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The Antiseptic

A Monthly Medical Journal

Dr. U. Rama Rau, M.L.C. & U. Krishna Rau, M.B., B.S.

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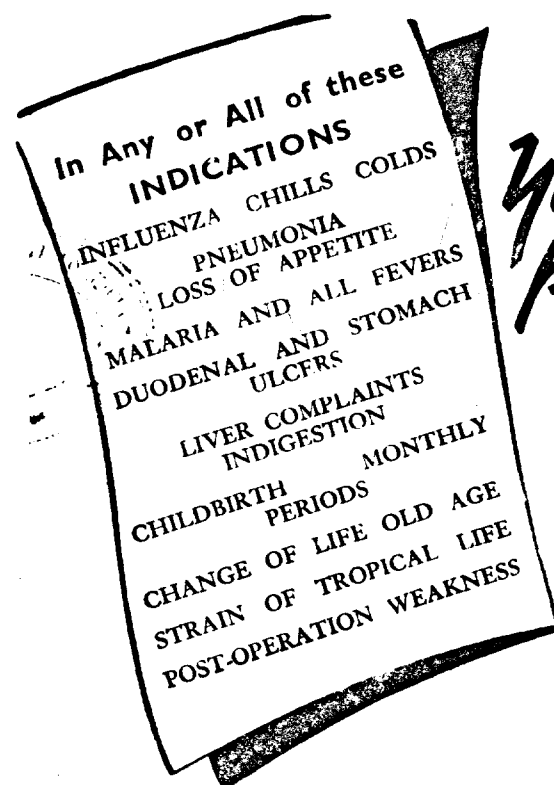
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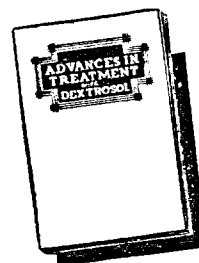
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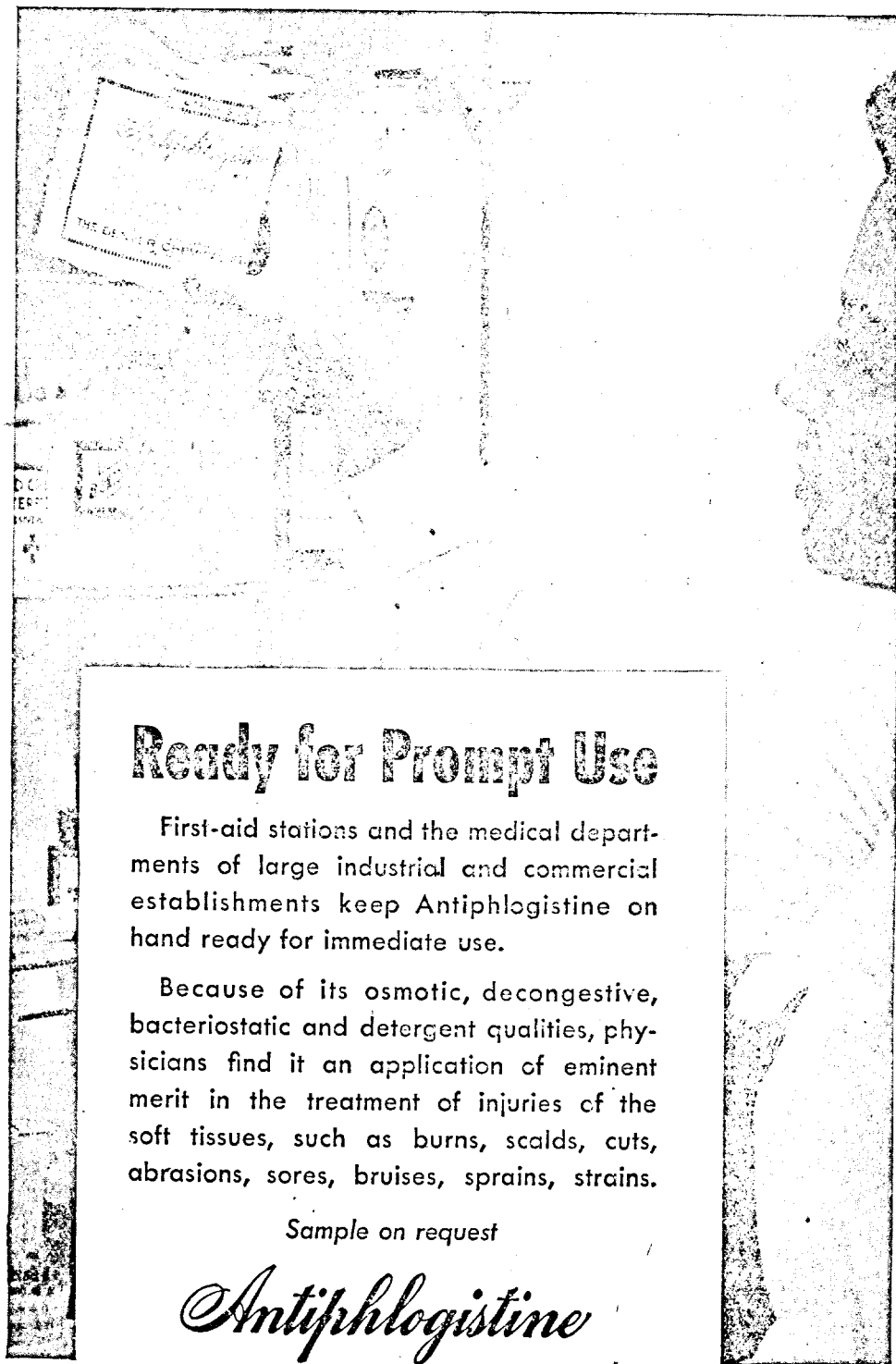
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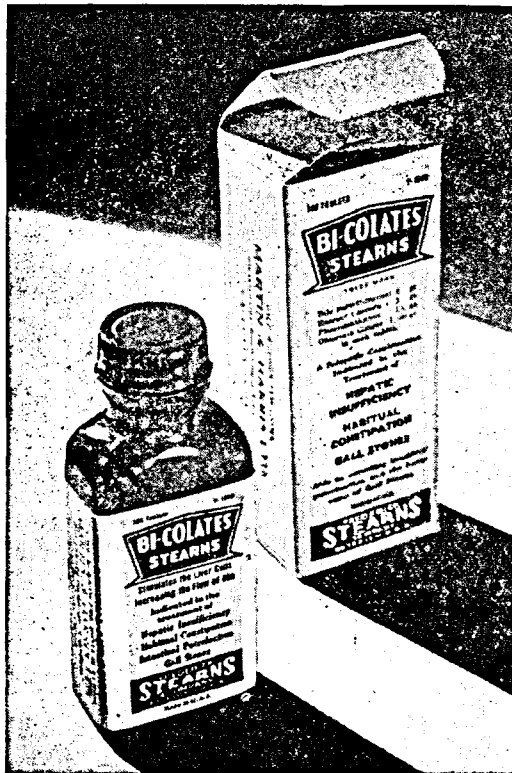
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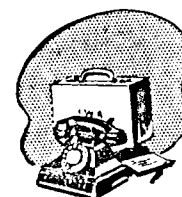
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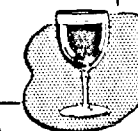
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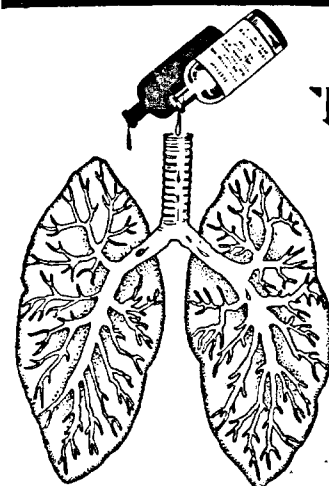


