaneous manifestations and angular stomatitis and loss of pigmentation of the lips and sides of the tongue.

The onset of eruption is insidious and some of the patients are not at all aware of its existence till we draw their attention. A dry and sometimes rough skin may precede the appearance of the typical papules.

There are no subjective symptoms, no pain, no itching, no tenderness.

Vol. 49, No. 7] THE ANTISEPTIC [July '43

MA—K. NARAYANAMURTHI

345

Phrynoderma

Dr. K. N. Murthi, M.D.



DORAIKANNU—25-2-43.

Marked cutaneous depigmentation noticed on the lips and the sides of the tongue.

Note: There is no xerophthalmia and the girl is healthy.

of the papules are of the pilo-sebaceous follicle—a big pin's head to the size of a mustard semispherical in outline—a sharply defined papule is darker than the rest of the skin and is felt by the fingers over the papules we feel as though it is a bump.

children of both sexes as well as in adults. Heat and cold are normal. There is no tenderness or pustules.

Histo-pathological features of the disease, no appreciable changes in the stratum lucidum, and stratum granulosum. The epidermis and the stratum corneum is hard—some places shows moderate hypertrophy. Histological evidence of hyper-pigmentation was not observed in the papules. The sebaceous glands are normal. Changes are found in hair follicles in all cases but the degree varied in every instance. The mouths are widened and plugged by dense masses of keratinized tissue the horny tissue is found to consist of cells which there were no nuclei. Pustulation was not observed. Follicular plugs. Varying degrees of cellular infiltration, mono-nuclears, and lymphoid cells are seen. The histo-pathological feature of this disease is hyperkeratosis of the epidermis with marked dilatation of the sweat ducts with cornified tissue, atrophy of the sebaceous glands and impairment of the function of the sweat glands.

Conditions such as lichen spinulosus, lichen planus, ichthyosis, keratosis supra follicularis and ichthyosis.

Phrynoderma is a entity by itself as it is found in close association with keratomalacia. The beneficial results obtained with cod-liver oil make the lesions disappear. Therefore the deficiency of avitaminosis of 'A' cannot be a coincidental inflammatory condition of the lesion, hyperkeratosis and the involvement of the cutaneous glands all point to a deficiency of vitamin A. However it is very difficult to establish the cause of the disease. Among children in whom xerophthalmia is common phrynoderma does not seem to be any bearing between the two and ceratine intake. There is no relation between a fat-free diet rich in vitamin A and the occurrence of the dermatitis in rats does not seem to be affected. It cannot be also due to the absence of the

divergent opinions held regarding its ætiology. Giving large doses of vitamin 'A' and normal diet does not cure the condition, but these should be given for a month.

Parathyroid Deficiency

BY
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THE parathyroids, the smallest of the endocrine glands, are four in number and are located in pairs on the posterior aspect of the thyroid lobes. The two upper parathyroids are found on the middle third of the posterior border of each lateral lobe of the thyroid, in which they are embedded, but the two lower parathyroids are placed very close behind the lower pole and are very loosely attached to the thyroid. In some subjects accessory parathyroid glands are present and, when present, they are usually distributed equally between the upper and the lower portions. In some, accessory parathyroids are found in the main thoracic thymus or its involuted remains but very rarely they are present actually in the thorax also. The average size of the parathyroid is $1\frac{1}{2} \times 3\frac{1}{2} \times 1\frac{1}{2}$ m.m. and their total weight 132 mg.

The parathyroids are developed as an outgrowth from the third and fourth bronchial pouches and are composed of masses of epithelial-like cells, sometimes more or less divided into lobules by bands of connective tissue. The cells contain granules, some of which are fatty. The blood vessels are numerous and form sinus-like capillaries which come in close relationship with the epithelial cells of the glands. Two types of cells are distinguished, the 'oxophil' and the principal. The two types do not appear to be of distinct nature and it is not known which cells give rise to the active substance and extracts of the gland.

Parathyroid extract is prepared from the fresh parathyroid glands of healthy animals by being extracted with hydrochloric acid. The standard unit now adopted is the amount necessary to raise the blood calcium of a dog weighing 20 kilograms by 1 mg. per 100 c.c.

During the last century it was found that after thyroidectomy, the laboratory animals went into a state of convulsion and spasm of the hands and feet, more marked in carnivorous than in herbivorous animals. EUGENE GLEY of Paris demonstrated that this condition was due to the removal of the parathyroids along with the thyroid, and the apparent immunity of herbivorous animals to this condition after thyroidectomy was due to the fact that the lower parathyroids in them are situated at some distance from the thyroid and thus escaped removal. The first symptoms of convulsion appear 36 hours after parathyroidectomy. The animals become depressed, the limbs are stiff and fibrillary twitching later develops. Later, clonic spasms of the limb and facial muscles occur, or there may be tonic spasms of the extensor muscles. If clonic movements predominate, snapping of the jaws with barring of the teeth is seen and co-ordinated automatic movements such as shaking of the paws occur. The animal is semi-conscious. In certain percentage among dogs, spasm of the abductors of the larynx occur, the expiration then being noisy and high-pitched. The blood shows a reduction in the calcium content. There is a rise in the plasma inorganic phosphorus.

Relation of Parathyroid to Vitamin D and Ultra-violet Rays.—Absence of vitamin D was considered the widely recognised cause of rickets. The factors at work include the ratio between calcium and phosphorus in the body. If the ratio is not correct, vitamin D will correct it. When this ratio is correct and no vitamin D is present rickets occur. But when rats kept on diet deficient in vitamin D or calcium and phosphorus are exposed

to ultra-violet rays, rickets do not develop. If the diet is absolutely deficient in vitamin D or calcium and phosphorus, ultra-violet rays do not prevent the formation of rickets. Ultra-violet rays only rectify a relative deficiency in the above constituents of the food. It is experimentally proved that rickets is prevented by the ultra-violet light by its activating the ergosterol present in the fat of the skin. Parathyroid by increasing the blood calcium greatly helps in the prevention of rickets. There is no definite association of parathyroid with rickets but parathyroid administration by increasing the blood calcium, along with ultra-violet rays, certainly does prevent the onset of rickets. Parathyroids are entirely associated with calcium metabolism only.

Diseases due to Parathyroid Deficiency.—*Tetany* is characterised by a combination of intermittent painful cramp in the muscles usually of the extremities or larynx and associated with hyper-excitability of the neuromuscular apparatus.

Symptoms.—There is muscular spasm which may affect any muscle of the body, even the ocular and the bladder muscles, but are common in the muscles of the forearms and hands and the larynx and less frequently the muscles of the lower limb. The spasm is tonic and is nearly always bilateral. The small muscles of the hand are first affected. The thumb is adducted into the palm, the fingers are adducted, flexed at the metacarpophalangeal joints and extended at the interphalangeal joints, thus producing the "accoucheur's" position or "pen-holding" position. The wrist and elbow joints are also strongly flexed. The feet are commonly held in a corresponding attitude, the toes and feet are dropped and in-turned and flexed at the knees and hip with adduction. But the lower limbs frequently escape. Rarely trismus, risus sardonicus laryngo-spasms or opisthotonus is present as also diplopia due to spasm of the eye muscle. The attack lasts from 15 minutes to 3 hours sometimes. There is tingling of the arms due to sensory nerve involvement. There may be present a tingling sensation, the distribution of the sciatic nerve. The central nervous system may be involved leading to coma and death.

Trousseau's sign:—Pressure upon the vessels or nerves of the limb will produce a spasm. Typical carpal spasm can be brought about by pressure on the upper arm. This is best done by inflating a sphygmomanometer bag, wound round the arm, to a pressure just higher than that of the systolic B. P. The spasm is produced if absent, or is augmented if present, the thumb can be separated from the palm only with great difficulty. This is a reflex phenomenon due to irritation of the peripheral sensory nerves.

Chvostek's sign:—CHVOSTEK first pointed out the readiness with which mechanical stimulation induces spasm in the muscles in patients suffering from tetany. This is best elicited by tapping the facial nerve close to the ear lobe, when twitching of the upper lip or even of one half of the face occurs. It is not a very reliable sign since occasionally patients suffering from tuberculosis, gastro-enteritis and myxoedema and even some healthy people show this sign.

Erb's phenomenon:—Fibrillation and spasm are induced by cathodal stimulation of the muscles by currents insufficient in its effects on normal subjects. Other diseases due to parathyroid deficiency. There is no definite evidence that paralysis agitans and Thomsen's disease are due to lesions of the parathyroids.

1. *Diet*:—Diet low in phosphate content as meat, yolk of egg and milk must be restricted to a few ounces a day.

2. *Drugs*:—Calcium Lactate 60 grs. (1 dram) six times a day must be given. Calcium Gluconate can also be given but Calcium Chloride 30 grs. six times a day is more effective. Intravenous injections of 20 c.c. of a 5% solution of CaCl_2 is very useful to relieve the symptoms during an attack. The solution must be injected slowly into the vein, subcutaneous infiltration must be avoided since necrosis is thereby produced. It is usual for the patient to feel momentarily a sense of warmth all over the body, some patients describing it as a burning sensation. When the parathyroid tissue has been extensively damaged, 30 units of parathyroid extract must be injected intramuscularly two to four times a day. But when giving parathyroid extract injections, serum calcium must be estimated. But, after injections of parathyroid extract are repeated for several months, an apparent immunity is established and then huge doses have no effect. In such cases where parathyroid extract cannot be continued indefinitely either due to its cost or due to its action becoming slow or inert, vitamin D should be given. Calciferol is a good drug for administration. A fraction of irradiated Ergosterol called A. T. 10 is given in doses of 5 c.c. by mouth on alternate days. While giving this A.T. 10, serum calcium must be estimated, as otherwise grave complications due to hypercalcaemia are liable to occur. Thyroid extract given in 2 to 3 grain doses daily augments the effects of parathyroid administration. A serum calcium level of 10 to 12 mg. per 100 c.c. is alright but above that level it is not desirable. Parathyroid grafting is of no value.

3. Removal of foci of infection.

4. Rest in quiet environment.

5. Mental and physical health must be attended to. Patients must be encouraged to work and attempts at introspection discouraged.

6. Sedatives as luminol to produce sleep.

7. Pain of the cramp can be alleviated by the application of cold to the affected limbs.

Symptoms of overdosage of Parathyroid or Hyper-calcaemia.—Hypotonia, muscular weakness, anorexia, nausea, vomiting, abdominal cramps, polydipsia, polyuria and renal calculi are present. The bony changes are as follow: The bones are soft and cut easily with a knife. The great resorption of bone leads to deformity in any part of the skeleton. There may be spontaneous fractures. Histologically there is lacunar resorption, apposition, fibrosis of the marrow, and the formation of osteo-clastomata and cysts.

Parathyroid deficiency is a definite entity in the causation of tetany, which can be easily recognised by its characteristic symptoms and signs and which, though causing alarm to the patient and his relations, need not alarm the medical attendant, since it is very amenable to treatment.

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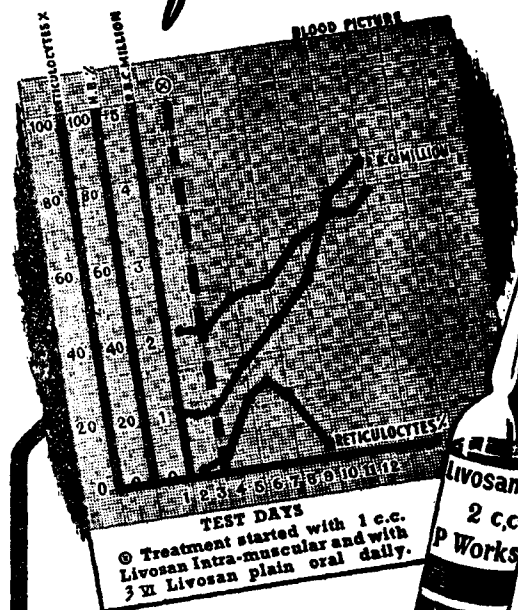
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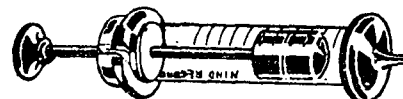


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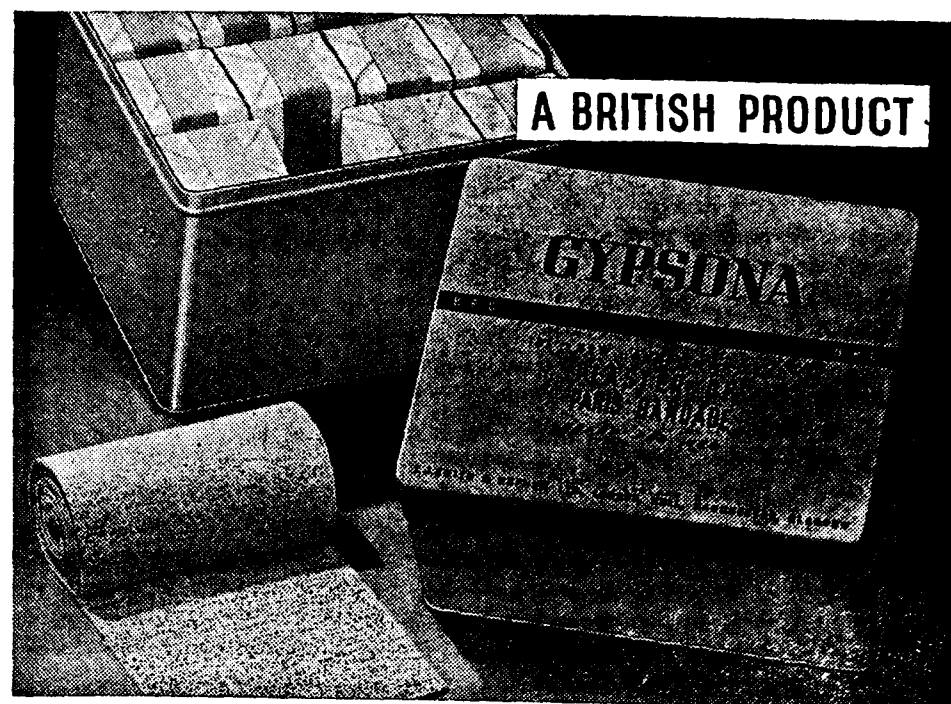
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Deficiency Anæmia

BY

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THE deficiency anæmias are due to the lack of one or more of three factors :—I. Castle's hæmatinic principle ; II. Iron, Copper. III. Vitamin C, Thyroxin.

The deficiency may be due to the lack in the diet of these elements necessary for blood formation or the body may be unable to utilise them when present. These anæmias are of two types.

1. *Macrocytic, hyperchromic anaemia* which is due to :—(1) deficiency of intrinsic factor ; or (2) deficiency of extrinsic factor ; or (3) inadequate storage of Castle's hæmatinic principle in the liver ; or (4) improper utilisation of P.A. factor which is adequately stored in the liver.

The following are examples of this type : (1) Addisonian anæmia, (2) sprue anæmia, (3) tropical macrocytic anæmia, (4) anæmia due to bothrioccephalus latus infection, (5) pregnancy anæmia, and (6) anæmia of coeliac disease of children and idiopathic steatorrhea.

2. *Microcytic, hypochromic anaemia* due to : (1) Deficiency intake of iron in food ; or (2) defective absorption of iron taken which occurs in chronic gastritis, cancer etc., where there is deficiency of Hcl in gastric juice or (3) vitamin C deficiency.

The following are examples of this second type : (1) chlorosis, (2) primary hypochromic anæmia (Witt's), (3) anæmia after gastro-enterostomy, (4) nutritional anæmia of children, (5) anæmias of prematurity, (6) anæmia of scurvy, and (7) pregnancy or lactation anæmia.

A knowledge of erythropoiesis and its control is essential for a proper study of the etiology, pathology and treatment of deficiency anæmia.

Erythropoiesis.—In early foetal life blood is formed by the liver and spleen but after the 5th. month this is taken on by bone marrow. In the child all the marrow is red but in the adult it is confined to flat bones. Low oxygen tension stimulates the endothelial cells lining the intersinusoidal capillaries of red bone marrow to divide repeatedly to give rise to immature cells (*megaloblasts*), whose cytoplasm at first is entirely basophilic. If the endothelial cells fail to proliferate, Aplastic anæmia develops. Then Hb makes its appearance in the cell, the cytoplasm stains a slaty colour and the nucleus becomes smaller ; thus the megaloblast becomes converted into *erythroblast* (early and late) and then into *normoblast* (drop of ink appearance).

Castle's Hæmatinic Principle.—Maturation of the megaloblast to normoblast is dependent upon Castle's principle, which is produced by the interaction of an enzyme—*intrinsic factor*—which is present in normal gastric juice (produced by oxyntic cells of pylorus probably) and a substance found in ox muscle—*extrinsic factor* in the nature of vitamin B₂ complex—marmite and yeast. This hæmatinic principle consists of 2 parts. (1) Hemopoietic factor, necessary for the maturation of the megaloblasts and (2) neuopoietic factor—(Mallenby), absence of which causes sub-acute combined degeneration. This principle is absorbed from the gut, stored mainly in the liver of mammals—thus explaining the therapeutic

value of liver extracts in macrocytic anæmia. This might not be available for use by bone marrow in conditions of deficiency of either factor or of improper absorption or utilization of P.A. factor due to abnormal state of intestinal tract or presence of parasites (tape worm). The lack of this Castle's principle is responsible for the *macrocytic, hyperchromic anaemia*.

Then the normoblasts lose their nuclei and become erythrocytes (R.B.C.). For this maturation iron, copper, magnesium, vitamin C and thyroxin are all necessary; and deficiency of any of these is responsible for *microcytic, hypochromic anaemia*.

Fate of R.B.C.—The average life of R.B.C. is 30 days. The R.B.C. which have become worn out are destroyed by a process of fragmentation by cells of reticulo-endothelial system of spleen, bone marrow and Kupffer cells. The Hb of the red cells consists of globin (histone) united with hæmatin which is an iron derivative of porphyrin (consisting of 4 pyrrol groups), and it is broken down into 2 moieties: (1) Hematoidin (iron free) and (2) hemosiderin containing iron. The former is converted to bili-rubinogen which is carried to the liver to be excreted in bile and the latter is retained within the body for building up of fresh R.B.C. The Hb is essential for oxygen carriage in the body, in the transport of CO₂ and in the regulation of blood reaction. It is 13.8 grams per 100 c.c. of blood. The percentage of Hb is an index of the oxygen-carrying power of blood.

Metabolism of Iron.—Green leafy vegetables which are rich in chlorophyll contain large quantities of Iron, (*amaranth, coriander, mint, manathakkali, and lettuce*); greener and thinner the leaf greater is the iron content. Iron is contained also in *amaranth* stem, bitter gourd, drumstick and in animal food. Ordinary diet contains enough of iron, i.e. 5-10 mgms. but only part of iron in the food is available for building Hb. 70% of iron in liver, 50% in beef, 50% in cereals and 20% in the vegetables. Milk is low in iron content and so when milk is the sole article of food, as in infants, anæmia is produced. The iron is absorbed in the upper part of the small gut, depending on the presence of Hcl in the gastric juice and then carried to the liver for storage, whence it gains access to bone marrow for Hb formation. The unabsorbed iron of the food is excreted in fæces. Daily minimum requirement for an adult is 6-12 mgm., pregnant women and suckling mothers require more.

Symptoms of Anæmia.—(1) Failing strength and languor of insidious onset and considerable duration, (2) pallor of lips and conjunctiva; sclera are bluish, (3) dyspnœa on exertion, palpitation, giddiness, fainting, œdema of ankles and hemic murmurs, (4) loss of appetite, discomfort after food, (5) vertigo, headache, nervousness, neuralgia, irritation or depression of spirits, and (6) amenorrhœa.

Addisonian anaemia is a true deficiency disease due to the lack of the intrinsic factor. There is gradually progressive anæmia with insidious onset and its course is marked by spontaneous remissions and exacerbations. The symptoms clear up and blood picture becomes normal during the remissions. The patient is plethoric, above middle age; the skin is of a lemon yellow tint. There are glossitis, chronic gastritis and achlorhydria. All the formed elements of blood are diminished in number, Hb % is diminished and colour index is greater than one. Average size of R.B.C. is

Bilirubin content of serum is raised and there is latent jaundice. The one and only pathognomonic finding is megaloblastic hyperplasia of bone marrow. Though the bone marrow is exceedingly active and full of megaloblasts, the further maturation is arrested, due to the lack of the P. A. factor. The result of liver therapy is dramatic.

Sprue anaemia is a tropical disease with passage of large bulky stools with a high fat content; achlorhydria is common but 75% cases respond with Hcl secretion to histamine. There is no hemolysis as in Addisonian anæmia. There is atrophy of tongue and intestinal mucosa, causing defective absorption of Castle's principle. The anæmia is macrocytic, hyperchromic.

Tropical macrocytic anaemia is the most common type of anæmia in the tropics affecting, especially, pregnant women. It is macrocytic in type responding specifically to marmite and liver, and is probably due to deficiency of extrinsic factor (Vitamin B₁₂ Complex)—Riboflavine (B₂), Adermin or Pyridoxine (B₆), Nicotinic Acid (P-P factor) and Pantothenic acid (filtrate factor). Pure liver extract is not useful in this condition but a new substance found in crude liver extract which retains all the factors of Vitamin B Complex and in autolysed yeast, and in marmite is effective. Purification of liver extract to isolate the anti-pernicious anæmia principle destroys the Vitamin B complex. In 1931, marmite in sufficient dose was found to be curative alone in this condition.

Macrocytic anaemia due to fish tape worm is due to interference with absorption of the hæmatinic principle. This is common among fish-eating people.

Pregnancy anaemia.—In pregnancy mild anæmia is of almost common occurrence. Factors at work are:—(1) Iron deficiency in the diet, poor secretion of gastric juice leading to defective iron absorption and great depletion of iron from the system for foetal blood production. (2) Intrinsic factor may be lacking, thus producing macrocytic anæmia; tropical macrocytic anæmia may complicate pregnancy and even end fatally unless effective treatment is instituted.

Deficiency anaemias of childhood: Types:—(1) *Nutritional A* is due to deficiency of iron either from deficient ante-natal storage or post-natal supply. This arises due to prolonged breast feeding and in infants fed by anæmic mothers. This is microcytic, hypochromic.

(2) *Anaemia of prematurity:*—The premature child becomes anæmic a few weeks after birth, due to deficiency of iron.

(3) *Anaemia of scurvy:* Due to vitamin C deficiency (Hypochromic A).

(4) *Anaemia of coeliac disease:*—It is hyperchromic and macrocytic due to absence of extrinsic factor.

Primary (idiopathic) hypochromic anaemia (Witt's A) occurs exclusively in middle-aged women, remarkable for its chronicity and hypochromic in type. The bone marrow undergoes normoblastic reaction. The average size of R.B.C. is less and colour index less than one (microcytic). Tongue is red, angry and glazed; at times it is associated with dysphagia (Plummer Vinson Syndrome). Nails are brittle, dry and spoon shaped (koilonychia). The chief cause is achlorhydria and chronic gastritis due

Hypochromic anaemia following gastro enterostomy and gastrectomy : The stomach empties so rapidly that there is no time for adequate digestion and absorption of iron. This responds well to iron.

Treatment.—1. Rest in bed ; removal of septic foci ; blood transfusion in severe cases. Superimposed infection must be first eliminated and then the specific therapy started.

2. The treatment resolves itself into the adequate supply of the deficient factors. This, like all other deficiency diseases, must be treated on quantitative basis, enough being given to supply his needs and to furnish an adequate reserve. The improvement in the condition must be kept up by maintenance dose.

(1) *Liver*:—The simplest way of administering liver is to include it in the menu, but large amount is not palatable. It can be given as fresh liver $\frac{1}{2}$ lb. daily—very lightly cooked or made as liver soup. But parenteral injection is simple and cheap. In mild cases give the liver extract—a sterile aqueous solution obtained from mammalian liver, 1 c.c. representing the potency of 200 grams of oral liver—in doses of 1-2 c.c. daily for a week and later continue with 2 c.c. once a week. Dosage depends on the clinical progress and by reference to blood examination. Maintenance dose: 1-2 c.c. a week or even longer.

(2) *Dessicated stomach* ($\frac{1}{2}$ ounce twice daily) or *Ventriculin* (10 grams daily) is also given in macrocytic type.

(3) *The extrinsic factor* is given as marmite, 30 to 60 grains thrice a day in warm water or milk.

(4) *Acid hydrochloric dil* in 60 minim doses with meals in cases of achlorhydria.

(5) Iron has been known for centuries as a valuable remedy indispensable for Hb formation. The degree of success of therapy depends on the form in which iron is administered. Iron is not absorbed in the ferric state but must be converted in the small gut to the ferrous state, especially Ferrous Chloride by the action of Hcl of gastric juice. But in anæmic patients, there is usually Hypochlorhydria which interferes with proper and adequate absorption of iron. So iron is best administered as *ferrous chloride*. Next in order comes *ferrous sulphate* which is the most readily assimilable of all forms of iron and the least likely to cause gastro-intestinal irritation. All ferrous salts are best given just after meals.

Ferri et Amm. Citras is well tolerated by even pathological gastric mucosa ; but must be given in large doses (90 grains a day). Another form of iron is *colloidal iron hydroxide* which causes little or no gastric irritation and which is available for immediate absorption.

Copper and Manganese:—It has been shown that the concomitant administration of these elements aids the assimilation of iron, intensifies the conversion of iron into Hb by acting as *catalysts*. Thus copper accelerates the therapeutic value of iron.

Arsenic has lost now much of the importance it had in the treatment of any secondary anæmia. It has no hæmatinic influence but acts as an irritant of bone marrow.

Pellagra in Children

BY

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PELLAGRA is being recognised more readily during recent years. Many workers have reported cases of pellagra from various parts of India. Pellagra in children has interested the writer. In this communication a typical case of pellagra is described, and recent opinions on pellagra in children are recorded.

Definition.—“Pellagra is a chronic relapsing disease occurring especially in maize eaters due to a deficiency in certain proteins and factors contained in the vitamin B₃ complex. Clinically it is characterized by buccal and gastro-intestinal disturbances, psychical and nervous features, and a symmetrical eruption especially affecting areas of skin exposed to the sun's rays or to friction.”

In India maize is not a dietary factor, but the diet is unbalanced and relatively rich in carbohydrate and poor in quality protein and protective substances and these points have been stressed by G. DINKER RAU and T. K. RAMAN. “The more distinguishing features are:—(a) a remitting dermatitis of the exposed parts of the body ; (b) marked emaciation ; (c) profound depression alternating with manias ; and (d) a terminal confusional insanity” (PHILIP H. MANSON-BAHR).

Case Record.—A female child aged 9 years, (and coming from a poor family) was admitted on 26-6-1941 for swollen limbs of five days duration.

Clinical:—The patient is an ill-nourished child with scabies over the hands and feet, and a soft swelling of the extremities. Conjunctival xerosis was present. Angular stomatitis present. Tongue raw and denuded of epithelium. Skin scaly and pigmented symmetrically over the dorsum of the hands and feet. Nails and conjunctivæ pale.

Abdomen:—Liver and spleen not palpable. No masses felt inside the abdomen. No signs of free fluid inside the abdomen elicited. Bowels move twice or thrice a day. Heart—Nothing noteworthy. Lungs—Nothing noteworthy. Nervous system—The child looks extremely depressed and irritable. Cranial nerves—Nothing particular. Motor system—Low muscular power. Co-ordination normal. Sensory system—sensation not impaired anywhere. Deep reflexes—Present. Superficial Reflexes—Present. Plantar responses flexor on both sides. Visceral reflexes—Normal.

Investigation:—Motion—Semisolid—no mucus or blood. Nil particular under the microscope ; Urine—Sp. gravity 1005. No sugar. No albumin. Oxalates and phosphates present. No organisms. Blood—No malaria parasites present. Red cells—2.1 millions per c.m.m. Hæmoglobin 35%. C. I. 0.83. White cells total 100,800 per c.m.m.

Differential count of W. B. C.—Polymorphus 76%—Lymphocytes 22.51%—Monocytes 1.5%—Eosinophiles 0.1%—(Hypochromic, normocytic anæmia ; leucocytosis present). Khan's test of patient's blood—Negative. Gastric analysis—complete achlorhydria present. Mucus, bile and starch present in every specimen. X-rays of Lungs—Nil particular—No signs of active tuberculosis. X-ray of wrist—six carpal bones seen. Generalised rarefaction of bones present.

This case was diagnosed as pellagra on the basis of skin changes œdema of hands and feet, mental picture, irregular diarrhoea, an

achlorhydria. Mental depression and œdema of extremities have been stressed by PHILIP H. MANSON-BAHR as being characteristic of child pellagrins.

Treatment :—Adexolin (m. 3 B. D.), Marmite (3 ss B. D.), Nicotinic acid 3×50 Mgms. per day by mouth, and Liver extract 2 c.c. intramuscularly on alternate days, were administered. Egg albumin added to diet. Danish ointment for the skin. The child developed a terminal pyrexia and died in a state of exhaustion.

Etiology.—Pellagra is believed to be caused by a deficiency of vitamin B₂ (nicotinic acid and riboflavine), vitamin B₆ (adermin), and also proteins of high biological value.

Pellagra is said to be common between the ages of 20 and 40 years. Box has recorded a case at the early age of 20 months. Females are said to preponderate. Spring and early autumn are seasons when symptoms aggravate. The disease is endemic in the Southern States of U. S. A., Lower Egypt, Turkey, Rumania, the Balkans, Spain and Italy. Cases have been reported from India, China, Japan, parts of Africa, Mexico, West Indian Islands, etc. Poverty, chronic malaria, tuberculosis, syphilis and alcoholism are all debilitating conditions wherein pellagra may also develop.

Pathology.—Emaciation and atrophied viscera are seen. The dermal lesions consist of initial erythema of the surface layers ending in exfoliative or exudative dermatitis. Orolingual ulceration and lingual atrophy ensue. Small round ulcers of jejunum, colon and rectum are described. Mesenteric glands may be enlarged.

There may be a sub-acute combined degeneration of the cord (posterolateral sclerosis and damage to the Clarke's column). Degeneration of the anterior horn cells in the lumbar cord, inflammatory change in ganglion cells in the posterior roots, meningeal thickening and adhesions, cerebral atrophy, and hydrops of ventricles and subarachnoid space are all described. Histamine fast achlorhydria associated with secretion of intrinsic factor of Castle are found. Total W. B. C. count is normal but lymphocytosis and secondary anæmia are found. Cerebro-spinal fluid is normal. Low co-enzyme content of blood is found in severely ill pellagrins.

TROWELL recorded amongst child pellagrins in Africa, microcytic and macrocytic anæmia, fatty stools, and neurological signs and low blood sugar curves. He found also, post-mortem, remarkable atrophy of the thymus.

Symptoms.—A pellagrin exhibits in spring, anorexia, nausea, vomiting or diarrhœa as also oro-lingual ulceration, and excessive salivation. Erythema and itching of areas of the skin exposed to sun or subject to friction may be noticed. Older children may complain of numbness or cramps in the legs, and headache, giddiness or insomnia. In children retardation of growth occurs and neurological complications are rare.

The backs of fore-hands and fore-arms, the fore-head, a butterfly area of the face and the dorsum of the feet are regions where the skin changes are generally manifest, and these are symmetrical and resemble sunburn with definite edges. In children swelling of hands and feet may be present. Vesicles may form, and in summer scaling starts as colour fades, and finally after years, the skin becomes thinned out. Seasonal recurrences are common. The tongue is raw and red (beet tongue). Angular stomatitis may be present. Blood and mucus may be seen in the motions. The

temperature is usually normal. A febrile and typhoidal form is also described and carries on bad prognosis. Pellagra sine pellagra is of doubtful etiology.

Differential Diagnosis.—Typical pellagra is diagnosed on the presence of characteristic triad of dermatitis, diarrhœa, and dementia. In the diagnosis of early and sub-clinical cases, a reliable history of dietary deficiency, and the clinching urinary list for nicotinic acid excretion (HARRIS and RAYMOND) are very important. Amongst adult pellagrins, sprue and ergotism may cause difficulty. Erythema multiforme and dermatitis venenata have also to be remembered. Pink disease is distinguished by the characteristic tachycardia, cold and pink extremities, and mental condition which is sometimes very pronounced.

Course and complications :—Modern treatment of pellagra with vitamin B₂ complex and high protein diet tend to reduce the mortality. In the past, a large percentage of adult cases died of dementia in asylums.

The fulminant typhoidal form leads to death. Secondary pellagra needs appropriate diet and treatment of basic alimentary trouble.

Treatment :—Prophylaxis lies in a balanced diet. A normal adult requires about 18 milligrams of nicotinic acid daily.

Curative treatment :—Rest in bed needed for acute cases as also heavy dosage of nicotinic acid and balanced food containing meat, milk, eggs and fruits.

In a series of 73 patients including 36 children, treated by SPIES, the dosage of nicotinic acid was from 100 to 200 milligrams daily. In the hands of TROWELL, early cases of pellagra in African children responded to massive doses of marmite, liver, increased animal protein and elimination of maize from diet.

For oro-lingual ulceration and angular stomatitis, riboflavine 1 mgm. is given thrice a day. For diarrhœa, and achlorhydria diluted Hydrochloric acid is given orally. Liver extract by injection or stomach extract by mouth is needed. Vitamin B is given when neuritis is present. The skin is protected from the sun. Moist dermal lesions are painted with 1 in 5000 Potassi-Permanganas solution. Good results have been claimed with intravenous Sodium Thiomephate (10 c.c. 10 per cent.) daily for 20 to 60 days.

I am greatly indebted to Prof. G. R. DINKER RAU, M.D., first physician, Stanley Hospital, Madras, for permitting me to study and report on this case under his care. My thanks are also due to Prof. M. G. KINI, M.C., M.Ch. (Ortho.), F.R.C.S. (Edin.), F.R.S. (Edin.), Superintendent, Stanley Hospital, Madras, for permitting me to utilize the hospital records while preparing these notes.

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Vitamin B₁ Deficiency and Cardio-Vascular Disturbances

BY

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Introductory.—The old concept that a diet composed of carbohydrates, proteins, fats and salts supplied all the necessary requirements of the human machinery, was revolutionized during the first two decades of the present century. Gradually it became apparent that physiological evolution had rendered the higher mammals dependent on food constituents, other than the major ones. In 1881, LUNIN, working in Basel, had considered this possibility. In 1905 PEKELHARING described experiments which indicated that milk contained some unrecognised substance or substances which were presumably necessary for nutrition. It was HOPKINS (1906, 1912) who showed that young rats could not flourish on a 'synthetic diet' composed of proteins, carbohydrates, fats and salts, unless some fresh materials like milk are supplied, and these, as HOPKINS pointed out, "contain countless substances other than the proteins, carbohydrates and salts," which go by the name of vitamins.

The number of vitamins is increasing everyday. At the present day vitamin B₁ seems to hold a conspicuous position. RUDRA (1939) says that in the medical literature of this ancient land of India (CHARAKA and SUSRUTHA), for symptoms now ascribed to vitamin B₁ deficiency, the diet prescribed includes rice, barley, kodo rice, pulse and pulse juices which are rich in vitamin B₁. It appears therefore that their diet prescription was quite sound even in the light of latest researches. Empirical observations on the nutritional disturbances date from the latter part of the sixteenth century.