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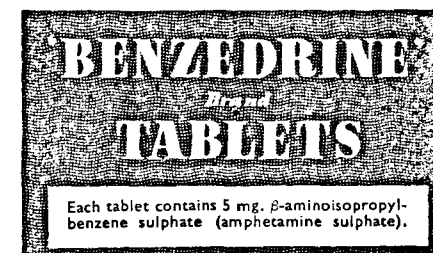
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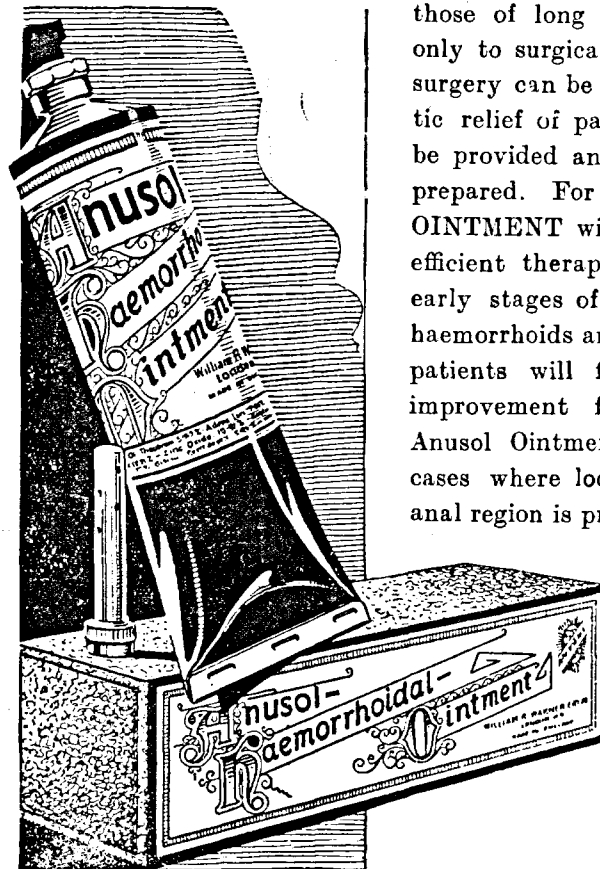
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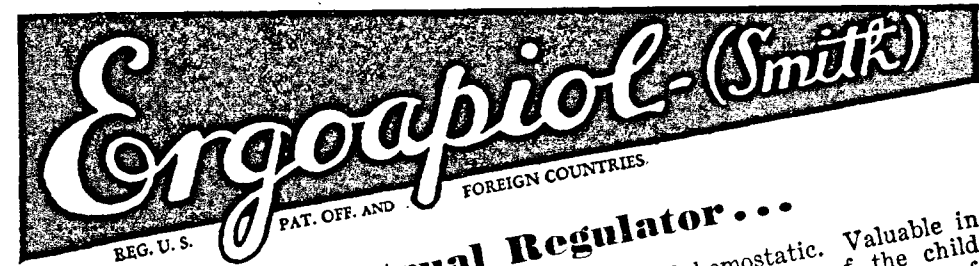
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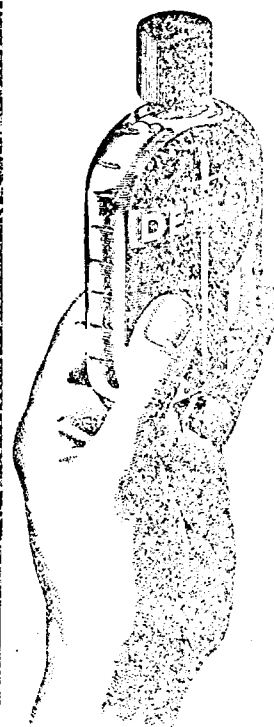
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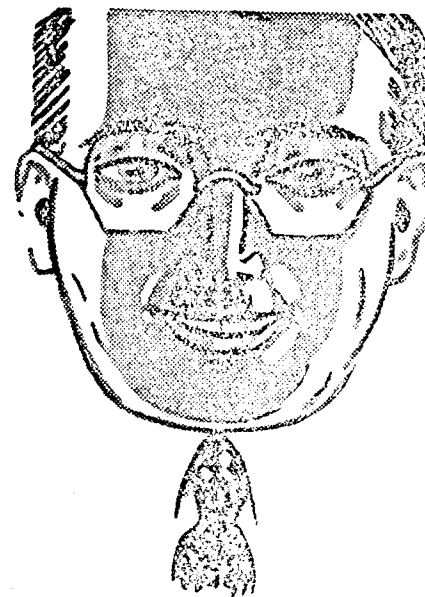
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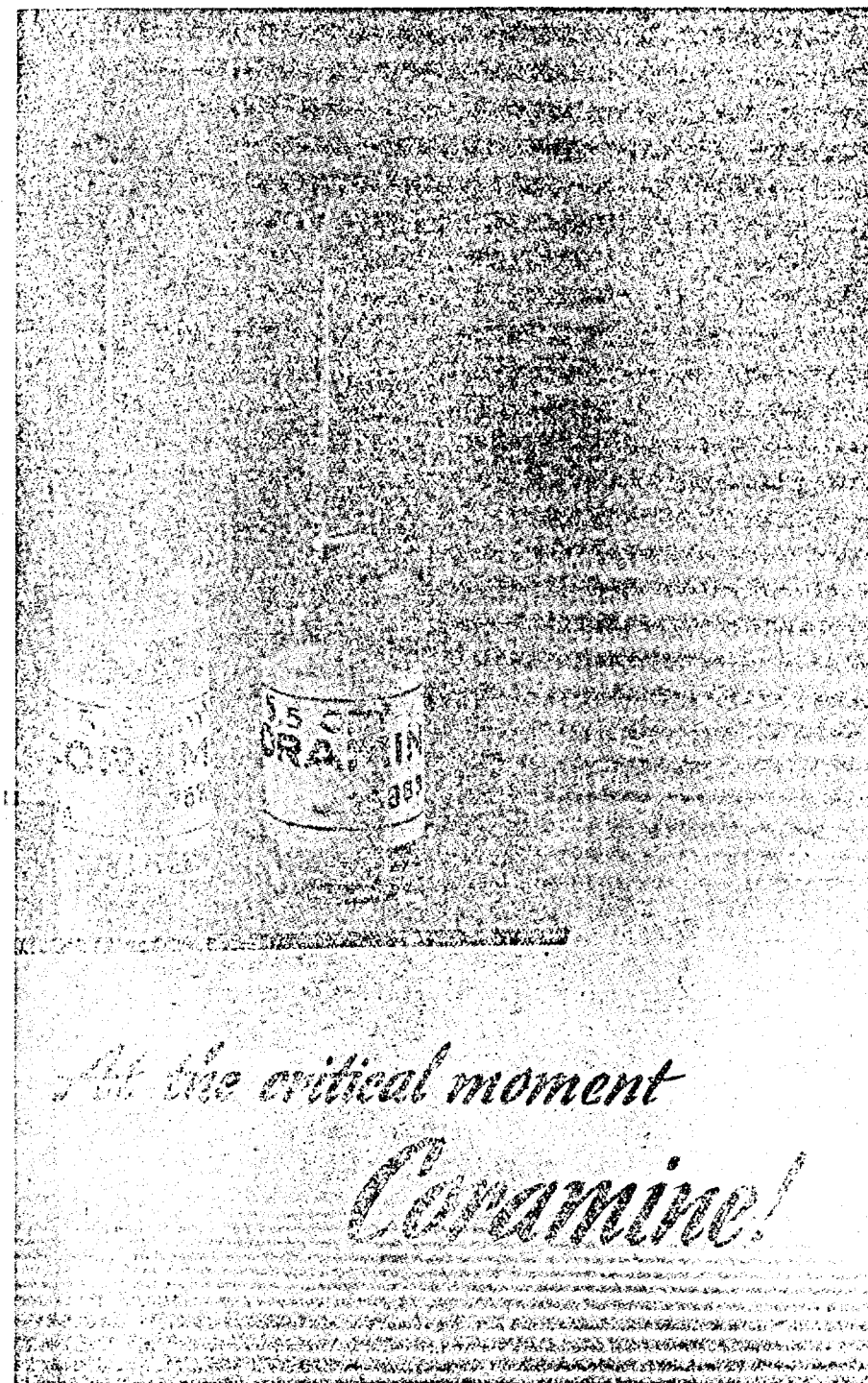
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AUGUST, 1941.

No. 8.

Original Articles

Radiological Appearances of Osteoarticular Lesions in Leprosy studied through Radiology*

BY

MARIO SOARES DA VEIGA,

Director of X-Ray Institute of Portuguese India, Nova Goa.

THE present study concerns the radiological images of hundred patients interned in our Leprosarium Central of Macasana. We have taken four hundred roentgenograms—two hundred of the hand and two hundred of the feet. Our attention was drawn to the leprotic changes in distal extremities of the upper and lower limbs. Amongst the hundred leprosy patients, who constituted the object of our research work, 58 were males and 42 females.

For the convenience of our study, we have divided the patients into three main forms: tuberculous, nervous, and mixed. However, in order to follow the classification arranged by the International Congress, held in Manila in 1931, we have again subdivided the three main groups into five sub-groups.

The first sub-group is formed by the patients showing only skin involvement and according to the extension of the lesions, we divided it into C_1 , C_2 and C_3 .

The second sub-group is formed by the patients showing marked cutaneous involvement besides the slight changes of nervous system. Hence the designations C_3N_1 , C_2N_1 and C_3N_2 .

The third group is purely nervous.

The fourth group is formed by the patients generally with widespread nervous and slight cutaneous involvement.

The fifth group is characterised by cutaneous and nervous involvement nearly at the same degree of intensity. Hence the division into C_1N_1 , C_2N_2 and C_3N_3 .

* This paper was read at the twenty-eighth annual meeting of the Indian Science Congress, held in Benares in January 1941, and specially sent to 'The Antiseptic' for publication.

As regards the first sub-group, purely cutaneous, which is distinguished by the presence of small and big tubercles, spots, infiltrations, ulcers, and nodosities, we were able to select a very small number of only seven patients whose age ranges from 19 to 46 years and the age of disease from 3 to 8 years. Radiologically, no marked lesions were seen. Only slight changes of the bones constituted by the growths of mushrooms appearing in phalanges, local destruction, and enlarged nutritia foramina (84 % of cases) were seen, the last ones, due, according to MURDOCK and HUTTER, to leprous lesions of nutrient vessels. But such lesions, excepting the enlarged nutritia foramina, are found in other diseases of chronic, infectious, nervous and endocrine glands such as nodosum erythema, syringomyelia, morbus Raymond, scleroderma, sclerodactylia and others. In this sub-group, we rarely found signs of osteoporosis, so frequent in the sub-group quoted below.

The second sub-group is characterised by the prevalence of the cutaneous lesions described in the first sub-group with slight changes of peripheral nervous system consisting of limited anaesthetic patches. The present sub-group is formed by 18 patients of age ranging from 17 to 58 years and the age of disease from 1 to 20 years. The principal characteristics are the lesions already described in the former sub-group and which are more marked here. These local destructions, which we are going to describe, consist of bone destructions of somewhat roundish and irregular appearance, localised near the distal extremities of the first and second phalanges of the hand, and directed either to the radial or ulnar side—not however, showing signs of periostitis. Besides these bone destructions (11 % of cases of this sub-group), we found other lesions such as cystic osteitis, which are detected generally in the distal metaphyseal areas of the proximal phalanges of the hands and the feet, rarely in metacarpals and metatarsals and more rarely in middle phalanges. The osteitis, in these cases, is shown like solitary, well circumscribed cysts of several sizes. Besides these forms of circumscribed osteitis (39 % of the cases), we found in this sub-group a small percentage (11 % of cases) of multiple cysts which, by analogy with the tuberculous multiple cystic osteitis, described by JUNGLING, is called by the American authors—MURDOCK and HUTTER, as leprous multiple cystic osteitis. Besides these lesions, we found destruction foci of spongy bone, under the forms of roundish areas occupying a great part of the diaphysis of the affected bones due to their big sizes. They are found generally in phalanges of the feet—chiefly in proximal phalanges (11 % of cases). These foci occupying great parts of the diaphysis often cause fractures, shortages, deformities and destructive arthritis. We found also another type of lesions at the level of the distal extremities of the proximal phalanges, in the shape of transverse destructions at right angles to the diaphysis and some cases of ossificans periostitis consisting of a small percentage of our patients (6 %). *Vide Fig. 3.*

And finally, what distinguishes this sub-group is the osteoporosis (61 % of cases) that is found in the hands and the feet and that, besides its local characteristics, giving remarkable appearance of spots, may be shown under diffuse forms. *Vide Fig. 9.*

The third sub-group, with purely nervous manifestations, counts 42 patients whose age ranges from 12 to 80 years and the age of disease from 3 to 26 years. In this sub-group, we verified that the patients with short duration of disease showed only slight destructions of terminal

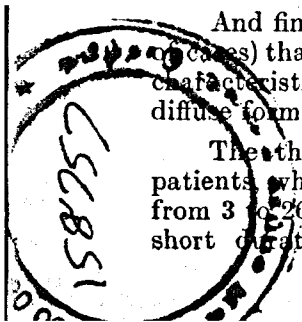
phalanges, whilst patients with advanced lesions of peripheral system and with long duration of disease showed wide mutilations of the hands and the feet. These mutilations are due to tropho-neurotic disturbance of the hands and the feet produced by peripheral leprous interstitial neuritis, possibly accompanied by the lesions of corresponding centres of spinal cord. These centres should share the process of increasing tropho-neurotic disturbance. It appears to us that the intensity of the osteoarticular changes depends chiefly on leprous involvement of the nervous system. Our remarks agree with the opinion of some eminent leprosy specialists such as PROF. JANSELM and PROF. FROILANO DE MELO.

Besides these purely leprous lesions, there are secondary infections and trauma, giving, in radiograms, shadows of severe mutilations, chiefly in the feet.

Moreover, the abnormal mobility of the joints, which results from lack of elasticity of their ligaments and muscular atrophy, is a factor which tends to develop destructive arthritis. In our cases, in which the destructive arthritis was found in a percentage of 62 %, it was found more frequent in the feet than in the hands. The particularity of this leprous arthritis is its rare tendency to ankylosis. As regards the osseous, circumscribed and solitary cysts, they differ from the ones described in the second sub-group, in their big sizes and well limited and sclerosed borders. *Vide Fig. 16.*

The fourth group is formed by 11 patients, with age ranging from 24 to 59 years and the age of disease from 6 to 28 years. In this sub-group, besides the changes described in the previous group, we found slight cutaneous involvement with osteoarticular lesions showing a great deal of different kinds of mutilations at different stages of development. So, besides marked mutilations, we found a gradual destruction of bone tissue of the proximal phalanges which begin at their diaphysis developing afterwards their atrophy and fractures, and may end in complete destruction of the bone tissue of the same phalanges, while the distal ones may remain relatively normal radiograms or the destruction may begin at distal phalanges and spread gradually to the middle and proximal ones. The combinations of these two kinds of mutilations are frequent. It is interesting to note that in leprosy cases, fractures of the bones unnoticed by the patients are not generally predisposed to osseous callus which, however, we found in some cases even under hypertrophic forms. Finally, the symptoms more characteristic of this sub-group are the muscle contractions which develop as a result of the peripheral nerves and which give the hands and feet the typical form of claw hand and foot (73 % of cases). The contractions of the hands and the feet are more marked and more frequent in the last two fingers. These contractions do not give generally osseous ankylosis nor show changes of joint surfaces confirming our examinations as has been stated by the LA CAMP; DEYKE—PASHA. *Vide Figs. 23 to 25.*

And finally, the fifth sub-group is formed by 22 patients with age ranging from 14 to 67 years and the age of disease from 3 to 22 years. In this sub-group, the osteo-articular changes are nearly the same which were described in other sub-groups, chiefly in the third one, where prevails principally nervous pathology and so the diversity of mutilations depends on long life of the disease. This is the principal characteristic of the sub-group C₃ N₃.