Vitamin B₁ deficiency has been associated with a variety of disorders, notably anorexia (COGWILL), polyneuritis including alcoholic polyneuritis (STRAUSS, MINOT and COBB), polyneuritis of pregnancy (STRAUSS and MACDONALD, WINANS and PERRY, and others), certain cardio-vascular disturbances (WENCKEBACH, KEEFER, WEISS and WILKINS) and finally beri-beri, a symptom complex which embraces these and other manifestations.

The present paper deals with the cardio-vascular disturbances caused by vitamin B₁ deficiency.

Clinical features.—WEISS and WILKINS (1936) have concluded that a special type of cardiac disease, caused by a deficiency of vitamin B₁, is not uncommon in the United States: the symptoms include circulatory disturbances, excessive arterial pulsation, loud sounds over the arteries, rapid circulation rate, vasomotor collapse, exaggerated sensitivity of carotid sinus and cardiac enlargement. There is tachycardia, or vagus reflex irritability with bradycardia, sometimes with a systole and syncope.

JONES and SURE (1937) discuss the thesis that "certain cardiac diseases may be due to accumulative effects of vitamin B₁ deficiency extending over a period of years, as it is a known fact that the general American diet is deficient in vitamin B₁. JONES and SURE feel that their

and in some of the cases commonly reported in western countries as idiopathic hypertrophy of the heart. ABT concludes that vitamin B concentrates should certainly receive a trial in babies with unexplained cardiac enlargement.

LION and FACQUET (1938) have written on the association of heart failure with arterial hypotension resulting from alcoholism. In support of their thesis they have published two cases. They have described five characteristics of the clinical syndrome. They are: heart failure manifested as dyspnoea, painful distension of liver, and oedema round the ankles; (2) hypotension; (3) normal or accelerated circulation rate; (4) alcoholic polyneuritis or hepatic fibrosis, and (5) withholding of alcohol results in a complete disappearance of the symptoms. They agree with the other observers that the syndrome resembles that ensuing in beri-beri and that a deficiency in vitamin B₁ provides a specific etiological factor.

STEPHENSON, PENTON and KORENECHEVSKY (1941) have said that they have not noticed well-developed symptoms of the cardio-vascular system as a result of vitamin B₁ deficiency. In two, however extra-systoles which had been present for a long time before the treatment disappeared after the administration of vitamin B complex. No constant or significant changes have been observed in the 'resting' blood pressure and pulse rate. Although varying in individual cases, the average figures obtained in all of their forty patients were remarkably close. They have not noticed any change in the degree of arteriosclerosis of the treated patients. Some definite improvement has been indirectly recorded, however, in the function of the cardio-vascular system, because the shortness of breath and oedematous eye sacs and ankles completely disappeared or were much improved in a large percentage of cases.

The common early symptoms are oedema, shortness of breath, and weight. The oedema is, however, not typically cardiac in that it is widespread (the face often very puffy) and sometimes precedes the dyspnoea, the reverse of the usual order in heart failure. It has, moreover, been noted that the degree of oedema is excessive in proportion to the venous congestion. Normal rhythm with tachycardia is the usual rule, though auricular fibrillation has been found in three of a series of 98 cases by WEISS and WILKINS (1940).

The following changes in the electro-cardiogram have been observed—low voltage waves, lengthening of the Q-T interval, deviation of the R-T segment, flattening or frank inversion of the T waves in the limb leads although tending to be less conspicuous in the chest lead. The electro-cardiographic changes disappear rapidly after treatment.

Pathogenesis.—Alcoholic neuritis is now believed to be due to deprivation of vitamin B₁, but it is important to recognise that alcoholic poisoning may cause cardio-vascular disturbances, which are usually accompanied by a peripheral neuritis but may occur independently. Alcoholism causes vitamin B₁ deficiency in three ways: first through anorexia and deficient ingestion; secondly by way of achlorhydria the gastric contents become alkaline and inimical to the vitamin; and thirdly, utilisation of the vitamin may be adversely affected by liver damage. Apart from alcoholism vitamin B₁ deficiency may occur due to other factors and cause the same cardio-vascular disturbances.

Diagnosis:—In the diagnosis of avitaminosis-B₁ heart, the chief points are a history of alcoholism and deficient diet, the absence of any other adequate cause, the age and sex of the patient who is, most often, a man in the early middle age, a low blood content of the vitamin, and finally the therapeutic test.

A word of caution may be sounded here that before associating a symptom to vitamin B₁ deficiency the possibility that avitaminosis may be no more than a precipitating factor in chronic cardio-vascular disease has to be borne in mind.

Prognosis:—It is usually very good with adequate and proper treatment.

Treatment:—If the patient is addicted to alcohol then it must be withheld. The patient must have proper amount of rest. Digitalis and mercurial diuretics will accomplish little, but parenteral injection of vitamin B₁ will generally dispel the symptoms in a few days.

Comments:—There is a convincing amount of literature demonstrating a causative association between cardio-vascular disturbances and partial deficiency of vitamin B₁. These are promptly cured by vitamin B₁. The extreme—"cardiac beri-beri"—is very dangerous; but even the milder forms call for the closest medical attention.

Acknowledgment.—My thanks are due to Dr. HABIB KHAN, and Dr. E. N. GUPTA, my house physicians, for their valuable suggestions.

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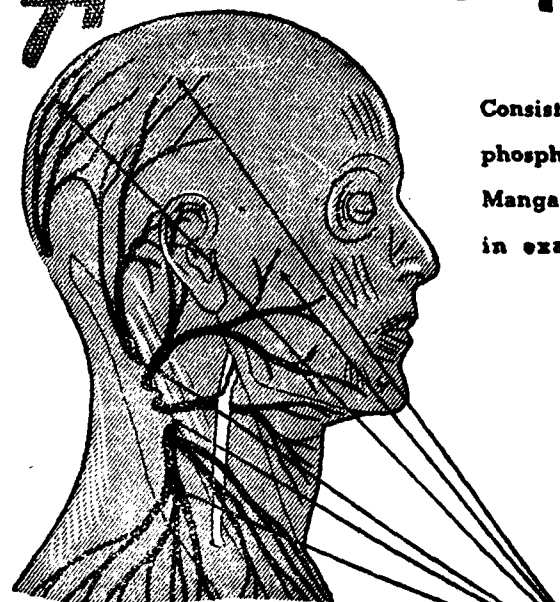
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Some Observations on Vitamin B Complex

BY

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VITAMIN B during recent years is subdivided into numerous component parts chief of which are B₁ and the anti-neuritic vitamin commonly known as the beri-beri vitamin. It is, however, not quite correct as the absence of this vitamin is the cause of any peripheral neuritis whether due to beri-beri or due to any other cause like alcohol or arsenic, or due to a metabolic disorder like diabetes, or due to a toxic process following dysentery, diphtheria or any infectious disease like malaria, influenza, pneumonia or enteric fever. Every case of beri-beri is a case of peripheral neuritis but every case of peripheral neuritis is not one of beri-beri. Beri-beri is a specific form of multiple peripheral neuritis due in part to deficiency of vitamin B₁ and partly perhaps to a toxin of an infective nature. Every case of beri-beri must have a cardiac involvement, viz., dilatation with vagal neuritis. Nearly 40 years ago a number of medical students in a medical hostel on the ground-floor in Vizagapatam suffered from typical beri-beri while those on the top-floor escaped and both groups had identically the same diet in the hostel mess. Again about 10 years ago in one hospital where I was in charge, a typical case of beri-beri was admitted in a ward amongst other patients and in about six weeks both the patients on either side of this beri-beri patient showed signs and symptoms of beri-beri and they were originally the one a case of trachoma and the other a case of arthritis. The presence of this infective toxin appears to be more common in areas where the soil is brackish and moist as in Masulipatam and less common in island areas like Avanigadda or fresh water deltas. The presence of salt in the soil has something to do with it. These perhaps throw some light that there is some infective origin besides a deficiency in the causation of beri-beri.

The association of some of the B complex factors B₂ (1) riboflavine generally and B₂ (2) nicotinic acid, pellagra preventing factor, sometimes with deficiency of vitamin A, is a common feature in adolescents. Although vitamins A and B are two distinct and separate entities producing different train of signs and symptoms, deficiency of A producing night-blindness, xerosis, kerato-malacia, defective resistance to infection and diminished growth, and deficiency of vitamin B₂ (1) ariboflavinosis, producing angular conjunctivitis, blepharitis, superficial keratitis, angular scarring of nose, angular ulceration of mouth, glossitis and phrynoderma, we often find them combined in children and adolescents of defective nutrition. Fifteen years ago, while in charge of a Junior Certified School, I found nearly 50% of the boys varying from 7 to 14 years of age suffering from this combined deficiency. The complex component parts of vitamin B were not known then and as there was xerosis and the boys complained of night-blindness, in nearly every case, cod-liver oil, green vegetables and home made buffalo's butter milk without butter being removed was added to their regulation diet of par-boiled rice. The boys improved remarkably in a few weeks putting on weight every fortnight and they got quite alright in less than 2 months. But again when the additional factors stated above were withdrawn from their diet and when they were restored to the regulation diet of rice and *dhal* and vegetables, they

used to suffer from the same deficiencies after a few months. Very recently I had a case, a report of which I had sent to *The Antiseptic*, (a case of B complex deficiency) in which there were all the factors of vitamin B deficiency with attendant signs and symptoms viz., B₁, B₂ (1) and B₂ (2) neuritis, ariboflavinosis and pellagra. It is a common feature to find this combined deficiency of vitamins B and A in almost every out-patient department amongst children and adults of defective nutrition.

In our country where the standard of living is necessarily low from economic distress, it is not possible to suggest yeast, marmite, wholewheat bread, milk, eggs, butter &c., as the chief sources to cure these deficiencies of A and B complex type. Something that is cheap, efficient and within the reach of men of average means must be the solution for these ills. I have used with good results germinating *dhal*, bran tea, hand-pounded rice, ragi cakes or porridge and butter-milk and green vegetables with medicines necessary in these cases. 1 to 2 ozs. of any cereal gram like green gram, Bengal or Punjab gram, can be soaked in water, germinated and eaten with jaggery or lightly cooked and taken with rice or porridge or cakes. Bran tea is made by taking 1 or 2 ozs. of fresh milled rice polishings in a cup and about 6 ozs. of hot water added and kept for about half an hour, then it is strained through a piece of muslin, and taken with jaggery added to it and a few ounces of milk, if necessary. This can be given twice a day. I have used this prescription in all cases of peripheral neuritis for several years with gratifying results.

There seems to be some affinity between vitamin B₁ and E as in cases of muscular dystrophies and organic nerve disorders. A combination of this therapy is more efficient in amyotrophic lateral sclerosis, progressive muscular atrophy, psuedo-muscular hyper-trophied paralysis, etc.

Diseases of Vitamin C Deficiency

BY

R. K. BHATTACHARYYA, M.B.,

Nabadwip.

SCURVY is the prime deficiency disease, as it was the first to be recognised as such, and led to the discovery of the wonderland of vitaminology. For a long time it was a dread to sea-farers, and took a very heavy toll of life, over and above much disablement and suffering.

Early explorers must have been seriously hampered by the scourge. After about four hundred years of suffering, reckoning from the time of the earliest explorers, about 1200 A. D., the remedy was empirically discovered. But it took another three hundred years for the underlying truth to crystallise. When the East India Company was floated in the year 1600, out of the first four ships that set sail on the maiden voyage of the company, three suffered heavy loss and disablement by scurvy while the ship of the Commodore totally escaped the disease due to the Commodore personally administering three table-spoonfuls of lemon juice to each person on board daily. The want of fresh fruits and vegetables was hinted at by many as the probable cause of the disease. (*Theory No. 1*).

Yet it was not until 1795 that the British Admiralty issued orders making it compulsory for every ship to carry an adequate supply of lemon juice, for prevention of the disease.

CAPTAIN COOK (1728-1779) is credited with having first been able to make a voyage an all-round success, by managing the ration of his crew adequately with the help of anti-scorbutic elements, and he was awarded a medal by the Royal Society in recognition of this. But what was the actual etiological defect and exactly how the remedy acted, remained a matter of controversy till very recent times.

We find NANSEN (1871-1930), the famous Arctic explorer, refuting the fresh vegetables and fruit juice theory, as he and his party were not affected by scurvy, although they had for a long time to live on raw meat and blood of the Polar bear, the seal and the rein deer. Necessarily, his view was that the disease must be due to some toxic substance formed in tinned and preserved food. (*Theory No. 2*).

In addition to the above two theories there was a third view about the causation of the disease, held by many, as late as the first quarter of the present century, the theory of infection by an unknown micro-organism. (*Theory No. 3*).

Extensive research on food factors and food values, gradually led to the discovery of that mysterious vital spark factor, that has since been named vitamin. Research in biochemistry is fast progressing, and in the span of roughly a quarter of a century, biochemistry has become a full-fledged branch of science. Innumerable obscure disease conditions are being fished out into the net of deficiency diseases; and vitamins are being discovered in rapid succession, with the exact formula of many of them. Prospects are not yet exhausted, and what was only the other day in the form of a controversial theory, has now developed into a definite "ology."

form now-a-days, except under very exceptional circumstances. In the last great war it was not uncommon among Indian troops in Mesopotamia.

It is no use dilating on the classical description of the disease in adults or in infants and children as there are ample descriptions in text books. There are obscure border line cases that can be benefited by setting the defect right. The insidious primary stage shows loss of weight, progressive weakness and pallor, and a certain stiffness and tenderness in muscles, specially of the legs. The gums may bleed easily. There may be palpitation, mental depression and headache. In infants, in the insidious stage, it is marked by failing nutrition, increasing pallor, fretfulness and pain on movement. Often tenderness of the legs is the very first symptom noticed. The child cries whenever handled. Some swelling may be noticed in one or both legs, and this may throw the doctor out of his track, who suspects some injury, or new growth or the like. The swelling and deep purplish colour of the gums and the tendency to bleeding soon develop and these should put the medical attendant on his guard in diagnosis. Often signs may be so slight only to suggest that the hip or the knee-joint is somehow or other affected and thus causing the pain and tenderness.

Since the discovery of the chemical composition of vitamin C, research work is progressing towards the estimation of the degree of vitamin C saturation in the body in normal individuals, and under abnormal conditions of disease, stress and strain. It has thus been found that about 40% (400 mgms.) of a one gram dose of Cevitamic acid, administered intravenously, is excreted in the first five hours and 10% more in the first twenty-four hours. It has been determined that the normal range of concentration in the blood plasma of ascorbic acid is between 0.6 and 2.0 mg. %. In a series of diseases, it has been discovered that the vitamin C concentration in the blood plasma falls below the normal level. In gastric or duodenal ulcers the level comes down to 0.2 mg. % or less. Conversely it has also been asserted that animals in an atmosphere of vitamin C deficiency are apt to be affected by gastric or duodenal ulceration. The prospects of ultimate healing of the ulcer, the complication of bleeding, and the chances of surgical intervention are now-a-days considered with reference to this vitamin shortage. Disease conditions with hypo-vitaminosis as above may gradually be grouped together under the heading "asymptomatic scurvy" or "sub-scorbutus," considering that scurvy is the prime disease of "C" vitamin deficiency.

The blood plasma and spinal fluid of confirmed drunkards with or without peripheral neuritis or alcoholic psychoses have been examined for concentration of ascorbic acid and it has been found that while those without symptoms have a low range of concentration, those with symptoms are all below par in percentage. It indicates that vitamin C probably plays an important role in the nutrition and proper function of nerve matter, although scurvy cannot claim any such nerve complication as its own.

It has also been shown that a deficiency in concentration of this vitamin contributes to delay in wound-healing in general.

The bleeding tendency in scurvy is attributed to fragility of blood capillaries. Since the discovery of ascorbic acid, it has been found by experiment that the employment of pure vitamin C in various hæmorrhagic diseases does not yield as good results as were expected. But whole lemon juice and other natural sources of vitamin C contain anti-hæmorrhagic

properties to a great extent. This has led to the discovery of an accessory anti-permeability vitamin in the natural sources. This has been called vitamin P (permeability), hesperidin, or citrin. Injections of vitamin P are used in hæmorrhagic diseases, especially purpura.

In heart failure it was found by W. EVANS that vitamin C produced an increase in urinary output in all cases, greater than in the case of digitalis, but less than Theobromine, Diuretin, and Ammon. Chloride. On account of this diuretic property of vitamin C it has been advised that in all cases of heart failure the maintenance of a proper concentration of vitamin C should be aimed at, by lemon and orange juice at least if not by medication.

In arsenobenzene intolerance it has been found that the administration of vitamin C in abundance, increases the tolerance to the arsenicals. It has been tried by actually mixing an ampoule of vitamin C to the solution for injection. But perhaps this reduces the efficacy of the arsenical while reducing its toxicity. Yet the addition of 0.05 to 0.1 gm. of ascorbic acid to an injection of arsphenamine has increased the tolerability of sensitive patients to larger doses while preventing skin reactions.

Experimental work in infectious diseases, by measuring the excretion of vitamin C in the urine, lead to the observation that in acute disease conditions there is often a marked drop in the amount excreted, showing that more vitamin C is needed by the body in such affection. Vitamin C therapy in such acute diseases as pneumonia, typhoid fever, etc., is therefore reasonable. The tendency to vitamin C retention occurs also in chronic infections and cachectic conditions.

Vitamin C has been discovered to be an essential factor in the manufacture of red cells. Hence the anæmia, in scurvy, appears before hæmorrhagic complications, and it at first is of the macrocytic type, and later after hæmorrhages the secondary type becomes superimposed.

Vitamin C was first named ascorbic acid. It was later also called cevitamic acid. The formula is $C_6H_8O_6$. PROFESSOR SZENT-GYORGYI first isolated Hexuronic Acid in 1928, and concluded that it was identical with the anti-scorbutic principle in vitamin C concentrates. Hexuronic acid is largely present in the suprarenal cortex. So the suprarenal cortex is the natural laboratory for developing the vitamin C resources of the organism. When sufficient supply is not received in food, great stress falls on this laboratory for supply of the deficiency and hence the adrenals are hypertrophied in such cases.

The properties of vitamin C are in short the following:—

- (1) It is essential for proper cellular metabolism and helps in tissue respiration.
- (2) It maintains the integrity of endothelial cells of blood capillaries.
- (3) It takes part in the manufacture of red cells from the bone marrow.
- (4) It has anti-infective properties, and acts as an antitoxic against arsenicals, and in certain allergic conditions.
- (5) Its associate in nature, vitamin P, is necessary for normal coagulability of the blood.
- (6) It favours appetite and promotes growth and maintains the normal tone of the body.

Besides in outspoken scurvy, vitamin C is now employed in the following conditions:—Pregnancy and lactation—beneficial both to mother and child; growth of infants—should be employed from the sixth month; all infectious diseases and acute illnesses; peptic ulcers; allergic states; surgical operations; bleeding tendencies and anæmias; alcoholism; cardiac insufficiency; intolerance to arsenicals and gold salts; stomatitis, gingivitis, etc. Whenever a state of sub-scorbutus is diagnosed vitamin C should be freely prescribed at least in food. NANSEN escaped scurvy because raw meat and fresh blood contain an abundance of vitamin C. Other natural potent sources of this vitamin are the following:—

Oranges, lemons, pineapples, cabbages, lettuce, green peas, spinach, tomato, swedish turnip, raspberries, etc.

Summary.—1. The history of Scurvy and the discovery of its cure is traced.

2. Signs and symptoms in the insidious primary stage both in adults and children are given, and it is hinted that with the help of modern discoveries about the properties of vitamin C, it may be possible to group together diseases under the title "asymptomatic scurvy" or "sub-scorbutus."

3. Extracts from modern research work with vitamin C are given as the following:—(a) Normal and abnormal concentrations in the blood. (b) Disease conditions in which the normal level is not maintained, such as,—gastric and duodenal ulcers, the neuroses of alcoholics, delay in wound-healing, etc. (c) The discovery of vitamin P as a specific anti-hæmorrhagic element. (d) Vitamin C as a diuretic in heart failure. (e) It reduces intolerance to arsenobenzene therapy. (f) Studies in the excretion of the vitamin in the urine lead to the discovery of its great use in infections, (acute and chronic), all acute diseases and cachectic conditions. (g) Its part in the manufacture of red cells from the bone marrow. (h) The elaboration of vitamin C in the form of hexuronic acid in the suprarenal cortex.

4. The chemical composition, action and diseases in which it is employed are detailed.

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Some Minor Disorders Due to Vitamin Deficiencies

BY

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NORMAL health shades off into abnormal health imperceptibly. The border-land instances of ill-health are often missed. Dietetic and nutritional defects are mostly of this variety. The patient may not complain and the doctor may not notice the defect. In times of famine and in times of war, these cases may come into relief and crowd the hospitals. During these times, if a health survey were to be conducted, infectious diseases mortality and the incidence of such diseases like tuberculosis would be found to have increased. To the general practitioner falls the duty of spotting out these incipient deficiency disorders and correct them in order to prevent any gross consequences supervening therefrom.

The deficiencies more often start from infancy. Infants and small children suffer from the effects of nutritional deficiencies sooner than adults. Subacute infections creep in as a result of lowered vitality. Vitality depends on the adequate supply of the basic elements of nutrition, proteins, carbohydrates and fats and the vitamins.

The potential capacity for assimilation and growth with which an infant comes into the world along with the immune bodies derived from the mother during lactation is responsible for the comparative freedom from infection, which an infant may enjoy. As long as a sufficient amount of the essentials of nutrition is supplied, the resistance to infection may continue. The capacity for assimilation and growth and adequate supply of nutrition are interdependent. For instance, a diminution in the protein intake, or in calories, may gradually arrest the growth. A nutritional deficiency in vitamins, especially A and B, can interfere with the proper absorption of nutrition and arrest the growth. Even though the child may appear to be in normal health, weight may not increase, and the general activity may become diminished.

Vitamin A is said to possess the property of resisting infection. The three chief channels of infection are the upper respiratory and oral passages, and the skin. A deficiency of vitamin A results in an unhealthy condition of these passages and the skin. Accumulation of epithelial debris takes place in these passages, the skin becomes vulnerable to infection. Vitamin A deficiency may be responsible for the incidence of scabies in the poorer classes who cannot be expected to get a sufficiency of vitamin A. Long continued vitamin A deficiency has been responsible for papular keratosis. When infective material gets access to the upper respiratory passages in a normal person it is quickly removed by the activity of the ciliated epithelium lining these passages and by the free passage of mucus there. Such a healthy elimination of infective material is prevented by the accumulation of mucus and debris. It is a recognised method of infecting small animals with pathogenic organisms for experimental purposes by an initial introduction of mucin into the respiratory passage or into the peritoneal cavity, along with or before the infecting organisms are put in. Mucin and mucus must be behaving in a similar way in allowing the organisms to multiply without being acted on by phagocytes, thus encouraging infection.

Mild grades of xerophthalmia are a common feature among the children of the poorer classes. Mild grades of night-blindness often may go unnoticed. Such instances may be present even among better classes sometimes.

Achlorhydria has been known to be sometimes associated with vitamin A deficiency. This may be due to a collection of mucus in the stomach. A similar condition of the intestines and the urinary passages may lead to inefficiency in the function of these organs, and infection may easily take place. Dysentery, diarrhoea and pyelitis may result. An adequate supply of vitamin A has been found according to McCARRISON to be capable of affording protection against stone formation.

Nasal catarrh in children has been found to improve and prevented by the adequate administration of vitamin A. A chronically low supply of this vitamin can maintain a chronic nasopharyngeal catarrh. It is not an improbable deduction from this that adenoids and tonsils, apart from the few hereditarily predisposed cases, are really the result of such chronic nasopharyngeal catarrh. Chronic discharge from the ear is often closely related to adenoids and tonsils. A chronic accumulation of cerumen in the ear can be the outcome of vitamin A deficiency and can later on lead to defective hearing.

The accumulation of epithelial debris and secretions in the sebaceous glands may be also the result of vitamin A deficiency. Such choked glands can be noticed over the nose where such glands are abundant. For a similar reason, acne can be benefited by the administration of vitamin A.

Defects of dentition start in very early infancy. Vitamin A deficiency affects dentition first. Delay in dentition, defective enamel formation, even the beginning of pyorrhoea may also be due to a similar defect.

Though severe scurvy is comparatively rare in India, mild cases are often allowed to go unnoticed. Slight pain or slight swelling or more pain in the growing ends, weakness in the limbs, and soreness of the gums may be due to a deficient vitamin C intake. Bleeding from the gums after fevers is the result of a general deficiency which occurs after fevers.

Vitamin D deficiency may not be expected in a country where the people suffer from too much sunlight. Surprisingly enough, varying degrees of vitamin D deficiency are not infrequently met with among infants and even adults in towns and cities in India. The causes may be found in the insufficient provision of ventilation in small tenement houses, insufficient provision of parks where mothers could give daily doses of sunlight to their children, and the dust-laden atmosphere which cuts off the beneficial rays of the sun. Cow's milk obtained in the city is notoriously deficient in vitamin D, due to the deficiency of open grazing grounds. If a careful examination of children during the first three years of life is made, a fairly large number of them will be found to have pigeon chest and other evidences of rickets. Instances of sleeplessness and restlessness in children can be traced to a deficiency of vitamin D. Carious teeth and dental abscesses in children are more frequent than one would expect and are the result of mild vitamin deficiencies. Cases of back pain in women have been traced to deficient vitamin D. Women clothe themselves heavily

excepting the labouring classes, especially those living in the cities and towns, are more often confined indoors during greater part of the day.

Vitamin B deficiency is not merely a disorder prevalent among the poor but is becoming apparent even among the richer classes on account of the adoption of civilised food habits. The use of refined sugar, lozenges and polished rice, have been responsible for a generalised vitamin B deficiency. Adequate supply of vitamin B is necessary to complete the digestion of carbohydrates. Absence of this vitamin can cause delay in digestion due to two causes, incomplete digestion and want of muscle tone of the gastric muscles. Thus many of the mild gastric disorders can be traced to this cause. In an attempt to find a suitable carbohydrate of an easily soluble kind the necessity for adequate supply of vitamin B is forgotten, and such foods as glucose, and dextrinised foods are given to children and infants. Digestion is thereby upset, gaseous distension of the abdomen with atonia may result.

Absence of vitamin B complex has been found to be responsible for various grades of anæmia, retardation of growth and want of general muscular tone. Along with those have to be included sub-clinical varieties of pellagra, mild neuritis, and conditions that predispose to such diseases like leprosy. The storage of vitamin B is very low in cases of diabetes. Symptoms of neuritis are common in diabetics as a result of this deficiency. Vitamin B has to be supplied as continuously as insulin and in large quantities. The benefit that diabetics derive from substituting ragi for rice is probably more due to the high vitamin B content of ragi than to the reduction in the quantity of carbohydrate taken. A similar reason can explain the relative advantage of wheat over rice for the diabetic, for there is not much to choose regarding starch between rice and wheat. Experiments in infant feeding have shown that the addition of an abundant supply of vitamin B to the food in the form of Brewer's yeast concentrate had enabled thin restless infants to grow up, to become happy, rosy cheeked babies with excellent appetite and with very good increase in weight. Growing infants have therefore to be supplied regularly with vitamin B along with orange juice and cod-liver oil.

The purpose of these notes has been to stimulate some interest in the study of vitamin in the causation of mild disorders which often go unnoticed, or which get ineffectually or symptomatically treated by the use of drugs. The timely warning in dietary may prevent the incidence of many of these ailments.



Syphilotherapy in Deficiency Diseases

BY

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THE deficiency diseases occur due to the lack in the diet of vitamins; this is of particular importance in the tropics because the tropical diet is not balanced, and specially so at the present moment, due to the war when prices of commodities have gone very high; this combined with mental strain a large number of deficiency diseases like scurvy, rickets, beri-beri, pellagra and the like develop in an increasing number than at other times; this is the experience of every clinician in every country. Vitamin deficient subjects react very badly to syphilotherapy.

I have found that the incidence of sub-clinical pellagra and arsenical intolerance due to sub-clinical scurvy (LAHIRI, 1941, 1942 and 1943) are seen in larger number of syphilitics with antisyphilitic treatment than formerly and is certainly due to vitamins Bpp and C deficiency in the diet of those patients.

I have shown (1942 and 1943) in a large number of syphilitics that they develop symptoms of intolerance to drugs when the blood vitamin C content is low, and those untoward symptoms can be avoided by the administration of vitamin C and liver extract. It has also been found that patients developing typical pellagrous skin conditions after the anti-syphilitic therapy show avitaminosis Bpp and the condition clears up by giving nicotinic acid.

The estimation of vitamin Bpp and vitamin C nutrition of patients is very important before giving antisyphilitic therapy. The close symbiosis in the liver of vitamin B and C have been found by many workers.

The estimation of vitamin Bpp is done by finding out porphyruria, the presence of which in urine will indicate vitamin Bpp deficiency. This is done by mixing 20 p.c. 50 c.c. sulphuric acid with 50 c.c. urine and examining the colour of urine after 12 hours.

If there is any deposit found or the colour of the solution is reddish brown the test is positive for porphyruria and shows deficiency of vitamin Bpp nutrition in the subject.

For the estimation of vitamin C nutrition the estimation of blood vitamin C is the best method and found by mixing 2.5 c.c. blood in 20 p.c. 25 c.c. trichloroacetic acid and filtering. The filtrate is titrated against 2:6 dichlorophenolindophenol at O_2° (LAHIRI and RUDRA, 1943).

When there is normal vitamin C nutrition the blood vitamin C content will be 0.6 mgm. per 100 c.c. blood (LAHIRI, '42). If the blood vitamin C content is found to contain below 0.6 mgm. there is vitamin C deficiency in the subject.

I have shown that when these vitamin deficient syphilitics are previously treated with vitamin C, liver extract and in certain cases with vitamin B complex, they develop no untoward symptoms with the anti-syphilitic treatment (LAHIRI, 1943).

Many patients who developed symptoms of dermatitis, jaundice and nitritoid crisis with syphilotherapy have been successfully treated with vitamin C before or with the syphilotherapy and fatal complications have been avoided, thus making the syphilotherapy available to a large number of people.

Summary.—Avoiding syphilotherapy in syphilitics showing symptoms of deficiency diseases is not wise and the continuance of syphilotherapy in such cases is of great import. The simple estimation of vitamin deficiency tests in doubtful cases before instituting the specific therapy should be a routine procedure to avoid untoward symptoms and ultimate disaster in some cases. Vitamin-liver therapy acts specifically in developed cases of intolerance which has been found in a large number of cases and have been reported.

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Deficiencies in Amoebiasis

BY

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IT is a well-known fact that Indians, particularly of the hospital class, live on the verge of starvation. It will therefore be easily surmised that deficiency conditions must be very common amongst them and so it is. Their height, weight, general build, colour, energy, efficiency all speak out in one voice about a universal deficiency present amongst them.

But I shall deal with only the peculiar deficiencies met with amongst them as a result of amoebic infection. One should however always assume the presence of multiple deficiencies and take it for granted that there is no parallel between human beings and the experimental condition amongst animals in every case.

Vitamin 'A'.—This substance is present in such materials as never or very rarely form any part of the dietary of the hospital class. Still the adult population amongst them generally do not show gross manifestations of this deficiency. We necessarily suppose that though they do not get an optimum quantity of the substance they somehow manage to get a fair proportion from the green leaves which they consume.

The carotene contained in these green leaves and vegetables, *Beta* carotene particularly, has been shown to be an effective substitute for vitamin 'A' of animal fats, and it has also been shown that the liver stores and metabolises carotene in such a way as to convert it to vitamin 'A' (MOORE). The liver, however, certainly requires some years before it becomes competent enough to deal with this material efficiently—this we gather from our clinical observations. Many facts are still unknown about the metabolism of this substance beginning from digestion absorption to conversion into the vitamin. Though gross manifestations of deficiency

of this vitamin is infrequent amongst adults suffering from amœbic infection, they are pretty common amongst children. I have noted that children living on the same diet in a family but not suffering from any bowel disorder, do not show any manifestation; whereas any bowel disorder, particularly amœbic dysentery, in a single individual of the same lot of children consuming the same diet precipitates manifestations of vitamin A deficiency.

I have also noted manifestations of vitamin A deficiency amongst adults who have become prematurely old as a result of some infections though they are consuming the diet shared by the other members of the same family who, however, have escaped these infections, particularly amœbic dysentery.

The age of the children who manifest vitamin A deficiency varies from 5 years to 16 years and most of them are males. The age amongst adults varied up to 68 amongst one and most of the adults are also males. The cause of this preponderance of males over females may be that the male organism requires more vitamin, or that they protect themselves less with leafy vegetables, or that they are more exposed to infections. COWARD has shown that male rats are more sensitive to vitamin A deficiency.

Besides the particular manifestations of vitamin A deficiency the children are generally under-developed for their age, show a pasty colour due to (sometimes severe) secondary anæmia, are loathe to any physical activity and it appears as if the very life itself has gone out of them. They are generally thin but a few are plump with protuberant bellies. Manifestations of rickets are not altogether uncommon amongst them though they cannot cover up their bare skin from the healthy action of the sun's rays with any vestige of cloth.

The adults are very much under-weight and they also show the same pasty unhealthy pallor and a constant tired and hungry look.

The first case that led me to suspect that amœbic infection has a great deal to do in bringing about deficiency manifestations was a boy aged about 12 who complained of night-blindness mainly. The other children of the same family, though younger ones were also present, did not show any manifestations of vitamin A deficiency though living on the same diet. This boy improved remarkably well on avoileum and eggs, green leaves and milk. But he relapsed soon after stopping avoileum though he continued the improved diet. So I had to think of some complicating factor. I learnt from him that he would occasionally suffer from diarrhoea, abdominal pain, flatulence and indigestion. His stool examination showed the presence of *Ent. Histolytica*. Emetine injections and the same improved diet without avoileum cured him quickly and he has not relapsed since.

I like to mention another case of a man aged about 65 who had lived well in his young days but has now recently fallen on bad days. He complained of severe night-blindness, xerostomia, diarrhoea and some pain in the region of both the parotids. No xerophthalmia was present but a dry lack-lustre appearance of his sunken eyes with Bitot's spots was prominent. As he could not afford avoileum in his present circumstances I advised him to take plenty of green leaves. This however did not improve his condition even after a few weeks. But his condition remarkably improved

when he could use avoileum after these weeks. Examination of his stool showed *Ent. Histolytica* and cysts and a large number of pus cells. This case easily would lead us to think about some difficulty in his or similar cases about the proper handling of carotene but none with vitamin A.

From these two cases we find that (1) there is a difficulty in absorption as because of the diarrhoea giving little time for absorption; (2) the quantity of vitamin A supplied through leafy vegetables, eggs and milk, though absent from the diet of other children of the family yet not suffering from vitamin A deficiency, did not suffice in a case suffering from amœbiasis to prevent a relapse of vitamin A deficiency; (3) leafy vegetables though sufficient to prevent any manifestation of vitamin A deficiency in a family could not prevent the appearance of night-blindness in a man suffering from amœbiasis; and (4) a highly concentrated product like avoileum of vitamin A was quickly curative.

We necessarily have got to presume that amœbiasis as an infection or as a condition producing diarrhoea brings about changes which require more vitamin A than is ordinarily necessary to prevent deficiency.

But we observe that the large majority of cases of amœbiasis does not show any vitamin A deficiency. These are generally adults. But when this infection occurs amongst children who live on a bare minimum of this accessory factor it precipitates manifestations of vitamin A deficiency.

It is well recognised that children suffer more from vitamin A deficiency than adults or infants. I could add that old people also suffer from this deficiency and that adults are not exceptions. It is also recognised that many conditions like infections and gastro-intestinal disorders require an extra allowance of vitamin A.

CLAUSEN finds that the vitamin A content of livers of children coming to autopsy were found to be low in severe infections, liver disease and dysentery when badly fed etc. He also finds that vitamin A is well absorbed when supplied as Halibut-liver oil than as carotene and that during diarrhoea even that is slight.

It would also seem highly probable that carotene though it supplies vitamin A to healthy organism cannot supply the proper quota even when plenty is supplied to children or old people when they suffer from amœbiasis producing both diarrhoea and liver disease.

We also observe that the infection and the vitamin A deficiency form a vicious circle one aggravating the other.

It is well known that vitamin A in the form of fish-liver oil cures sub-normal dark adaptation and even night-blindness very quickly even within a matter of hours. But such a happy result is not obtained with carotene when given orally. I did not try it parenterally as it is not available. If it is however found that parenteral administration does not help as quickly as fish-liver oil one can certainly surmise that the proper conversion of this substance into vitamin A cannot be done by the liver when it is diseased as in amœbiasis. CLAUSEN's finding regarding comparative absorption of vitamin A and carotene is corroborated by case No. 2. FRAZIER and LI have administered carotene 1 to 2 mg. intramuscularly in a patient showing vitamin A deficiency dermatoses. It took a long time for improvement to set in. I cannot say whether this case had any diarrhoea or amœbiasis.

It may be that different sources of carotene may show different degrees of activity regarding its absorption and conversion. About that I cannot say anything. I had administered the juice of grass freshly extracted to a night-blind child with very little effect who however reacted promptly to a single intra-muscular injection of cod-liver oil as such—which proves extremely painful and so unsuitable in most cases. This boy aged 5 was almost a complete atlas of deficiency manifestations of vitamin A and had so bad a type of diarrhoea that vitamin A as cod-liver oil could not be given by mouth; so I tried the intra-muscular injection which quickly helped him to see at night. Examination of his stool showed vegetative forms of *Ent. Histolytica*. His brother and sister do not show any manifestation of A deficiency, nor do they suffer from diarrhoea though they do not get any other richer diet than their unfortunate brother. Their mother though suckling a baby has no difficulty to see at night but their father suffering from diarrhoea proved to be amœbic has some difficulty with his vision at night. He seemed to have aged prematurely. Though he was only 45 he looked about 60.

I have also observed a few mothers suffering from amœbiasis show manifestations of night-blindness and xerophthalmia while suckling their babies, while mothers of similar age and suckling babies and living on the same standard of diet but not suffering from amœbiasis showed no manifestations of A deficiency. It may be that the female organism requires less of vitamin A but the stress of suckling and pregnancy should have made deficiency manifestations more common amongst them. But I do not find it to be so.

The manifestations of vitamin A deficiency are too well-known. I found the following as diagnostic:

- (1) The earliest one is a subjective symptom of diminution of the vision at night. This can be tested by the methods for determining dark adaptation on the part of the subject. I had not carried out any such test for want of proper instruments.
- (2) The dry finely-scaling skin which the subject feels to be too dry.
- (3) Particularly the phrynodermia or toad-skin as described by NICHOLLS and which is highly diagnostic. I have seen this, in a child, cover up the whole skin of his body except the face. I have noted this even amongst a few of the medical students.
- (4) Nyctalopia or night-blindness.
- (5) Xerophthalmia.
- (6) Bitot's spots. Advanced eye changes which are uncommon such as keratomalacia, blindness with the destruction of the globe may occasionally be seen in a few unfortunate. One will be simply astonished to note the appalling degree of incidence of these mild eye changes as noted above amongst children of the poorer classes when they suffer from bowel disorders specially amœbiasis.
- (7) Xerostomia, an unusual dryness of the mouth, is sometimes complained of.

Besides the above recognised forms I am of the opinion that:
(1) Diarrhoea is itself a sign of A deficiency as I have noted that in

children suffering from amœbiasis and A deficiency, a few intra-muscular injections of cod-liver oil cured not only the night-blindness but greatly improved and sometimes "cured" the diarrhoea though no measures were taken against either diarrhoea or amœbiasis; (2) some non-inflammatory hardness, tenderness and pain in the parotid glands; (3) though I did not find urinary lithiasis common amongst these patients yet certain symptoms like a pain in the loin passing toward the groin, some pain and burning sensation during micturition, the urine showing nothing unusual except some desquamated renal epithelial cells, which disappeared after cod-liver injection, lead me to believe that they are due to vitamin A deficiency; and (4) I believe that the nail shows certain changes which may be due to vitamin A deficiency but I have not as yet tested the effect of cod-liver oil on these nail changes so that I cannot state definitely this to be so.

The liability of these children suffering from A deficiency to catch infections of the lungs, skin and the kidney is very well-known.

About the pathology of vitamin A deficiency I only refer to the work of WOLBACH and HOWE. I mention this only to state that MELLANBY found change in the nervous system in experimental animals on an A deficient diet and excess of cereals—almost parallel to the condition obtaining in many poor children—and noted mental changes, ataxia, rigidity and weakness of extremities and even convulsions—symptoms which are pretty common due to vitamin B deficiency. I have never noted any of those in my cases though severe deficiency signs and symptoms were present in a good proportion. This leads me to doubt whether a parallel can be established between man and experimental animals. Wheat germ was however a great difference between the diet of his animals and that of my cases. Besides, it may be questioned seriously whether vitamin A deficiency leads in the child or man to any nervous system changes.

I can say that though intra-muscular injections of cod-liver oil were started by me with fear and doubt—fear as to the consequences of such an unwarranted and unorthodox procedure and doubt as to the absorbability of vitamin A when a local inflammatory reaction is always brought about—I am now in a position to say that it is comparatively harmless, and extremely speedy in action. The great difficulty is the pain it causes, so that children are very loathe to take it. When diarrhoea is really bad precluding the use of oil per mouth I found injections help not only the eye condition but also diarrhoea. Of course there cannot be any question about the effect of oil on the protozoa and which must be nil.

The skin conditions take much longer time to disappear but not certainly as long as taken by carotene injections (FRAZIER and LI).

Vitamin B Complex.—Vitamin B₁ deficiency is pretty common as a result of amœbic infection. This hypovitaminosis is common even under western conditions of diet and it is needless to say that it will be very much more common in India where most of the people are ignorant about dietetics, suffer from various infections and have to undergo hard labour for their living and whose staple diet consists of a huge amount of cheap carbohydrates, i.e., exactly those conditions which favour the development of a hypovitaminosis of B₁. For we know that the requirement of B₁ is proportional to the calories derived from carbohydrates and the amount

of exercise and infections, diarrhoeic conditions, liver disease, pregnancy, lactation, etc.

The vitamin B complex is getting more and more complex from day to day. Hypovitaminosis according to each different component of this complex is being worked out by various workers, notably SPIES, WILLIAMS, SETRELL, BUTLER, etc. If we simply remember that the components are interrelated with the intracellular system of enzymes of our organism it will be easy to understand that the whole complex, with special emphasis on some, is extremely necessary for the economics of our body processes.

We know about deficiency symptoms of thiamin (aneurin) riboflavine and nicotinic acid, and work has been done on pantothenic acid, pyridoxine, adermin, biotine, inositol and adenylic acid.

But we may easily believe that a single component deficiency is not at all common amongst us; so also a single vitamin deficiency. It may be stated categorically that it is always a multiple deficiency in any single case.

Vitamin B₁ deficiency has been induced in human beings by WILLIAMS and SPIES. Symptoms which developed were anorexia, fatigue, loss of weight, disturbance of gastric juice, constipation and intermittent tenderness of the calf muscles. Prolonged moderate deficiency as is common in most of my cases showed capricious appetite with nausea and vomiting, faint cardiac sounds and low blood pressure, giddiness on standing, disordered cardiac rhythm, achlorhydria and anæmia with later development of a depressed, irritable, quarrelsome, fearful condition. Insomnia was also common.

SPIES has shown that adermin (B₆) used for human nutrition can cure extreme nervousness, insomnia, irritability, abdominal pain, weakness and difficulty in walking. Deficiency of pyridoxine (B₆) brings about some scaly condition of the skin, a dermatitis of the groin and genitalia and about the eye lids. Pyridoxin has been used therapeutically in muscular rigidity, myasthenia and epilepsy.

The symptoms of beri-beri and pellagra are well-known but ariboflavinosis, as shown by SEBRELL and BUTLER and SPIES amongst human beings, manifests by Cheilosis, a 'shark skin' appearance over the nose, erosions around the eyes, and granulation and extreme redness of the lower lids, with itching, burning and photophobia of the eyes.

Pantothenic acid and adenylic acid are still not found to be very useful in human beings except as a synergist to nicotinic acid as like riboflavine.

The signs and symptoms which I have found in cases suffering from amœbiasis suggestive of B deficiency could not be placed under any single one component deficiency but comprise of a motley which could better be placed under the whole complex. They are as follows:—

(1) Anæmia is always present but only seldom is it very severe. The hyperchromic macrocytic type I have never seen.

(2) Loss of appetite.

(3) Loss of weight is certainly present but many seem to be flabby and hypothyroid. Personally I had lost within the course of one month about 10 lbs. of body weight when suffering from amœbiasis.

(4) Belly-aches, cramping pains in the stomach.

(5) General fatigue with particular stress on the leg muscles which ache, irritability of temper, insomnia, want of energy, etc., as described before, are very common combinations.

(6) Giddiness and neuro-circulatory asthenia are very common and even pain in the precordium may occasionally be noted.

(7) Blood pressure is frequently found low. Only a few cases show hypertension, but that is in advanced ages when essential or renal causes may independently bring this about.

(8) The first heart sound in the mitral area is weak and resembles more or less the 2nd sound and the intervals are so changed as to resemble a pendulum rhythm. This is not very frequent.

(9) The cardiac rhythm shows disturbances with frequent extrasystoles. The pulse rate is more frequently quick but a few cases show bradycardia.

(10) The knee-jerk and ankle-jerk are found highly exaggerated. I demonstrated one case before the students in which the ankle-jerk had almost become clonic and whose heart sounds were as described above.

In beri-beri or B₁ deficiency it is taught that jerks are lost. But WALSHE says "the tendon reflexes in the limbs diminish and disappear very early but there is often an initial stage of increase of short duration." This initial stage is probably what persists in these cases of amœbiasis producing chronic B deficiency.

I have seen only one case who had amœbiasis and peripheral neuritis affecting the lower extremities with all manifestations of the same.

I believe such an advanced condition is the exception rather than the rule in amœbiasis and exaggeration of deep reflexes is the rule.

(11) Calf muscles are generally painful in most cases.

(12) Cramps in muscles throughout the body and particularly with toes and fingers are not uncommon.

(13) Burning sensation and "pins and needles" in the lower extremities are very common.

(14) Cheilosis is not uncommon.

(15) The eye symptoms or ariboflavinosis are common and commonly supposed to be due to "liver." The erosions around the eye are rare but the granulations are frequent. The vision is sometimes found defective, which the subject appreciates.

(16) Certain neuralgias, e.g., rectal and intercostal are also occasionally found.

(17) Evidence of nicotinic acid deficiency as mental symptoms or symmetrical dermatitis, porphyrinuria etc.

(18) A smooth atrophic tongue which is found also to be fissured and occasionally pigmented at the borders.

(19) I have not done gastric analysis, but I am certain that hypochlorhydria will be pretty common.

(20) I have however found that patients suffering from amœbiasis show a deeper shade of colour like sun-burn on the dorsum of the palms and feet and particularly during the winter. No actual dermatitis except in one single case, did I see.

(21) Scrotal pruritus is very frequent. Whether there was or was not any dermatitis as in pellagra I did not see for reasons of delicacy. One of my patients J.P., aged 30, stated the curious coincidence of increased scrotal pruritus (which was sometimes embarrassing for him) with increased bowel disorder particularly a flatulence in the right side of his belly. I wonder whether there is any relation of segmental representation in the spinal cord between the cæcum and the scrotum.

(22) *Oedema*:—This may be general anasarca or limited to the legs. The kidney is not involved as there is no albuminuria or cylindruria in these cases. The actual pathology of œdema in these cases—nutritional œdema—is much too complex to enter into. I can only refer to the articles on MEHLNÄHRSCADEN and KWASHIORKOR in Vol. IV of British Encyclopædia of Medical Practice. The result of treatment is sometimes anything but flattering.

I have detailed the symptoms of these cases suffering from amœbiasis and it will be easily appreciated how very strong a combination they form to make one certainly believe that they are due to vitamin B deficiency.

I have, however, carried out Meyer's test in some cases of amœbiasis proved by stool examination. This test was found positive in all these cases. The test is carried out by giving an injection of about 5 to 7 m. of adrenalin and then auscultating over the cubital fossa when a continuous hum is heard. I have found that auscultation over the femoral artery is better in this respect. The hum was noted in every case over the femoral artery. I have demonstrated this to students. If Meyer's test is diagnostic of B₁ deficiency it was certainly present in my patients with exaggerated knee-jerks and ankle-jerks.

Volhard's diuresis test, though said to be a better one, is not suitable for outdoor work and so I could not carry it out.

On X-ray examination of the gastro-intestinal tract "deficiency pattern" or a flat glucose curve on blood sugar estimation indicating defective absorption, may also be detected. The electro-cardiogram may show changes as described by SOMA WEISS. Other chemical tests may be carried out.

Others: Though there is the probability of a C or K deficiency I did not pay particular attention to detect them. I found various bony changes like rickety rosary, Harrison's Sulcus etc., of D deficiency in some of the poor children but they are not common. Dental caries—amœbiasis or no amœbiasis—is very frequent amongst all and sundry but I believe that amœbiasis may have some influence on its incidence.

Treatment is certainly not wholly satisfactory as in most cases of amœbiasis there is a relapse. But it is always advisable to administer some vitamin B with the treatment for amœbiasis like yeast (Brewer's) or marmite.

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Vitamins and the Eye

BY

S. SANYAL, M.B.,

H. O. R. Institute, Calcutta

VITAMINS are micro-constituents of diet and are necessary for the growth and maintenance of the normal health of the individual. They may be regarded as exogenous hormones which the body takes from outside: of the vitamins, A, D, E, are fat soluble, others are water soluble: Excepting E all of them are essential for the eye. If any of them is absent from the food, it may produce characteristic symptoms which clear up rapidly under the particular vitamin therapy.

Vitamin A.—Sometimes called the "Ophthalmic" vitamin—because of its importance in ocular economics. It is elaborated from carotinoids, which are fat soluble hydro-carbon pigments widely distributed in plants, and animals—specially in their liver. It is an alcohol derived from semi-B-carotene. It is liable to be inactivated by the vitamins of the B group and vitamin C, and should therefore not be administered together.

Functions.—In general, it is necessary for the normal growth of young animals and maintenance of the normal condition in adults, tissues of ectodermal origin. In the eye, its deficiency or absence produces chiefly the well recognised condition of xerosis of the cornea and the conjunctiva, nyctalopia or night-blindness, some congenital anomalies and degeneration of lachrymal and tarsal glands and cataract.

Xerosis:—The chief change that this avitaminosis causes is one of metaplasia and consists of conversion of columnar epithelium to stratified epithelium; clinically, the initial symptom is a lack of lustre distributed irregularly as small patchy areas. This lack-lustre appearance is easily detected if the eye is kept exposed to air and light for a few seconds. In the affected areas, the blood vessels, as they course through them, take on a peculiarly blush appearance. Associated with this, there is wrinkling of the conjunctiva, specially marked in the palpebral region, due to loss of elasticity of the conjunctiva. The top of the folds are dry and moistureless, furrows are moist and are in a direction concentric with the limbus.

Appearance of Bitot's Spots:—These are small white circumscribed spots with a foamy top not moistened by tears. They are at first oval, then triangular in shape, and are situated in the palpebral apperture with base at the limbus. The foamy deposit is easily removed and contains xerosis bacillus.

Sometimes, a peculiarly yellow colouration of the whole conjunctiva may be seen, with deeper brownish black areas of pigmentation, scattered throughout. The pigment is melanin. In the pigmented areas the melanoblasts with dendritic processes are generally found in the basal cell layers. In severe cases, the conjunctiva bulbi becomes thick, dark, epidermoid, leathery and has a peculiar waxy appearance. The most affected part is the area round the cornea, next fornix; tarsal conjunctiva takes little or no part.

In the Cornea:—The earliest changes are: (1) Loss of lustre, (2) dryness when exposed to air, and (3) a reduced sensibility over the whole cornea. When the condition has fully developed Bitot's spots appear in the palpebral areas either as a prolongation of the latter, running concentric with

the limbus like arcus senilis or occurring in isolated areas in the centre of the cornea. If true kerato-malacia develops, cornea becomes dull, infiltrates appear, sometimes like dots in the cornea. Vascularisation of the cornea occurs from all sides. There is exfoliation of the epithelium with formation of hypopyon ulcer, perforation and pan-ophthalmitis. From beginning to end, there is practically no inflammatory symptom. Clinically even in the earliest cases under corneal microscope corneal nerves may be seen to be thickened (PILLAT, 1929).

Pathological changes that occur in a case of xerosis may be described as under: (1) pre-xerosis stage, (2) fully developed stage. In the pre-xerotic stage, according to KREIKER, (1) disappearance of the goblet cells, and (2) hyaline degeneration of the protoplasm of the epithelial cells occur. In the fully developed stage we notice (1) thickening of the whole epithelium, (2) flattening of the superficial cells with disappearance of their nuclei, (3) appearance of prickly cells in the deeper layers, interspersed with leucocytic collections, (4) loss of regular arrangement in the superficial layers which become wavy and irregular bands, (5) the outermost layers lose their cellular appearance giving the straining reaction of kerato-hyaline, (6) the cells immediately beneath them contain granules of the same material, thus resembling the stratum granulosum of epidermis, and (7) fatty globules stained deep black by osmic acid are common in the superficial and deeper layers. The superficial cells are being constantly cast off and contain xerosis bacillus. Section through Bitot's spots show the same characteristics, but here fatty infiltration is not particularly in evidence. The foam-like appearance is due essentially to vesicular formation and irregularities of the superficial layers of the keratinised epithelium upon the deeper layers.

The intimate mechanism whereby the fat soluble deficiency affects the epithelium of the conjunctiva and the cornea is not exactly known. Various suggestions have been made from time to time *e.g.*, (1) dessication following loss of lacrymal secretion, (2) loss of lysozyme in tears, (3) it is neurotrophic, as with development of xerophthalmia, there is synchronous occurrence of changes in the myelin sheath of the trigeminal nerve in experimental animals. If the epithelial condition can be brought back to normal, the nerve also assumes a normal appearance. In severe cases Wallerian degeneration and degeneration of the cells of the Gasserian ganglion occurs. CIRIMICIONE (1891) found cells of the ciliary and Gasserian ganglion degenerated, at an autopsy in these conditions.

Ætiology.—In mildest form, it generally occurs in children, specially boys associated with night-blindness. The children need not be particularly ill-nourished. It occurs during the summer months. After 10th year of life the gross eye changes are usually rare. They suffer from some debilitating diseases especially affecting the intestinal tract (HILDRETH, 1870). In this connection the experiments of SHERMAN and BURTIS (1928) are interesting. They found that if rats were fed on a diet deficient in vitamin A temporarily in youth and subsequently fed adequately, autopsy at a later stage showed an infection incidence of 75% as against 25% in controls. Other factors in the ætiology, specially in the East are, alcoholism, hepatic affections, parasitic diseases, malaria, intestinal disorders, etc., to a small percentage. It may also occur in epidemic form in conditions of mal-nutrition specially in children in the tropics. Some

evidence has been brought forward to show the effect of strong light in the ætiology of this condition.

In addition to frank cases of xerophthalmia, it is quite possible that partial deficiency over a long period of time may produce milder grades of symptoms, and persistent blepharo-kerato-conjunctivitis in children remind one of such a possibility.

Treatment.—This resolves itself into the treatment of the eye and general treatment. Locally (1) application of cold to the eye. If the eye condition warrants, powdered ice kept between two pieces of lint may be applied over the closed eye-lids for a minute or two. This should be repeated 6 times a day, specially between 3 to 6 p.m. It should be given every three hours beginning from 9 a.m. In severe cases alternately, several pieces of lint soaked in cold water, may be placed over the closed eye-lids for about 5 minutes every three hours. Sterilised liquid paraffin or cod-liver oil or Haliverol may be dropped into the eyes 3 or 4 times a day. If the case is not too severe a local Sulphanilamide ointment (4%) may be used along with it because of its stimulating effect on the ectodermal cells (SANYAL, 1942). General measures consist in: (1) keeping the patient in darkness, (2) ice to the head or a lint soaked in ice-cold rice water to the forehead. Frequent washing of the head with cold water and cold-mud poultice to the abdomen. To stimulate through the nasal mucous membrane the sympathetic system, scented oils should be put on the head. If the patient is diarrhœic, a watery emulsion of cod-liver oil, or cod-liver oil washed in several changes of ice-cold water may be rubbed over the abdomen, groin and axillæ. Orally vitamins A & D together with calcium should be given. If necessary these may be injected. Sometimes vitamin B is necessary to help the system and may have to be injected. In addition, undermentioned powder may be necessary:

Pulv. Ipecac	... gr. 1/12.
Hydrag Cum creta	... gr. ½.
Ephedrine Sulph	... gr. 1/12.
Atropine Sulph	... gr. 1/800.
Sulphanilamide	... gr. 1/12.
Calcium Lactus	... gr. ii.
Aspro	... gr. ½.

Mft. Pulv. one for a child one year old. This should be given before each feed after sponging the head, arms and the legs with cold water.

The diet should consist of orange juice, whey, if tolerated, fresh goat's milk, barley-water, decoction of roasted green mangoes; decoction of tamarind stored for several years may also be given. The treatment outlined above is for moderately severe cases. In milder cases avoidance of sun, Sulphanilamide ointment to the eyes, vitamin A along with emetine may be injected. Frequent baths are beneficial. Foods containing carotins are necessary, such as carrots, oranges, green fresh vegetables. Animal diet should be avoided. Bowels must be kept free. "Charnosol" a proprietary medicine (S.O.C.W., Calcutta) is useful.

Night-Blindness.—It is a condition in which vision in feeble, illumination is defective. It may occur in various conditions of the eye and is generally classified into six varieties:

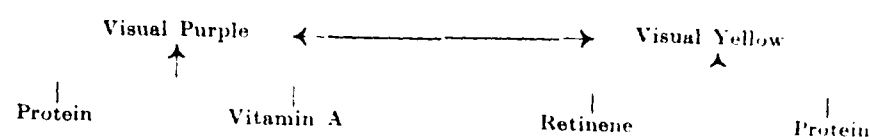
- (1) As a symptom of various eye-diseases.

- (2) As a congenital and hereditary condition.
- (3) Diseases of the liver.
- (4) Over-exposure to light.
- (5) Where pathological changes and nutritional disturbances are absent.
- (6) As a result of vitamin A deficiency.

It is with the last that we are here concerned.

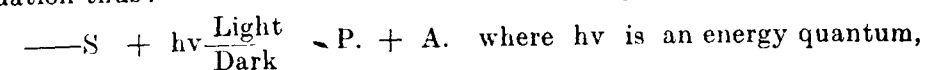
Symptoms:—The main symptom is a defect in dark adapted eye. It takes several forms (a) light, minimum, may be normal at first, but there is very little or no increase in sensitivity with the progress of dark-adaptation; (b) the initial light threshold may be high and subsequent adaptation may be subnormal or absent; (c) least disturbance occurs with red light, but the long waved lights may be affected to a greater extent (Best); (d) light difference is also affected, (e) in change from photopic to scotopic vision, as the illumination is diminished, the red becomes darker and blue becomes relatively brighter. This is the normal Purkinjee's phenomenon, when the illumination is increased (about 0.25 metre-candle), a reversed Purkinjee's effect occurs, reds becoming brighter and blue relatively darker. In night-blindness this phenomenon develops slowly or not at all; or the reversed Purkinjee's phenomenon may be observed or the Purkinjee's after-image may not be seen. The photo-chromatic interval, i.e., the interval between recognition of light and recognition of colour during which the objects appear colourless may be shortened or absent.

(7) Some degree of blue blindness may occur. The whole clinical picture suggests either a defect in the red or functional deficiency of visual purple whereby there is an impairment or absence of scotopic mechanism. The permanent forms are those where the reds are destroyed, and the temporary groups include a deficiency of this pigment, as in long continued exposure to strong lights or defects in the liver precluding the elaboration of vitamin A. That vitamin A plays an important role is substantiated by the fact that in albino-rats fed on a diet deficient in vitamin A, a delay in the regeneration of this pigment occurs. The intimate relation is shown by WALD, according to whom the light acting on the visual purple, changes it to visual yellow which is subsequently converted into a protein and a carotinoid retinene. This retinene is changed to vitamin A which recombines with protein to regenerate rhodopsin. This cycle, according to WALD (1934) is as follows:—



This regeneration is independent of central nervous control (STATGARD 1915). The process is, however, greatly retarded by a deficiency of vitamin A and does not occur at all if the rods are separated from pigment epithelium. It is interesting to note that visual purple has rarely been observed in human beings, proof of its presence is indirect. It is said to have been observed entoptically in the human eyes. The primary chemical action of the light on the retinal cells is the production of acid and the degree of acidity is greatest with yellow green, the part of the spectrum

which appears brightest. This acidity destroys the visual yellow and is probably a factor in dark adaptation. The energy quantum for any light is 2×10^{-12} ergs., the limit of retinal sensibility. The intimate nature of the photo-chemical reaction is one involving the splitting up of a single molecule into two components while the rate of reformation follows the kinetic of a bimolecular reaction. The reaction may be expressed in an equation thus:—



P and A decomposition products of the photo sensitive substances. It is interesting to note that visual purple has never been demonstrated in cones, although similarity of photo-chemical reactions suggest that probably they are present in such dilute solutions as to be invisible. The assumption of the presence of dilute solutions of the pigment receives a strong support from the researches of WEIGENT (1921) who showed the extreme photo chemical properties of very thin layers of dilute solution dyes. But it must also be remembered, that as the chemical composition of rods and cones differ (LA CASCIO, 1923) some other photo-chemical substance other than visual purple may be present in these cases (FRANCOIS and PIERRON, 1924). Since the chemical changes seen in the rods are of the same nature as those occurring in the corneal cells, it is not clear why nature has put in a pigment for the intimation of the process. That the bleaching is intimately connected with the photo-chemical reaction, there is no doubt, but why in a tissue which is chiefly dealing with the spectral colour, a non-spectral pigment has been put in a cell (for purple is a combination of red and violet and is non-spectral) it is not known.

It is to be remembered that the primary functions of the cones is to send the message of arrival of light to the brain in the shortest period of time and to have acuity of vision and that there is a certain minimum intensity of light below which vision is not possible, that is, a certain minimum amount of energy stimulus is necessary to evoke vision, energy quantum rising or falling according to the requirements of vision. For the first purpose a coloured light (yellow) which has the lowest latent period and the highest capacity for initiating the photo-chemical change has been chosen by nature, and each cone is separately connected to a nerve fibre, so that in the shortest possible time, maximum energy may be transmitted to the brain.

Apparently for the second purpose the sum total of energy obtained in the cases, is either not sufficient under ordinary circumstances to give sufficient stimulus for acuity of vision or its continuity for any length of time is not possible. So a yellow pigment has been put in the macular area, permeating all the cerebral layers with a capacity to absorb yellow colour, and transmit the energy through the numerous inter-connections to the proper nerve channel. In point of time it comes next to cones. But a greater area, from which sufficient energy may be drawn, lies outside the macular area, and as the yellow light has the highest capacity for initiating energy and as the yellow pigment has the highest capacity for absorption of yellow colour, the visual purple is changed to yellow, and having served its purpose, may be changed to a colourless product; and this conversion requires a greater time than in the macular region. Thus a photo-chemical process initiated in the cones is successively helped by the macular pigment and rod pigment.

In the scotopic eye, the same method of functioning of the retina may be observed, the difference being quantitative, not qualitative. Cones having the lowest threshold for colours even in the dark adapted eyes, transmits the message of light as quickly and in as short a time as in the photopic eye. In point of time, probably the rod comes next, this being due to lowest latent period for green light and as visual purple is complementary to green light, very minimum time and energy are necessary for the change of visual purple to a colourless product, thus allowing the rods with minimum injury to act like a colourless cell similar to cones, so long as scotopic vision continues, so that light may act directly on the cell protoplasm without the necessity of intervention of a pigment, thus obviating the loss of energy of a very dim illumination.

It may be because of the numerous inter-connections in the cerebral layers, the little time lost by the yellow pigment in changing light energy into nerve energy, is more than compensated by the arborisations in the cerebral layers and the order of sending impulse is the same as in photopic vision.

The mechanism of initiating vision seems thus to be the same, both for the photopic and scotopic vision, and this is further supported by the luminosity curve in which the only change observed is that the curve is shifted from yellow to green. But all these are suppositions and require confirmation by experiments.

It is interesting in this connection to observe that we are only aware of a unidirectional pathway from the retina to the eye, vision being regarded as a psychological phenomenon. Whether the stimulation by light of the cortical area liberates any energy as a reflex phenomenon and if it does what is its pathway and what is its function, we have no knowledge; even admitting the actual seeing to be a function of some higher force, in the physical plane, to every action, there should be a reaction. Some simple experiments seem to indicate that such is the case, but further investigations are necessary before one is sure, and, if possible, will be the subject matter of another paper later on.

Treatment of this condition lies in giving sufficient vitamin A to the patients both in foods and in concentrated form in drugs and if necessary it should be given by injection. Avoidance of sun and strong light, administration of sympathetic tonic drugs like ephedrine to make firm contact with the pigment epithelium and allow "fusin corpuscles to migrate into the nuclei of epithelial cells (YAMANKA, 1924). Because so many factors contribute to night-blindness it would be useful if vitamin A deficiency were measured by some such instruments as devised by N. G. DEY and N. K. BOSE or G. SANKARAN.

Other diseases due to deficiency of the vitamin include:

(1) Superficial keratitis—STOCKER considers that superficial keratitis is improved by vitamin A.

(2) Kerato conus has been experimentally produced in rats (MUCH and RICHARD).

(3) Anterior and posterior chamber affection leading to gradual blindness (WRIGHT).

(4) Development of cataract has been observed by PILLAT (1929) clinically, and V. SZILY and ECKSTEIN produced cataract in suckling rats by depriving the mother of vitamin A.*

(5) Degeneration of the retina occurs, Ganglion cells and bipolar cells being specially affected (STOCKER, 1936).

(6) Retro-bulbar neuritis—pallor and blurring of the edge of the optic disc (WRIGHT, 1936).

(7) Congenital defects—In pigs a deficiency of this vitamin may result in an ophthalmos. The author observed complete blindness in a newborn child whose mother suffered from epidemic dropsy of moderately severe type during the last 5 months of pregnancy. This was probably due to exhaustion of vitamin A reserve which may occur in pregnancy and defective absorption due to epidemic dropsy. Clinically the iris was inactive, there was generalised congestion of retina and slight veiling of the disc. Blindness continued for a month before treatment was instituted. It improved under vitamin A after a month and the sight was restored.

(8) Hordeolum and meibomitis have been observed by SEN, and BAHADURI considers recurrence of Morax-Axenfeld can be prevented by vitamin A.

Vitamin B Complex.—Vitamin B has been divided into (1) anti-neuritic, thermolabile B₁ and (2) thermostabile growth factors—B₂, B₆, B₁₂, B₁₅, B₁₆. (1) Vitamin B₁, also called aneurin or thiamine, is a crystalline powder and is a thiazol derivative of 2-methyl-4-aminopyrimidine and occurs naturally in the form of a chloride. It is stable in acid or neutral solutions. Oxidation, yields a yellow pigment with a blue fluorescence, thiochrome. Rapidly inactivated by alkalis. An acidified 50% mixture of water and alcohol is the most effective solution for its extraction. Thiamine pyrophosphate acts a co-enzyme in carbohydrate metabolism in nervous and other tissues and is necessary for the degradation of pyruvic acid, the oxidation derivative of lactic acid.

Effects of avitaminosis B₁ (1) beri-beri—a polyneuritis characterised by loss of muscular co-ordination, gradual paralysis of limbs, alimentary disturbances, degeneration of heart muscle, general emaciation and dropsy. Nerve degeneration is claimed by ENGEL and PHILLIPS to be due to or aggravated by a deficiency of flavin or vitamin A; (2) gastro-intestinal functions impaired appetite, decreased motility of stomach, loss of tonus, sub-normal temperature, hydræmia, decreased resistance and fatigue; (3) pyruvate accumulation—the pyruvate content of blood, urine and cerebro-spinal fluid rises; (4) avian polyneuritis; and (5) brady-cardia in rats.

In the eye, the following complications may be noted: (1) Retro-bulbar neuritis, corneal and conjunctival dystrophy. Paresis of ocular muscles associated with beri-beri (KNUSE and McCOLLUM 1932).

Vitamin B₂.—It consists of two portions, one an antidermatitic water-soluble growth factor, the riboflavin, and the second B₆ or Y factor. It is a member of lyochromes.

Functions:—Riboflavin phosphate when combined with protein takes part in dehydrogenation of glucose, lactic acid and other metabolites. It is concerned both in amino acid deamination and in urea formation. Liver and kidney are very rich in riboflavin. The human requirements for normal health is about 40 to 50 of riboflavin per 100 kilo calories of food and is proportional to food intake. It is probably required for the growth and maintenance of every animal tissue.

In the Eye—it is present in the normal lens. The fluorescence of normal lens is claimed by THIEL (1926) to be due to riboflavine. With development of cataract it gradually diminishes, till it disappears altogether in cataract. Some authorities failed to associate this fluorescence with riboflavine. Experimentally in young rats, cataracts of various types have been produced by various observers in a diet deficient in riboflavine, while others have not obtained an uniformity of results. Probably the development of cataract in rats is a local process. Probably, it acts as a cataract-preventing factor by keeping up the normal oxidative metabolism of the lens. But evidence is not sufficient to attribute avitaminosis a primary aetiological role in the formation of cataract. The essential factor involved is unidentified, therefore uncontrolled.

Riboflavine deficiency has been claimed to produce the following diseases in the eye:

(1) Kerato-conjunctivitis with development of opacities in the cornea, alopecia and in some cases of cataract. (SHERMAN and SANDALS and others, 1931).

(2) Diffuse superficial keratitis in Japan (epithelial). It is a bilateral affection characterised by minute grey epithelial flakes covering the entire cornea and accompanied by slight ciliary injection and photophobia. It may continue for years. There is frequently a slight Uveitis. TAKEDA, considers it to be due to a deficiency of vitamin B₂.

(3) Kerato-conjunctivitis with development of opacities, alopecia round the eye-lids, opacities in the vitreous, inflammation of the lachrymal gland.

(4) Vascularisation in layers of cornea (WODRACH and BESSEY 1937).

(5) As complications in pellagra; night-blindness, amblyopia, optic atrophy, ptosis asthenopia.

(6) DAY, LANGSTON and O'BRIEN (1931) obtained cataract in 100% of cases in rats fed on a diet deficient in B₂. The diet must start on the weaning of the animals, in order that the deficiency may be complete (YUDKIN 1933, O'BRIEN, 1932).