Diagnosis.—When the radiograms of the hands and the feet reveal localised destructions in terminal phalanges, growths of mushroom appearance, mutilations partial, with collar-button appearance (*bouton en chemise*) of French authors, somewhat roundish destruction of the diaphysis opening to the radial and ulnar side, enlarged nutritia foramina, grave mutilations in phalanges of the hands and the feet that may go up to metacarpals or metatarsals, localised or diffuse areas of osteoporosis, solitary cysts, either of JUNGLE's or SHURER-WALDHEIM's type, destructive lesions with ossificant periostitis, transverse linear destructions, osteomyelitis, and destructive arthritis, then leprosy is to be suspected in diagnosis.

Differential diagnosis.—There are diseases that may give, in radiograms, shadows similar to the leprosy ones, and they are: RAYNAUD's disease, tuberculous osteitis, syphilis, coccidiosis, sclerodactylia, scleroderma, BESNIER-BOECK's disease, lupus pernio, syringomyelia, tabes and certain forms of osteolysis.

(1) RAYNAUD's disease gives, in radiograms, shadows similar to the leprosy ones. For the differential diagnosis of RAYNAUD's disease, one has to use other means of diagnosis in order to exclude leprosy.

(2) The tuberculous multiple cystic osteitis of JUNGLE's type may be mistaken to be leprosy multiple cystic osteitis. In this latter, the foci of osteitis are localised principally in metaphysis, whilst in tuberculous osteitis they occupy generally the whole diaphysis of phalanges giving a honey-combed appearance to spongy bone. In both the diseases, no periosteal reaction is ever noted. Moreover, in leprosy osteitis, is detected a small quantity of sub-periosteal cysts.

(3) Osseous sarcoid or BESNIER-BOECK's disease or bone disease of BOECK, produces bone lesions which may be mistaken for leprosy. They are found in tubular bone of the hands and the feet. They are of pure pseudo-cystic polydactylitic type or nodular pseudo-cystic poliosteitis of the extremities—PERTHE's disease—Iunghing. This disease, first described by German authors, is purely a radiological one and is localised in the extremities of the hands and the feet. The disease is constituted by multiple nodular neoplasms formed by osteomyelitic mesenchyma. There are wide bone destructions giving pseudo-cystic roentgen appearance. It is a rare disease which we came to know through monographies published by PROF. POITRIER of Venereal Diseases Department, Strasburg Medical College.

Lesions of this kind have been noted also in different types of lupus, but rare in vulgaris lupus. HUTCHISON's lupus frieiro may sometimes show clear spaces in epiphysis. However, it is the lupus pernio which produces wide bone changes of pseudo-cystic polydactylitic type. That only direct examination of nasal mucus or gland puncture can clear out the leprosy as even histopathological examination, as it is dealing with granuloma, shows a uniformity common to other chronic specific granulomatous diseases, including leprosy.

(4) The syphilis and coccidial granuloma show similar proliferative periostitis of the hands and the feet. However, its localisation is more frequent in long bones. The coccidial granuloma is developed speedily and produces slight atrophy of the neighbouring bones.

(5) In syringomyelia, the absorption of phalanges never goes up to a degree which the leprosy may show. Moreover, in syringomyelia, the large

Radiological Appearances of Osteoarticular Lesions in Leprosy
Studied through Radiology—By Mario Soares da Veiga.

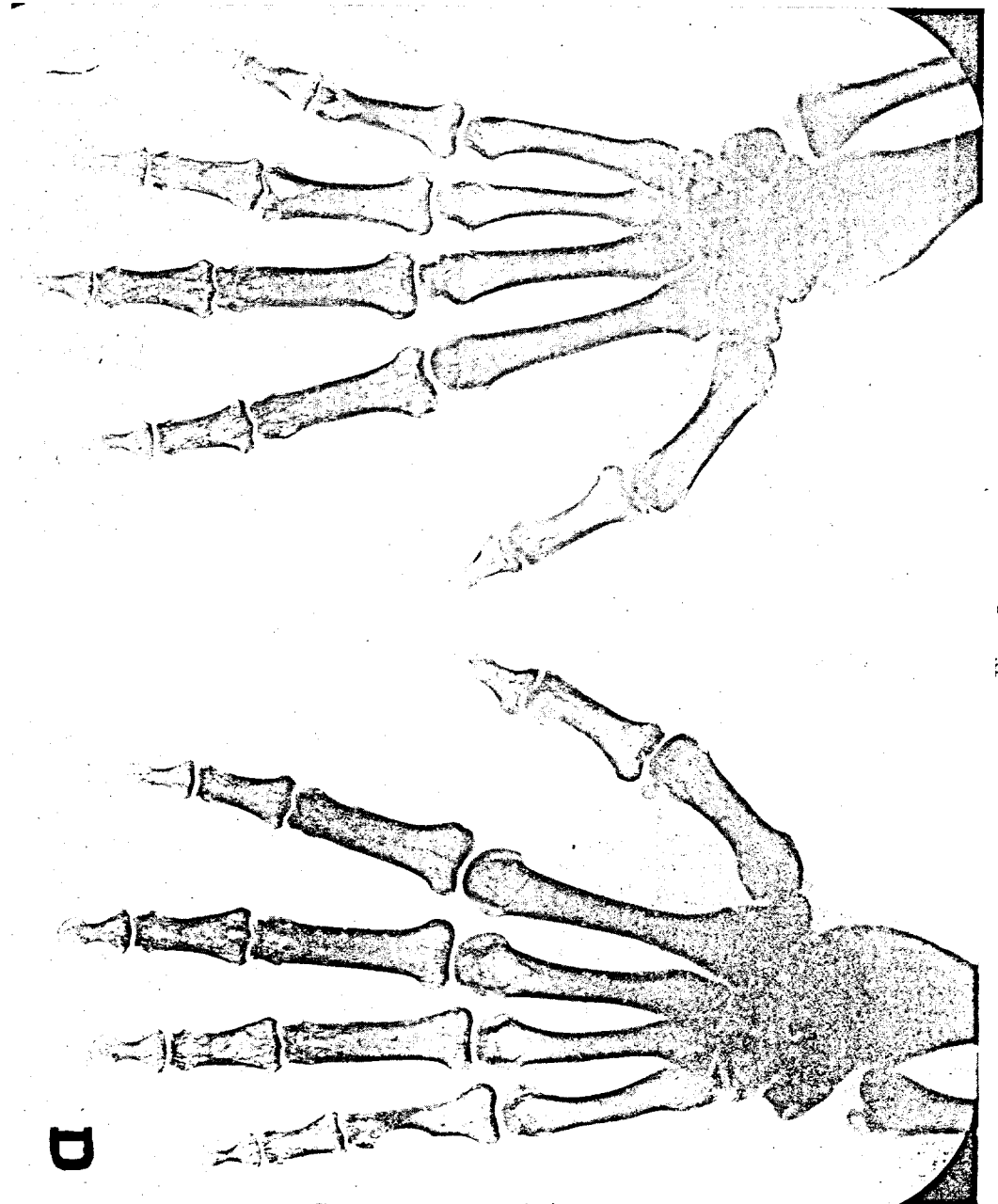


Fig. 3.

Fig. 3. Right-hand—Terminal phalanges under mushroom appearance. Local destruction of the diaphysis under somewhat roundish appearance, proximal phalanges fifth finger.

Left-hand—Cysts. Local destruction directing to the cubital side, middle phalanx fifth finger.

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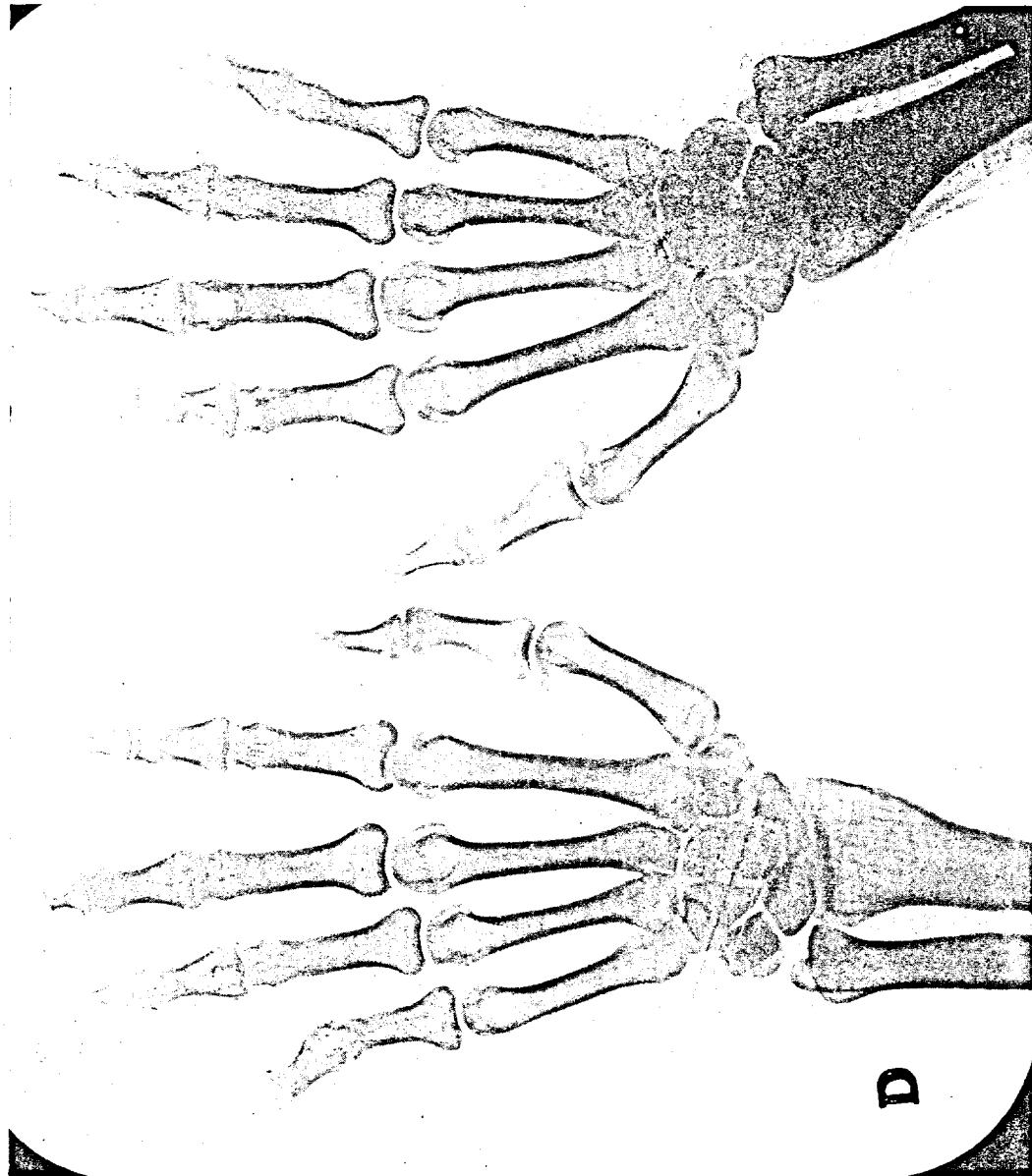


Fig. 7.