Vitamin C.—This is ascorbic acid. It is a colourless compound. The melting point is 192. It is very soluble in water producing strong reducing properties. It is the most unstable of all vitamins and readily undergoes reversible oxidation, followed by hydrolytic destruction, specially in warm alkaline solutions. It can probably act as hydrogen transport agent in cell metabolism and is probably concerned with mechanism for synthesising carotinoids in plants. General symptoms of avitaminosis—include:

(1) Infantile scurvy.

(2) Adult scurvy, many of the symptoms of scurvy are attributable to increased permeability of blood vessels.

(3) Dental disorders—It appears to be necessary for the activity of odontoblasts, ameloblasts and osteoblasts and pyorrhœa, spongy gums and caries have been cured by it.

(4) Rheumatism:—Symptoms of subacute prolonged cevitamic deficiency are similar to rheumatic states and it has been suggested that this deficiency is a factor in this disease. It is not specific.

(5) *In the eye*:—Cevitamic acid is present in the lens in concentrations that is about 10 to 20 times that of the blood plasma, its value being about 30 mg. and is more abundant in cortex than the nucleus. In old age it is diminished and in cataract it falls considerably. In the aqueous also it is in higher concentrations than the cerebro-spinal fluid or blood plasma but the value is considerably lowered in aphakic eye. In cataractous and aphakic eyes it occurs in reversible oxidised form. Its presence in such large concentrations is supposed to depend on an actively functioning lens and that it is manufactured from dextrose whence it diffuses through capsule into the aqueous according to the equation—dextrose → oxidised glutathione → cevitamic acid → reduced glutathione; others consider that as the concentration of oxidised cevitamic acid is generally equal to the concentration in the blood, it is probably dialysed from blood in the eye, where it becomes converted by dehydrogenation into the reduced form, so that lens act as a sort of preservative for this vitamin.

As regards its importance in the formation of cataract, it is suggested that it is caused by disturbance of blood-aqueous barrier in the background of a vitamin insufficiency. But, at present, it is difficult to say cevitamic deficiency is the cause of cataract.

Although even in extreme cases of scurvy, either clinically or experimentally, no cataract is usually found, MONYUKOVA and FRANKIN (1934-35) obtained consistent changes in lens when aqueous was depleted of cevitamic acid by paracentesis. Other ocular complications that have been attributed to vitamin C include (1) a kerato-conjunctivitis associated with hæmorrhage in the lids, deficiency of lacrymation and a tendency to ulceration, (2) hæmorrhage in the eye-lids, conjunctiva and retina, (3) proptosis—outwards and downwards, (4) hæmorrhages in the orbit, (5) thickening and pigmentation of conjunctiva, (6) discolouration and pigmentation of the skin of the lids, and (7) night-blindness (DUGGAN).

Vitamin D.—This is the antirachitic vitamin. It is formed by the action of ultra-violet rays on sterols and is usually associated with vitamin A in nature. It is present in the leafy parts of most plants, but is absent from vegetable oil and occurs freely in liver oils and animal fats. The pro-vitamin ergosterol occurs chiefly in yeast and higher fungi. Synthetically, the vitamin may be obtained from ergosterol by irradiation with ultra-violet rays and is known as vitamin D₂ or calciferol. There are two other vitamins D, one is vitamin D₃ obtained from irradiation or chemical activation of dehydrocholesterol. It occurs in colourless crystals m. p. 114.5 to 117°C. The solutions show an intense absorption band at 26 m.μ. It gives none of the carotinoid reactions. Other is D₄.

Effects of Vitamin D:—It is a calcio-kinetic vitamin, i.e., calcium distributing vitamin regulating the absolute and relative absorption of calcium and phosphate solutes from the intestinal tract. Deficiency in this vitamin produces (1) subnormal phosphate and calcium level in the plasma, (2) decrease in CAXPO₁ product in plasma, (3) decrease of excretion of phosphate and calcium by the kidney, (4) increased excretion of phosphate and calcium by the intestines. Pathological conditions associated with vitamin D deficiency are:—(1) rickets, (2) osteomalacia (3) dental caries. The decalcifying process in the bones is imperfectly understood, a decalcifier, termed calciovium, has been found in oat meal and it is suggested that excessive consumption of cereals should be prohibited in these conditions.

In the eye the exact role of calcium and this vitamin is not known. This vitamin and parathyroid are the two regulators of calcium metabolism. It has been observed in growing children, that if this vitamin is lacking, rickets, tetany and zonular cataract develop. If parathyroid is injected there is a rise of serum calcium which is drawn from the skeletal tissues and if this rise is allowed to go on till it is double the normal amount death may occur. In parathyroidectomised animals a drop in serum calcium is associated with development of opacities in the lens and there occurs a hyperexcitability of nervous tissues. Embryo lens fibres growing in vitro are poisoned by a slight reduction in calcium content of the culture media but were unaffected by increase (KIRBY, 1931). Clinically, it has been associated with development of myopia, conical cornea, blue sclerotics, and disturbances of the supporting tissues without any strong evidence.

Deposition of large amount of calcium in cataractous lens led to investigation of serum calcium with the idea that hypercalcemia might be the factor in the production of cataract, but as the serum calcium generally remains normal throughout life and patients are in perfect balance, its association with cataract is difficult to assay, although occasionally such conditions have been reported (ADAMS, 1929). Nor does its deficiency lead necessarily to cataract, except in cases of dysfunction of parathyroid; although it must be admitted that deficiency may lead to changes in the normal electro-chemical condition with consequent disturbances in the labile colloid system of the lens protein. One thing must, however, be remembered that it is very difficult to say what is normal for a particular individual, and while the metabolism of calcium may be within normal limits, there may be actually a slight deficiency. The serum calcium level at the expense of the skeletal tissues, and the slight changes in the bones are not easy to detect even by X-rays. As the experiments of KIRBY show that slight calcium deficiencies may be toxic, it is possible that such deficiencies as stated above may lead to cataract by increasing the permeability of the subnormally functioning lens fibre. Further, the irritability of the nervous system produced by its deficiency may indirectly lead to interference with the normal metabolism of the lens with the result that a cataract forms. As vitamin D is one of the regulators of the absorption, excretion and metabolism of calcium, avitaminosis D may be a factor in the causation of cataract. Besides this, hyper-irritability may lead to defective absorption of other vitamins which have some influence in cataract formation.

Vitamin K.—This is the anti-hæmorrhagic vitamin. It is a fat-soluble, alkali-labile, thermostable vitamin. It is an yellow oil soluble in fat solvents. The lipid fraction of green vegetables is the principal source. Its deficiency leads to reduction of prothrombin content of the plasma. Two forms are known K₁ from alfalfa K₂. It does not influence hæmophilia.

In eye, its deficiency may lead to recurrent intra-ocular hæmorrhage.

Vitamin E.—This is a factor in human subjects in habitual abortion, sterility in male and female and excessive post-partum hæmorrhage. It is without any ophthalmic interest.

Vitamin F.—These are the unsaturated acids necessary for growth, reproduction and lactation in rats. Its deficiency in human beings is not yet known.

Comments.—The vitamins are something like endocrine glands and in a general way are sympathetic stimulants with special function attributed to each. They are the endocrine glands of the plants concerned with its cellular metabolism in all its stages. With food, if properly taken, they are transported to the animal cells where they help the metabolic activity. Difference from animal endocrine glands are that while the glands are situated in particular situations from which secretions permeate the cells of the whole body, these come in a ready made form and at once enter each cell and begin their action, of course with local depots in places where it is most needed. For example vitamin A in liver to protect it against the large amount of toxic material, which it has got to deal; vitamin B in kidney and liver and so on. In plants these are manufactured and therefore there is no deficiency there; in human beings, supply comes from outside and therefore the term deficiency has to be coined. Nature has so ordained, that with food, the means of its proper utilisation is also supplied at the same time, helping the purpose of food in growth and nutrition.

Vitamins may be divided into :—(1) Ectodermal, e.g., vitamin A and B by their action on nerve which is also ectodermal. (2) Mesodermal, vitamins D, and K, C, E, and F.

All these are sympathetico-tonic, the most active of which produces symptoms characteristic of vagus stimulation, e.g., vitamin B₁.

Hence the symptoms of deficiency may be produced in conditions where loss of sympathetic tone occurs, for example prolonged exposure to heat, sunlight, cold; in prolonged mental worries, stress and strain. So in labelling a disease as due to a particular deficiency, all these factors must be taken into consideration.

Even in cases where a particular vitamin is biologically or bio-chemically found to be deficient, it is necessary to judge, whether the clinical picture is a primary avitaminosis or it is a depressed nerve condition due to some other causes.

Hence we find that, even in frank cases of avitaminosis, the clinical condition is not improved in many cases, even after the administration of large amounts of the particular vitamin, specially in adults, e.g., failure of improvement in vitamin deficiency diseases in adults. In children, where stress and strain play a minor role, improvement is more satisfactory, supposing both are deficient to the same degree. Importance of nervous factor may be seen in villagers who live on perfect health only on salt and rice, but have complete mental peace. The perfect health of the Indian 'Yogis' who live from day to day on a starvation diet, is an extreme example.

By this the importance of vitamins is not minimised, but is meant to state that the accessory food has become essential due to modern life of hurry and high ambition, without in many cases any means to fulfilment.

What is most essential, is a full stomach with free air and light and a quiet life free from greed, avarice, jealous and pride.

In places like India, specially Bengal, if all the vegetables of the season are taken enough vitamin may be found provided they are taken properly and concentrated forms are not necessary and before any national work on vitamins is planned, the nation should see that its individual people get sufficient food, otherwise accessory food factors will be mere accessory.

But its importance as a sympathetico-tonic effect in diseased conditions other than deficiency must not be forgotten.

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7B, Beaulon Street,
Calcutta.

Proceedings of Associations

The Kistna District Medical Association, Masulipatam.—The monthly meeting of the Association was held on the 2nd of July 1943, with Dr. N. N. Sujeer in the chair. The President complimented Dr. T. S. Chowdary, L.M.P. of Pedakapalle on his getting a Commission in the I.A.M.C. Dr. T. S. Chowdary read a paper on "The Treat-

ment of Sprains by Lerriches' Method." Dr. Mrs. P. Janaki Bai followed with a speech on "An Interesting Case of Tumour Abdomen." Dr. M. V. Subba Rao read his notes on two interesting cases of Hernia. The President brought the proceedings to a close after making some observations on the salient points contained in the papers read.

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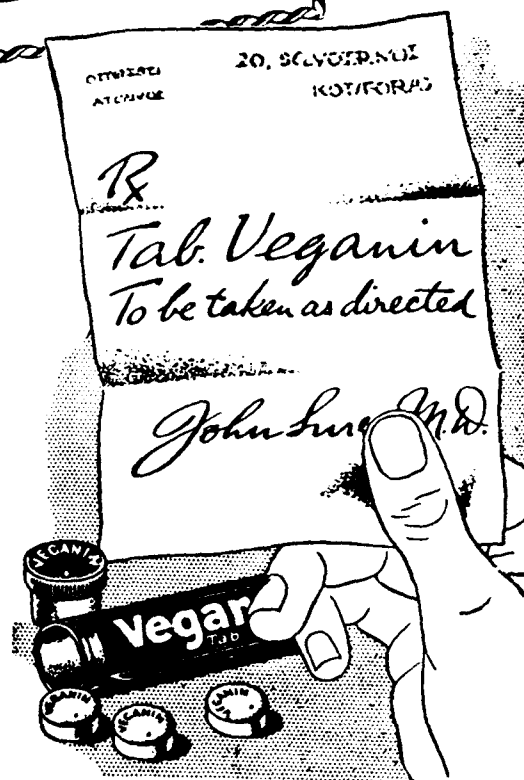
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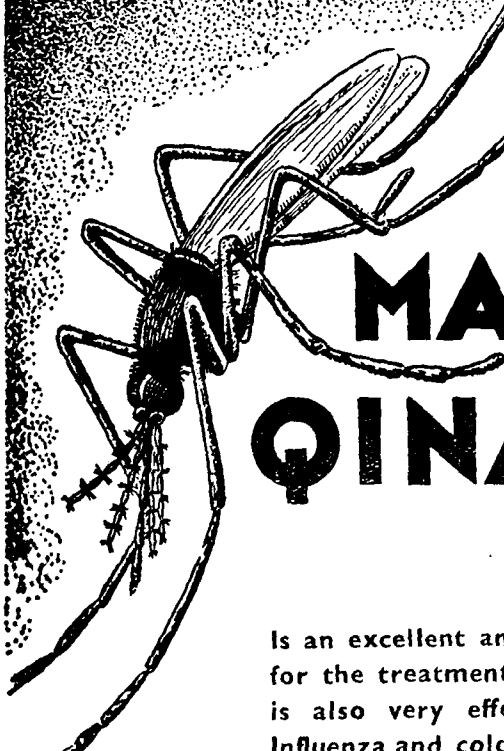
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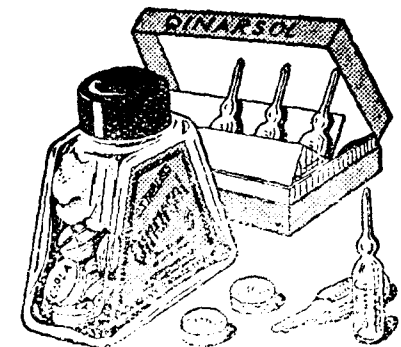
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INDEX TO ADVERTISERS, JULY '43

	PAGE		PAGE
Alembic Chemical Works Co., Ltd.	X, XXX	Ilford-Selo (India) Ltd.	IV
Allen & Hanburys' Ltd.	XXXVIII, LVI	Indian Health Institute	LII
Andhra Pharmaceutical Works Ltd.	III	India Syringe Works	XVI
Anglo-French Drug Co., Ltd.	XXVI	Jai Singh & Co.	IX
Allied Drug Industries Ltd.	XIII	Jammi Venkataramanayya & Sons	XIX
Associated Drug Co., Ltd.	XXXIII	John Wyeth & Brother Ltd.	XXI
Associated Drugs & Chemical Concern	IV	Joshi Pandit & Co.	XXII
Atlantis (East) Ltd.	L	Lister Antiseptics & Dressings Co.	XXVII
Banerji P.	VI	Malarial Research Institute	LI
Behar Chemical Works	XXVII, L	May & Baker Ltd.	XXIV
Bengal Chemical and Pharmaceutical Works	Inside of Back cover	Medical Research Laboratory	XXXVI
Bombay Surgical Medical Agency	XXXV	Menley & James Ltd.	XX, XXXIII
Boots Pure Drug Co., Ltd.	XXXII	Navaratna Pharmacy	IV
Brand & Co., Ltd.	XLVIII	Organon Laboratories Ltd.	VIII
British Drug Houses Ltd.	XXXVII	Orient Laboratory Ltd.	XL
Burroughs Wellcome & Co.	XXIII	Oriental Pharm. Industries Ltd.	XLV, LIII
Buyers' Bureau	LI	Oriental Research & Chemical Lab. Ltd.	XXIX
Calcutta Clinical Research	XVI, LI	Oriental Scientific Works	LI
Calcutta Polyclinic Ltd.	LII	Parke Davis & Co.	Outside of Back Cover
Chemo-Pharma Laboratories Ltd.	II	Petrobagar Laboratories Ltd.	XI
Ciba (India) Ltd.	XV, LIV	Pharmaceutical Labs. of India Ltd.	XLI
Cipla Laboratories	XXV, XLIX	Polyclinical Laboratory Ltd.	VI
Corn Products (India) Ltd.	XXXI	Rendell, W. J., Ltd.	XIV
Deodhar Bros.	II	Scientific Glass Blowers Ltd.	LI
Director Genl., I.M.S. VII, XVI, XVII, XXII, XXXIX		Scientific Indian Glass Co. Ltd.	XXII
Dutta Glass Blowing Concern	LI	Sinha Medical Stores	LI
East India Pharmaceutical Works	XIX	Smith Stanistreet & Co., Ltd.	I
Eli Lilly & Co.	Inside of Front Cover	Smith T. J. & Nephew Ltd.	XXVIII
Glaxo Laboratories Ltd.	XLII, XLIII	South Indian Eye Laboratory	LII
Great Bengal Pharmacy	LI	Standard Pharmaceutical Works Ltd.	XII
Hall & Kay	L	Suren & Co., W. T.	V
Himalaya Drug Co.	XLVII	Tapissier & Co., H.A.	VI
Hind Surgical Works	XXVII	Thacker Spink Ltd.	LV
Horlicks Malted Milk Co.	XXXIV	Union Drug Co., Ltd.	XX, XLVII
		William R. Warner & Co., Ltd.	XLIV, XLVI
		Zill & Co.	XXXVI

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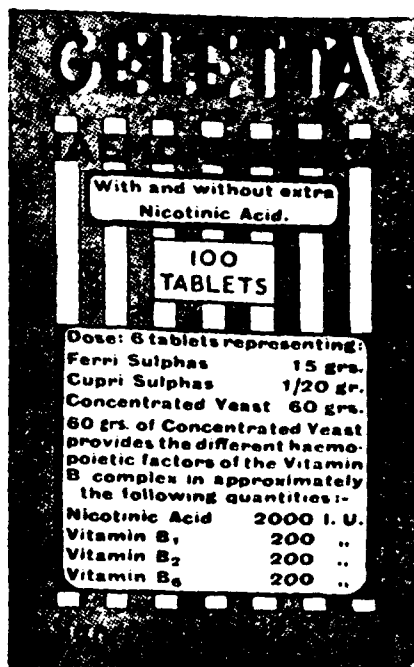
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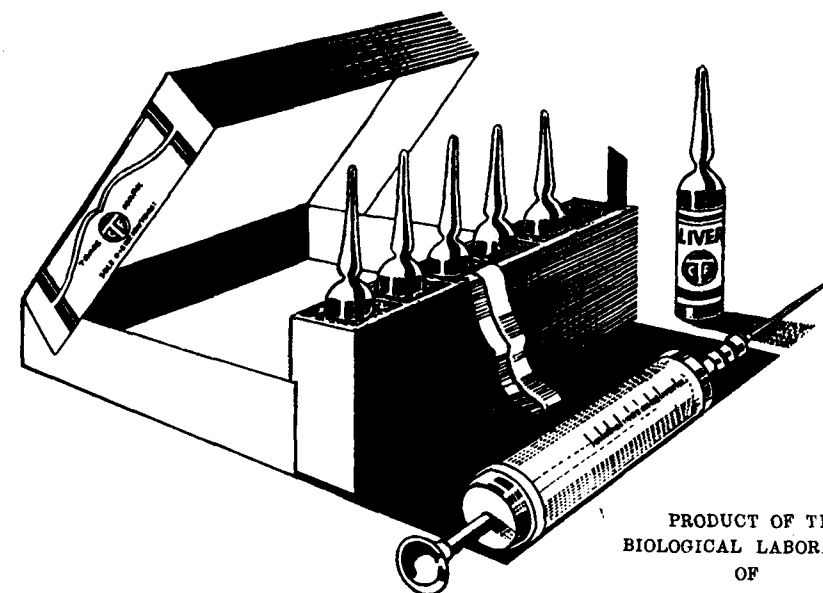
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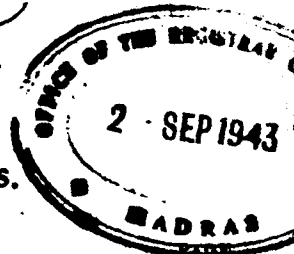
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CONTENTS

Original Articles :-

THE SEX HORMONES. By D. N. Banerjee, M.B., (Cal.), M.D., (Berlin), Carmichael Medical College, Calcutta. ...	391
THE STUDY OF TWINS. By L. P. Varma, M.B., B.S. (Pat.), P. W. Medical College Hospital, Patna. ...	398
ON EARLY MANIFESTATION AND IMMUNITY IN PULMONARY TUBERCULOSIS OF ADULTS. By K. Eisenstaedt, M.D., T.D.D., State Hospitals, Jamnagar. ...	403
SOHISTOSOMIASIS. By V. V. ANKALIKAR, M.B., B.S., (Bom.), Medical Officer, Shibam-Hadhramaut, Via Aden, Arabia. ...	408

Cases and Comments :-

REVIEW ON 32 CASES OF THROMBO ANGEITIS OBLITERANS—Treated at the Prince of Wales Hospital, Bhopal, with reference to various modes of treatment advocated. By Major Khan Bahadur Ahmed Bakhsh, O.B.E., I.S.O., Senior Surgeon and Supdt., Prince of Wales Hospital, Bhopal. ...	411
A REPORT OF TWO CASES OF HARADA'S DISEASE. By P. K. Bhatt, M.B., B.S., D.O., Ophthalmic Surgeon, Bhavanagar. ...	416
A FOREIGN BODY IN THE VAGINA OF A SMALL CHILD. By Captain Dr. Khushdev Singh, M.B., B.S., P.M.S., Medical Officer, Civil Hospital, Sunam. ...	417
CASES OF DERMATITIS DUE TO IDIOSYNCRASY. By S. Mansur Hussain, B.Sc., M.B., B.S., Canning Street, Lucknow. ...	419
A CASE OF HYSTERIA. By A. C. Roy, M.B., Balagaria Charitable Dispensary, P.O. Balagaria, Midnapore. ...	421

Editorial :-

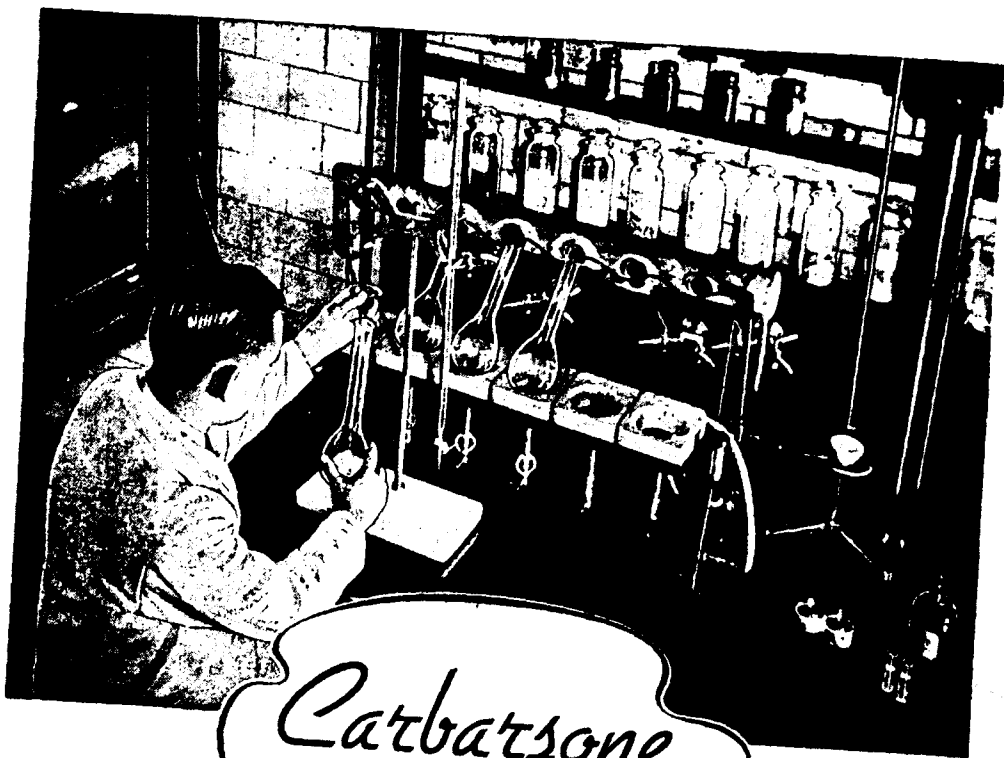
SOME PROBLEMS OF TUBERCULOSIS IN EARLY CHILDHOOD. ...	424
Gleanings from Medical Press	
MEDICINE AND THERAPEUTICS ...	427
SURGERY ...	432
OBSTETRICS AND GYNÆCOLOGY ...	434
DERMATOLOGY ...	435
OPHTHALMOLOGY ... (Detailed Contents on Page LIII)	436
Proceedings of Associations ...	437
Book Reviews ...	438
Success of Aerial "Hospital Ships" ...	438
Errata ...	438

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Index to Advertisements, see page LIII



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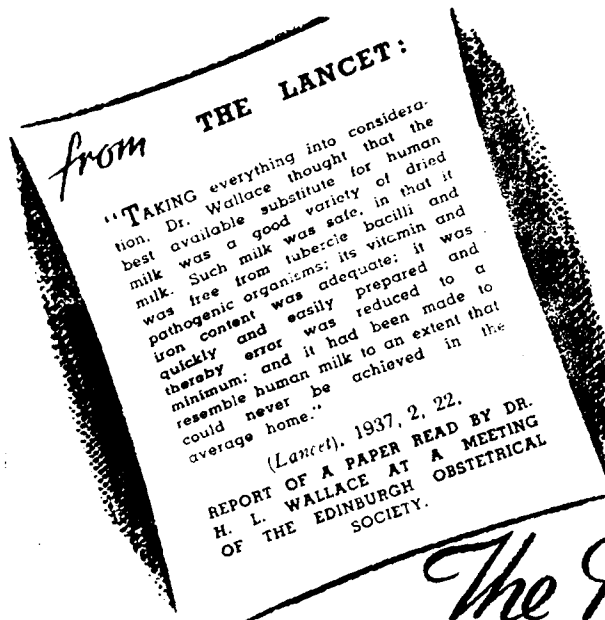
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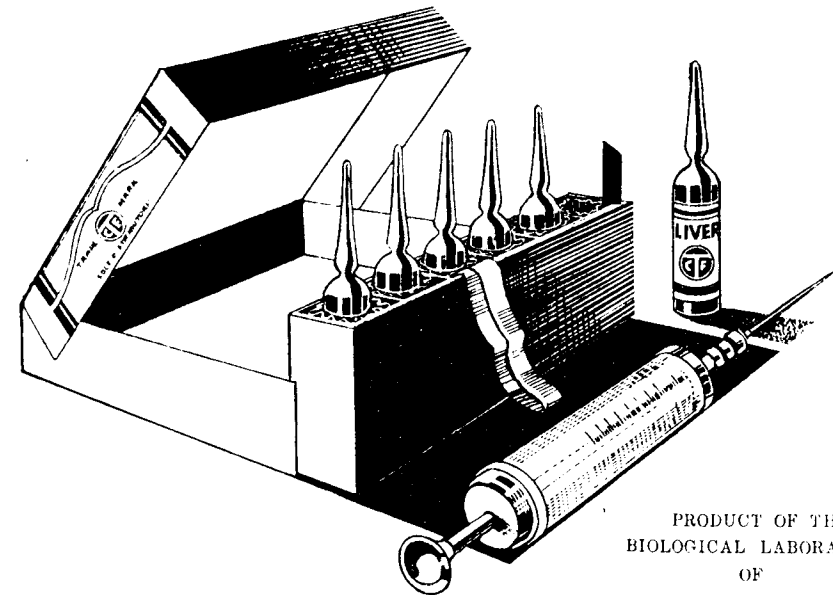
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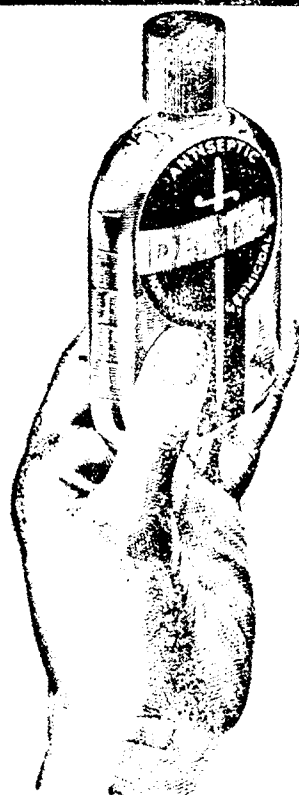
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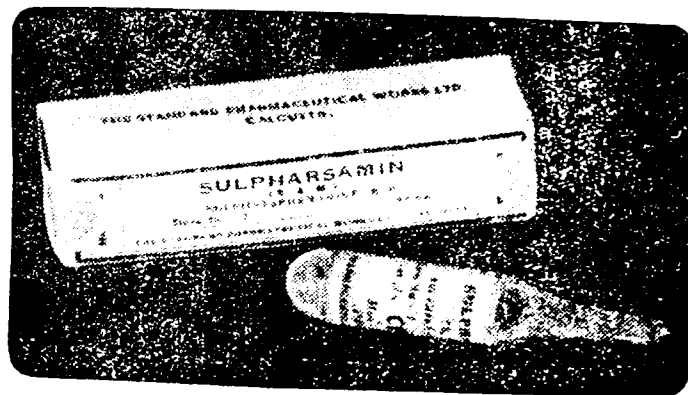
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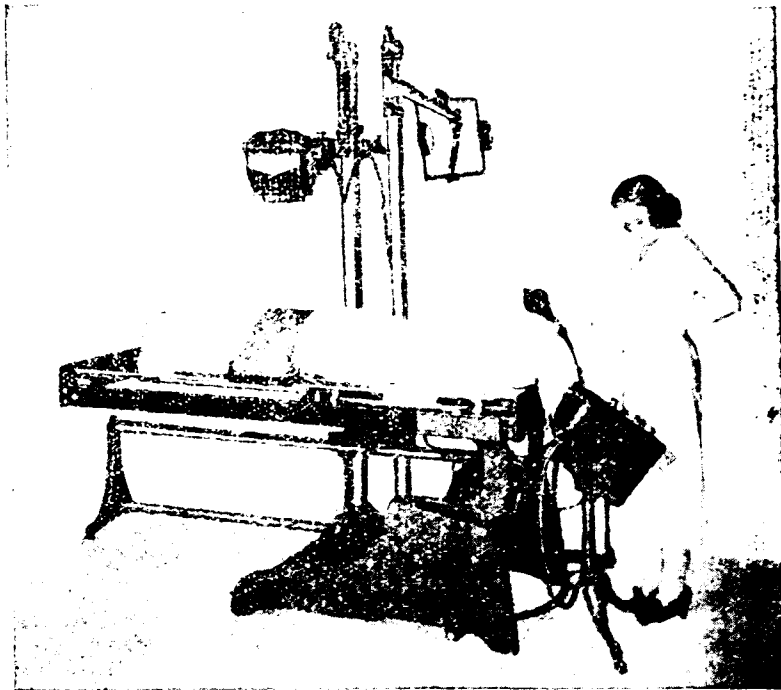
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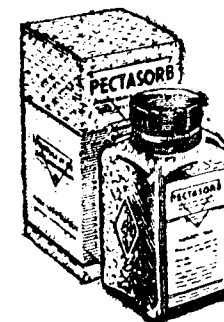
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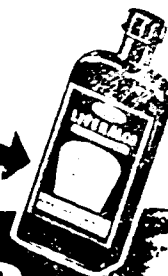
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Vol. 40.

AUGUST, 1943.

No. 8.

Original Articles

The Sex Hormones*

BY

D. N. BANERJEE, M.B., (Cal.), M.D., (Berlin),
Carmichael Medical College, Calcutta.

A HORMONE (derived from a Greek word meaning, 'to excite') is a chemical substance formed inside some glands of the body and circulating into the blood stream influences one or more distant organs or functions of the body. The hormone can be obtained as an extract from the gland and many of them can be obtained in the urine and even produced synthetically.

I. **Male sex hormone.**—Male sex hormones or androgens have the property to develop the male organs and characters. The growth of the prostate, seminal vesicles, Cowper's glands, Vas deferens are also maintained by the androgens. The androgens, in their turn, depend on the gonadotropic hormone of the anterior pituitary. Testosterone is the hormone extracted from the testes. Androsterone is the hormone found in the urine of both the male and female but more in the male. Synthetic products having the androgenic activity have been prepared. It appears thus that both androgens and oestrogens represent in the urines of both sexes, the only difference is that androgen predominates in the male urine while the oestrogen in the female urine. The androgen oestrogen ratio as excreted per day is from 2-5 times higher in males than in females.

A great amount of stir has taken place in recent times in which the man in the street has also been profoundly affected, and that by the brilliant researches of VORONOFF in France and STEINACH in Germany. The gland-grafting—the all important monkey gland—rejuvenation of the old—the triumph over senility—all these phrases have come to be very common with even the laity. But every action has an equal and opposite reaction. It gradually came to be known that senility is the result of a degenerative process of all organs and is beyond the influence of the hormones liberated

* Specially contributed to 'The Antiseptic'.

from grafted testes. Absence of testes, even, is not necessarily the cause of premature senility as shown in the eunuchs who show no premature senility. Further the females after menopause, *i.e.* with the regressive changes in the ovaries, do not show hastening of the degenerative changes incident to age.

Male sex hormones or androgens are sterols and hence readily soluble in oil, and in water only in low concentrations. Being sterile in nature they have low molecular weight and is capable of absorption through the intestinal mucosa in an active form and that in greater quantity as compared with pituitary hormones. There is one derivative of the androgen group, which though active when given by mouth but produces progesterone-like effect and not androgenic.

Testosterone, a hormone extracted from testes, and androsterone a similar substance excreted in the urine of both sexes but more in the male and their esters are the preparations available for clinical use. Both of them have been prepared from cholesterol. These have been found useful in the following conditions:—

In males: (1) In male castrates, the testes of whom have been removed surgically, (2) delayed sexual development, delayed puberty with or without cryptorchidism. The effect is seen by the development of secondary sexual characters *e.g.*, size of external genitalia, growth of hair, deepening of voice, development of manly appearance, etc. (3) premature senility with impotency, (4) male sterility and defective spermatogenesis. (5) eunuchoidism in adult life, and (6) hypertrophy of prostate. In females, on account of the anti-œstrogenic property, androgens are often found efficacious in the treatment of chronic mastitis and menorrhagia. The basis for its use in menorrhagia is the temporary atrophy of the endometrium which is caused by androgen; thus amenorrhœa is induced which may be followed by normal menstrual rhythm.

II. Female sex hormones.—The development of the secondary sex characters of the female occurs from the follicular hormone of the ovary. The secondary sexual characteristics are growth of the breast, increase in size of the genital tract, *e.g.*, uterus, ovary, vagina etc., and other changes of the pelvic organs. The hormone produces growth and reconstructs the mucous membrane of the uterus after each menstrual period. But it exerts no influence on the premenstrual decidua. The formation of decidua is made by luteal hormone. Pituitrin, the hormone of the posterior lobe of the pituitary gland, usually stimulates the uterine muscle, thereby the activity of the follicular hormone is increased. Oestrin is the main hormone which is actually secreted by the follicle. But this hormone can be extracted from other body tissues in larger concentration than from the ovarian follicles.

The follicular hormone—Oestrin:—The functions of the follicular hormone are—(1) In immature animals, in adult ovariectomised animals or in adult normal animals during the œstrus period, administration of œstrin produces hypertrophy of uterus with proliferation of its glands, rhythmical contraction of the uterine muscles, sensitization of the uterine muscle to the action of pitocin, changes in the vagina, growth of the breast and psychic changes. (2) Restoration or prevention of the otherwise inevitable changes in ovariectomised animals, particularly with respect to menstrual changes, atrophy of accessory reproductive organs, water retentions

and increase of blood volume. (3) It acts as a “primer” for the action of corpus luteum hormone and antagonistic to progesterone in large doses. (4) It inhibits the gonadotropic activity of the anterior lobe of pituitary, thus making “antimasculine effect” when injected into adult male rat inducing retrogressive changes in the genitalia and sexual immaturity. (5) It has a synergic action with the oxytocic principle of posterior pituitary. (6) It exerts no effect on the ovaries themselves nor on the thyrotropic activity, *e.g.*, basal metabolism or on body growth, circulation, respiration etc.

The follicular hormone (œstrin) occurs in various forms of which three are most important.

(1) Oestradiol—commercially known as œstroform B (British Drug House), Dimenformon (Organon), Progynon B (Schering), Ovostab (Boots).

(2) Oestrone—Oestroform (BDH), Menformon (Organon), Progynon (Schering), Theelin (Parke Davis & Co).

(3) Diethyl stilbœstrol—a synthetic product, similar but more simple in chemical structure to the œstrogens.

The composition of the follicular hormone, is peculiar in that it contains a phenanthrene ring which has been found to produce carcinoma experimentally. Compounds of benzanthrane possesses carcinogenic properties and it is curious that the most potent synthetic œstrin-producing agent is a derivative of the carcinogenic benzanthrane compound. Coal tar benzenethracene compounds petroleum, etc., are also œstrogenic. Experimentally mammary carcinoma in male mice, which are not susceptible to carcinoma ordinarily, has been produced by injection of menformon. In women, however, no growth suggestive of malignancy has been observed by therapeutic administration of the estrogenic hormones. During œstrous state, however, there is considerable proliferation of cells which reminds the condition taking place during early stages of malignant tumour.

The œstrogen can be obtained by extraction of solid parts of ovary, from the placenta, from the urine of both men and women, but in larger amount from the urine of women at the time of ovulation and in still larger quantity from the urine of pregnant females. The substance is almost insoluble in water and when injected subcutaneously to oophorectomised mice, œstrin is induced in the course of the second day. A mouse unit is the amount required to cause cornification of the vaginal lining epithelium in 10 out of 20 oophorectomised mice. The œstrogen (œstrin) is not species-specific, and is known chemically to be sterols and belongs to three different forms known as œstrone, œstriol and œstradiol. Oestrone can be obtained in a pure crystalline form and gives the formula $C_{18}H_{22}O_2$. (Estrol is also crystalline and is a trihydroxy-substance. Both of them are found in the urine of normal men (20-100 units daily) and women (100-600 units daily) and in pregnancy urine (150,000-500,000 units daily). (Oestradiol is found in ovarian tissue and is most active, being 6 times the potency of œstrone. Several synthetic substances *e.g.*, diethyl stilbœstrol have œstrogenic properties.

The corpus luteum hormone—Progesterone:—The function of the luteal hormone is to create a favourable field in the endometrium for possible reception of a fertilized ovum. The hormone progesterone is secreted by

However, substances like anterior pituitary have been manufactured from sources, *e.g.*, placenta, pregnancy urine and serum of pregnant animals. Commercial preparations are—a type substance—(the follicle-stimulating factor) Antostab (Boots) obtained from serum, B type substance—(the luteinizing factor)—Gonan (BDH), Pregnyl (Organon), Antruitrin (P.D.&Co.) obtained from pregnancy urine and is the substance responsible for the ASCHHEIM-ZONDEK test of pregnancy and is supposed to be derived from the chorionic elements of the placenta.

Gonadotropic hormone—“castration cells”:—The anterior portion of the pituitary contains 3 groups of cells scattered throughout the substance. These are—(1) acidophil or alpha cells having granule showing affinity for acid stains, and constitute 35% of all cells, (2) basophilic or beta cells having granules showing affinity for basic stains and constituting 15% of all cells and (3) chromophole cells which show no granulations and stain poorly and constitute 50% of all cells. The acidophilic and basophilic cells are called the chromophil cells and are supposed to be the source of the hormones of the anterior pituitary while the chromophole cells present an earlier stage in the development of the chromophil cells.

The basophilic cells or beta cells are probably the source of the gonad-stimulating hormones. After castration of the animal these cells show considerable increase in number and size, hence called “castration cells” with increase of the gonad stimulating principle in the gland. On the contrary, injections of oestrin reduces the number of both the basophil and acidophil cells with their ultimate disappearance and a proportionate increase in the chromopholes. These observations show that while the anterior pituitary influences over the gonads by the gonadotropic hormones the gonads, in their time, exert normally a restraining influence upon the gonadotropic functions of the pituitary.

Deficiency of the gonadotropic hormones of the pituitary leads to atrophic changes of the testes disturbing the internal secretion producing hypogonadism or eunuchoidism resulting in failure of development of accessory male sex organs and of the secondary sexual characters. Hypogonadism also takes place as a result of primary affection of the testes *e.g.*, destructive tumours, syphilis, typhoid fever, mumps etc., or from prolonged and severe inanition or of vitamin B₁ deficiency.

In certain tumours of the testes, in young boys, hypersecretion of the male hormone takes place which often produces development of the secondary sex characters—growth of hair upon the pubis, over the face, in the axillæ, deepening of the voice and enlargement of the penis. These subside as soon as the tumour is removed. Hyperfunction of adrenal cortex also leads to hypergonadism with similar features.

IV. The Lactogenic hormone of the anterior pituitary—Prolactin, also called galactin.—In addition to the function of the gonadotropic hormones A and B on the ovarian tissue to stimulate maturation of the follicles or to produce a luteinization in the existing mature follicles the anterior pituitary produces prolactin or lactogenic hormone which stimulates production of milk in the breast while the growth of the breast during pregnancy is due to various other factors. Administration of the gonadotropic hormones occasionally restores the balance and normal function to an obviously disturbed group of glands and that particularly at the stage

when the activity of the ovaries and pituitary gland undergo considerable physiological alteration. The lactogenic hormone from the anterior pituitary controls the establishment and continuation of lactation. In lesions of the pituitary, failure of lactation may take place. In clinical medicine, again, the use of pituitary hormone is often found to be very useful.

That anterior pituitary exerts an influence on sexual development has long been observed in diseases of the anterior pituitary, *e.g.*, acromegaly, Frohlich's syndrome, pituitary infantilism and also after hypophysectomy. In animals where atrophy and disturbance of sex functions take place. It has, further, been observed that anterior pituitary possesses hormones having different functions—(1) growth promoting and luteinizing functions, (2) stimulating follicle maturation and causing liberation of oesterin. The follicle stimulating hormone was known as Prolan A and the luteinizing hormone Prolan B. These hormones are found both in the extract of anterior pituitary as well as in pregnancy urine. In order to distinguish the source, it has been now become customary to use the term “prolan” for the gonadotropic hormone in the pregnancy urine while those of the anterior pituitary extract as follicle stimulating and luteinizing hormone.

The gonadotropic hormone also influences the male sex organs. Thus the follicle-stimulating hormone also stimulates the epithelium of the seminiferous tubules of the testes leading to the gradual development and maturation of the spermatozoa while the luteinizing hormone stimulates the interstitial cells of the testes thus producing the testicular hormone, the androgens. A deficiency of the gonadotropic hormone thus results in atrophy of the gonads and modification of secondary sex characters. In clinical medicine, however, the use of gonadotropic extracts has proved disappointing.

V. Placental Hormones.—Human placenta contains 3 hormones—(1) oestrone, (2) oestriol, the two forms of oestrin and (3) anterior pituitary-like substance which is not identical with the gonadotropic principle of the anterior pituitary but possesses ovary-stimulating, mainly luteinizing, principle. The gonadotropic principle found in pregnancy urine is derived from the placental (chorionic) tissue and not from the ant. pituitary, although the manufacturers give trade name suggestive of being pituitary extract.

The gonadotropic hormone (prolan) is secreted by the placenta and is found in the urine in cases of chorion-epithelioma and hydatidiform mole in much greater amount than in normal pregnancy. There is, however, a synergic action between the placental hormone and the ant. pituitary hormone. Follicle-stimulating hormone has been obtained from pregnant mare's serum but this has no influence in the treatment of pituitary infantilism. A good amount of clinical trial has been given to this hormone as it is easier to collect, while gonadotropic extracts from the pituitary itself are found generally contaminated with other factors *e.g.*, thyrotropic hormone, thus precluding the use in large doses.

The Study of Twins'

BY

L. P. VARMA, M.B., B.S. (Pat.),
P. W. Medical College Hospital, Patna.

"The Scientific Journey has no end. It has only halting place—points at which the traveller can look round and survey"—SHERINGTON.

TWINS have attracted the attention of mankind since the very dawn of civilisation and references to it have been made in the Bible and Shakespeare (WASH and POOL, 1941). The striking similarity in their facial features, complexion, colour of the eyes, character and diseases has always been regarded with curiosity by their fellow-brothers and has been the cause of many comedy of errors. DR. ABBOT, one of the past Presidents of the American Psychiatrists' Association, was once a victim to such an error. He had a young woman with schizophrenia at McLean Hospital whom he planned to transfer to Butler Hospital in Providence. On subsequently visiting Butler Hospital, he thought that he saw his own patient there and enquired when she was transferred from the McLean Hospital. He was told that no such transfer had been made and it subsequently developed that the young woman, whom he saw at the Butler Hospital was an identical twin of his own patient at McLean Hospital (KESANIN, 1934). The experience of DR. ABBOT is not rare and many similar reports have been made by other students of heredity and mental disease.

The origin of twins.—The attention of the medical profession was first drawn to the study of twins by GALTON (1883) who collected a number of cases of psychological resemblances in twins. He classified the twins into two groups, viz., monozygotic and dizygotic. He pointed out the difference between the two and postulated their origin. Monozygotic, uniovular or identical twins are products of a single ovum, and have the same placenta or chorion. They divide into two in course of development and are of the same sex. The dizygotic or binovular twins, on the other hand, develop from two ova, independently fertilized and may be of the same sex or of different sex. They have separate foetal membranes.

Striking resemblances of the twins.—Cases of striking resemblances among twins have been described in literature, the most beautiful example of which, has been furnished by MORANDON DE MONTYEL (1906). Both the twins were girls with marked physical and psychological resemblances. Both of them developed colds, intestinal derangements, measles, mumps and varicella together during childhood. The diseases always appeared simultaneously and their symptoms were exactly of the same nature and character. They married and lived in different districts but became pregnant at the same date. Both of them suffered from acute mania during the fourth month of their pregnancy. The mania was attended with hallucinations. The contents of the hallucinations were similar in both. Each of them saw the virgin Mary and the Devil. The Devil lifted her clothes and got into bed with her. Each was afraid of having a devil as her child. Both of them were delivered of a male child at an interval of forty-eight hours. They recovered in less than a month after it. Cases of schizophrenia and manic depressive psychoses have also been reported in twins to show that both the twins developed the disease at the same

• Specially contributed to 'The Antiseptic.'

AUG. '43]

THE STUDY OF TWINS—L. P. VARMA

399

age and had delusions and hallucinations of the same nature and character. Even when such persons have committed suicide, they have done it in the same way.

Developmental anomalies.—The study of twins has recently crept into the scene of scientific work and is now considered to be the principal gauge for estimating the importance of hereditary factors in the production of physical or mental disease. Most of the work on this subject has been done during the past twenty years, and the recent stress on the part played by heredity, constitution and diatheses, in the causation of certain diseases, makes its study all the more imperative for us. It is by far the most accurate method of estimating the importance of heredity in the production of a disease. It is supposed that the uniovular twins, which are the products of the same ovum carry the same hereditary factors with them and thus are equally equipped for a struggle in this world. Nowhere is this fact more evident than in a study of developmental anomalies, such as cleft palate, spina bifida, patent ductus arteriosus, congenital pyloric stenosis, polydactyly, anencephaly, phocomelus, cyclopia, pseudo-hermaphroditism, hypospadias, epispadias, epiloia, and inguinal hernia have been found in both of the uniovular twins, (COCKAYNE, 1938). The absence of frontal sinuses (PASTORE and OSLEN, 1941) and the association of undescended testes, urethral valves and dilated ureters (BOROVSKY, 1934) have recently been reported.

General Medicine.—The study of the twins in certain pathological and clinical conditions have enabled us to form an accurate estimate of the part played by heredity in these diseases. The origin of diabetes mellitus has always been explained on constitutional basis and the study of binovular twins has confirmed it. Cases of diabetes mellitus have been reported in 13 pairs of uniovular twins by CURTIS (1929). MAY (1914) MICHAELIS (1926) and LEHMAN (1941) have also reported cases of diabetes mellitus in both pairs of uniovular twins. The question whether tuberculosis is a hereditary disease has always been discussed, but as yet nothing definite has been established. Recently cases of tuberculosis in both uniovular twins have been reported by KITTEL KAMP and STRANBRO (1941) and COCKAYNE (1938). Similarly cases of pituitary dwarfism (HACKGIST and SAUNDERS, 1940), Simmond's Disease (BROM, 1940), toxic goitre (NEFF, 1932), thrombosis of the pulmonary artery (GUNTER, 1941), bronchiectasis (PASTORE and OSLEN, 1941), pseudo-leukæmia infantum, aplastic anæmia, paralysis agitans (COCKAYNE, 1938) celiac disease (COLLISHAW, 1940), osteosclerosis (HAGENS, 1941) subarachnoid hæmorrhage (O'BRIEN, 1942), congenital syphilis affecting identical lobes of the lungs (COCKAYNE, 1938) and cases of pneumococcal pneumonia of the type 1 variety (JACOBS and SABOL, 1940) in both of the uniovular twins have been reported in literature. Then again a case of adenocarcinoma of the rectum has been reported by MACLIN and MACLIN (1939) and one of fibroadenoma of the breast, by COCKAYNE (1937).

On the other hand, KELLET (1937) has published a case of acute myelocytic anæmia, in which only one of a pair of uniovular twins suffered from this disease. NETTLESHIP (1912) has cited another instance where only one of the pair of uniovular twins suffered from red green blindness while the other was perfectly normal. SPENCE (1938) came across a case of pink disease in only one of the uniovular twins though both of them were similarly fed and kept in close contact with each other.

So we find that many new beginnings have been made but still the work as yet does not lead us to any definite conclusions. It is expected that in future, when the literature on twins accumulate, we shall be in a position to estimate the value of heredity in the production of certain pathological or clinical entities.

Psychological Medicine.—Psychiatry has always struggled to emancipate herself from the clutches of heredity. Her attempts in this direction have been mostly futile. Heredity is still believed to play a considerably important role in the causation of most of the mental disorders. Schizophrenia, manic depressive psychoses and a host of other disorders are still believed to be the outcome of defective germ plasms. Time and again, evidences have been put forward, both in support of and against this hypothesis, but the methods of approach to the subject, at our command, have been so elusive and vague that it is neither possible to prove nor to reject them altogether. Geneticists, in this field, have always felt the difficulty of having to deal with two sets of variables, viz., the inborn tendencies and the environmental factors and thus their results have been mostly vague and confusing. The study of twins offers us the possibility of eliminating at least one of these variables, viz., the hereditary endowments. We have seen in the foregoing discussions that the hereditary endowments are the same in the uniovular twins. We are therefore left to deal with the only other variable, viz., the environment when we are dealing with cases of uniovular twins. It is thus possible for us to make an accurate estimate of the relative importance of the two factors in the causation of any mental disease. It has been said that the identical twins resemble each other extraordinarily in their intelligence (RODGERS, 1938) and a pair of twins have been reported by COCKAYNE (1938) where both of them had low mentality and mental derangement. They suffered from delusions of a religious nature at the age of twenty-one and then developed pulmonary tuberculosis of which one died at the age of twenty-eight and the other at the age of twenty-nine. Cases of suicide among the twins have reported by LOWENBERG (1941) occurring in the same way and under the same psychological setting.

Different environments.—Recently many painstaking efforts have been made to find out and study cases of identical twins reared apart and an attempt has been made to discover the extent to which environment has been successful in bringing about differences in individuals, identical in genetic constitution. MULLER (1925) was the first to describe such a case and he was followed by NEWMAN (1937) who collected nineteen cases of this type. KESANIN (1934) reported a case of schizophrenia in one of the uniovular twins and attributed this difference to a change in environment. Another case of a pair of uniovular twins, where environment has been held responsible for the differences, has recently been reported by YATES and BRASH (1941). One of the twins is decidedly superior in height, weight and general health, while the other scores higher on the scale of the BINET-TERMAN-MERRILL intelligence tests than his fellow twin. Still the radiological and ophthalmoscopic examinations revealed strikingly similar stomachs and deep physiological cups in both pair of eyes. The similarity between the sets of teeth was amazing. All these facts clearly show the importance of environmental factors in the causation of a mental disease and in changing our temperament and character inspite of our genetic constitution.

Schizophrenia.—Some work of a tangible nature in this direction has been done by studying cases of schizophrenia in uniovular twins. SOUKHANOFF (1900) reported of a pair of uniovular twins who suffered from dementia præcox. PARKER (1926) collected a number of such cases and came to the conclusion that the occurrence of schizophrenia in uniovular twins pointed to the germinal nature of the disease. He had still some doubts in his mind that cases of uniovular twins, where only one of them was affected, was not reported in literature. KESANIN (1934) reported of a case of schizophrenia in only one of the uniovular twins and suggested that this change was due to a change in environment. ROSANOFF ET AL. (1934) studied 1014 pairs of twins with some mental disorder, of which 142 pairs represented cases of schizophrenia. 41 pairs of twins were of uniovular type, in which one or both of them were cases of schizophrenia. In 61% of these cases both the twins suffered from schizophrenia and so heredity was held responsible for their causation and for the rest some other factor had to be sought for.

Manic depressive psychoses.—ROSANOFF ET AL. (1935) studied 91 pairs of twins with manic depressive psychoses, of which 24 were monozygotic twins. In 69.6% of the monozygotic twins and 16.4% of the dizygotic twins both the pairs were affected. This suggests that in a majority of the cases of manic depressive psychoses, heredity played an important role. Still, heredity in itself, cannot explain the causation of this disease as there were 30.4 per cent. of the cases studied, where only one of the monozygotic twins was affected.

Epilepsy.—In ROSANOFF ET AL.'S (1934) group of cases there were 23 pairs of monozygotic twins, where one or both of them were patients of epilepsy. In 52.2 per cent. of the cases heredity was held responsible for the causation of epilepsy. CONRAD'S (1935, 1936) work is more precise for he studied cases of idiopathic epilepsy in 22 pairs of uniovular twins. Both the twins were affected in 19 pairs of the uniovular twins while only four pairs were similarly affected out of 97 pairs of binovular twins. Thus he concluded that idiopathic epilepsy was hereditary.

Other psychoses.—Cases of preanoia delusions in both the uniovular twins (HOFFMAN 1940) and of juvenile paralysis in one of the uniovular twins (IRONSIDE 1940) have been published. COCKAYNE (1938) cites of a pair of uniovular twins, where both of them suffered from paroxysmal hæmoglobinuria when exposed to cold and had their W. R. positive. Both of them developed general paralysis of the insane at the age of thirty years.

Conclusion.—The foregoing collection of case reports and discussions show us the importance of the study of uniovular twins to estimate the relative importance of heredity and environmental factors in the causation of a disease. Many good beginnings have been made in this direction. Still the data collected is so meagre that one is not justified in arriving at any conclusions about any certain disorder therefrom. It is expected that in future practitioners in India, will also publish such case reports and thus add to the work which is being carried on in this direction in other parts of the world.

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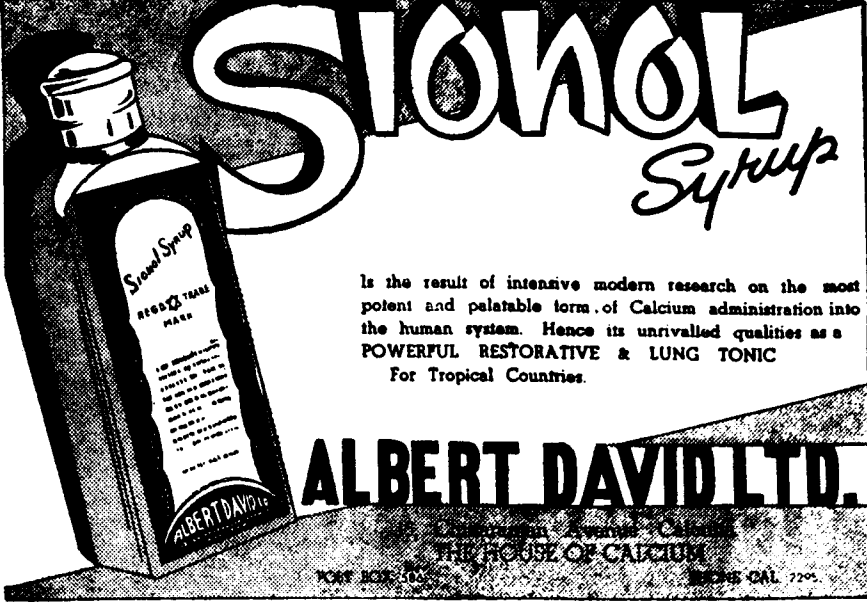
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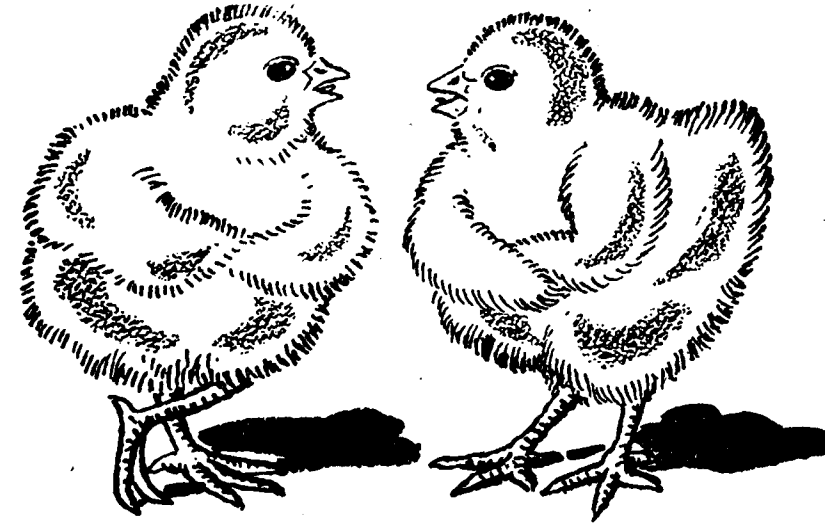
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from tuberculosis in India, even 4 to 6 times more than elsewhere (R. A. SHAH). All these cannot die from additional complications as pregnancy, diabetes etc., which are more or less exceptional. It is a plain fact that death from tuberculosis occurs through deterioration by successive, irregular, acute, subacute or latent exacerbations. Whatever the clinical or anatomical feature of such an *activation* may be, it is always a manifestation of *impaired immunity*. These periods of activation or disturbed immunity are the real causes which lead the patient to the doctor's consulting room. A patient in stationary condition, if ever he is aware of his disease at all, rarely cares to ask for a doctor's advice.

Tuberculosis in adults, as it appears in general practice, is a vast collection of various clinical, anatomical and allergic manifestations. This variety does not allow to speak about "adult type" (or types) which term actually has no meaning and, therefore, is purposely avoided in the headline of this discussion. There are, of course, certain forms which occur more frequently than others. Widely differing from western experiences among people of the white race, we learnt that the acute, exudative exacerbations are predominant in this country. Real "early infiltrations" (ASSMANN, REDECKER) are, unfortunately, mostly overlooked and rather rarely detected. This fact is amply proved by numerous reports from outdoor services, hospitals and sanatoria. Institutional reports are the more convincing because of their policy to admit curable cases rather than those who are beyond hope; nevertheless their figures show the predominance of far advanced patients. On the other hand, the figures of chronic-fibrotic forms fall far behind the others, because they generally do not appear in general practice so long as their stationary condition is not disturbed by acute exacerbation. It is quite possible, and even probable, that by the intensification of organised *detection work* (depistage) the percentage of fibrotic and similar cases might increase but, so long and even then, the general medical practitioner as well as institutional services will have to deal mostly with cases of impaired immunity.

This opinion is shared by many co-workers from all over the country, some of them expressing their views as follows:

"That the disease prevailing among our people is predominantly acute in type seems to be well established." (R. A. SHAH).

"In Indian patients the disease is of a very serious type; it is acute, rapidly developing, with little tendency to show a natural resistance and healing." (P. V. BENJAMIN).

"Generally speaking, the population of India is highly susceptible to tuberculosis and among most of them the disease takes a virulent form and runs a rapid course." (Public Health Commissioner with the Government of India, 1934).

"Various theories have been brought forward in order to explain the prevalence of violent, exudative forms in India. For some time, western opinion was inclined to believe in the hypothesis of the "virgin soil," viz., absence of hereditary tuberculation in certain races. If primary infection occurs in these cases, some unknown hereditary factors are supposed to be missing so that no immunological defence reaction takes place, and the patients succumb after a fulminant course of the disease. Tuberculosis in India, however, is known since olden times warranting a certain extent of tuberculation of the population, especially since the era of growing urbanisation and industrialisation. Yet, there are wide areas of remote villages and small cities where tuberculation has not taken place at the same rate so that the theory of the "virgin soil" may still hold good to some extent, as I personally could see many a time why in areas of wider tuberculation, exudative cases nevertheless are prevalent, is still an open question and should be a matter of careful investigation." (R. A. SHAH).

In the last annual survey of our own Tbc-Section 209 cases of lung tuberculosis were registered by the outdoor service. Out of this number only 85 could be admitted in our Tbc-Wards for want of beds. The selected patients were those who

offered the best possible chances of improvement. In spite of this selection the following were the figures obtained after complete examination:

Age:			Activity:	
Under 15 years	...	4	Regressive condition	... nil
From 16 to 19 years	...	12	Stationary ..	... 32
" 20 to 29 ..	...	42	Progressive ..	... 53
" 30 to 39 ..	...	20	Clinical Diagnosis:	
" 40 to 49 ..	...	5		
" Over 50 ..	...	2	Primary lesion	... 1
Stage of Disease:			Hæmatogenous-metastatic tbc.	... 1
I (slightly affected)	...	6	Acute miliary tbc.	... 1
II (moderately advanced)	...	6	Chronic ..	... 3
III (far advanced)	...	73	Apical tbc.	... 3
Extent of Disease:			Early infiltration (with cavitation)	... 2
Unilateral, non-cavernous	...	5	Lobar & broncho-pneumonic tbc.	... 29
" cavernous	...	19	Chronic fibro-caseous tbc.	... 2
Bilateral, non-cavernous	...	13	" " " cavernous tbc.	... 33
" unilateral cavernous	...	22	" " " cirrhotic tbc.	... 7
" bilateral cavernous	...	26	" " " cavernous tbc.	... 3

It may be worthwhile to remark that the term "chronic" of the last 4 items should be understood only with respect to the long standing duration of the disease which, none the less, by repeated acute exacerbations led to far advanced condition (see above stage and extent).

Out of numerous other reports the following ones may be mentioned:

P. V. BENJAMIN (1938):			R. A. SHAH (1942):		
	Number	Percentage.		Number	Percentage.
Fibrotic	... 108	5.3	Exudative	... 633	65.0
Exudative	... 1,879	93.0	Fibrotic	... 216	22.2
Pleurisy	... 34	1.7	Miliary	... 18	1.9
			Pleurisy	... 106	10.9

Annual report of the Union Mission Tuberculosis Sanatorium, Arogyavaram, for 1940-41:

I Stage or early cases	... 25 (or 7.4%)
II Stage or medium severe cases	... 42 (or 12.5%)
III Stage or advanced cases	... 270 (or 80.1%)

P. V. BENJAMIN (1942): 3,945 patients (registered from 1916 till 1940) were grouped as follows: I Stage 876; II Stage 1,084; III Stage 1,985.

I think that these figures speak for themselves and need no further comment.

It has been objected sometimes that these staggering figures are not conclusive for a general survey because of a possibly great number of chronic-fibrotic cases who do not care for institutional help. But, as I pointed out earlier in this exposition, it seems just as unlikely that these cases consult the general practitioner until and unless some exacerbation compels them to do so.

We all are aware of the possible fallacy of statistics due to geographical, environmental and selective conditions. In the here given surveys, however, we are allowed to assume some "*bona fide*" objectivity, because there is no plausible reason for exaggeration favorizing any particular interest or purpose. I personally have no doubt that further surveys will lead to similar results. The fortunate occurrence "*per se*" of chronic-fibrotic forms amongst others *should not dim our critical outlook nor encourage us to foster too optimistic generalisations.*

Conclusion 1.—(1) There is neither age nor any particular type (or types) of disease vouchsafing the anticipation of any stage of immunity.

(2) Exudative cases, being prevalent in this country, warrant the inference of a low degree of immunity among the population.

almost out of question. If these cases are left alone or, what amounts practically to the same, are merely treated symptomatically, the environment of the patient is continually endangered. We may concede that the prognosis of the patient "quoad vitam" is quite favourable, let alone the possibility of exacerbations. But there remains the medico-social question of preventing the outspread of the disease which cannot always be tackled by mere hygienic measures. Operative collapsotherapy in these cases offers a fair chance of closing a long standing potential source of infection. In fact, these cases give generally the best operative results for the benefit not only of the patient himself but of his surroundings as well.

(b) Apart from collapsotherapy, there are various so called *specific* medical treatments in use, viz., tuberculin, gold etc. These treatments are not without risks, and very experienced clinical observation is necessary, lest more harm than good might be done to the patient, let alone the question of wasting money and time.

Any *specific* treatment, therefore, medical or collapsotherapy, should be carried out by specialists. This suggestion implies, as a matter of course, that the indication and selection of treatment should be left to the specialist too. As tbc. diagnosis is a "flowing" one, treatment also is flowing according to the actual condition of the patient.

Conclusion II—Thus, it may be asked, if the general practitioner will not be out of the picture for good? Not at all! Generally speaking, there are three ways through which the general practitioner can contribute his share.

(1) Being the first to come into contact with the patient, the detection of a tbc. case as such would be a fine achievement indeed. By scrutinising all suspicious signs and symptoms, he should guide the patient in such a way that not a stone should be left unturned in order to make a proper diagnosis.

(2) In cases which do not need institutional treatment (or are unsuitable for such) it is up to the general practitioner to carry out the symptomatic routine treatment and general supervision of the patient. In certain circumstances even specific medical treatment may be possible, if he is temporarily guided by the advice of a specialist.

(3) The last but not the least task consists in the general supervision of the patient's surroundings, housing, family, segregation, disinfection, etc. By his close connection to the family, and their confidence in his advice, he is certainly the proper person to enforce all necessary hygienic precautions, thus maintaining his indispensable share in our common anti-tuberculosis campaign.

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Schistosomiasis*

BY

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THIS is a disease produced by nematode worms of the family schistosomidae of which there are three distinct varieties:—

- I. Urinary schistosomiasis (endemic hæmaturia)—caused by *schistosomum hæmatobium*.
- II. Intestinal schistosomiasis caused by *S. Mansoni*.
- III. Far Eastern schistosomiasis caused by *S. Japonicum*.

Historical.—The results of researches are briefly as follow:—The disease is known to have been present in Egypt some 3,000 years ago, as RUFFER found the characteristic ova in ancient mummies.

The worm of urinary form was discovered by BILHARZ in 1851 in Egypt and was named *Bilharzia hæmatobia* after him but now it is called *schistosoma hæmatobia*.

LEIPERS (1903) confirmed the fact that the lateral spined ova cause the disease of intestinal variety.

Spineless were found by KATSURADA (1904) in Japan which produce the third variety or 'Katayama' as the Japanese call it.

FUJINAMA (1910) infected mice by immersing them in water of rice field which was supposed to be dangerous. MASTUDA found that man can also be infected that way. MIYARI and SUZUKI (1923) succeeded in infecting snails. ATKINSON (1925) traced the full development of the worm in mesenteric vein of mice and established finally the distinction between the three varieties. CHRISTOPHERSON (1919) was the first to prove the usefulness of tartar emetic in destroying the infection.

Geographical Distribution.—*S. Haematobium* is widely distributed in Africa along the northern and eastern coasts, also in Palestine and Iraq. It has been also found in Hadhramaut and in some parts of Rajputana and Sindh.

S. Mansoni is also widely distributed in Africa and in some parts of Egypt, Sudan, the Congo, West Africa and in the whole north coast of South America.

S. Japonicum is found in Japan, the Phillipines, Singapore and Burma.

Etiology.—The male adult worm is flat and is about 15 m.m. in length and 1 m.m. in breadth. The female one is round, 25 m.m. in length and 0.25 m.m. in breadth. Leaf-like lateral borders of the male are folded in front to form gynæcophoric canal in which the female is embraced. Both the male and the female have two ventral suckers by which they adhere to abdominal veins.

Ovum of *S. Hæm.* is 160 m. × 50 m. with a sharp terminal spine; that of *S. Mansoni* is 150 m. × 70 m. with lateral spine or spines; that of *S. Japonicum* is 70 m. × 40 m. with no spines.

Life history in short:—Ova are discharged in fæces, or urine-ciliated miracidium comes out of the shell and enters appropriate water snail in

which sporocysts, daughter-sporocysts and cerceria are formed which come out of the snail and reach the water again—these pierce the skin of man while bathing, wading or drinking the infected water and enter the portal vein in about six weeks time to form adult worms. These migrate towards the veins of rectum or the bladder according to the variety. In small veins the female leaves the male and migrates to the smaller veins and ova are extruded which penetrate the wall of the vein and make their way to the sub-mucous tissue and then the mucous membrane of the rectum or bladder and are finally discharged in the fæces or urine.

CLINICAL FINDINGS:

I. **Urinary S.**—Incubation period—one to two months.

The invasion of the parasite is rarely accompanied by toxic signs which appear after a month and consist of urticaria and malaise.

After some months the patient comes to you complaining painless hæmaturia at the end of the micturition. The accompanying burning and scalding and pain in the perineum usually occur after a long period of three years or even more. The majority of these come to you for sexual debility. On examination: In proportion to the loss of blood the patient is not pale and carries on his routine work as usual. Urine shows ova, R. B. Cs. and sometimes pus cells. There is an increase of eosinophile leucocytes from 12 to 24%. Anæmia is slight except in extreme cases.

Diagnosis.—Painless hæmaturia at the end of micturition is characteristic but confirmation must be made by microscopical examination of the last few drops of urine. Ova will be found out easily in the deposit after centrifugalisation. Gonorrhœa is sometimes mistaken for Sch.; urinary stone may be associated with the disease. Fairley's complement deviation and intradermal test are not reliable.

Course.—If not treated the disease pursues a chronic course and in the absence of complications gradually it becomes less evident but complete cure is unusual.

Complications.—Cystitis, stone in the bladder, hydro or pyo-nephrosis, papillomatous growth in the bladder, pseudo-elephantiasis of urethra, urethral fistula or stricture. Prognosis is good in cases which come to you before complications arise.

II. **Intestinal S.**—About three months after the infection the symptoms begin to appear, viz., diarrhœa with mucous and blood. Diarrhœa may resemble acute or chronic dysentery. In advanced cases tenesmus is increased due to the presence of polypoid growth in rectum. But in many cases there may not be any definite symptoms at all except occasional attacks of mild diarrhœa. On examination typical lateral spined ova are found in the fæces.

Diagnosis can only be confirmed by finding ova in the stools. Polypoid growth may protrude through anus and resemble piles.