Fig. 7. Destruction of collar button appearance in terminal phalanges, cysts. Partial destruction of the diaphysis, middle phalange, left hand second finger, opening to the radial side. Similar lesion at middle phalange, fourth finger, but opening to the ulnar side.

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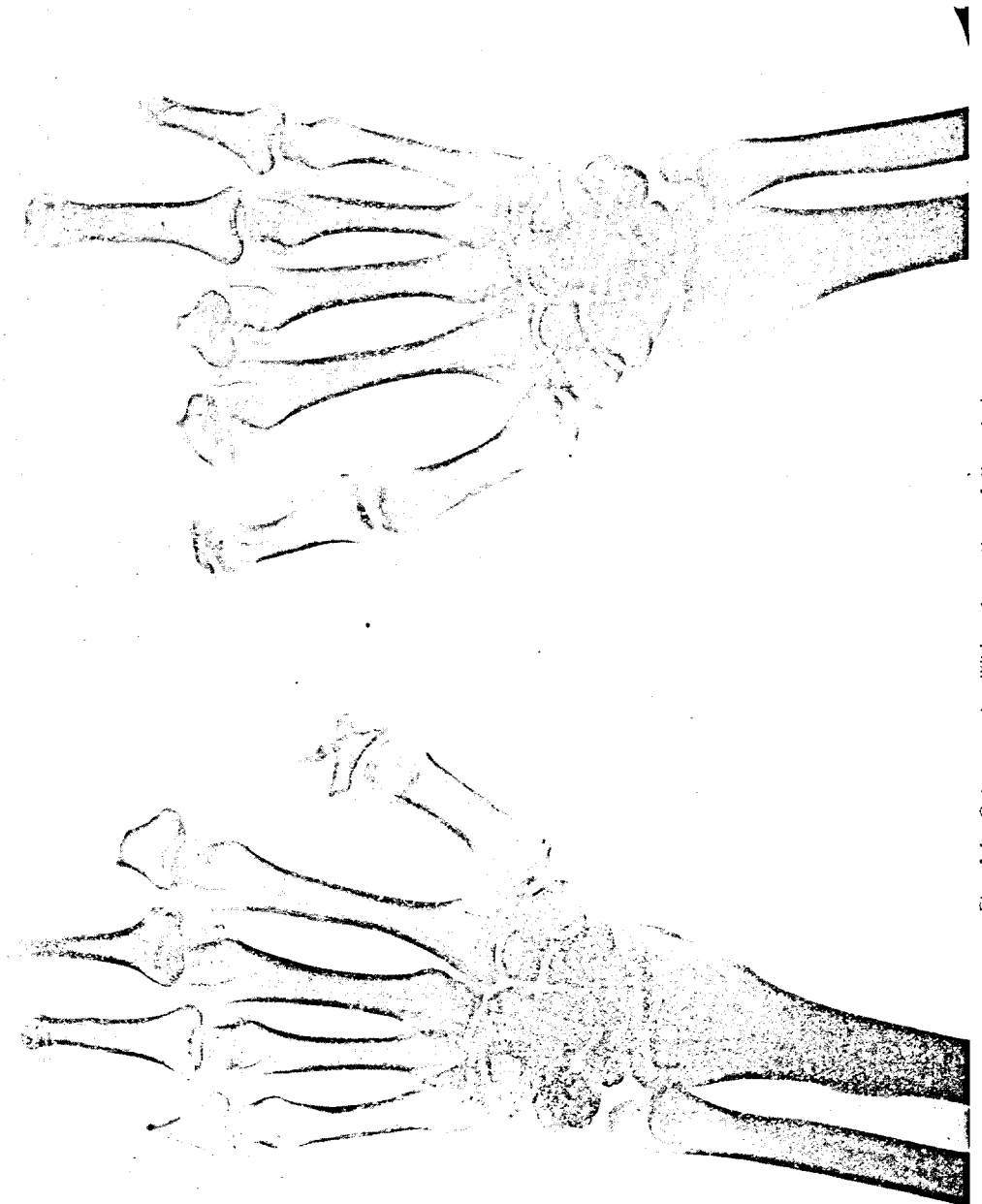
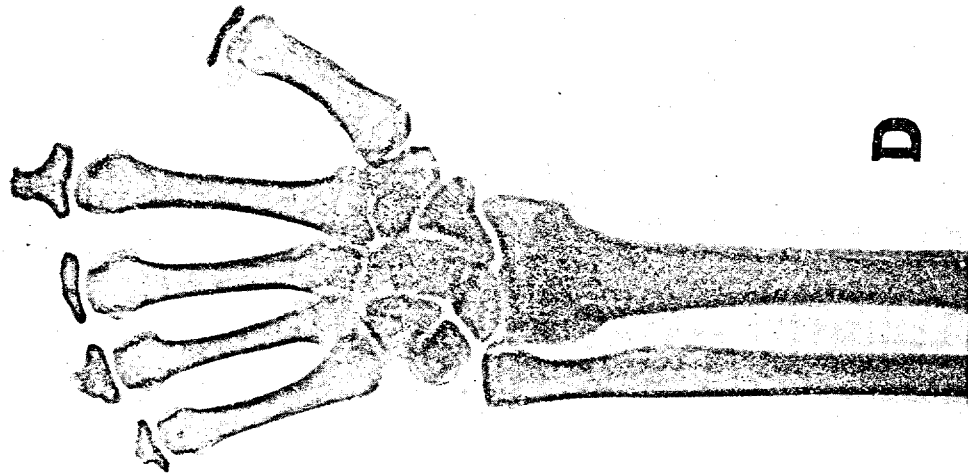


Fig. 14. Osteoporosis. Wide absorption of the phalanges.

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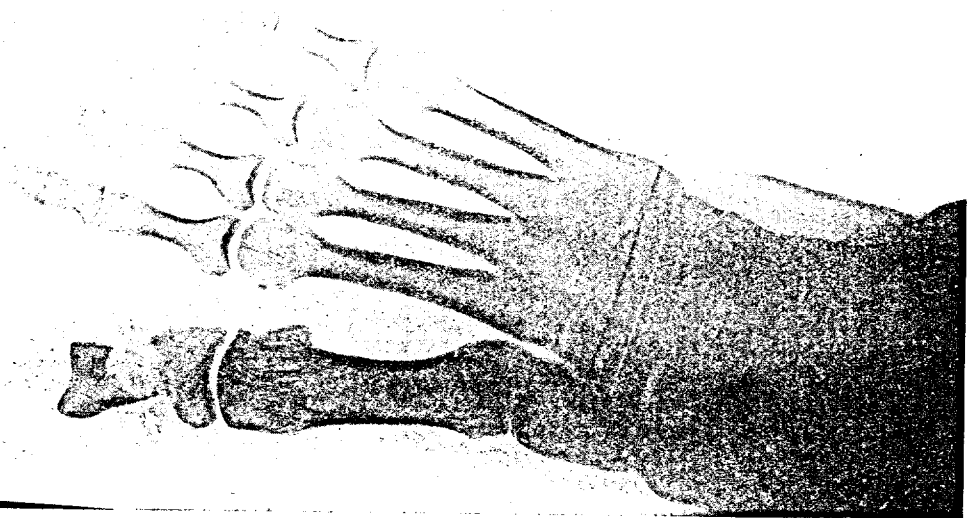


Fig. 9. Large focus of Shurer-Waldheim type in the proximal phalange, leading to shortage of the same phalange. Absorption of the middle phalange at the same side.

Fig. 16. Grave mutilations of the phalanges with proximal extremities showing collar button appearance.

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Fig. 24. Claw-foot.

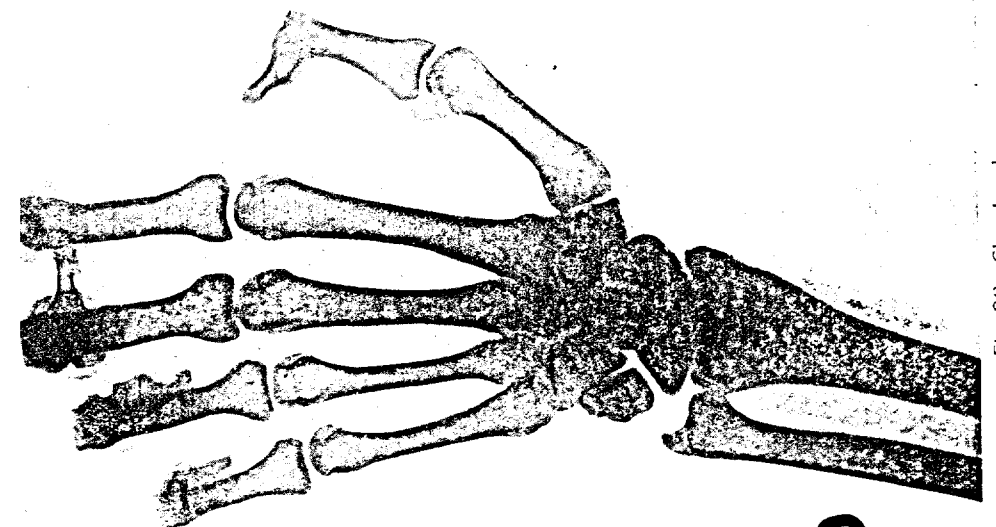


Fig. 23. Claw-hand.