Complications.—Cirrhosis of the liver, splenomegaly, perennial fistula. Prognosis is good before cirrhosis of the liver appears.

III. **Eastern S.**—In early stages, resembles the 'intestinal variety but later enlargement of liver and spleen occur.

Complications.—Cirrhosis of the liver, ascites, cancer of liver. Prognosis is not good as the hepatic cirrhosis usually develops rapidly.

* Specially contributed to 'The Antiseptic'.

There is blenching of the part upon elevation (ischemia) and rubor upon lowering it, with diminution or loss of pulsation either in the anterior tibial, dorsalispedis or posterior tibial arteries. This is followed by trophic disturbances in the part i.e., the skin is smooth, shining, thickened, red and glazed and the limb is cold when compared with the sound limb. In course of time the patients notice fissures, ulcers, scaling, etc., which ultimately lead to gangrene of the toe or finger. This process extends upwards and is attended with severe pain. The patient is restless, cannot sleep for nights unless he is given morphia, etc.

To sum up the signs and symptoms :—(1) Intermittent claudication, (2) rubor or cynosis of the limb in the pendant position, (3) ischemia or blenching on elevation, (4) loss of pulsation in the anterior tibial, dorsalispedis or posterior tibial, (5) trophic disturbance leading to ulceration which terminates in gangrene of the part.

The treatment adopted for such ailments varies with the condition of the part and the stage of the disease, as cases do not come to hospital early for treatment.

Preventive measures are out of question, but in suitable and early cases it is worth trying and would save many a limb from sacrifice. In early and suitable cases, before resorting to surgical treatment, medical treatment is tried and it greatly ameliorates the symptoms, specially pain, and in early stages the condition of the limb improves.

(1) I give 5% saline solution intravenously, beginning with 150 c.c. and increase it upto 250 c.c. twice a week. This greatly ameliorates the trouble of intermittent claudication and improves the condition of the limb. After 4 or 5 injections the signs and symptoms improve except the gangrenous part. In addition to this, padutin has also been tried with a fair amount of success in six cases who could afford it.

(2) Measures to promote circulation of the limb, Philip's exhaust box (Edinburgh) appears very efficient but has not been tried here.

Surgical Treatment.—When the above measures fail, as they do, when the disease is advanced, surgical treatment is desirable, as the disease is of the nature of vaso spasm; sympathectomy, either peri-arterial or ganglionectomy lumbar, or crushing the tibial nerves are advocated. Their results in my hands have been the same with ganglionectomy or crushing the nerves, peri-arterial sympathectomy. I do peri-arterial sympathectomy on femoral artery, either in Hunter's canal or Scarpe's triangle for the lower extremity. The following 3 cases will illustrate the condition of the patients when they left the hospital as they all had much improved :—

1st Case :—Male, aged about 65 years, caste Mahomedan. duration of illness 3 months. The patient complained of neuralgic pain in the 2nd and 3rd toes of the right foot, which radiated upwards along the foot and leg as far as the knee. He was unable to walk on account of pain and had to rest to relieve it. There was numbness of the part. In the beginning he felt some itching over the 2nd and 3rd toes. He used to scratch it. This lasted for a week or so and was followed by pain in the area mentioned above. It was slight in the beginning, but gradually got worse, so much so that he was unable to walk, stayed in bed for sometime which relieved him. It was severe, of a shooting character, and was first noticed at the toes only and thence radiated upwards along the posterior aspect

of the leg as far as the knee. A fortnight after this there was a little swelling over the toes. About 1½ months later he noticed he was losing sensation also in the part. No history of venereal disease. B.P. 140-80 m.m. H. G., urine normal, Wasserman negative, knee jerks normal, no ankle clonus. The foot and leg were quite cold. No pulsation was felt either in the anterior tibial dorsalispedis or the posterior tibial arteries.

12-6-1934. Peri-arterial sympathectomy in the Scarpa's triangle.

13-6-1934. Pain passed off, the foot and leg were felt equally warm on both sides, marked pulsation in dorsalispedis but not in the posterior tibial.

On the 17th he got fever and cough and developed pneumonia. His convalescence was thus protracted. This cleared up in about 15 days and he was discharged with no pain, etc., and the colour of the skin &c., was normal.

2nd case :—Male, aged about 30 years, with gangrene of the 5th toe of the left foot which had fallen off. Duration one month. He had pain in the toe which radiated upwards along the leg. Four days after he noticed the toe was getting black and was very painful. It lasted for 25 days and then it fell off by itself. Four years back he had a similar trouble with the great toe of the right foot, which was dis-articulated. He had syphilis ten years back. Wasserman was slightly positive, urine normal. He was of a weak constitution and had an ulcer on the 4th metatarsophalangeal joint of the left foot. There was severe pain in the foot and leg, no pulsation in the posterior tibial and dorsalispedis. He could not sleep due to pain.

20-11-1934—Peri-arterial sympathectomy in Hunter's canal.

21-11-1934—Pain passed off, the foot and leg felt warm, colour was returning, very feeble pulsation in the dorsalispedis. Condition gradually improved, ulcer healed up and the patient was discharged on 12-12-1934. There was no pain.

3rd case :—Male, aged about 35 years, caste Hindu of Bhopal State. (1) Came in with a sinus at the level of the 5th metatarsus bone of right foot for the last 12 months, (2) throbbing in the sole of the foot and calf muscles for 15 months and (3) weakness and numbness in right leg and foot for 12 months. H. P. C.

He noticed his right leg was getting weaker, gradually, with pain in the sole of the foot, calf muscles, etc. He also noticed that walking even for a short distance would cause spasmodic contraction of the muscles of the leg and induce severe pain, so that he had to take rest and then walk again. The pain gradually increased and was later on felt all day and night. Three months after this a small blister appeared close to the base of the 5th toe. It went on increasing and turned into gangrene of the part which was dis-articulated, leaving a sinus. No history of syphilis, Wasserman negative. On examination the whole knee limb from knee downwards was cold and blue, with severe pain so that he could not sleep. The skin of the foot became glazed, shining and hard. No loss of motion, pulsation in dorsalispedis and posterior tibial not felt. He was worn out with pain.

5-2-1934. Peri-arterial sympathectomy in Hunter's canal.

6-2-1934. Pain much less and the patient could sleep.

7 & 8-2-1934. Limb was warm, colour returning to normal.

9-2-1934. The limb was slightly cold. There was pain in the calf muscles but not severe.

10-2-1934. Foot and leg again resumed natural heat, pain disappeared.

16-2-1934. Discharged cured. The sinus was healed, pain etc., disappeared and he could go back on foot to his home in the district.

1st patient:—Aged 58 years, caste Hindu male, gangrene of great toe left side, lumbar ganglionectomy was performed left side on 5-11-1936, toe was not removed as there was no line of demarcation.

6-11-1936. Pain in the foot and leg was much better, he could sleep without morphia.

7-11-1936. Condition of the limb much improved and was equally warm on both sides.

8-11-1936. Comfortable. Gangrenous process showing line of demarcation at the metatarsal pharyngeal joint.

12-11-1936. Gangrenous toe came away by itself, leaving the head of the metatarsal bone exposed.

18-11-1936. General condition much improved except for the head of the bone which was exposed and he left the hospital without undergoing further treatment.

2nd patient. Aged 25 years. Came in with signs and symptoms of thromboangitis obliterans in left foot and toes. He was in extreme pain of a burning character with trophic changes. Lumbar sympathectomy on left side was performed on 7-2-1939.

8-2-1939. He was much relieved of burning sensation, but trophic changes and temperature of the limb remained the same.

9-2-1939. He has been comfortable.

12-2-1939. Condition of the foot and toes improving, shining appearance was passing off and wrinkles began to appear on the skin.

19-2-1939. Condition much improved and he left the hospital of his own accord.

Out of 32 cases I have done peri-arterial sympathectomy in 11, lumbar ganglionectomy in 3, and crushing of both of the anterior and posterior tibial nerves in 5. The results achieved in my cases by peri-arterial sympathectomy have been similar to those, if not better than, with lumbar ganglionectomy or by crushing of the nerves. The remaining 13 cases had to submit themselves to amputations of the limb, in addition to saline treatment and hence no peri-arterial sympathectomy had to be resorted to.

Table showing incidence as regards age, sex, caste etc.:

Age: 12	between	20-30	years.	Sex: Males	32.	Female	Nil.
.. 10	..	30-40	..	Upper extremities	...	2	
.. 5	..	40-50	..	Lower ..	...	30	
.. 5	..	50-60	..	Bilateral	...	1	
Caste: 24 Hindus.	8 Mahomedans.			Unilateral	...	31	

I am thankful to my Assistant, Doctor RAM NARAIN PRADHAN, for all these figures and for sorting the record.

A Report of two Cases of Harada's Disease

BY

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HARADA'S Disease, the symptom complex of which consists of bilateral uveitis associated with bilateral detachment of retina. Its characteristics are sudden onset, headache, vomiting, and acute diffuse choroiditis or uveitis with detachment of retina and accompanied by alopecia, poliosis, vitiligo and deafness.

It is not always that all the symptoms are present. Atypical cases do occur.

Total number of cases reported in the ophthalmic literature up to now are 29 in number. There is marked resemblance to sympathetic ophthalmia. Both sexes are equally affected. Most common age period is after the age of 40 years.

Etiology.—There is difference of opinion in regard to whether the uveal tract is first affected causing a change in the other pigmented structures from an anaphylactic toxin formed as an antibody, or whether all the cardinal symptoms have a common cause. Tuberculosis is mentioned as the cause but all the tests of tuberculosis have been found negative and no tuberculous lesion have been detected in the lung or other organs by radiographic examination. Syphilis is also considered but Wassermann test and Kahn's test have always given negative results. The etiological factor is unknown and the typical onset, the extensive detachment of the retina, and the relatively favourable prognosis of the spontaneous reattachment are features of special entity. Of course all cases do not recover but most of the cases after a period of a year or two get some favourable vision.

Case No. I:—Pt., a male aged 41 years, history of sudden headache and vomiting on the night of 18th May 1941 and blindness of both eyes. The patient came under my observation after the 25th day of the attack and on examination the following picture was present: Vision both eyes: P. L. Ciliary Injection: Moderately present.

Both the pupils were occluded with thin membranous exudate. Iris showing advancing atrophic changes, the stroma of the iris being clearly seen. Both the lenses were not affected. In the vitreous there was floffy exudate and both the retinas were extensively detached. On slit lamp examination:—Both the corneas were clear. There was fine membrane filling both the pupils and fine strands of the membrane were drawn out into the stroma of the iris. The pigment epithelium on the iris was atrophied and in his eye it has completely disappeared. Tuberculin test was negative. Wassermann and Kahn both negative.

There was partial loss of hair and deafness but other symptoms of the disease were absent. The patient was under observation and treatment for about six months but no reattachment of the retina took place and gradually both the iris and ciliary bodies were atrophied and gradual reduction of the eye balls took place.

Case No. II:—Female, aged 44 years, referred for diagnosis by another oculist from another town. History was sudden onset of headache, vomiting and blindness.

Examination of the eyes showed: Vision both eyes: $\frac{1}{2}$ foot centrally. Perception and projection: absent except centrally. Ciliary injection: moderate. Tension: low.

Both the pupils were occluded with a fine exudate stretching into the iris stroma and extensive exudate into the vitreous. Both the retinas were extensively detached. There was no alopecia but deafness was present. Other symptoms like vitiligo were absent. Wassermann was negative. Tuberculin tests were negative.

Sudden onset with headache and vomiting and typical picture of uveitis with detachment were quite sufficient to class the case as one of Harada's disease.

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A Foreign Body in the Vagina of a Small Child

BY

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A MAHOMEDAN female child, aged about two years, was brought in the out-door with the history of discharge of pus from the vagina for over twenty days, and irritation and itching all over her private parts.

The child was carefully examined, both the labia were red and swollen, hymen was found slightly red and intact, and discharge was present at the mouth of the vagina.

A smear was stained on a slide and was examined under a microscope. No gonococci were seen but pus cells were found in abundance.

The parts were well cleaned and the vagina was douched with a dilute Condy's lotion of 1/6000 strength by means of a narrow rubber tubing as nozzle, and the labia were fomented with warm boric lotion and ointed with warm olive oil. One third of a Septanilum tablet was prescribed thrice daily for three days.

On the fourth day I examined the vagina again and found a small whitish sort of membrane in the posterior part of it. As this whitish speck was very deeply situated, I put in a probe and was surprised to find that it struck against a solid body. I passed the scoop of a director behind that solid body, and on drawing it out found it was a cowrie, which was covered with pus and mucous discharge.

The vagina was douched for three or four days and the patient recovered completely. On further investigation I learnt that about a month ago this cowrie was put in the vagina of the child by her elder sister while they were playing, and the later did not tell anybody as she was afraid of being beaten.

The point of interest in this case is the small age of the child, when the patient cannot tell much, and the doctor and the parents cannot suspect of such a happening.

Cases of Dermatitis Due to Idiosyncrasy

BY

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I HAVE come across cases which show peculiar idiosyncrasy to certain articles of diet or some drugs. I cite below a few cases which are of interest on account of the selective action of various articles on particular individuals.

1. M. M. of 38 years was suffering from scabies in 1933. He applied phenyle diluted with water and developed an acute dermatitis which was confined to the parts to which phenyle had been applied. After a day vesicles appeared on the inflamed parts and on bursting were discharging a sticky, slightly yellowish fluid. It took him nearly one month to regain his health.

2. In 1934 M. M. aged 46 years applied diluted phenyle to his hands to get rid of intense itching due to scabies. He too developed vesicular dermatitis (eczema medidans) not only on his hands but also on different parts of the body to which no phenyle had been applied. He suffered from high temperature and bodily pains. He complained of burning and itching in the parts affected. He too required about one month's treatment to be restored to his normal health.

NOTE:—It may be mentioned that scabies was very common in those days and there was a general opinion among the people that phenyle was a specific for it and it was generally used by many persons without any obvious harm. These two cases which came under my observation, however, reacted intensely to it showing individual idiosyncrasy. Phenyle is also commonly used in disinfecting drains and urinals and does not cause any trouble to those who touch it.

3. A M. M. of 42 years touched a stick with which a snake had been killed. The same evening he developed a weeping type of eczema on his hands. The trouble started from the fingers which had come in contact with the stick and soon spread and extended up to his arms. It was accompanied with fever, constipation, headache and bodily pains. Becoming secondarily infected it resulted in ulceration and abscesses with adenitis of the axilla.

4. A. M. M., about 40 years, of age went to a compounder in a hospital for the treatment of an ulcer or abrasion on his chin and the latter applied an ointment containing idoform. Knowing his own idiosyncrasy to idoform he at once wiped it off. This momentary application was enough to start his trouble of which he had previous experience and he had been warned by a doctor never to allow idoform to be applied to any part of the body as it was a poison for him. The whole face became swollen, the swelling extending to his neck and pinna. There were extensive patches of dermatitis on the extremities and trunk ranging from a diameter of $\frac{1}{2}$ " to 2". This was accompanied with fever and restlessness and locally by itching, burning and discomfort. Later on a sticky fluid began to ooze out from the affected parts. The dermatitis ran a protracted course and it took him nearly 6 weeks to recover. This case shows the extraordinary idiosyncrasy and his reaction to idoform (which was more commonly used before than now) was similar on all occasions.

5. H. F., 50 years of age, was a permanent employee in a tobacco factory. She always suffered whenever she handled cinnamon. Once however she had reluctantly to crush and powder it in a pestle and mortar on account of a temporary shortage of hands. The same evening she suffered from itching on both the hands and feet. Later on patches of eczema developed with swelling, exudation and raised temperature. It is to be mentioned that both the hands and feet were exposed to the action of cinnamon in crushing and powdering it. Examination showed the presence of oval or round patches varying from 1" to 3" in diam. They were swollen and much raised above the level of the skin, angry-looking and exuding a yellowish fluid. These patches extended from the fingers almost to the shoulders (the arms and forearms being left uncovered by the dress) and from the toes to the knees. Itching and burning were intense. Her temperature ranged between 100° and 101° and the pulse was frequent. When she had recovered, as a test cinnamon oil diluted with olive oil 1 in 10 was applied to a small area on the forearm. The repetition of the symptoms and signs in a mild form confirmed her idiosyncrasy to cinnamon. Cinnamon is consumed as one of the spices and is relished by many. It however showed a poisonous action in this particular case.

6. M. F., aged about 35, suddenly developed general dermatitis with high fever in 1930. The skin of practically the whole body was erythematous and swollen. She used to be delirious in the evenings when her temperature was high. There were generalised discomfort, itching and feeling of heat and tension all over the body. Gradually the acute condition subsided and at places eczematous patches appeared discharging thin copious fluid. In about a month's time she recovered completely. She gave a history of having suffered before in a similar manner.

A second attack similar to the first occurred a year later at about the same part of the year. This directed the attention to the possibility of idiosyncrasy to some seasonal article of diet. On questioning it appeared that the unusual thing consumed was purslane (khurfa). No one believed it to be the cause of so serious a trouble. Experimentally she was asked to take it again and the whole phenomenon repeated itself confirming it to be the cause of her fever and dermatitis. The other members had partaken of the same dish without detriment to their health. Purslane is commonly used but in her case it violently disagreed with her. She was asked to abstain from taking it and she has remained free from those attacks for over ten years.

7. An unusual case of Iodide eruption: Iodism is commonly seen in varying degrees of severity. Iodide eruption is infrequent. When it occurs it assumes the form of either papular erythema or acneiform rash. Less commonly it is bullous and rarely it may be solid at first to break down later on. In this case a young patient of 25 or 26 on taking only one dose of a cough mixture containing only 3 gr of Pot. Iodide per dose developed within 4 hours a papular eruption on the face, forearm and legs. The trunk, arms and thighs were not affected. The papules were of various shapes and sizes and markedly raised. The eruption on the face was copious particularly on the forehead. The forearms, hands, legs and feet had fewer papules and the increase in number was noticeable in a regular fashion from the proximal part to the distal end. Another most noticeable and unusual feature was the swelling of the tips of all the fingers and toes which was painful and hard. There was no rise in temperature, no running of the

nose, no swelling and redness of the mucous membrane of the nose and throat. A small erythematous papule also appeared on the conjunctiva of the left eye. The withdrawal of Pot. Iodide and administration of Mist. Alba and Sod. Bicarb in large doses relieved the symptoms. The skin eruption cleared up in two days but the swelling of the tips of fingers and toes took another two days to subside completely. The interesting features of this case were:

- (1) Swelling of the tips of fingers and toes which were painful.
- (2) The small quantity of Iodide taken.
- (3) Rapid development of the eruption, i.e., within 4 hours.
- (4) Appearance of an erythematous papule on the conjunctiva.
- (5) Absence of the familiar symptoms of iodism.
- (6) Rapid cure.

A Case of Hysteria

BY

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Balagaria Charitable Dispensary, P.O. Balagaria, Midnapore.

In June 1940, a female patient aged about 18 years came to the dispensary for the treatment of her constipation. Her bowels moved every 4 or 5 days. She had practically no discomfort for this but thinking that the condition might give rise to some future complications, she came to the dispensary for the treatment of the condition which was of two months' duration.

The patient was in perfect health and nothing particular was found in any system. Menstrual history—the periods were regular and the flow was good lasting for 4 or 5 days. There was no pain before, during or after the periods. Patient was nullipara. She did not give any history of any fit.

Her condition was diagnosed as due to atony but later on the cause of the condition became evident as due to hysteria. General tonic with a purgative was prescribed for her which she took irregularly for nearly two months without any improvement.

In the middle of August 1940, she menstruated and with the onset of menstruation she had a severe attack of fits. This was the first attack in her life. The fits were like those of the gradual type of epilepsy. She was fully unconscious. As soon as a fit came the head was drawn back, the hands were clenched and legs extended. The chest rose above the ground at the height of the fit as is seen in a case of tetanus—the patient assuming a position of opisthotonus. With the passing off of the fit the body fell on the bed suddenly making a distinct sound as in a case of fall from a height. During an attack it was impossible to hold her down and it took 6 persons to control her. The fits occurred in rapid succession and the patient was fully unconscious throughout.

The cause of the violent attack could not be elicited except that she did not pass any urine for nearly 24 hours. She had also no motions for the same period.

She was given an injection of Morphia-gr. $\frac{1}{2}$ but to my utter surprise it was of no effect. After an interval of an hour an injection of Hyoc

Hydrobrom-gr. 1/100 was given which also failed to stop the fits. Later on an injection of Valerian-1 c.c. was given and the fits stopped after a few minutes and she gradually regained her consciousness. The bladder was relieved. The fit lasted for about 8 hours.

She was given Trivalerian pill (Iron, Quinine and Zinc Valerian-gr. 1 each) to take. A few injections of Valerian-1 c.c. were also given. As the bowels were not regular purgatives had to be given occasionally.

In the third week of September 1940 it was noticed that she had lost the power of speech and could not utter a single word. She understood everything and answered questions by signs. The condition is continuing since then. The bowels were irregular as before and she had no other trouble and did all her household duties as a normal person.

Early in November 1940, she had her periods and a second attack of fits exactly similar to the previous attack in August. The fits were controlled with Morphine and Hyoscine and Valerian injections and the patient regained her consciousness within a short time. The fit lasted for nearly four hours. It may be mentioned that both the attacks were in the evening.

The next day she had profuse hæmatemesis which was controlled with Adrenalin. Later on (48 hours after the fits) she became fully unconscious and remained in that condition for nearly two days.

She was quite well upto the middle of November 1940, when it was noticed that she could neither walk nor stand on her legs. She could not even move her legs as if completely paralysed. There was nothing with her upper limbs. Trivalerian pill was continued and the condition returned to normal in a fortnight and she could walk and move about freely as before.

Early in December 1940, she had another fit less violent than previous ones. The fits were easily controlled by Chloral Hydras and Hyoscine Hydrobrom tablet.

In the third week of December 1940 she had her fourth and last attack. The fits were less severe but the patient remained unconscious for nearly 3 days.

By the end of December 1940 it was found out that in addition to her usual constipation she passed urine only once in 24 hours. There was no discomfort or any trouble. She had no further fits and the valerian treatment was continued upto March 1941. Her bowel and urinary condition became normal by this time. She had a motion daily and passed urine 4 to 5 times in 24 hours. The treatment was discontinued as the patient did not like to take any further medicine. No improvement was noticed of her dumbness.

Upto the time of writing these notes (May 1943) she had no further fits and no trouble with her bowel or urinary condition. She has given birth to a son and is now in perfect health. The only symptom that is persisting is her dumbness.

A case of hysteria with the following manifestations is described:

- (1) Constipation. (2) Fits of hystero-epilepsy type. (3) Hæmatemesis.
- (4) Astasis and abasia. (5) Passage of urine once in 24 hours. (6) Persisting dumbness.

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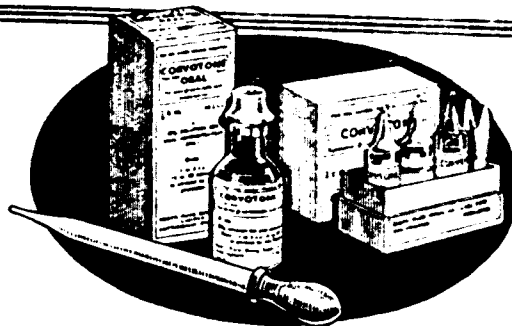
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Some Problems of Tuberculosis in Early Childhood

OF recent years much has been learnt of tuberculosis as it affects the child. It has long been recognised that the disease presents itself in a way very different from that which it assumes in later life. Apical localization in the lung with fibrocaseous changes and cavitation though found in adults was not found in young children. On the other hand, gross enlargement, caseation and calcification of the tracheo-bronchial glands and caseous broncho-pneumonia are forms peculiar to childhood. It was generally believed that tuberculous infection in early childhood was peculiarly virulent and fatal. To-day, however, it is known that tuberculous infection in early childhood was peculiarly virulent and fatal. Today, however, it is known that tuberculous infection in children can be, but usually is not, of grave significance and that the so-called primary infection is controlled with uniform success. Only in the youngest children is the danger of dissemination of foci, and therefore of meningitis, considerable during or soon after the primary infection. To have to say of a child that he has under-gone infection with tuberculosis, is to make a statement which carries with it far less grave implications than would a similar pronouncement in the case of his mother. The disease in the child is likely to be that of the primary infection; in the mother it is almost sure to be of the re-infection type. Primary infection without allergic reaction may occur at any age and always tends to have the same benign and relatively symptomless course with rapid healing and cicatrization. Yet among adults the primary infection is seldom encountered, the re-infection type often; whereas in children the order of frequency is reversed. The older conception of the extreme gravity of tuberculosis in the child arose because the detection of the primary infection, owing to the non-availability of X-ray, had not yet been learnt and only very advanced diseases of the re-infection type showing itself in very fatal terminations were recognised and treated. In a very learned and clear article, in our distinguished contemporary, the *Practitioner*, DR. H. C. CAMERON has discussed at length these problems of tuberculosis in early childhood and we would commend our readers to read it for a more detailed account, as this leader is only a summary of this very excellent article.

To-day some of the reasons which determine this selective difference between tuberculosis in the child and in the adult are beginning to be understood. In many ways tuberculous infection offers contrasts with most other specific infections. The first implantation of the bacillus, the primary infection, is insidious, with minimal local reaction and minimal constitutional disturbance. There is then a delay of from three to seven days before the development of what is called sensitivity. During this period, the tendency is to localisation and repair. At the point of localisation of the tubercle bacilli, there is the usual phagocytic response of the polymorphonuclear leucocytes. In turn these are ingested by the large mononuclear cells which by their aggregation, determine tubercle formation. Encapsulation, fibrosis and cicatrization are the rule. The body deals with the invading bacillus much as though it were a particle of indifferent matter such as carbon or silica. No conflagration results. This latent period before sensitivity occurs is the safeguard which accounts for the benign course of so much of the primary infection. Sensitivity, when it has developed, implies that renewed infection whether exogenous derived from some other person, or endogenous from an extension of the primary focus, will be accompanied by tissue reactions far severer than those provoked by the original invasion. In other words, in sensitized tissues, the reaction is allergic. Such a division into two phases is rarely met with in any other disease. In most specific infections one single invasion with one lasting immunity has to be dealt with. But in tuberculous disease the invading organism is prone to survive in the host for years. The immunity developed is at the most partial. The dormant focus retains the power to again become active and to spread, setting up in tissues which it has made sensitive, the violent processes of the re-infection type of the disease. Thus it is not a single infection and one immunity that has to be dealt with: the body has to devise means to cope with renewed invasion or with reactivation and spread.

Not all primary infection takes place into the lungs. The ear, eye, nose, throat, skin and intestinal tract may all be its seat, but, wherever situated, the type of response is the same. Primary infection of the intestinal tract is in a somewhat different category because the source of infection is in the main bovine and milk-borne. The extensive pasteurization of milk now practised in large cities has to a great extent diminished the frequency of abdominal tuberculosis. Under present conditions pasteurisation or boiling of the milk for infants and young children must never be omitted.

We have referred already to renewed infection. This, whether endogenous or exogenous, is always accompanied by severe reaction. Danger is at a maximum at two periods of the child's life, the first immediate and the second remote. The immediate follows close upon the primary infection and is inversely proportionate to the age of the child. From a study of available statistics, DR. CAMERON has come to the conclusion that dissemination with terminal meningitis is a grave danger in young children from the fourth to the sixteenth week after primary infection. It is also clear that the great majority of children pass through the primary infection apparently unscathed. Other forms of the re-infection type of the disease occur throughout childhood. Tuberculous pneumonia and pleurisy with effusion may also occur as forms of re-infection tuberculosis. Re-infections disease occurs also in joints, bones,

glands, viscera and skin. The second danger period is that of puberty, adolescence and early maturity. In the sensitized tissues of the lung the response to infection takes the form known as phthisis with caseous, fibrotic and ulcerative processes in different degrees. It is not known whether this form of pulmonary tuberculosis developing in later life is the result of the reactivation and spread of long dormant foci or the result of renewed infection derived from contact with an infected person. Both sources of reinfection are common and must be reckoned with in practice. What percentage each contributes to the common total is still doubtful and this point is much debated.

The diagnosis of the primary infection usually in the lungs is one of the great problems. Symptoms are minimal or absent. Cough, expectoration and loss of weight are not to be found. The pyrexia is slight. The physical signs are not helpful. What then remains? DR. CAMERON relies on the following aids: (1) The raising of the sedimentation rate. This is not pathognomonic of tuberculous infection. It is a guide to treatment and gives timely warning of extension or retrogression; (2) changes in the differential count of leucocytes may be suggestive; (3) outbreaks of erythema nodosum. This also is not a specific response; (4) an X-ray examination conducted by an expert; and (5) an examination of the sputum sometimes demonstrates the tubercle bacillus. Sometimes washings from the stomach reveal the bacilli in the swallowed sputum.

The question of producing immunity against tuberculosis has also engaged the attention of workers. CALMETTE and his colleagues in France have claimed for their attenuated bacillus, the "Bacillus Calmette-Guerin" or B.C.G. the capacity to produce a true immunity. CALMETTE's work and claims have been subjected to much criticism. His statistical facts and deductions alike have been subjected to much destructive criticism. These have led to a decline, in recent years, in interest regarding his claims. Moreover it is not certain that the procedure is without risk. The GOTHENBURG experiment is claimed to be an improvement on the methods of CALMETTE. Instead of the oral method used by the latter, WALLGREN in the former used hypodermic inoculation producing accurate control of dosage as well as the certainty that the B.C.G. vaccine had been absorbed. It is claimed in these experiments that the controlled doses of the B.C.G. vaccine given hypodermically indicate a relative immunity, conferring protection against tuberculosis even when exposed to it, which in some cases lasted ten years.

Protection of children by means of inoculation as mentioned by the two above named tests do not yet prove that the immunity thus acquired is more efficient or more permanent than that produced by the disease itself. Reliance must be placed on public health activities, working in two directions. First, the detection, notification and supervision of infected adults must be secured. Secondly, all child contacts must be examined to identify those that have undergone primary infection. Children so infected must be cared for during the first danger period and thereafter kept under constant and systematic supervision.

Treatment of Diabetic Coma.—Joslin and his co-workers have recently reviewed their results in the treatment of diabetic coma since 1923. Since that time 525 patients have been treated, with a total mortality of 11%, and, since 1940, 62 consecutive cases without a single death. These striking results deserve close study. The American workers find that the commonest cause of coma is laxity in treatment either through dietary indiscretions or through failure to take the proper amount of insulin; the next is infection; in a third group coma is the first recognized sign of diabetes. Death cannot be correlated with any single symptom or sign, and the prognosis can be assessed only by the consideration of many factors, particularly age, degree of shock, blood pressure, depth of acidosis, blood sugar, plasma carbon dioxide, and blood non-protein nitrogen. Of these the single most important consideration is the depth and duration of unconsciousness before the institution of treatment.

The treatment of diabetic coma is essentially a hospital procedure, and preparations must be based on the realization that it is acute medical emergency for which early, prompt, and vigorous treatment is absolutely essential. The medical, nursing, and laboratory staff should be organized to this end. As coma is due primarily to insulin insufficiency, adequate insulin dosage is the first consideration, and the first 50 units should often be given at home or on the way to hospital. On arrival the first dose should be between 20 and 100 units subcutaneously, and if the patient is unconscious and shocked a similar dose may be given intravenously. Further doses are then given half-hourly or hourly, the average patient needing at least 200 units in the first two or three hours. Subsequent dosage depends on the initial blood sugar response. If this is slight, larger doses of insulin should be administered more frequently, and it may be necessary to give 100 or more units at half-hourly or hourly intervals. The level of the blood sugar, the urinary sugar, and the clinical conditions, especially the mental state and the type of breathing—are the most valuable guides.

Next in importance in treatment is the administration of fluids and salt, and isotonic sodium chloride solution, either intravenously or subcutaneously, should be begun at once, four to five litres in the first 24 hours being an average amount. Relatively more should be given by the mouth as recovery takes place. Other important measures include gastric lavage (which should be performed carefully on admission in almost every case), enemas, and small feeds such as broth, gruel, or tea with sugar by the mouth after about the first six hours. Dextrose is contra-indicated during the first few hours of treatment because there is already excess in the body fluids,

and to give more masks the response to insulin. Alkalis are also considered unnecessary. Patients with prolonged unconsciousness and peripheral circulatory failure with very low blood pressure respond best to large doses of insulin and continuous saline infusions, with additional transfusion of plasma or whole blood in selected cases.

In summing up, Joslin and his colleagues emphasize that coma should be prevented, and that the occurrence both of coma and of death due to it shows that someone has blundered. The key to success lies in the education of the diabetic patient and of his doctor. These conclusions and their application are of profound importance. However good the actual technique of treatment in hospital or clinic, the incidence and mortality of diabetic coma will not be appreciably reduced until every diabetic patient is taught how to look after himself and when to seek further medical advice, and until every doctor knows how to manage diabetes in the home. In this country, especially in rural districts, delay in making the diagnosis and getting the patient to hospital is sometimes so considerable that the best treatment comes too late. Diabetic coma is as grave and as dangerous an emergency as an acute abdomen, and must always be treated as such.—*British Medical Journal*.

Sulfadiazine in Pneumonia.—Ensworth and his colleagues employed sulfadiazine for a group of 239 patients with typed pneumococcus pneumonia. The fatality rate was 10.9 per cent, or only 7.8 per cent, if the deaths occurring in less than twenty-four hours were excluded. There was only one death among the 113 patients who were under fifty-one years of age (1 twenty-four hour death was excluded). In spite of the fact that the blood levels (usually 7 to 9 mg. per 100 cc.) during sulfadiazine therapy were higher than those obtained with either sulapyridine or sulfathiazole, the incidence of toxic reactions was lower. Of the 239 cases, only 5 vomited and 2 of these had vomited before admission as well as afterwards. There were no instances of agranulocytosis. The number of complications of the disease was small: there were 8 cases of empyema, 5 otitis media, 5 pleural effusion and 2 of pneumococcus endocarditis. Five other patients also had miscellaneous other complications. It was concluded from the study that sulfadiazine appears to have about the same degree of effectiveness as sulapyridine and sulfathiazole in the treatment of pneumococcus pneumonia while at the same time causes fewer toxic reactions.—*United States Office of War Information*.

Comparison between Psittacosis and Atypical Virus Pneumonia.—"A cute atypical pneumonia" is defined as a form of interstitial broncho-pneumonia, not usually

accompanied by secondary bacterial invasion and characterized by brady-cardia, headache, dry cough, low or normal white count, and failure to respond to the sulfonamides. While the term may well be used where no etiologic agents have been identified, when they can be isolated from cases of this type, they are found to belong to a related group of viruses. The first isolated example of this type of pneumonia is psittacosis or ornithosis. Its history is traced from its initial description in 1879, through the discovery of its virus etiology in 1930 during the international epidemic, to the ecologic investigations of the last few years. Interstitial pneumonia has been described in cases of influenza, measles, pertussis and varicella, and in these instances may be due entirely to the action of either a virus or a bacterium, or frequently to a combination of the two. It has also been observed in the course of acute rheumatic fever (rheumatic pneumonia) and after irradiation of the thorax, and for that reason probably represent a pathologic response to a number of types of pulmonary irritants. Comparison of the incubation period, symptoms, clinical course, pathologic and laboratory findings in ornithosis (psittacosis) with those in the human atypical pneumonia shows very striking similarities. The group reveals virus studies and serologic tests results of virus studies and serologic tests in these diseases also indicate this close relationship. One probable and 2 definite cases of ornithosis observed in the course of eighteen months in Boston are presented by Favours. He emphasizes the frequency with which ornithosis virus may cause atypical pneumonia in man. Careful questioning about contact with varieties of birds known to harbor the virus, and complement-fixation tests on acute and convalescent sera should be performed in order to make the diagnosis. He suggests that cases of human atypical pneumonia from which no etiologic agents have been isolated may be due to a virus of the psittacosis group which has become fixed in man and is usually incapable of heterogeneous parasitism.—*U.S.O.W.I.*

Hemorrhage from the Trachea, Bronchi and Lungs of Non-Tuberculous Origin.—According to Jackson and Diamond, 50 years ago hemorrhage from the trachea, bronchial tree or the lungs was regarded as being pathognomonic of pulmonary tuberculosis. To-day, while tuberculous pneumonia still continues to be responsible for more cases of blood-spitting than any other disease, the prominent roles played by cardiovascular diseases, neoplasms and other non-tuberculous processes in the etiology of non-tuberculous bleeding are being more broncho-pulmonary bleeding are being more clearly recognized. The study and more clearly recognized. The study reported is based on a survey of patients with hemoptysis observed in the bronchoscopic clinic at Temple University Hospital in Philadelphia from January 1, 1930 to July 1, 1941. The number of patients finally selected for study was 436. Only patients seen by the bronchoscopist were included in the analysis. Inflammatory

processes were found responsible for the hemorrhage in the majority of the cases, the most common etiologic agent being bronchiectasis. Bronchial dilatations were demonstrated in more than one-third of the patients who had massive hemoptyses and in fully two-thirds of those whose initial attacks of blood-spitting occurred before they were twenty years of age. Nevertheless, in a study of the relative hemorrhagic tendencies of various non-tuberculous bronchopulmonary lesions, it was found that expectoration of blood occurred actually in a smaller percentage of bronchiectatic patients than in patients with primary carcinoma of the bronchus, in those with pulmonary abscess and in those with bronchial adenoma. Fatal hemoptysis occurred in but three of the patients in the series, each of whom had a pulmonary abscess. A hemorrhagic focus could be seen on bronchoscopic examination in 25 per cent. of the patients. Nearly all of the neoplasms causing hemoptysis arose in major bronchi and thus were readily apparent endoscopically. In only a negligible number of inflammatory lesions, however, was it possible to detect a definite bleeding point. There was no endoscopic evidence of recent or active bleeding from a vascular lesion in the tracheo-bronchial tree in any of the patients. The precise cause of the hemoptysis could not be determined in nearly one-fourth of the patients. These were patients in whom no parenchymal disease was demonstrable on roentgenography and no bleeding point evident on bronchoscopy. The likelihood of existence of ulcerative lesions in the smaller bronchi of these patients must be considered. Despite the fact that in fully 90 per cent. of the patients with hemorrhagic bronchial cancer the neoplasm was situated in one of the larger, bronchoscopically accessible bronchi and that bronchoscopy was performed in these patients soon after the occurrence of the first episode of blood-spitting, the process was found to be too far advanced to permit surgical cure in all but one. Taking into consideration the fact that expectoration of blood is the initial manifestation of carcinoma of the bronchus in only a very small percentage of the patients, it is obvious that bronchoscopy must be done and the diagnosis made early in the course of the disease, before the symptoms have reached the hemotysic stage, if a successful therapeutic result is to be achieved in these cases.—*U. S. O. W. I.*

Dilantin Sodium and Phenobarbital Combined for Epilepsy.—Merritt and Brenner report observations on 100 patients with convulsive seizures whose attacks had not been satisfactorily controlled by the administration of dilantin sodium, who were then treated by a combination of dilantin sodium and phenobarbital. Thirteen patients were markedly improved on the combination as compared with dilantin sodium alone, 21 patients were moderately improved, while the remaining 66 patients showed no significant

improvement. All of the patients with exclusively Petit mal or Psychomotor seizures or with Jacksonian seizures were in the group which showed no significant improvement after the addition of the phenobarbital. Thirty-seven patients with convulsive seizures who had never before received dilantin sodium but whose attacks had been unsatisfactorily controlled by other forms of treatment were treated by a combination of dilantin sodium and phenobarbital. The results in this small group of patients were roughly equivalent to those reported by Merritt and Putnam for similar larger groups treated by dilantin sodium. Patients with convulsive seizures whose attacks are not satisfactorily controlled by dilantin sodium alone should be tried on a combination of this drug with phenobarbital. To obtain satisfactory results, they say, with the combination of phenobarbital and dilantin sodium, full dosages of both drugs must be administered.

—U. S. O. W. I.*

Virus Pneumonia.—Dr. Kamin (Illinois Medical Journal, January, 1943) says that the incubation period varies from five to fifteen days. The onset may be sudden with a rigor, but usually is gradual, with lassitude, marked weakness and severe headache. Hoarseness may develop during the first few days. After the third or fourth day a dry cough makes its appearance. This later becomes paroxysmal in character, somewhat productive, and may last for several weeks after the patient's temperature has become normal. The patient is markedly prostrated, but does not appear to be very ill or toxic. The breathing is neither increased nor laboured. There is usually no cyanosis present. The temperature varies between 100° and 104° Fahr. The pulse is only slightly increased in rate. The temperature persists from seven to fourteen days. In rare instances it may last as long as from three to six weeks. Temperature relapses are common. Physical examination of the chest reveals very little in the early stage of the illness. Later on slight impairment of resonance may be detected in one or more areas. This is usually associated with some fine inspiratory rales. By the end of the second week the findings, although not marked, are diffuse and bilateral as a rule. Only in unusual cases do we find definite dullness and marked bronchial breathing. Practically all patients recover and complications are rare. Chemotherapy has no effect on the course of the disease.—Medical World.

Treatment of Asthmatic Paroxysm with Nicotinic Acid.—Since June, 1941, a group of 30 severe ambulatory patients have been treated with nicotinic acid by Fred. E. Maisel and Eugene Somkin (Journal of Allergy, May, 1942). These 30 patients were classified as follows: In 10 the asthma was associated with a sinus, bronchial, or pulmonary infection; in 3 it was due to various

inhalants and pollens; in 4 it was due to a combination of infection plus a sensitivity to inhalants and pollens; in 5 the asthma was associated with a cardiac deficiency. In the remaining 8 asthmatic patients no definite etiologic factors could be ascertained.

In order to study the effect of nicotinic acid on the asthmatic paroxysm, 21 patients received intravenous injections of 0.1 gm. of nicotinic acid while 9 patients were given only oral treatment. The oral treatment consisted of doses of 0.2 gm. of nicotinic acid before meals and on retiring. Supplementary oral therapy to be taken at home was also administered to 18 of the 21 patients who received intravenous therapy. During the period of treatment no other form of medication was given.

Analysis of the results obtained in the 21 patients who received intravenous injections of nicotinic acid for the relief of asthmatic paroxysms indicates that 16 or 76 per cent. were definitely improved from varying periods of 3 to 15 hours. Although all the rhonchi in the lungs did not disappear following the administration of nicotinic acid, in most of these patients the degree of subjective relief that ensued was marked.

Of the 9 patients receiving only oral therapy, 5 were likewise benefited. Freedom from attacks was maintained or attacks became definitely less severe and recurred with less frequency.

Of the 18 patients who received intravenous therapy followed by oral therapy, 11 showed decided improvement in their asthma.

Nicotinic acid orally was therefore efficacious in about 60 per cent. of the patients both for the control of acute attacks and for the continuous wheezing.

The clinical manifestations following the administration of nicotinic acid are characterized by a flush and sensation of heat. The flush occurs 3 to 5 minutes after an intravenous injection and is confined mainly to the face, neck, chest, and upper arms. The sensation of heat precedes the flush and is of shorter duration. The patients who experienced relief following the appearance of the flush breathe more freely. They often begin to cough and expel tenacious mucous plugs. In the group studied no patient developed any disturbing symptom other than the flush and the drug was well tolerated. Important likewise is that the efficacy of nicotinic acid was not affected by repeated and continuous use.

While nicotinic acid is not offered as a substitute for ephedrine or epinephrine, it is suggested as an adjuvant in the therapeutic armamentarium in the treatment of asthma. It may serve as a substitute when ephedrine is ineffective or when the patient becomes epinephrine fast.—G. P.

Vitamin Therapy.—O'Leary points out that certain cutaneous diseases are due to avitaminosis and that after the supplemental use of the necessary vitamin or

vitamins, either by proper diet or by the administration of a synthetic form, the cutaneous signs of the disease disappear. The diseases include pellagra, ariboflavinosis or cheilosis, phrynodema, pityriasis rubra pilaris, keratosis follicularis and the so-called seborrheids (nonpellagrous eruptions) recently described by Gross. Another group of diseases in which the results of vitamin therapy are less pronounced, do not appear in all cases and are less conclusive includes lichen spinulosus, keratosis pilaris, senile vaginitis, monilia infections, rosacea keratitis, psoriasis, acne cachecticorum and "dry skin." The evidence now indicates that under certain conditions vitamins to be effective must be in harmony or in combination, as, for example, in pellagra, in which nicotinic acid, thiamine and riboflavine is necessary to control the disease. The author believes that other diseases, such as psoriasis, may be completely controlled when the proper combination and ratio of vitamins is found. This would probably explain some of the discrepancies in the literature and differences of opinion as to the value of vitamin therapy in dermatology. No significant benefit has been obtained with vitamins in the various cutaneous manifestations of syphilis. The most pronounced benefits occur when a dermatosis is associated with constitutional signs of avitaminosis.

—J. A. M. A.

Chest Pains in Patients with Mitral Stenosis.—A. M. Burgess and L. B. Ellis (New England Journal of Medicine, 226: 937, June 11, 1942) report 6 cases illustrating the different types of chest pain that may occur

with mitral stenosis. The four types of pain that occurred in these cases were a cardiac neurosis, the pain of acute rheumatic carditis, angina pectoris, and so-called hypercyanotic angina. The pain of cardiac neurosis is of a very variable nature, usually felt in the apical region, but sometimes over the precordium; it has no relation to exertion and often persists for hours "or even days." when this type of pain occurs with mitral stenosis it is associated, with an anxiety state due to the patient's knowledge of heart disease and its dangers and with other neurotic symptoms. Patients with mitral stenosis who develop active rheumatic carditis during acute recurrences of rheumatic fever may develop chest pain during the acute attack, which is relieved when the acute symptoms subside; one such case is reported. True angina pectoris is rare in mitral stenosis, but may occur; one case of this type is reported. A severe type of precordial pain, sometimes called "hypercyanotic angina," usually occurs in advanced cases of mitral stenosis; the pain clinically resembles that of acute myocardial infarction, but is not accompanied by other symptoms of infarction—fever, leukocytosis and electrocardiographic changes. The accompanying cyanosis is often extreme and dyspnea and orthopnea severe, but the pulmonary congestion may be slight as the attacks of pain are not necessarily associated with congestive heart failure. In the 3 cases of this type reported, the patients died during or immediately after an attack of pain of this type, but not at the time of the first attack. Autopsy was not permitted in one of these cases; in the other 2 cases there was no evidence of myocardial infarction.—Medical Times.

Surgery

Crush Syndrome.—By John Almedyda, M.R.C.S., D.P.H., Senr. Clinical Assistant, Royal Chest Hospital.

Definition.—It is a condition of renal impairment or failure following a crush injury. The recognition of this entity in the present war has been made easier for three reasons:

- (1) Aerial bombing has produced many instances of pinning beneath wreckage, with persistent compression of lower limbs for many hours.
- (2) It tends to be masked by, and is regarded as, a sequel of shock.
- (3) It could be looked upon as resulting from the transfusion of unsuitable blood.

Historical.—The condition was well recognized in Germany in the last war (Frankenthal, 1916) and a large literature has existed there. A full description was written by Minami (1923), who also pointed out the analogy with equine paralytic myohemoglobinuria. No reference to the syndrome in the English literature had appeared until Bywaters and Reall (1941) published four cases of air-raid casualties under the title

"Crush Injuries with Impairment of Renal Function."

Our knowledge of the syndrome is still meagre, but during the past two years, interest in it has considerably increased. It is now appreciated that if a person has a pinning accident in which a limb becomes crushed for some hours, he may upon release show little evidence of shock, and yet some days later, whether he has had a transfusion or not, die rather suddenly from renal failure. A similar picture can be experimentally produced in animals, by placing a tourniquet around a limb for a time and then releasing it. The autolytic products of the crushed muscles produce either a purely mechanical (blocking) or else a toxic effect on the renal tubules.

Pathogenesis.—The precise pathogenesis of the condition is still unsettled and various views have been put forward by different investigators. Bywaters and Reall (1941) consider the condition due to products of muscle necrosis. T. R. Gilmour (1941) attributes the syndrome to myohemoglobin and toxic substances formed from the crushed muscles. Patey and Robertson (1941-42) do not think that the toxic bodies liberated

*United States Office of War Information.

from the damaged muscles are the cause, but that there is loss of "substance," a depletion of plasma protein, from the circulation into the damaged and swollen limb. They agree, however, that this view does not explain readily the renal failure. *Minami* (1923) has pointed out the analogy with equine paralytic myoglobinuria; a view which *Gilmour* put forward later. *Workers at the London Hospital* (1941) believe that this may be due to pressure of deep fascial compartments, producing tissue damage. They concluded that muscles in strong fascial compartments which are non-contractile show necrosis in 33 hours after release, while other muscles, though pale and watery, which contract normally, showed moderate necrosis only. *H. Miller* (1941) considers that the liberation of lactic acid into the circulation from the damaged muscle is a major factor in this phenomenon. At the *Royal Society of Medicine* (1942) discussion, two views were expressed: (a) shock and renal damage, two aspects of the same primary lesion (i.e., muscle necrosis), although their obvious results are separated in time by seven days. The shock is due to the plasma leaving the blood stream, and entering the injured area, and the renal damage due to the substances such as myohemoglobin and acid metabolites leaving the damaged area and entering the general circulation as soon as the blood supply is re-established; (b) shock and renal manifestations, the result not of toxic bodies liberated from the damaged muscles, but simply of the loss of blood constituents into the injured limb. *Egginton, Richardson, Schild and Winters* (1942), in their recent experiments on dogs, suggest that the increase in permeability of renal tubules is due to released toxic agents and that the reduction in the creatinine clearance involves a reduction in the glomerular filtration rate by lowering the pressure.

Clinical Picture.—The patient gives a history of being pinned down for several hours with persistent pressure on one or more of his limbs. Upon release his condition appears good, although he is in the first stage of shock. There is slight swelling of the crushed limb, with some local patchy anaesthesia. After a few hours the second stage of shock sets in. The urine becomes diminished and slightly acid. The blood-pressure tends to fall, while the haemoglobin percentage, if anything, shows a rise. Transfusions of serum, plasma or blood at this stage raise the blood pressure.

All appears well with the patient for the next two or three days, until the urinary output, small at first due to the initial shock, becomes more diminished (30 ozs.) and acid. The urine passed is bloody and contains albumin, creatinine, and dark brown or black granular casts. The compressed limb is by now swollen hard and tense. There is patchy local anaesthesia, and the skin shows petechial haemorrhages, whealing and blisters. The circulation shows diminution of the arterial pulsation distally, accompanied by changes of incipient gangrene.

Pain in the loins, a sign of renal damage, soon appears, usually about the fourth to fifth day, and progresses even if the affected limb be amputated. The patient becomes by now, drowsy and anxious-looking. He has slight generalised oedema, thirst, and incessant vomiting, in other words, a picture of uraemia. The blood-pressure shows a rise by now and there is a progressive mounting up of the blood urea, serum potassium and phosphates.

The critical stage is reached at the end of the first week. If the injury is not severe, the patient may recover with sudden diuresis, with diminution in the blood-urea and an improvement in the urea clearance test. The crushed limb may show the pressure necrosis syndrome with or without infection, terminating in a restitution fibrosis, resembling a Volkmann's ischaemic contracture. If the injury is severe, the patient may show cardiac irregularity and electrocardiograms not unlike those of experimental potassium poisoning. Death will occur suddenly within a few minutes.

Pathology.—The post-mortem findings are those of necrosis of the crushed muscles and degenerative changes in the adrenals and the renal tubules. The latter are packed with casts containing brown pigments. There may be other traumatic lesions such as fat embolism, contusion or blast lung, and mid-brain haemorrhage.

The kidneys are enlarged, dark red and firm with dense capsules due to swellings of the cortices. On section, the cortex is widened, it wells up on the cut surface and is usually well demarcated from the medulla, the boundary layer of which is always deeply congested. It is either of a grey or pale grey-pink colour throughout. Microscopically, the main changes are noted in the tubules. The convoluted tubules and the loops of Henle are severely damaged. The cytoplasm of the lining epithelium is swollen, granular and vacuolated. In the loops of the Henle and the collecting tubules there are many casts of dense eosinophilic material of dark brownish colour, due to bile or blood pigments. The glomeruli show no obvious structural changes. The tufts contain relatively little blood, but the vessels of the medullary part are greatly engorged.

The renal lesion thus consists structurally of severe degenerative changes in the proximal convoluted tubules, and in the more distant parts of the nephron, brown pigmented casts like blood cells. The pigment can be accounted for by excretion into the lumen, from the blood stream, of haemoglobin, myohemoglobin or bile pigment, but not red corpuscles. The urine resembles a glomerular filtrate. There is no concentration of urea. No relative absorption of water, and some failure to re-absorb chlorides, i.e., a dysfunction of the convoluted tubules.

The compressed muscles show one or more types of change. Necrosis and friability of parts of muscle resembling fish flesh, due to direct pressure ischaemia; necrosis of

whole muscles, placed in tight fascial compartments; or patchy necrosis of isolated muscle fibres due to periarterial haemorrhage or direct arterial damage.

Prognosis.—The prognosis depends on the amount of muscle necrosis, the duration of compression and the temperature involved. After the initial shock, the prognosis will depend on the renal damage involved. Upper limb crushes have a better prognosis than lower limb crushes. The quicker treatment is installed the better the ultimate result. The degree of shock and uraemia are other factors that are guides in prognosis. About one third of the cases recover. The degree of haemoconcentration, oliguria, and blood urea rise, are other factors that are of prognostic significance.

Treatment.—As the renal damage occurs soon after the re-establishment of an adequate blood supply to the damaged limb, treatment should be aimed to prevent—

- The release of mass toxins present in the compressed limb, getting into the circulation.
- Shock.
- Renal failure.

(a) *Curphey* (1941) suggests that an Esmarch's bandage be applied to the affected limb, directly after the victim's extrication, in order to prevent the mass release of the toxins. To save premature amputation the bandage should be released inch by inch. *Patey and Robertson* (1942) aimed at restoring the extravasated fluid from the oedematous limb into the circulation, by intermittent positive pressure up to 60 mm. of Hg. applied through a specially made large blood-pressure cuff firmly bandaged to the limb, and connected with a Pavex motor. Because of the scarcity of these motors they are now investigating the possibility of developing a simple water pump, which fulfils the same purpose. This method has been confirmed experimentally by *Duncan and Blalock* (1942). It has been suggested by others that the crushed limb could be comfortably kept packed in ice, in order to diminish the rate of tissue breakdown, consequent upon enzymic action, which is accelerated by heat.

(b) The next step is to restore the circulation by combating shock. This can be done by giving plasma, serum or blood.

Early amputation of the compressed limb if oedematous is only indicated for purely surgical reasons. A case for such treatment must not be dictated, or made justifiable on the grounds of its association with the compression syndrome.

(c) Copious alkaline fluids should be administered by mouth, rectally, intramuscularly, or by intravenous drip, as early as possible to keep the urine alkaline, and reduce the liability to precipitation within the renal tubules. This will promote diuresis and thereby combat uraemia. The substances used are 2 per cent. (200/300 gr.

in 1,000 cc. of fluid) of sodium citrate, sodium sulphate or sodium chloride with glucose. —*Medical World.*

Treatment of Ischaemic Contracture.—Volkmann's ischaemic contracture was originally thought to be due to circulatory obstruction resulting from the over-tight splinting or bandaging of fractures in the region of the elbow-joint. This theory was upset by the clinical evidence, however, because many cases were reported in which neither splints nor bandages had been applied. So the more modern version of the mechanical obstruction theory was propounded. This supposed the obstruction to be due to internal pressure from a subfascial effusion or haematoma in the antecubital fossa, and to be venous rather than arterial. *Brooks* showed experimentally, as later did *Middleton*, that if the entire venous drainage from a muscle were ligated the muscle developed a contracture. This they regarded as being identical with Volkmann's but *Griffiths* demonstrated conclusively that the histology of the two conditions was entirely different, and in the same paper produced further evidence against the venous occlusion theory by recording three cases in which the venous system was completely unaffected.

What, then, is the essential pathology of Volkmann's contracture? According to *Griffiths*—and his clinical and experimental evidence is most convincing—it is due to segmental spasm of the main artery to a limb, accompanied by reflex spasm of the collateral circulation. If the main artery alone is occluded and the collateral circulation functions, then no ill effects are observed. If the collaterals go into reflex spasm, however, the result is either gangrene or Volkmann's contracture according to whether the spasm is complete or incomplete. *Foix*, reviewing the early operative findings in 30 cases, reported from many sources during the last two years, points to their striking similarity. In all, the lower end of the brachial artery was reduced by spasm to a string-like structure in which pulsation had ceased. *Barnes and Trueta* have now brought forward additional experimental evidence in support of the earlier work of *Griffiths*. By applying a wire tourniquet for four hours they were able to produce a persisting spasm of femoral artery in rabbits, and showed by arteriographic studies that the collateral circulation was also in spasm. They further found that the spasm of the collaterals could be abolished by lumbar sympathectomy, though the main vessel remained in spasm, and that in the rabbits in which the collateral circulation was not released in this way changes occurred in the muscles identical with those seen in Volkmann's contracture.

It is obvious, therefore, that the crucial factor is the reflex spasm of the collateral circulation. It seems established that the impulses causing this spasm originate in the walls of the occluded main vessel, but their subsequent path has not been precisely

determined. The travel by way of the sympathetic system, but in human being, at all events, they do not seem always to be relayed through the paravertebral ganglia. Successful blocking of the stellate ganglion, as evidenced by the development of a typical Horner syndrome, has failed to release the collateral circulation, so there must be a more peripheral reflex path. The most certain method yet known of abolishing the reflex is to interrupt it at the source by resecting the segment of main artery in spasm, a procedure originally proposed by Leriche. This operation (arteriectomy) has proved reliable in all the published cases and is the one of choice, but it must not be delayed too long. Tight plasters or bandages, or overflexion of the elbow-joint, are factors that cause additional circulatory embarrassment and should be removed. Moreover, in some cases, notably in gunshot wounds, in which the damage is confined to the soft tissues, the reflex spasm of the collaterals may pass off spontaneously. But faced with the possibility of such a severely crippling condition as Volkmann's contracture the surgeon should not wait long now that he has this valuable weapon to hand.—*British Medical Journal*, Jan. 23, 1943, Page-108.

Anæsthesia for Laryngofissure.—In view of the growing popularity of surgical treatment of carcinoma of the larynx it is important that an adequate anæsthetic technique should be evolved. In the past preliminary tracheotomy, with its attendant risk of chest complications, has usually been performed. The suggested method does away with the necessity for a tracheotomy and ensures post-operative hæmostasis. In a recent series of 8 cases performed by Mr. Charles Keogh there were no post-operative chest complications.

Method:—Premedication: omnopon 1/3 gr. and scopolamine 1/150 gr. 1 hour before operation. Induction: anæsthesia is induced in the normal way with N₂ O, O₂, and trilene using a Boyle apparatus. When the patient is well into the third stage of anæsthesia a stiff gum-elastic intratracheal catheter, St. Bart's pattern, size 12-14, is passed through the larynx into the trachea under direct vision. Great care is taken to avoid the growth during this manoeuvre. The proximal end of the catheter is now connected to the machine and anæsthesia is maintained with O₂ and trilene. Owing to the small bore of the tube it is usually necessary to supplement the volume of oxygen reaching the lungs by gentle intermittent pressure on the rebreathing bag with the expiratory valve fairly tightly screwed down.

Operation:—As soon as the incision has been made and the larynx opened the space between the catheter and the trachea is thoroughly packed off with narrow gauze, leaving one end emerging from the lower part of the wound. This ensures that no blood leaks down the trachea during the operation. The catheter is now found to lie

in the posterior commissure of the larynx and usually interferes very little with the surgical procedure. If it proves to be in the way, then it may be retracted one way or the other. In only one case, where the growth was quite extensive, had this proved necessary. Anæsthesia is maintained as before with O₂ and trilene, aided by manual pressure on the rebreathing bag at each inspiration. Some of the gas percolates back through the packing in the trachea. As trilene is non-inflammable the diathermy may be used if necessary.

Hæmostasis:—Following excision of the growth it is often very difficult to achieve perfect hæmostasis. After removal of the tracheal packing and catheter the anæsthetist may play his part by passing a large-bore Magill tube, size 8-9, with inflatable cuff, through the nose and into the trachea under direct vision. When the cuff lies in the larynx it is inflated through the pilot tube, until it occupies the lumen of the larynx and presses up against the field of operation. The laryngofissure is now sewn up, and the patient returns to the ward with a clear airway and a guarantee of hæmostasis.

Post-operative Treatment:—The intratracheal tube is left in position for 8 hours. During this time the patient is given morphine, and a little 2% decaline solution is sprayed down the tube from time to time. In only one case did the tube have to be withdrawn earlier than was desirable. In this case it had been passed through the mouth. Provided it is passed through the nose very little discomfort is experienced. Eight hours after the operation the laryngeal cuff is deflated. After waiting a few minutes the patient is asked to cough. If blood is coughed up through the tube, then the cuff is re-inflated and the same procedure repeated in 2 hours. If no blood appears, then the tube is gently but firmly withdrawn. Even this procedure causes, remarkably little discomfort to the patient.—*British Medical Journal*.

Operation for the Orchitis of Mumps.—The orchitis caused by the virus of mumps may be as acute as the epididymo-orchitis caused by the gonococcus. Within a few hours the testes swells to three or four times its normal size and becomes so acutely sensitive that the patient cannot bear to have it touched. The infection is often associated with a high temperature and sometimes even with delirium. What is still more important is that the acute infection is followed in 54.7% of cases by testicular atrophy due to cell necrosis brought about by the great rise in intratesticular pressure. In 1864 Henry Smith advocated that an acute epididymitis due to gonorrhœa should be treated by multiple punctures of the tunica albuginea. A. Wessel hæft and S. N. Vose recommend that the same method should be applied to the severe orchitis in mumps. It would indeed seem that relief of the increased intratesticular pressure is more necessary in the case of mumps than in that of gonorrhœa.

because the risk of subsequent atrophy in the former case is far greater. The writers emphasize the necessity for intervention before the inflammatory process has reached its peak. In the 9 cases in which they operated sufficiently early the testicle recovered without undergoing subsequent atrophy. In the one instance in which incision was postponed some degree of atrophy followed. For multiple punctures they substitute complete exposure of the swollen organ and a longitudinal, or sometimes a crucial, incision of the tunica albuginea. The operation not only affords an immediate relief of pain but also brings about a rapid fall in the temperature. It is only necessary in the fulminating type of cases, which constituted 10 out of the total of 24 cases reported upon. Atrophy is only likely to follow the very acute infections, and the less severe cases need not be subjected to operation.—*British Medical Journal*, Jan. 30, 1943, Page 138.

Acute Mechanical Intestinal Obstruction.—Romano (*New Orleans Medical and Surgical Journal*, 1941: 94) summarizes his observations on the management of acute mechanical intestinal obstruction: 1. Acute mechanical obstruction demands immediate operation. 2. Pre-operative decompression continued over a long period of time by means of nasal tubes is a dangerous procedure. 3. During the hour or two of delay necessary for setting up the operating room and preparing the patient, gastric suction with a Levin tube or a Miller-Abbott tube should be employed, infusions of sodium chloride and glucose should be given and the patient's blood should be matched for transfusion. 4. The obstruction must be released or removed, but the principle of doing the least possible amount of surgery should be followed. The non-viable bowel must be removed but primary anastomosis should seldom be attempted. 5. Unusual care must be exercised in handling the dilated bowel to avoid tearing with consequent spillage. 6. Enterostomy is contra-indicated; ileostomy in the proximal loop has no advantage over a Miller-Abbott tube in the same place and jejunostomy has no advantage over gastro-duodenal suction drainage.

In obstruction of the large bowel due to carcinoma, colostomy and cecostomy should be done to relieve pressure on the cecum. Ileostomy is not indicated as a decompressive measure for obstruction of the large bowel. 7. Post-operative decompression by means of the Miller-Abbott tube is beneficial regardless of what is found and of what procedure is performed. Nasal oxygen should be administered post-operatively, both for its decompressive effect and to combat anoxæmia and shock. Sodium chloride and glucose solution should be given routinely. Transfusion of blood or plasma will save many patients who have strangulated obstructions.—G. P.

Treatment of Shock by Action on Nervous Centres.—Stern points out that the ordinary methods of combating shock generally do not give positive results because they do not influence the condition of the central nervous system. These negative results may be explained by the presence of the hæmatencephalic barrier. Medicinal substances introduced into the circulation have no effect on the central nervous system, whereas the introduction directly into the cerebrospinal canal, particularly into the ventricles, often has a decided effect. To obtain a positive effect the substance should be introduced intrathecally. This applies particularly to substances normally present in the cerebrospinal fluid and the blood, such as potassium, calcium salts and certain hormones. The antagonism between the central and peripheral parts of the vegetative nervous system, as regards their reaction to the same substance, explains the absence of a positive antishock action of medicinal substances when introduced directly into the circulation. Actually, if these substances do penetrate the central nervous system, particularly the vegetative centres, they may produce an opposite effect and thereby neutralize the immediate action which they have produced on the peripheral vegetative organs. To produce a direct action on the vegetative nervous centres the substance should be administered directly into the cerebrospinal fluid. The given substance should be introduced into the ventricles of the brain, as its introduction into the subdural or subarachnoid space is usually without effect because it passes rapidly from the cerebrospinal fluid into the blood stream and does not come into contact with the nervous centres. In the last phase of traumatic shock there is first a considerable fall in the tone of the sympathetic nervous system and a partial rise in the parasympathetic tone. The most effective way of combating traumatic shock in its later phase is to raise the tone of the sympathetic centres by direct action of the vegetative centres; potassium phosphate does this simultaneously. By direct action on the nervous centres the ionic potassium causes an excitability of the sympathetic nervous centres, whereas the ionic phosphate, by concentrating the ionized calcium, decreases the tone of the parasympathetic nervous centres. Potassium phosphate as an antishock remedy is indicated in all those cases in which the tone of the sympathetic nervous centres is decreased. Potassium phosphate must never be used in cases in which the excitability of the sympathetic nervous centres is increased, such as sometimes occurs in the first stage of shock. If hæmorrhage is present it must be arrested, because the rise in blood pressure from potassium phosphate will definitely increase it. To avoid a repetition of shock after potassium phosphate is given, the actual cause of shock, in particular painful stimuli, must be removed. Potassium phosphate is introduced directly into the ventricles by cisternal puncture.

The fluid (1 cc. of a solution of potassium phosphate in a concentration of 1/6 gram molecule with a pH of 7.6) must be introduced with a definite pressure and the direction of the needle must be correct. The patient must be in the horizontal position preferably on the left side, with the head lower than the body.—J. A. M. A.

Dissolution of Urinary Calculi by Citrate Solution.—A report has been made on the dissolution of phosphatic urinary stones in patients in whom a citrate solution containing magnesium ions was employed. The solution contained citric acid (monohydrated) 32.25 g.m., magnesium oxide (anhydrous) 3.84 gm., sodium carbonate (anhydrous) 4.37 gm. and water q.s. ad 1000 cc. The citrate solution in this formula is rendered less irritable by the addition of magnesium. This solution was found practical for the dissolution of urinary calculi composed of calcium phosphate, calcium carbonate or magnesium ammonium phosphate. Six selected cases are reported in which such a solution was used successfully for the partial or complete dissolution of stones; in 2 cases the solution was introduced through ureteral catheters, in the remaining four through nephrostomy tubes. One case is reported in which attempted dissolution of a large stone was without success because the solution was prevented from coming in contact with the stone by a thin coating of unidentified material, possibly old blood clot. The choice of apparatus depends on the individual case and the ingenuity of the operator. Ideally the hydrodynamics should be such that the pressure in the kidney is great enough to get the solution around the stone, but sufficiently intermittent so that a pyelonephritis is not produced by a constant back pressure. For determining whether a stone is in contact with the kidney pelvis and for following the course of dissolution, the advantages of an air pyelogram are stressed. The importance of the roentgenogram in determining whether the stone is of the soluble variety is pointed out; a case is cited in which the centre of the stone was calcium oxalate and hence indissoluble, a finding that could have been determined by suitable x-ray technic.—U.S.O.W.I.*

Ruptured Intervertebral Disk.—The term "lumbago" is rapidly falling into disuse as a nondescript designation for ill-defined pains in the lower spinal region. Unknown as an entity but twelve years ago, ruptured intervertebral disks are recognized to-day as the cause of a remarkably high percentage of hitherto intractable and incurable low back pains and sciaticas. Neurosurgeons have now precisely defined the

symptomatology and pathology, and perfected an operative technic that not only relieves but cures virtually all back disorders in this category.

The symptoms are low back pain in recurring attacks, some very severe and disabling, the pain radiating down the posterior aspect of the legs. An interesting as well as a diagnostic feature is the intensification of the pain on sneezing, coughing and straining of the abdominal musculature. The spine is stiff, relatively immobile, for movements aggravate the pain. Lasague's sign is usually positive. Neurologic manifestations must be carefully sought, as the Achilles reflex and occasionally the knee jerk, may be diminished or even be absent. Sensory disturbances are not uncommon, consisting of hyperaesthesias of a segmental distribution, and paraesthesias. Previously, contrast media were used to localize and demonstrate the lesion radiologically, but this procedure is now deprecated, for extensive investigations have demonstrated that 96 per cent. of these cases are situated in the region of the fourth and fifth lumbar vertebrae. This significant finding obviates the necessity for contrast x-ray studies, with the attending disadvantages. Needless to say pelvic, rectal, and neighbouring osseous pathology must be excluded before a diagnosis of ruptured intervertebral disks can be safely made.

The operative technic has been simplified so that the original bilateral laminectomy has been replaced by a method in which the neural arch is either preserved or untouched, with rare exceptions. After adequate exposure, the necrotic intervertebral disks, the result of trauma and not of infection, are curetted. In about 25 per cent. of the cases the necrotic intervertebral disk does not protrude and hence is not easily visible, but is concealed and must be searched for and emptied. Not all of the pathologic disk need be gouged out, but free egress for its ultimate protrusion should be provided, as in the adequate opening and drainage of an abscess. After the operation, a brace should be worn for three months. Recurrences are unusual after operation, so that once a cure is attained it may be regarded as final.

A crippling and disabling ailment is now amenable to surgical therapy. For those who habitually suffer from agonizing back pain with sciatic symptoms an avenue of cure is now opened. As in all disorders that cause disability and painful inefficiency, the cure of ruptured intervertebral disks will not only make many persons happy but will also make them more useful.—*New York State J.M.* 42: 2197-2198, Dec. 1, 1942. N. W. Medicine.