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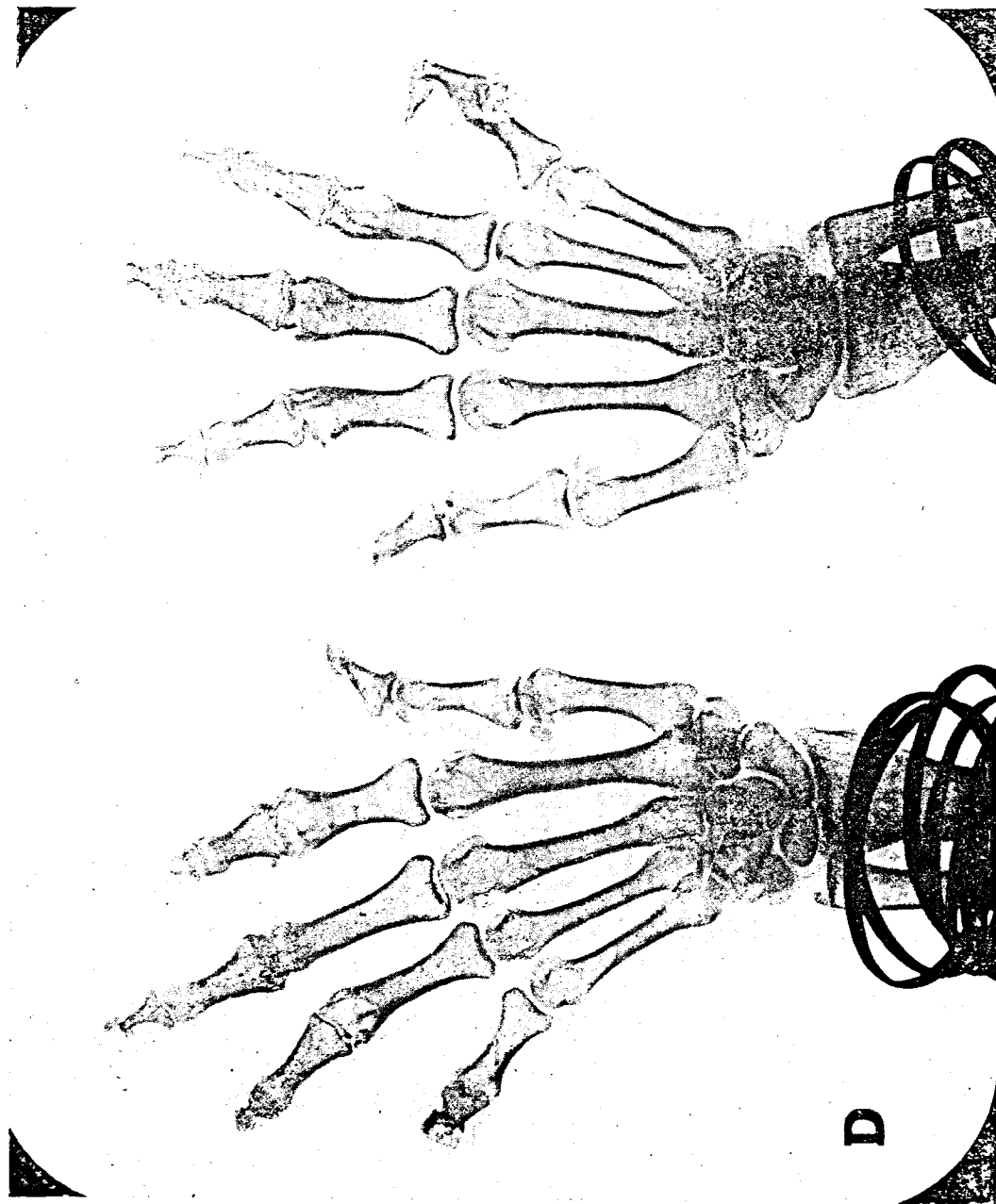


Fig. 25. Bilateral cysts. Reduction of interphalangeal joint spaces, chiefly of right hand, with shifting to the ulnar side "en coup de vent".

joints are affected while only in leprosy are affected the small joints of the hands and the feet and generally are symmetrical. In syringomyelia are seen the spontaneous fractures in long bones whilst in leprosy, they are bound to the phalanges, metacarpals, metatarsals, and rarely to tarsal bones, astragalus and calcis bone. One never sees in leprosy cyphoscoliosis that is frequent in syringomyelia. This cyphoscoliosis is localised at cervical spine and upper part of dorsal spine giving a typical deformity of thoracic cavity (navicular thorax).

(6) In sclerodactylia, the radiograms of the hands may show nearly the same appearance, but the absorption of the bones never goes to such a high degree as in leprosy.

(7) In tabes, according LEVY, bone destructions of metacarpophalangeal joints of first fingers are constant. Together with these lesions, it is not rare to observe destructive process in the remaining metatarsals and finger bones, which resemble manifestations developed by leprosy. To diagnose the tabes, we have to apply for clinical and serological findings. Finally, there are other diseases which may develop marked osteolysis of the hands and the feet. It is necessary to search for their aetiology.

Conclusions.—Osteoarticular changes of leprosy were found in our patients in a percentage of 98% of cases.

These changes have the following characteristics:

(1) They are seen in distal extremities of the upper and lower limbs;
 (2) The process of these changes is essentially of a destructive character; it is based on progressive destruction of bone tissue (osteoporosis, rarefaction);
 (3) Leprous changes of the bones are seen, according to our examinations, under three principal appearances: local osteoporosis, leprous osteitis and severe destructions leading to mutilations. Practically, we found many combinations of these fundamental forms.

(4) The local destructions of somewhat roundish form, solitary and multiple cysts of JUNGLING's type, and forms of SHURER-WALDHEIM's type are found generally in patients that show cutaneous involvement and slight changes of nervous system.

(5) The osteoarticular changes of the leprosy under the forms of wide destructions leading to mutilations are found in advanced cases of nervous leprosy;

(6) Comparing radiological findings with the clinical ones and principally basing on the study of the dynamics of bone changes from radiological viewpoint, we can state two fundamental points of the genesis of leprotic osteoarticular changes, viz.: (a) Bone involvement (osteoporosis, rarefaction) as a result of local infection through vessels. (b) Osteoarticular changes in leprosy of neuro-dystrophic type are the result of leprous involvement of nervous system and are revealed under the forms of bone destructions, diffuse osteoporosis and mutilations often with symmetrical localisations.

(7) The intensity of the destructive process is more marked in the feet not because it may appear to us that this particularity may be due only to mechanical cause, but it must be rather considered as a clinical modality of leprous process.

Acknowledgments.—We have to thank DR. FROILANO DE MELO, Dean, Medical College, and DR. LOIOLA PEREIRA, Director of Leprosarium Central of Macasana, for the facilities given for the present research work.

Treatment of Corneal Opacities*

BY

MAJOR K. RAI, I.M.S.,

Ophthalmic, Specialist, Indian Military Hospital, Meerut.

IN general, opacity can be defined as something that interferes with the transmission of light through a transparent medium.

Commonly, it is used for relucet, or faceted regions of optic heterogeneity as estimated by direct illumination rather than for impaired transparency by retro-illumination. The latter is in accord with the definition of "Opacity" as the ratio of the intensity of incident light to the intensity of light transmitted. Opacity as understood to imply the end results of disease of the cornea after subsidence of the active process, is called a *nebula* if tenuous and ill-defined, a *macula* if localised and small, and a *leukoma* if it is a white disfiguring scar, (Basil-Graves).

For our purpose, they are classified according to the extent and site of cornea involved, as given below:—

- (1) Thick opacity covering almost the whole of pupil—tattooing and optical iridectomy can be undertaken in any hospital.
- (2) Thick opacity, more or less localised and situated anywhere preferably away from pupillary area.

Localisation has been done by the following methods singly or in combination:—(a) Direct illumination; (b) Retro-illumination—direct and indirect; (c) "Sclerotic scatter"; (d) Retinoscopic mirror; (e) Electrical Ophthalmoscopy.

Slit lamp microscopy has not been tried as it is not available anywhere in the District.

Before giving treatment, it is made certain both from history and by examination of the ocular structures, that the receptive elements are alright and that there are no anterior synechiae. The latter, if present, must be divided.

The common causes of the opacities in the patients, so far treated, have been:—1. Trauma from a foreign body. 2. Trachomatous ulcers. 3. Small-pox ulcers.

Treatment.—1. The eye is bathed thoroughly with normal saline lotion and then cocainised.

2. Median basilic vein of the arm of the same side as of the opacity, is cleaned and painted with iodine. After pressure has been applied on the arm, about 2-3 c.c. of blood is taken with a sterilised syringe.

3. With a fresh needle attached and after removing air bubbles, 2-3 c.c. of blood is injected subconjunctivally about 2-3 m.m. away from the limbus but in the same line as of the corneal opacity.

If the conjunctiva bulges too much out of the palpebral aperture, a little massage to disperse the blood is applied.

4. Sterilised Vaseline is inserted and the eye is bandaged.

* Specially contributed to 'The Antiseptic'.

5. From the next morning, the eye is bathed twice a day and 10% Dionine two drops b.d. are put in. The bandage is kept for 3 days and then an eye shade is given. Dionine drops are continued for 10-12 days till the eye becomes almost white again.

6. Ung. Quinine Bisulph, 2% b.d. (size of a split pea) is used for massage of the cornea for 6 weeks. About the end of the 8th week, a 2nd injection of blood with all other routine treatment is repeated.

The results have been very successful both amongst the Military and the Civil poor patients; in the latter, however, the standard of vision being low, the patients have expressed a great enthusiasm even after one course of treatment. The results have been gauged by subjective tests *i.e.* improvement in the patient's vision. The great advantage of this treatment is that the patient need not be admitted, as he can daily attend the O.P.D.

Previous to the above mentioned outlines of treatment, subconjunctival injections of normal Saline or Mercury Oxycyanide along with local applications of Yellow ointment and Dionine drops or ointment had been tried. The last two drugs are being still extensively used by various medical men, but the results of all these injections and drugs have not been so encouraging, besides being painful in case of Oxycyanide.

Notes on some of the cases treated are given below:—

Case 1. Sergeant D. F. E., aged 31, (K. G. V. O. S. & M. Roorkee).

Complaint:—Defective vision L. E., of 12 years' duration.

Examination:—Two opacities in the pupillary area near the outer periphery—bigger between 4 and 5 o'clock, and smaller at 10 o'clock—found.

Cause:—Traumatic ulcers from a foreign body.

5-4-40. Vision R.E.—6-6, D.—0.5. Vision L.E.—4-60, D.—2.0.

25-6-40. Vision L.E.—6-18 (two letters), D.—1 with difficulty, after one course of treatment. He was given a second injection on this date.

Case 2. L-Daffadar F. M. K. (pensioner, aged 40), 18th Cavalry.

Complaint:—Defective vision of both eyes, of over 10 years' duration.

Examination:—Scarring of lids, pannus more marked in R.E. than in L. E., opacities—large and extensive in R. E., almost blocking the pupil, but not so extensive in L. E., although situated in the pupillary area. The cornea in each eye was infiltrated with pannus vessels and signs of destruction by trachoma were too extensive to warrant any optical iridectomy, hence trial of this treatment was recommended to him.

25-5-40. Vision R.E.—1-60. Blood injection was given; Vision L.E.—6-60. Simultaneously in each eye, his eyes took much longer than usual to settle down.

20-8-40. Vision R.E.—6-60; Vision L. E.—6-18. He was given the same treatment again. Transferred to Rawalpindi in October and so not seen since.

Case 3. Turner L.S., aged 30, 22nd W/Shop, I.A.O.C.

Complaint:—"Granulids in both eyes", defective vision L. E. of over 6 years' duration.

Examination :—R. E.—pannus, scarring but no opacity ; L.E.—upper lid thickened, pannus. opacity large and irregular, almost blocking the pupil.

30-5-40. Vision R.E.—6-12 partial. Vision L.E.—4-60. He was put on Sulfanilamide by mouth, as well as given blood injection in L. E.

25-8-40. Vision R.E.—6-12 ; L.E. 6-18. No more injection was given as he was alright for his work.

Case 4. Signal-man K. F. aged 30, Lucknow Dist. Signals.

Complaint :—Defective vision L. E. of over 4 years' duration.

Examination :—A rectangular opacity from 12 o'clock spreading towards centre, as the result of an ulcer from a foreign body whilst in Egypt.

In June 1940, he had a fresh ulcer in the same eye at 10 o'clock from a foreign body.

31-7-40. Vision R.E.—6-5 ; Vision L.E.—5-60.

9-8-40. Vision L.E.—6-12. As he was under orders of transfer, he was tested earlier. He was asked to carry on with Quinine ointment.

The following notes are supplied by the Sub-Charge of Cantt. General Hospital :—

Case 5. Budhu, aged 20 years.

Complaint :—Inability to see with R. E., of 4 months' duration.

Examination :—R. E., P. L. present. L. E., Vision normal. A large opacity from 1 o'clock to 8 o'clock covering more than 3/4 of the cornea with fine haziness in the upper portion.

Cause :—Foreign body in R. E. about 5 months ago, when he was in his village where he could not get any proper treatment.

Admitted on 11-1-41 when he was given first injection.

30-1-41. Can see a man from 10 feet. R. E. had a second injection.

19-2-41. R. E.—Can see a man from 20 feet, and he was given a third injection.

1-3-41. Can see a man from 100 feet.

Case 6. Gurji, Hindu, aged 35 years.

Complaint :—Inability to see clearly with either eye (Patient was brought to the Hospital for treatment by another person).

Opacities from trachoma—duration 20 years, but he used to get about with the help of his left eye in which vision has become dim and hazy for the past one year.

Examination :—R. E.—Thick, white opacity covering almost the whole of the cornea except a small fringe at the upper part. Pigment present in the opacity. Anterior synechiæ. Red reflex but no details of fundus could be made out. P. L. when looking down to the toes.

L. E.—opacity in the lower quadrant of pupillary area with haziness and fine streaks over the remaining part. A comparatively thicker opacity outside the pupillary area at about 7 o'clock. Vision was present but everything appeared hazy and twisted.

Admitted on 28-1-41 and first injection given in both eyes simultaneously. A few days later, the report says: L. E., vision re-appeared, he can see things and walk about without anybody's assistance.

R. E. No appreciable change. 19-2-41. Injection repeated. The report reads: R. E. Very little improvement ; L. E. Can see from a distance more than 100 feet or so.

In Civil Government Hospitals, blood injections were repeated earlier than laid down in the course, as the patients being villagers did not want to stay in the town for a long time.

Conclusion.—The number of suitable cases for this treatment is necessarily small in the Army. This article is being sent for publication so that the treatment may be tried in the Civil Hospitals, where such cases will be available in sufficient numbers.

As seen from the notes, the results were quite encouraging, specially in cases where the opacities were small and localised. Although the vision improved in cases with deep opacities, the improvement was not so marked, nor so striking as in those with opacities involving only the superficial layers of the substantia propria.

Case No. 6 did not show any improvement in R. E. and it is worthy of note that this eye had anterior synechiæ which was not dealt with before the treatment of opacity was commenced.

It is possible that the synechiæ in some way interfered with the successful reaction to the treatment.

Yaws in Car Nicobar *

With Special Reference to the various Aetiological Factors
Influencing its Incidence.

BY

STEPHEN JONES.

Calicut.

THE following table indicates the number of cases of yaws treated at the Car Nicobar Mission Hospital, from 1936-1939.

Year.	Married Males.	Unmarried Males.	Married Females.	Unmarried Females.	Children under 10.		Total.
					M. C.	F. C.	
1936	16	8	12	3	4	2	45
1937	5	4	9	2	3	3	26
1938	4	2	5	Nil.	4	8	23
1939	6	Nil.	5	2	Nil.	Nil.	13
Total	31	14	31	7	11	13	107

"Yaws or Frambœsia" may be defined as a specific, tropical, contagious disease, endemic in warm and humid climate, running an indefinite and chronic course, affecting all ages and both sexes, caused by spirochæta

* Specially contributed to 'The Antiseptic'.

pertinui, characterised by skin eruptions, scaly macular patches, granulomatous and fungating ulcerative lesions, palmar and plantar hyperkeratosis, juxta-articular nodules, pigmentary changes, deformity and physical disability, and even mental, closely resembling syphilis in its clinical manifestations, serology and therapeutic response.

According to CASTELLANI, "Yaws is common in Burma, Malaya, Siam, Java, Batavia, Ceylon, present in Phillipine Islands, Samoa, New Hybrides and Fiji, and endemic in Nicobar Islands and among the aboriginal tribes of Australia." All these islands are known to have been connected up in ancient times and whether this points to any importance regarding the origin and spread of yaws is a doubtful question, although CHAMBERS regards this view as probable.

In Nicobars, especially Car Nicobar, the disease is definitely on the decrease and advanced stages of the disease with considerable deformity, etc. are not seen these years, probably owing to the vigorous injection treatment. Cases of yaws appear to have been mostly imported to Car Nicobar from islands of the Southern group, especially Chaura, where the disease was extremely rife some years ago.

Car Nicobarese stay in Chaura for varying periods of time during their yearly ceremonial visits, and the people of Chaura also make periodical visits to Car Nicobar for purposes of trade. Whether the disease is indigenous to Nicobars is a difficult question to answer.

Yaws may spread through a community with surprising rapidity under favourable circumstances. It is highly significant in affecting a large number of people without any resulting mortality. Some years ago, on two occasions, large numbers of patients including several entire families were brought from Chaura (a small island 40 miles to the south of Car Nicobar and about 2 or 3 miles square in area) to Car Nicobar hospital for treatment.

Infection takes place by contact. At least, a trivial abrasion or break in the skin appears necessary. Spirochaetes lodge themselves under the epithelial layers of the skin where they grow and proliferate causing slow and persistent irritation giving rise to acute inflammatory tissue reaction ending in degenerative changes.

Soles and palms by virtue of their thick distribution of the papillae and vascularity would encourage the deposition of organisms by blood. Owing to the thickness of the horny layer of the skin in these parts, the lesions take a longer time to develop than in other areas of the body. There may also be small fissures or cracks in the soles of the feet, where the organisms may usually be lodged.

Age and sex.—POWELL states that the disease is common between the ages of 2 and 14. It is suggested by CHAMBERS that adults are less liable to infection for the following reasons:—

- (1) Adults being less playful do not come in contact with one another to such an extent as children.
- (2) Adults and older children are better clad.
- (3) Few adults sleep together than do children.

Out of 43 patients brought from Chaura for treatment in Car Nicobar hospital in 1931, 22 were children under 10 years of age, and, from the

table given above, only in the year 1938 there was an unusual increase in the number of children affected. It is also said that, owing to increased activity of the boys, they are more liable to trauma than girls, hence the greater chances of incidence of the disease in boys. From the table, it can be noted that in 1938, out of the number of children affected, girls showed an increase. From this, then, definite conclusions regarding the affinity of yaws more towards male children, cannot be drawn. It can be further observed from the table that both sexes and all ages are equally susceptible to the disease under suitable conditions.

Race.—In Jamaica, it is said, the disease is mainly confined to the Negro population but, on the other hand, the United States of America has a large Negro population practically free from yaws. C. J. SCOTT says, "About seven white miners developed yaws while working in Johannesburg." SPITTAL states, "Yaws hardly ever attacks Europeans or, for that matter, any cleanly living individual whatever his race."

I have treated a few cases of ulcerative lesions in the extremities of Minicoy Mahomedan traders residing in Car Nicobar. All races appear equally susceptible to the disease under favourable climatic and other conditions.

Warm climate and humidity.—Warm climate and humidity are most important predisposing factors, and other factors such as rainfall, geological formation, vegetation and altitude are also important in so far as they affect humidity. RAMSAY points out that the hill tribes of Assam, who exhibit obscure lesions of yaws at high altitudes, break out into florid yaws when they come down to the plains.

C. J. SCOTT reported cases of yaws among miners working 2000 feet below sea level and pointed out that bare and moist bodies of men working 1000 to 2000 feet below sea level appeared suitable sites for spirochaeta pertinui.

Car Nicobar is a flat island with an average annual rainfall of over 100 inches, having a hot and extremely moist climate. Usually in damp and humid climates, vegetation is profuse and tends to increase humidity. Humid climate slows evaporation of moisture from the skin and is said to have some influence in the production of skin lesions, since the spirochaeta pertinui coming in contact with moist surfaces survive much longer periods, thereby having greater chances of multiplying.

Nicobarese are in the habit of shampooing their bodies with cocoanut oil usually after a bath and the skin, especially of children, is almost always oily to the touch. Will this increase the possibility of harbouring the organisms and help in the production of skin lesions if other conditions are favourable?

Soil.—Moisture in the surface soil increases humidity and the factors that tend to increase the dampness of the soil are likely to cause greater humidity and consequently increase the incidence of the disease. In Car Nicobar, hardly a month passes without at least a few inches of rain, whilst several marshy places and stagnant pools exist in the central portion of the Island all the year round.

According to Car Nicobar hospital records, the maximum incidence of the disease appears to be during the months of May, June, and July, and this points to the possibility of the rainy weather influencing the incidence of the disease.

Habits.—Usually, primitive people, especially mothers, who do not realise the contagiousness of the disease, kiss and embrace the children with juicy frambœseomata on the exposed parts of the face and limbs.

Children, who are suffering from the disease, are allowed to play and mix freely with healthy children, all sharing and biting one and the same fruit or other eatables. A person who has got the disease is not kept away, but eats and sleeps with other healthy members of the family, usually exchanging clothing such as short pants, underwear, vests etc. A Nicobari boy was once admitted for yaws contracted through wearing the pyjamas of a friend who was under treatment for yaws.

Unhygienic social life appears to be one of the most important predisposing causes in Car Nicobar. Sleeping in the same bed and using the same clothes are very dangerous. This is a common practice among the Nicobarese. When a patient is admitted for yaws in segregation huts, his or her relations may be seen sleeping with the patient on the same mat, sharing the same sheet and blanket.

If a man with definite diffuse lesions of yaws in various parts of the body, including the skin of the penis, indulges in sexual intercourse, is it possible for the disease to be transmitted to the female?

Flies.—SCHILLING, NIELON, DANCER, WILLIAMSON, and others mentioned the possibility of transmission by flies. "Small flies may gorge themselves with the *ishor* from an infectious ulcer, then alight on a non-infected ulcer or other abrasion and vomit up the material." KUMM in 1934 made further investigations in greater detail and concluded from his findings that transmission by flies, the "Hippelates Pallipes", was possible only within 7 hours after feeding on infectious lesions. Unhygienic surroundings with a tendency to breed and multiply this variety of flies which occur in areas with tropical humid climates is another contributory factor.

Poverty.—Poverty is put down as another factor influencing the incidence of yaws. Low standard of living, overcrowding and insanitary conditions are sometimes and in some places dependent on poverty.

Diet.—As a consequence of poverty or ignorance of a balanced diet, the constitution may lose the powers of resistance to disease, though improvement of diet alone is not likely to prevent the incidence and spread of the disease.

Treatment.—Among the Nicobarese, the disease is known as "To-choich Invah" (an incurable disease). The Nicobarese believe that the disease is caused by an evil spirit, and go, as a first step, to a witch-doctor to have the injurious substances extracted from their bodies. The witch-doctor, by gesticulations and ventriloquistic sounds, ostensibly extracts those harmful things which the spirits have caused to enter into the body. It is only after repeated and unsuccessful treatment at the hands of such witch-quacks that the patient reluctantly seeks hospital treatment. Owing to the growing belief in injection treatment, old custom of witchcraft is gradually dying out and in recent years the Nicobarese have been resorting more to hospital treatment. No efficient methods of prophylaxis or treatment are of any avail if the local conditions, customs and manners of the people and other circumstances are not carefully considered.