Obstetrics and Gynecology

Spontaneous Abortion.—Mason discusses the nineteen abortions that occurred among three hundred and eleven consecutive preg-

nancies cared for by him in private practice. Six of the abortions occurred in 2 patients, who had three each. This incidence of

6 per cent. is considerably less than is generally reported. All the women were in comfortable circumstances, usually reported early for antepartum care, had adequate diets and received thyroid or iodine if it was indicated by a basal metabolism test or the history, and foci of infection were cared for as soon as possible. If 50 to 80 per cent. of abortions are pathologic and if the chief cause is defective germ plasm, somatic factors should play no part. However, if the maternal environment is a major etiologic factor a comfortable life and preventive treatment ought to make considerable difference. The low abortion rate in the present series, together with an apparent considerable success in the treatment of abortion in these women, would seem to lend confirmation to the latter view. Among the series of pregnancies, thirty-four "threatened" abortions were treated. In 30, or 82 per cent. the abortion did not occur and the patients were carried to term. Four aborted in spite of treatment, but 1 of these went to term in her next pregnancy; prophylactic treatment was begun shortly after the first missed menstrual period. The other 3 of the 4 were lost sight of. In addition there were 17 patients who were treated prothylactically from the beginning of their pregnancies. These patients gave a history of having had one to three previous consecutive abortions. Of these 17 patients, 15 were carried to term the first time such prophylactic treatment was given. All gave birth to normal babies, who continued to develop normally thereafter. The two failures are represented by the 2 patients who figured in six of the total nineteen abortions. In addition to all other measures which constitute good antepartum care, the only other agent used in the treatment was progesterone (proluton). Morphine was not used. The progesterone was given in 5 mg doses (intramuscularly in oil) to patients with threatened abortion. If the symptoms were slight, this dose was given once a day; but if the symptoms were more severe, 5 mg. was given twice a day for several days. When cramps and/or bleeding ceased the progesterone was not discontinued immediately, but the 5 mg. dose was usually given every other day for several days. The dose was then reduced to 2 mg and continued for a week or more; then the intervals were lengthened and the injections were continued perhaps two more weeks. Patients with a history of recurrent abortion were given a history of recurrent abortion were given progesterone therapy as soon as pregnancy was diagnosed. The patient with a history of two previous abortions and no successful pregnancy should be treated through the seventh month of pregnancy.—J. A. M. A.

Endometriosis.—By this term is meant the lodgment of endometrial tissue in various parts of the genital tract. Dissemination is probably by lymphatic metastasis. The most frequent pathological lesion associated with endometriosis is fibromyoma. This tumour may represent the irritative

reaction to a preceding endometriosis. The symptoms met with in cases of endometriosis vary. There may be dysmenorrhoea, menorrhagia, metrorrhagia, lower abdominal pain, sacral back pains, bladder and gastrointestinal disturbances, a sensation of pressure in the pelvis and nervousness. Sterility is very common.

Dr. W. R. Holmes (*American Journal of Obstetrics and Gynecology*, Jan., 1942) states that the diagnosis of endometriosis is not always easily made; but the following symptoms, if present in the patient's history, are of diagnostic value: (1) dysmenorrhoea of an acquired type, or an accentuation of a pre-existing dysmenorrhoea; (2) premenstrual pain in the lower abdomen resembling menstrual cramps beginning a few days or even a week or more before the onset of the period; (3) abnormal uterine bleeding sometimes intermenstrual and consisting of a brownish-coloured discharge; (4) pain in one or both lower abdominal quadrants or suprapubic areas which may be constant, but which is often periodic, with a sensation as if everything were falling out of the pelvis; (5) pain referred to the groin, hip or thighs; (6) acquired dyspareunia; (7) obstipation and constipation, often related to the menstrual period; (8) sterility; (9) sacral backache; (10) menstrual type of headache; (11) reflex gastro-intestinal and bladder disturbances.—G.P.

Androgen Therapy in Gynaecology.

Giest and Salmon (*J. A. M. A.*, 1941: 117: 2207) from observation on 422 women treated with androgen during a period of five years write that androgens are effective because of their ability (a) to nullify or modify the action of oestrogens, (b) to suppress or decrease the production of oestrogen by the ovary, (c) to inhibit the proliferative process in the endometrium, (d) to inhibit the reactivity of the uterine musculature and (e) to inhibit the gonadotropic activity of the hypophysis. Androgens are used effectively in (1) functional menometrorrhagia (2) functional dysmenorrhoea, (3) premenstrual tension, (4) premenstrual mastopathies, (5) postpartum engorgement of the breast and (6) certain types of the menopause syndrome. Androgen is found effective in less than 50 per cent. of the cases in which menometrorrhagia is associated with uterine fibroids but of no value if the abnormal bleeding is due to sub-mucous myomas. An orally effective androgen is available in the form of methyltestosterone. The latter has also been shown to be absorbed in effective amounts when administered sublingually in solution in propylene glycol. Pellet implantations of androgen have been found to be therapeutically effective but they are not recommended for clinical use because of the greater advantages of parenteral and oral therapy. The effective dose is 300 mg. a month and a dose of 500 mg. or more a month may induce acne and arrhenomimetic effects, namely, hypertrichosis, deepening of the voice and slight

enlargement of the clitoris. A theory is propounded of the physiologic role played by androgens in the sex-endocrine balance of the normal female. In the light of the theory, functional menorrhagia, dysmenorrhœa and premenstrual tension may be

regarded as manifestation of a disturbance of androgen-gynacogen balance resulting in the abnormal dominance of the gynacogens. Administration of androgens in order to restore the normal balance would accordingly appear to be a rational therapy.—G. P.

Dermatology

Cutaneous Disturbances in Diabetes Mellitus.

Rudy and Hoffmann present 5 case reports which demonstrate that the cutaneous disturbances in diabetes mellitus are most frequently due to a vitamin deficiency, especially that of nicotinic acid. The authors have seen many more diabetic patients with similar more or less pronounced cutaneous eruptions. The skin of some of these diabetic patients is not unlike that of pellagrins. Its vulnerability to any kind of external irritation (rubbing or other mechanical or chemical trauma, perspiration, heat or sunlight) and to infections, especially those due to monilia, is increased. Vulvitis as well as pruritus vulvæ and in diabetes are proved signs of pellagra and are due to a nicotinic acid deficiency. The most frequent manifestation is a vivid, more or less indurated, erythema characterized by a violaceous or brownish colour and a definite shiny surface. Fine lamellar peeling or psoriasiform scalliness, more or less deep ridging of the papillary lines and fissuring, edema, blistering, maceration and even superficial ulceration vary the morphologic picture. Superinfection with monilia on predisposed regions suggests intertrigo or paronychia. Pruritus vulvæ in diabetes does not respond to local treatment and does not always clear up with the control of glycosuria. In the authors' patients improvement followed the administration of nicotinic acid or its amide with, and at times without, the addition of vitamin B complex. This occurred even in the presence of glycosuria and hyperglycemia. Nicotinic acid, like thiamine chloride, is essential for the satisfactory oxidation of carbohydrate foods. This explains why some cutaneous manifestations appear for the first time or become aggravated after the diabetes is controlled. In such cases the symptoms disappear with the addition of the nicotinic acid. The same happens with other vitamins, such as riboflavin and thiamine hydrochloride. It is not unusual for a neuritis of perleche to become manifest or aggravated following the control of the diabetes or diabetic acidosis. The vitamin deficiency in diabetes mellitus may affect the gastro-intestinal tract including the mouth, the central and peripheral nervous system and the genito-urinary tract. The degree of the cutaneous infection depends on the severity of the vitamin deficiency and not on the glycosuria or hyperglycemia. The predisposing factors in the development of deficiency in diabetes mellitus are a diet deficient in the components of the vitamin B complex, disturbance of absorption and assimilation, and impaired hepatic function.

The precipitating factors may be loss of weight, infection, gastro-intestinal disturbance, operations, acidosis and intemperance.—J. A. M. A.

Skin Diseases; Allergy. Roentgen Therapy of Hemangiomas. Fred M. Hodges, L. O. Sneed and R. A. Berger, *Radiology* 36: 700 (June) 1941.

In this article the authors report their results with roentgen therapy and give details of 3 cases, limiting their remarks to the strawberry hemangioma, the cavernous hemangioma and a mixture of these two types.

The first case reported was that of a 6 month old baby girl with a rapidly growing birthmark on the ear and face. It had been previously treated by a dermatologist who had used radium to the point of ulceration, but the lesion was still growing rapidly at the time the authors began to use filtered roentgen rays. With the surrounding normal skin protected with lead, roentgen rays were administered to the region. This treatment was repeated in two weeks, followed by small doses given over a period of thirty months by the method of contact roentgen therapy. By this time the bulkiness of the lesion and most of the discoloration had disappeared.

A second patient, also 6 months of age, had been previously treated by radium for a cavernous hemangioma of the cheek and the upper part of the left arm, in spite of which growth had continued. Treatment as for the former patient was carried out with filtered roentgen rays. A perfect result was obtained on the face, but a flabby fatty mass was subsequently removed surgically from the arm.

In the third case, a child 3 years old had a lesion 3.5 cm. in diameter on the forehead. It had grown slowly from birth. Large doses of roentgen rays were given, as in the 2 preceding cases, and a perfect result was obtained. Although the lesion extended into the eyebrow, the hair was not destroyed.

The authors believe that contact therapy offers radiologists an ideal method for the treatment of certain forms of birthmarks. They believe strongly that in the future this will be the procedure of choice in selected cases of hemangioma and lymphangioma, having observed that it is rapid, safe and efficacious. Contact roentgen therapy is the method of choice for superficial strawberry hemangiomas. Mixed

strawberry and cavernous types respond best to a mixture of filtered and unfiltered rays. By using these forms of therapy the authors believe that fewer cases will require supplemental surgery.—Anspach, Chicago.—A. J. D. C.

Lupus Erythematosus.—The treatment of lupus erythematosus by the injection of heavy metals, particularly gold and bismuth used either separately or in combination has over many years of close observation given sufficiently encouraging results to justify its further employment in this difficult condition.

In chrysotherapy the salt most generally used is gold-sodium-thiosulphate, preferably administered intravenously in an aqueous solution or alternatively by intramuscular injection in the form of an oily suspension. It is on record that a few observers have employed the drug by subcutaneous injection direct in the lesions, but this method apparently shows no clinical advantage over the intravenous and intramuscular routes, and is frequently objected to by patients on account of its painful character.

Dosage with gold-sodium-thiosulphate should be based largely upon toleration shown in individual cases, but the average patient rarely displays any toxic reaction to an initial dose of 0.05 gm.

Injections are given at approximately weekly intervals over a period of eight or ten weeks, gradually increasing the dose to a maximum of 0.1 gm. It is rarely necessary to exceed this strength of injection, though, where ready toleration is shown by the patient, a dose could be given as strong as 0.2 gm. or 0.25 gm. The initial course is followed by a few weeks rest, when in case of need, treatment can be repeated.

A combination of various case reports which have come to our notice from time to time shows that out of a total of 138 patients treated by chrysotherapy, 49 per

cent. were definitely cured, and of the remainder a high proportion were greatly improved. In a few cases the treatment was apparently unsatisfactory owing to intolerance and toxic reactions.

—Prescriber.

Chronic Psoriasis.—A novel form of treatment for patients suffering from chronic psoriasis is suggested by Bigham. The patient rubs a 2 per cent. crude coal-tar ointment in paraffin moll. into all the lesions every night. In the morning, with the ointment still applied, he is exposed to ultra-violet rays after the ointment is removed by means of olive oil, a thin film of which is left on. The patient is instructed to take 1000 units of vitamin B₁ daily. If there are many thickened lesions, 20 to 30 gr. of acid salicylic are added to the tar ointment and the patient is told to scrub off the scales with a stiff brush or pumice stone. In 53 patients so treated the condition in every case gradually disappeared. SAUNDERS states that the fact that psoriasis is an incurable disease is universally admitted. More women than men seem to suffer from it. Body-building seems to be an important consideration. The disease seems to respond to treatment most readily in patients who are slightly overweight or even obese. Psoriasis occurs characteristically on the scalp, and the daily use of 12 per cent. hydrarg. ammon. in a vanishing cream base is almost uniformly successful. A low-fat diet is desirable. Correction of gynæcological abnormalities appear to be beneficial. Vitamins are generally unsatisfactory. Arsenic is valueless. Main reliance, however, must be placed on local treatment in most cases. Chrysarobin in 2 to 10 per cent. concentration in ointment form for daily application still remains the most effective single therapeutic measure. Ultra-violet light is sometimes, but not always, helpful.

—Prescriber.

Ophthalmology

Epidemic Keratoconjunctivitis.—Andrew de Roeth, M.D., Spokane, Washington.

The author observed 12 cases of an eye disease within ten weeks, from December, 1941, to February, 1942. The significance of this disease lies in the fact that it causes total disability during its acute stage, which may last several weeks, and that it spreads in harbours and shipyards.

The initial subjective symptoms were foreign body sensation and lacrimation. Three patients had a slight malaise. After the eye got red, the clinical picture developed in four to five days to its fullness. They were swelling of the eyelids, acute conjunctivitis, sometimes with chemosis. The semi-lunar fold was swollen. The discharge was slight and of a mucoserous character.

About 8 to 12 days after the onset of the disease, when the conjunctivitis was definitely subsiding, eight patients developed superficial punctate keratitis. There were 3 to 30 or more tiny (1/3 to 2/3 mm. in diameter), slightly elevated infiltrates, mostly grouped in the pupillary area. Under slit lamp they appeared to be made up of several tiny gray dots, the tissue between the dots being opaque. They were located in the epithelium and immediately under it, perhaps even under the Bowman's membrane. There was no corneal vascularization. The keratitis caused a slight scratchy feeling, and the vision was 20/25 to 20/66. The keratitis remained unchanged for several weeks, when the infiltrates started to clear up, and in a month after keratitis started the cornea became normal.

except in three cases. The disease started in all cases in one eye only, but in five cases the other eye became involved in four to seven days, though the conjunctivitis was less marked. Swelling of the preauricular gland was a frequent occurrence. The bacteriologic and serologic findings did not disclose the nature of the disease. The majority of the modern authors agree that it is caused by an unknown virus.

The disease was identical with that observed lately in Oahu, Hawaii, 1941, in Germany in 1938-40, in Selangor, Federated Malay States, 1935-38, in London in 1933, and very probably with that of Madras, India, in 1928-29. According to private information from Drs. F. Cordes and M. J. Hogan, there were many cases in San Francisco in December, 1941, and January, 1942. Forrest E. Rieke, in his recent publication, "Epidemic Conjunctivitis," of presumed virus causation (*Journ. A.M.A.*, vol. 119, July 18, 1942), wrote about an epidemic in the Oregon Shipbuilding Corp. yards. There were 300 cases during the six weeks beginning last October 1941. The same condition under various names was reported as far back as 1889. There is an interesting point in the pathogenesis of the disease, the part of trauma suffered by the eye several days before the onset. Animal experiments failed to clear up the etiology of the disease.

The disease has no specific therapy, and so far local treatment failed to show results. Trachoma was the first virus disease in which sulfanilamide acted as a specificum. Having to deal with another, supposedly virus disease of the conjunctiva and cornea, sulfadiazine was ordered for these patients. There was in all cases a definite change, rapid subsiding of conjunctival symptoms in two to four days. But the outbreak of punctate keratitis was not impeded in any

way by this therapy.—*North-west Medicine*, 41:245 (July) 1942.—*Medical World*.

Treatment of Herpetic and Dendritic Ulcers.—The author discusses the literature from the time dendritic ulcers were first observed and described 72 years ago. It is the consensus of opinion in the literature that the presence of an unidentified virus or toxin is responsible for this type of keratitis. From the experiments of a number of investigators, one can conclude that there is a definite relationship between simple corneal herpes and dendritic keratitis, believing that the former is the forerunner of the latter. As no specific agent causing the condition has been determined, a wide range of therapeutic agents have been used, all having but slightly beneficial effects.

The author discusses twelve cases in each of which there was either herpetic or dendritic ulceration of the cornea and in which healing took place without recurrences after the elimination of foci of infection which were either in the sinuses, tonsils or teeth. All these cases resisted local treatment and made no definite progress until these foci were removed.

Local treatment, generally, consisted of anesthetics, antiseptics and atropine; and dressings were applied in all cases. The thermophore was used for five of the cases with less immediate improvement than would be expected. The time of healing was much shorter in the herpetic than in dendritic types.

The author concludes that an identified virus which may have its origin in infections in the teeth, nose, and throat or tonsils, should be considered as an etiological factor in the production of herpetic and dendritic keratitis.—*U. S. O. W. J.*

Proceedings of Associations

West Godavari District Medical Association.—The twelfth meeting for the year 1942-43 was held at Ellore, on 18th July 1943, with Dr. V. Venkataratnam, M.B., B.S., in the chair. Members were entertained to tea by Dr. C. P. Satyanarayana, at the commencement of the meeting. The following nominations were unanimously made for the offices of the President and 3 Vice-Presidents to the Central Association for the year 1943-44:—

President:—Dr. A. Lakshmanaswamy Mudaliar, B.A., M.D., Madras.

Vice-Presidents:—1. Dr. D. S. Ramachandra Rao, M.A., M.D., Bezwada; 2. Dr. M. R. Guruswamy Mudaliar, M.D., Madras; and,

3. Dr. B. Tirumala Rao, F.R.C.S., D.L.O., Vizagapatam.

It was also resolved to elect a Vice-President and a Secretary from the mofussil for conducting meetings and for organising and enlisting members for the association from the mofussil areas of the district.

Dr. P. G. Nityanandam Pillai, District Health Officer, West Godavari, delivered a very interesting and instructive lecture on "Malaria—its prevention and control" and exhibited several specimens from his collections. He also arranged to exhibit a talkie film on "Malaria" for the benefit of the doctors. The film was also shown to the public and school children, thanks to the organisation of Dr. B. S. Sastri, our Municipal Health Officer.

Book Reviews

Pain and Mechanism By Sir Thomas Lewis. Pp. 192; 27 figs., New York and London: The Macmillan Company, 1942.

The chief purpose of this book is to review modern ideas of the mechanism of human pain and to bring into perspective with other observations the work of the author's own laboratory during the last ten years. The two introductory chapters of this book form chiefly a record of plain facts without much discussion.

From the point of view of medical practice, the most significant part of the book is probably that dealing with deep pain and the associated phenomena of muscular tenderness and rigidity. The reader will appreciate that this is an important book, for pain and the associated phenomena of tenderness and muscular rigidity are the very foundations of the diagnostic art. It can be confidently recommended to medical men not only as a book which will arouse their interest but as one which ultimately cannot fail to help their understanding of disease. It is, moreover, a unique illustration of what can be achieved by a forceful mind with the simplest of apparatus and methods.

The Chemical Composition of Foods By R. A. McCance & E. M. Widdowson. (Medical Research Council Special Report Series, No. 235), H. M. Stationery Office, London, 1940. Pp. 150.

We now have before us, a third report in the series of "Chemical Composition of Foods" issued by The Medical Research Council, in which Dr. McCance and Miss Widdowson have gathered together in a new form all their earlier results, many of them revised and corrected along with a large number more that have hitherto not been published at all. The report gives the most accurate figures available to day for the composition of foods, both flesh and plant. It enables the medical practitioner immediately to look up the average protein, fat and carbohydrate contents of these foods, and in the conditions in which they are likely to be consumed. Besides these, the tables give figures for dairy produce, beverages, condiments, preserves and sweetmeats, and for a number of cakes, pastries and puddings, which have been made to standard recipes, all of them given in ten special pages of this report. With five pages of general introductory matter, a list of references, an adequate index and the recipes together constitute what must remain for many years the most authoritative and trustworthy collection of food analyses available. It will be found indispensable to the medical profession and also to many scientists outside the medical profession.

Recent Advances in Pathology By G. Hadfield and L. P. Garrod, Fourth

Edition, 1942, London. J. & A. Churchill, Ltd. Pp. viii and 346. 86 figures on 34 plates and 11 text figures.

One of the most successful of the series of 'Recent Advances in Pathology' by Hadfield and Garrod, now in its fourth edition. The principal topics discussed in this new edition are inflammation and infection, the reticuloses and reticulo-sarcomata, experimental cancer research, vitamin deficiency diseases, rheumatic endocarditis, arterial hypertension, pneumonia, nephritis, gastro-intestinal diseases, especially in their relation to anaemia, and diseases of the ductless glands. This last chapter has been contributed by a clinical colleague of the authors, Dr. E. F. Scowen.

In the chapter on inflammation, Menken's work is fully and critically reviewed and this is a good account of the Duran-Reynolds phenomenon. The chapter on the reticuloses and reticulo-sarcomata includes short but clear and up-to-date accounts of the Lipoidoses, Schaumann's disease, and regional ileitis. The three chapters on experimental cancer research include sections on chemical carcinogenesis, the filterable fowl tumours, tumour metabolism, immunity to transplanted tumours, the influence of heredity and the 'mother's milk' factor. Bittner Vitamin deficiency diseases are dealt with in a very satisfying manner, with full consideration of vitamin K, and its relation to the prothrombin content of the blood and to haemorrhagic states resulting from prothrombin deficiency.

Chapter IX provides a good and impartial discussion of the various aetiological theories of rheumatic fever. The chapter on Bright's disease includes an important and topical section on renal failure and ischaemic necrosis of muscle compression-anuria and the "crush syndrome" and another extra renal azotemia. In the chapter on ductless glands, first place is given to the sex hormones, the pathology of the adrenal centre, and necrosis of the anterior lobe of the pituitary and its relation to Simmond's disease.

The volume is thoroughly well documented and there are full subject and author indexes. Teachers of pathology as well as their pupils will find it of the greatest value as a labour-saving method of keeping abreast of recent pathological discovery.

Brompton Hospital Reports, 1941, (Vol. X) Published by the Research Department of the Brompton Hospital, London.

We have received a copy of the Brompton Hospital Reports, 1941, Vol. 10. It consists of several important articles which are of interest to all practitioners as well as to those who specialise in chest disease. The subject of empyema is excellently reviewed in two papers. The first of these is by R. C. Brock and

deals with the surgical indications for drainage of the pleura; these are given clearly and concisely and the article should be read by all general surgeons. Another article by J.G. Scadding presents the medical aspects of chronic empyema and describes very fully the causes and differential diagnosis of this condition. Clifford Hoyle contributes an authoritative paper on the treatment of traumatic pneumothorax. Another paper by Scadding is also included which gives a clear and original discussion of some aspects of closed wounds of the chest. This paper is particularly valuable for the practical description of the technique of aspiration, air-replacement and other measures used in the management of these cases. Both these last two papers owe much to the practical

experience which their authors have gained in the treatment of war casualties.

A. F. Foster Carter in his article on bronchial adenoma deals fully with all aspects of this tumour.

There are also articles by F. C. Ormerod on the treatment of carcinoma of the bronchus with radon seeds by Maurice Davidson on the aetiology of subacute pulmonary infections and by F. H. Young and E. Ganez on the persistence of lipiodol in the lungs following bronchography.

The general impression left by a survey of the contents of this volume is that the Brompton Hospital continues, in spite of war time difficulties, to play the leading part in the study and treatment of thoracic disease which has made its name familiar in many parts of the world.

Success of Aerial "Hospital Ships"

Eighteen thousand sick and wounded allied officers and men were flown from African battlefields in giant transport planes for treatment. This revelation was made by Brigadier General David Grant, Air Surgeon in the U.S. Army Air Forces. General Grant was speaking in New York. He said (reports the *Daily Mail*):

"Use of aerial evacuation of wounded will be greatly increased in future. Air evacuation is a great saver of lives. Because this is a mobile war, we soon discovered that the evacuation hospital of the World War days would not do in this present war. A large evacuation hospital is practically immobile and therefore vulnerable to capture by a quick breakthrough of armoured enemy forces.

"We converted giant cargo planes into flying ambulances equipped and staffed to carry the sick and wounded to hospitals back home. Our pilots flew their cargoes into the combat zones and then loaded human cargo into the same planes on the return trip. Aboard each of the plane ambulances there is either a flight surgeon or an army nurse, and also one non-commissioned officer of the

medical department. They provide medical attention, administering blood plasma and oxygen. Some days we moved as many as 600 patients—three times as many as can be carried by a hospital train on any one trip.

"Apart from medical efficiency brought about by the removal of wounded from fighting zones to relative peace and quiet, an important psychological element is involved. For a wounded man to know that he is soon to be in a safe and friendly area, clean and comfortable, with all the skill and equipment of modern medicine at his side, his will to recovery is much greater. For troops to know that means are at hand for their care if they are injured increases their morale.

"Planes apart from evacuation also flew a complete 250 bed hospital from the African battlefield to another when shifting of hospital facilities was necessary."

General Grant recalled that out of 91,000 U.S. casualties those dead were only 15,000. He went on: "There are four chief factors for this low death rate—blood plasma, quick evacuation to medical treatment, sulpha drugs, and surgery."

Errata

In the article "Some Observations on Vitamin B Complex" by Ch. Krishnamurthy, L.M.P., published in January '43 issue of THE ANTISEPTIC, the following corrections are to be noted:—

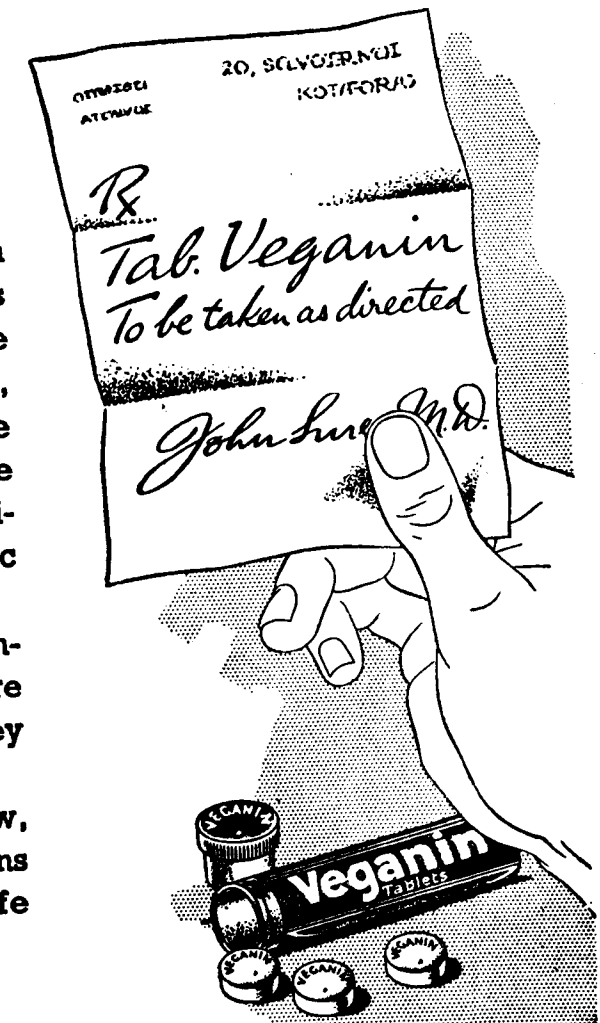
On page 361, 1st para	Line 2	read "chief of which is B ₁ the anti-neuritic vitamin"	for "chief of which are B ₁ and the anti-neuritic vitamin"
"	2nd para	Line 7	read "diminished growth and phrynoderma"
"	"	9	omit the word "phrynoderma"
"	"	"	after the words "glossitis and"

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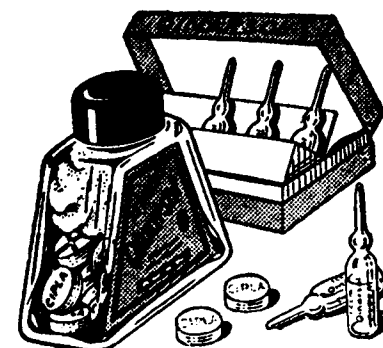
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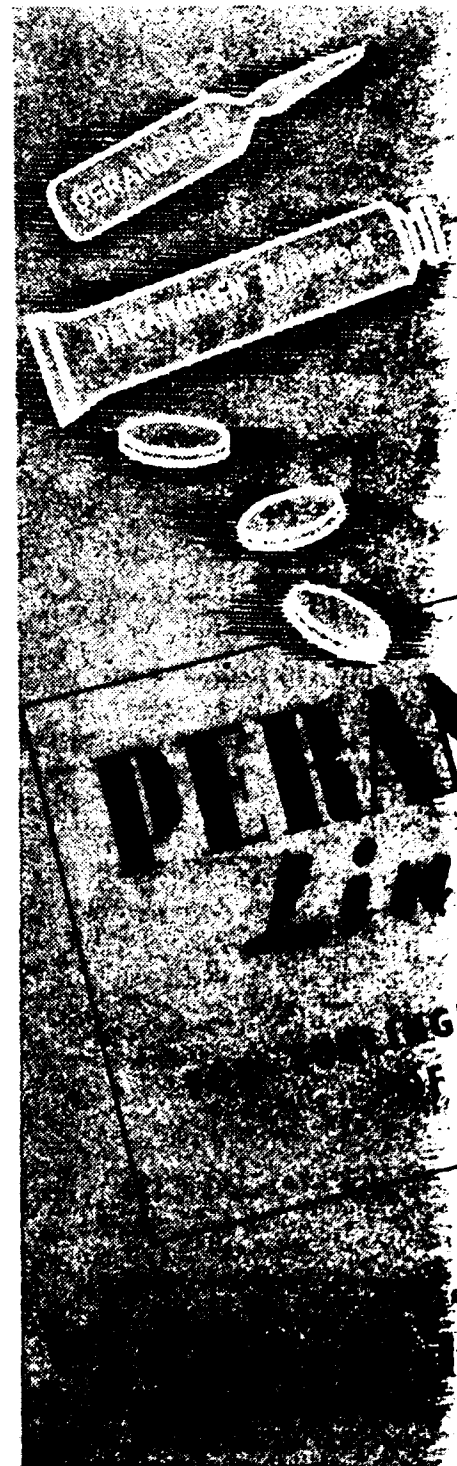
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Aqua (Bi-Distil and Sterilised) to	2 c.c.

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(ii) In anaemia arising out of repeated attacks of Malaria.

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CONTENTS.

Gleanings from Medical Press.

MEDICINE AND THERAPEUTICS :	PAGE		PAGE
Treatment of Diabetic Coma.	424	Treatment of Shock by Action on Nervous Centres.	431
Sulfadiazine in Pneumonia.	424	Dissolution of Urinary Calculi by Citrate Solution.	432
Comparison between Psittacosis and Atypical Virus Pneumonia.	424	Ruptured Intervertebral Disk.	432
Hæmorrhage from the Trachea, Bronchi and Lungs of Non-Tuberculous Origin	425	OBSTETRICS AND GYNÆCOLOGY :	
Dilantin Sodium and Phenobarbital Combined for Epilepsy.	425	Spontaneous Abortion.	432
Virus Pneumonia.	426	Endometriosis.	433
Treatment of Asthmatic Paroxysm with Nicotinic Acid.	426	Androgen Therapy in Gynæcology.	433
Vitamin Therapy.	426	DERMATOLOGY :	434
Chest Pains in Patients with Mitral Stenosis.	427	Cutaneous Disturbances in Diabetes Mellitus.	434
SURGERY :		Skin Diseases ; Allergy.	434
Crush Syndrome.	427	Lupus Erythematosus.	435
Treatment of Ischæmic Contracture.	429	Chronic Psoriasis.	435
Anæsthesia for Laryngofissure.	430	OPHTHALMOLOGY :	
Operation for the Orchitis of Mumps.	430	Epidemic Keratoconjunctivitis.	435
Acute Mechanical Intestinal Obstruction.	431	Treatment of Herpetic and Dendritic Ulcers.	436

INDEX TO ADVERTISERS, AUG. '43

	PAGE		PAGE
Albert David Ltd.	XXVI	Horlicks Malted Milk Co.	XXXV
Alembic Chemical Works Co., Ltd.	VIII, XXII, XXXIX	Indian Health Institute & Lab. Ltd.	XL
Allen & Hanburys' Ltd.	III	India Syringe Works	XVI
Anglo-French Drug Co., Ltd.	XIII	Jammi Venkataramanayya & Sons	VII
Allied Drug Industries Ltd.	XIX	John Wyeth & Brother Ltd.	IX
Associated Drug Co., Ltd.	IV	Kanthraj & Co.	LI
Atlantis (East) Ltd.	VI	Lister Antiseptics & Dressings Co.	XLI
Banerji P.	XVIII	Mahant Charan Dass & Sons Ltd.	VII
Behar Chemical Works	IV, XL	May & Baker Ltd.	XXIV
Bengal Chemical and Pharmaceutical Works	Inside of Back cover	Medical Research Laboratory	XXIX
Bombay Surgico Medical Agency	XLII	Menley & James Ltd.	XX, XLIII
Boots Pure Drug Co., Ltd.	XXXII	Morison Son & Jones (India) Ltd. J.L.	XXXVI
Brand & Co., Ltd.	X	Navaratna Pharmacy	IV
British Drug Houses Ltd.	XLV	Nonglay Modern Virtues	LIV
Burmah Shell	XLIV	Orient Laboratory Ltd.	XLIX
Burroughs Wellcome & Co.	XXIII	Oriental Research & Chemical Lab. Ltd.	XLVI
Buyers' Bureau	XLIV	Parke Davis & Co.	Outside of Back Cover
Calcutta Clinical Research	XXXIX	Petrolagar Laboratories Ltd.	XLVIII
Calcutta Polyclinic Ltd.	XXXIX	Pharmaceutical Labs. of India Ltd.	XXIX, XLIII
Chemo-Pharma Laboratories Ltd.	II, XLVII	Phillip Chemical Works	XXXVI
Ciba (India) Ltd.	XV, XXXVIII	Polyclinical Laboratory Ltd.	VI
Cipla Laboratories	XXV, XXXVII	School of Tropical Medicine	LIV
Corn Products (India) Ltd.	XI	Scientific Glass Blowers Ltd.	LIV
Deodhar Bros.	II	Sinha Medical Stores	LIV
Director Genl., I.M.S.	XVI, XVII	Smith Stanistreet & Co., Ltd.	XXX
East India Pharmaceutical Works Ltd.	XIX	Smith T. J. & Nephew Ltd.	XXVIII
Eli Lilly & Co.	Inside of Front Cover	South Indian Eye Laboratory	XLIV
Evans Sons Lescher & Webb Ltd.	XXXI	Standard Pharmaceutical Works Ltd.	XII
Galleno Pharmaceutical Works	XVI	Suren & Co., W. T.	V
Glaxo Laboratories Ltd.	I, XXI	Tapissier & Co., H.A.	VI
Great Bengal Pharmacy	LIV	Thacker Spink Ltd.	LI
Hall & Kay	XXXVI	Union Drug Co., Ltd.	XX, XLI
Himalaya Drug Co.	XLVI	Victor X-ray Corporation (India) Ltd.	XIV
Hind Chemicals Ltd.	XXVII	William R. Warner & Co., Ltd.	XXXIII, XXXIV, L
		Zandu Pharmaceutical Works	LII
		Zill & Co.	XL

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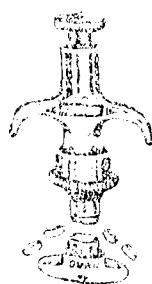
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