PROPHYLACTIC TREATMENT.—The following prophylactic measures are advocated :—(1) Patients who have yaws should not be allowed to mix with healthy individuals, sleep, eat, use the same clothes, etc., as is so common

amongst primitive people. (2) Isolation of those with the disease till they are declared free. (3) Immediate attention to all minor injuries in an endemic area. (4) Protection of feet and legs, particularly in rainy weather. (5) Suitable education of the people, especially the younger generation, by magic lanterns, describing the nature of the disease. (6) Destruction of badly infected houses by fire. (7) Prevention of water pollution by yaws-discharge.

Simple and primitive Nicobarese pay no heed to prophylactic measures and even when segregated, sympathetic relations come with food, staying and sleeping with the patient on the same mat.

LOCAL TREATMENT.—Dusting powder with Calomel, Mercurial ointments, simple ointments as Boric ointment have all been used. It is said that Mercury has no effect on yaws lesions but our experience is different. Sometimes no specific antiseptic dressing is found necessary except a sterile saline dressing to prevent secondary infection.

SPECIFIC TREATMENT.—I have often heard from medical men that one injection of N.A.B. or any other arsenical preparation is quite enough to establish a cure. I may be allowed to state that an indisputable case of yaws occurred in a woman who attended the hospital thrice with recurrence, each time receiving five injections. It is now a year since this patient has been discharged and so far is free from the disease. It is safe to say, one or two injections invariably clear the lesions but never cures.

The procedure adopted in this hospital is as follows :—

Adults:—Intravenous injections of N.A.B., commencing from 0.3 grammes, gradually increased up to 0.6 grammes. Five to six injections at weekly intervals are given to complete a course.

Children:—Intramuscular weekly injections of Sulphostab commencing from 0.1 grammes or 0.15 grammes, dose being gradually increased according to the age and susceptibility of the patient.

Sometimes, patients do not stay for the complete course of five or six injections and hence recurrence is common. Some leave the hospital, usually without permission, immediately after the skin lesions disappear thinking they are completely cured, and not until fresh lesions appear do they resort to further treatment. I have noted cases of recurrence due to both inadequate treatment as well as after one or two complete courses of treatment. Before injection treatment, the patient is examined for any organic disease contra-indicating the use of arsenic and usually a saline purge is given before the injections are started. Bismuth preparations (Bismo-stab) were tried in conjunction with N.A.B. but had to be discontinued as these gave considerable pain and discomfort to the patients who consequently refused to undergo treatment. Nicobarese prefer intravenous painless medication to painful intramuscular injections. This point is of importance when dealing with primitive people.

GENERAL TREATMENT.—Good nourishing food and tonics are of value. The following mixture is given three times a day during the interval between the injections.

R Pot. Iodide	...	grs. v
Liq. Hydrarg. Perchl	...	℥ x
Spts. Amin. Arom	...	℥ x
Aqua ad	...	℥ 1

C/o. Mr. D. Timothy,
Near Veterinary Hospl., Calicut.

Typhoid in Kashmir*

BY

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It sounds rather queer to name an article "Typhoid in Kashmir" as if illnesses are not the same all over the world. But that is just what I would like to show, the difference between typhoid in Europe and here in Kashmir.

How did we use to diagnose the typhoid-cases in Europe? The patients used to have a rise in temperature, every day a bit higher, till after three or four days, they developed a continua between 103 and 104 degrees. The pulse rate was relatively slow, the temperature and pulse charts showed a great discrepancy. The patients complained of severe head-ache, became somnolent, and in the second week, got diarrhoea, the so-called pea-soup stools characteristic of typhoid, and the whole typhoid-group. They showed a spleen tumour and leucopenia. The second week brought also the typhoid-spots, usually much more frequent in paratyphoid A and E than in typhoid; also the positive Widal test was found in the blood, and the positive Diazo reaction in the urine.

Whenever we saw a patient with a continuously high temperature headache and bradycardia, we suspected typhoid. The diarrhoea, spleen-tumour, typhoid-spots and leucopenia in the second week proved our diagnosis to be correct, even before we got the bacteriological proof.

How different in Kashmir! We have to learn our clinical features of typhoid once more, and I found it at first rather difficult to diagnose a typhoid-case. Kashmir is very fortunate in its climate, and we do not see any tropical diseases (no malaria) or any special diseases in the summer with the exception of typhoid and paratyphoid A and B. We see a fair number of them. I, therefore, had the opportunity of studying the symptoms and the course it takes. Instead of the continuously high temperatures, we have here swinging temperatures, in the morning between 99 and 100 degrees and in the evening between 103 and 104 degrees. The patients complain of headache, sometimes show a spleen-tumour, typhoid-spots and leucopenia, but never have I yet seen a case with diarrhoea; on the contrary, all our patients were suffering from constipation, and had to have an enema on alternate days as well as Paraffin oil by mouth. That means that two of the main symptoms are missing—the continua and the diarrhoea. The blood-agglutination test (Widal) shows the same result as in our cases at home, positive in the second week, but the Diazo reaction has been negative in all cases I have seen so far.

Several of the typhoid cases here in Kashmir have shown a complication which is extremely rare, and practically unknown in Europe. The patients got in the third or fourth week of their typhoid, a thrombosis in the vena saphena, either in one or both legs, a complication which prolongs the duration of the illness considerably. The percentage of hæmorrhages seems to be about the same.

I would like to add a few words about therapy. We are unable so far to influence typhoid by drugs. We tried Sulphonamide, but could not see any improvement, and it seems to have a toxic effect on the patients.

* Specially contributed to 'The Antiseptic'.

I had often to stop giving it because of very distinct cyanosis which disappeared quickly after stopping the drug. The prognosis of a typhoid-case is only and exclusively dependent on good nursing and feeding. We still have to fight against the idea of starving a typhoid-patient, a theory which really should belong to the past. The patients very often get nothing else but milk or glucose water and are in a very bad way, when they come to us. It is necessary to keep the strength of the patient up by giving him nourishing food: all sorts of soups, sago, gruel, jelly, eggs, honey, cocoa, chocolate, biscuits, butter, etc. By this method the patients do not lose so much weight, fight the infection better and have a quick convalescence.

We recommend inoculation against typhoid before coming up to Kashmir, as people are apt to get a typhoid-infection in the first two weeks after the inoculation; later on it seems to be quite a good protection.

Classification of Anæmia

BY

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AN apology is needed in classing anæmia as a disease; because it is not a disease by itself but a net-result of factors responsible for its production.

It was the usage to classify anæmia under two main heads—primary and secondary. Under the head primary, the following different types were known: (a) chlorosis (b) simple primary anæmia, (c) Pernicious anæmia (Addison-Biermer anæmia), (d) Aplastic anæmia, (e) Sickle-cell anæmia. The secondary anæmias were divided into two main groups—those following hæmorrhage (i) acute and (ii) chronic and—those secondary to various diseases. The story of anæmia is one of the romances of modern medicine and by investigation being carried out since 1920, the inadequacy of the old classification (primary and secondary) has been realised. Different hæmatologists have tried to supply a new classification that may cover the needs in respect of treatment and cure of the disease, but so far a perfect classification has not been achieved. With the advent of investigation, the disease chlorosis could not find its existence under the caption of primary anæmia.

One classification, however, that had recently been worked out (WINTROBE) was by measuring the diameter (*D*) of erythrocytes as follows:—(1) macrocytic (increased *D*), (2) microcytic (decreased *D*), (3) normocytic (normal *D*). The last one includes true secondary anæmia following acute hæmorrhage and certain aplastic anæmias. Microcytic anæmia is divided into simple orthochromic associated with chronic diseases and hypochromic in which are included achlorhydric anæmia and the anæmia following chronic hæmorrhage and parasitic infection. It may also result from deficient supply and utilisation of Iron and from lack of material other than Iron needed to build the Hb. molecule. Macrocytic anæmia is produced by a relative deficiency in important food factors probably associated with the vitamin B₁₂ complex, and also by a state of chronic malaria. The typical example of macrocytic anæmia comprises the pernicious type and all sorts of controversies centre round this type of macrocytic anæmia.

* Specially contributed to 'The Antiseptic'.

At one time again investigations were carried out under two main lines: (1) Hypochromic microcytic anæmia (HART, STEEN BECK, in laboratory side and HALLEN, MACKEY and WITTS on clinical side) (2) Hyperchromic megalocytic anæmias (MINOT, MURPHY, CASTLE & DAVIDSON). In dealing with them, a look into the following facts may be interesting:—Precursor of R. B. C. is named erythron in the reticulo-endothelial system and under abnormal condition the erythron may show hyper-or hypo-plasia or aplasia. In the case of aplasia, there occurs a functional failure of bone marrow and the resulting anæmia is normocytic. In the cases of hyper-plasia, two degrees of tissue-reactions are noted: (i) firstly, normoblastic reaction of tissues in which the number of normoblasts is greatly increased in peripheral blood and the erythron spreads to occupy the medullary cavity at the expense of fatty material. The aetiology of this group is heterogenous and the macrocytosis here is secondary in majority of cases, to prolonged stimulation of bone marrow. To prevent the normoblastic reactions in the bone marrow, the supply of Iron, Vitamin C and Thyroxine is necessary (WITTS), (ii) Secondly, we have the megaloblastic reaction of tissues, *i. e.* the cell differentiation is shifted back further and erythron occupies more and more spaces. It should be noted that whenever there is megaloblastic reactions *e.g.* in pernicious anæmia, the youthfulness of R.B.C. can be proved by the presence of a good number of reticulocytes in the blood demonstrable by vital staining. The supply of some specific anti-anæmic factor may be responsible for the cure of pernicious anæmia (on megaloblastic basis by altering the megaloblastic reaction. It follows then that this type of anæmia develops consequent on the deficiency of the specific anti-anæmic factor necessary for the development of the red cells at the megaloblastic level.

From a perusal of the above facts, possibly we shall not be very much mistaken if we classify all macrocytic anæmia under the heading "Deficiency anæmias".

It must be pointed out, however, that all megaloblastic anæmias are macrocytic, but all macrocytics are not megaloblastic in origin. It is necessary to emphasise that apart from the common factor of increased diameter, the macrocytic anæmias of the group, having normoblastic degree of blood formations, differ greatly from the true megaloblastic anæmia in their (a) aetiology (b) blood picture and (c) failure to respond to the specific anti-anæmic factor.

The specific anti-anæmic factor may be deficient in the system either by (A) failure in its manufacture in stomach (B) defective absorption (C) ineffective storage, mobilisation or utilisation. On this basis, megaloblastic anæmias may be classified as follows:—

Under head (A):—(1) Pernicious—Addisonian and Pregnancy anæmias—The loss of the specific gastric enzyme, normally present in gastric secretion in the former is permanent whereas in the latter it is temporary (Intrinsic factor of CASTLE), (2) Anæmia resulting from widespread destruction of stomach wall by cancer (Intrinsic factor), (3) Tropical macrocytic anæmia (WILLS)—This arises from a food deficiency and not from the failure of gastric secretion (Extrinsic factor of CASTLE). It is interesting to note again that NAPIER holds the view that "the recent works on treatment does seem to indicate that the deficient substance in this type is not identical with the extrinsic factor." Serious consideration again is to be given to the

food deficiency factor which is the deficiency of an independent substance or group of substances necessary for the development of red cells at the megaloblastic level just as iron is at the megaloblastic level.

Under head (B):—Dietary deficiency and impaired gastric secretion may also be considered along with the failure to absorption of the factor and this may occur in prolonged diarrhoea, sprue, etc.

Under head (C):—Are included anæmias as a result of severe liver diseases (Cirrhosis). Some authorities are of opinion that in these cases, the macrocytosis is really a false one which may be due to the flattening of the cells when spreading. It is thought that the physical state of the plasma in cirrhosis is conducive to this stage. The damage to the liver prevents it from functioning efficiently as a storage organ for the specific anti-anæmic factor thus allowing a megaloblastic blood picture to occur. Under treatment with the specific anti-anæmic factor, the blood picture of such cases may be altered to normal and the anæmia being cured, but really without altering the underlying disease of the liver.

Failure on the part of the bone-marrow to utilise the active principle whose absorption and storage being satisfactory is another cause for the occasional production of megalocytic anæmia (WILKINSON'S achrestic anæmia). Before this variety is finally accepted in the megalocytic head, investigation should further be taken up in the studies of the bone-marrow, partial aplasia of bone-marrow.

From the stand-point of treatment, however, it is not a sound practice to classify anæmias according to the size of the erythrocytes only. The classification, therefore, from the aetiological point of view, is possibly the best one at the present moment and the following manner of classification of DR. WITT'S, delivered in his Goulstonian lectures, will certainly be of much interest:—

(A) True secondary anæmia due to loss of blood from skin, mucous membrane or from serous membranes or surfaces may be:—

- (1) Acute (normocytic-orthochromic) or,
- (2) Chronic (microcytic-hypochromic) due to: (a) Following external or internal injury; (b) Associated with diseases of various systems *e.g.* gastric or duodenal ulcer, phthisis etc.; (c) Associated with congenital blood diseases *e.g.* hæmophilia; (d) Parasitic in origin.

(B) Anæmia due to errors of erythrogenesis and may be due to:—

- (1) Aplasia or hypo-plasia of toxic or mechanical origin *i.e.*, there occurs a functional failure of bone-marrow (normocytic—usually orthochromic).
- (2) Dysplasia—Here comes the deficiency anæmias either due to deficiency; (a) of iron metabolism or, (b) of hæmopoietic principle (CASTLE'S extrinsic or intrinsic factors) or,
- (3) Of supply of vitamin factors "Vit. C" or of Thyroxine.

This type of anæmia due to dysplasia may be either microcytic or macrocytic according to the stage at which hæmopoiesis is interfered with. In the pernicious anæmia (Addisonian and Pregnancy), by the absence of

CASTLE's intrinsic and vitamin B₂ factors, the blood formation is checked at the megaloblastic stage proved by the reticulocytes appearing in the blood and here we get the macrocytic anæmia. In anæmias caused by the deficiency of iron and vitamin C and as a result of sprue etc., where the blood formation has been checked at the normoblastic level, microcytic hypochromic anæmia results.

(C) Anæmia due to errors of erythrolysis may be due to:—

- (1) Defects in the red cells themselves affecting and rendering them more susceptible to normal lytic processes brought about by parasitization (malaria) and hæmolytic poisons (chemicals, toxins, snake venom).
- (2) Due to hyperfunction of erythrolytic tissues *e.g.* the reticulo-endothelial cells in the spleen and elsewhere.

The fragility of R.B.C. is increased in (1) and decreased in (2) but in both these cases, the anæmia is usually of normocytic orthochromic type.

If the erythrolysis is sudden, the reaction in bone-marrow will be great and the reticulocytes will appear in large numbers and sometimes erythroblasts (macrocytes and normoblasts) as well.

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Sasthikola, Jescore.

Vitamin A in Health and Disease*

BY

DR. S. GURUBATHAM

Schell Memorial Hospital for Eyes, Vellore.

THE subject of nutrition with special emphasis on vitamins is assuming greater importance and as our knowledg along this line grows, we find that many of our ideas about etiology of diseases have to undergo radical changes.

I have chosen for this paper 'Vitamin A', not because it is more important than other vitamins, but because of the closer realtionship between severall of the hitherto obscure diseases of the eye and deficiency of this vitamin and, as my own experience has been derived more from eye diseases, I had to pay special attention to this vitamin and that has led me to think about the effect of this vitamin on other systems of the body. When I touch the other systems in this paper, I dare not be dogmatic, and so I only throw out suggestions to provoke thought.

HIPPOCRATES has advocated the use of liver for curing night blindness, and in this country from time immemorial liver has been used in all cases associated with night blindness. Now we know that the liver is the store-house of this vitamin. After 1913, experiments on rats have proved that they could not grow when put on olive oil, almond oil, lard etc; but they grew rapidly if put on butter fat, codliver oil or egg yolk. Thus was proved that certain fats carried an essential nutrient which MC. CALLUM called "Fat-soluble A" and which is now designated as vitamin A. Later experiments have led to find that while vitamin A is found as such in animal fat, in the vegetable kingdom it exists in the form of hydrocarbon called carotene, precursor of vitamin A. It derived its name carotene from carrots the only root very rich in vitamin A. Red palm oil, leafy vegetables like drumstick leaf, agathi (*Sesbania grandi flora*), amaranthus, betel and corriander seeds and fruits such as ripe mango, pappaya etc., are very rich in carotene, which is converted into vitamin A in the liver. The mechanism of this process is not clearly known yet. This carotenoid substance is synthesised by plants more from its green outer leaves than from the white inner ones: a process similar to chlorophyll formation. From these, animals derive their supplies. Fishes perhaps derive their supply from sea weeds and algae.

Vitamin A is fat-soluble. It is not destroyed by ordinary cooking; but sunlight and high temperatures easily destroy it. Tamarind is a good preservative, a fact perhaps known empirically by Indians and hence used widely in the South Indian cooking. It is very essential to normal growth, normal function of the eyes and for normal function and structure of the epithelial tissues all over the body. Liver is the chief storehouse and to a great extent the lungs and the kidney. With a normal intake, even after a complete removal of vitamin A from diet, it requires a considerable period of time, perhaps from four to six months, to deplete the liver of its store of vitamin A. When the diet is poor in vitamin A, the normal level cannot be maintained. The storage capacity is relatively greater at the age of most rapid growth. Vitamin A being fat-soluble, the diet should be fat-rich; but if there be too much of fat, it is likely to pass down the intestines without

* Specially contributed to 'The Antiseptic'.

being absorbed in the circulation. Vitamin D also is very essential for its assimilation. What part it plays in the absorption is still a moot point. It is observed that in the absence of vitamin D, it is not absorbed. It is easily absorbed except in gross lesions of the liver and intestines. Liver plays a significant role in regulating the concentration of the vitamin through the body. Carotene is less efficient than vitamin A. Hence the preference of cod-liver oil to red palm oil though both have about the same quantity of it. If excess of it is taken, it is destroyed in the liver or oxidised in the blood stream. Excess has no bad effect on the system.

A normal adult needs from 3000 to 5000 International units per diem and this intake has to be doubled in the case of growing children, pregnant and nursing women and people affected with several infectious diseases, a classification of which has yet to be made in the light of the above facts. In this connection, I should like to mention two instances which came to my notice recently. My son aged 14, returned home after his examination last month. Three days later, I suddenly noted the conjunctiva of both the eyes, on the temporal side of the cornea, developing deep dirt-like patches, an early symptom of xerosis, a definite symptom of avitaminosis A. I immediately put him on Adexolin, vitamin A concentrate and Irradol, vitamin D concentrate.

Four days after the appearance, the conjunctiva being in the same condition, the boy went in for a severe type of measles, which gave me some anxiety also. But, by the time the infection subsided, the patch of dirt also disappeared; of course the Adexolin was continued throughout. The next is the case of a young man in whom I was very much interested. While in the college, he suddenly developed the same type of xerotic patch. His class-mates and one of his professors remarked about it. Soon after this, he went in for a mild attack of pleuritis, the acute symptoms of which lasted only three days. Later, on examination at the sanatorium, he was found to have T.B. lungs in early stage. He was also put on Adexolin in addition to his usual treatment. But occasionally, the patch was appearing. Since finding that vitamin D has an effect on the absorption of vitamin A, I put him on Irradol and the patch has not reappeared. These are clear cases where, in an acute infection, the system has drawn on its reserve of the vitamin—thus proving the need of greater intake of this vitamin in these infections. In all cases of keratitis following small-pox, the condition has been very intractable until put on an active vitamin treatment.

Though our knowledge at present is inadequate with regard to the physiological function of this vitamin—whether it is an integral part of the tissues or it works through blood vessels—we come to know more and more clearly what effect the absence of it has on different vital organs of the system, or on the course of certain infections. My own experience is that nutritional disorders are on the increase. Twenty years ago, working in an Out-Patient Eye-Clinic of 200 a day, I used to meet with three or four cases of keratomalacia per year. To-day, the daily attendance being the same, I meet with almost one a week. Between 1937 and 1940, I had been supplying a large number of clinical material for one of Dr. Aykroyd's assistants to investigate into different aspects of avitaminosis on the eye. Using a photometer of his own pattern, the sensitivity to light of the eyes of 500 school children was measured. On a subjective examination, about 5% of them were seen to be suffering from nutritional disorders as xerosis, angular conjunctivitis etc. In the dark room, about 20% of them

showed a lowered dark adaptation to light—the earliest symptom of deficiency. Night-blindness is a more advanced condition.

Vitamin A is a very important chemical component of the retina which has a substance called visual purple which is very sensitive to light. This visual purple in the retinal cones is bleached to an orange pigment called visual yellow, when light strikes on it. The yellow colour gradually fades and the retina becomes colourless. If sufficient vitamin A is supplied to the retina through the blood stream, it rapidly recovers its original appearance and function. In deficiency of vitamin A, there is delay which is measured by the photometer mentioned above.

Hence, the earliest symptom of vitamin A deficiency is hemerolopia. Even severe cases of hemerolopia may be present without any appreciable changes in the eye. The dry patch of conjunctiva, decreased secretion of tears, thickened white spot, Bitot's spot are all the manifestations of the next stage in this deficiency. Keratomalacia is the last stage of the eye manifestations. Infection of the cornea is favoured by the accumulation of keratinized cells to be described hereafter, and by decreased secretion of tears for washing the eye leads to hypopyon and panophthalmitis commonly seen following keratomalacia.

Some cases of retrobulbar neuritis, optic atrophy and retinal and choroidal affections of indefinite etiology are now treated as deficiencies. Other affections as the common sore eye, blepharitis, angular conjunctivitis, phlyctenular kerato-conjunctivitis some intractable forms of trachoma, especially in young girls, follicular catarrh associated with granular pharyngitis, keratitis after small-pox etc., hypopyon ulcers etc., all of which have some definite etiological factor—a bacterial infection—would not assume serious proportions, commonly met with in out-patient clinics, had it not been for deficiency in vitamin A. I always remember Dr. Aykroyd's statement once—how an epidemic of dysentery broke out in a whole group of monkeys kept for nutrition experiment. Among those on balanced diet, only two had a mild attack which recovered without any special attention, while, among the experimentally underfed ones, quite a large number of them—about 40%—developed dysentery and in spite of very careful attention mortality was very high. This experience of his made me change my treatment of the above eye conditions as nutritional disorders in addition to usual treatment, with often dramatic results.

In my experience, skin lesions due to deficiency of vitamin A were not always associated with eye lesions. Another friend of mine made his own observations working among plantation coolies. In his experience, he came across a large number of patients suffering from skin affections, and these were not always associated with eye lesions. This led us both to feel that both were due to vitamin A, this difference must be due to existence of two different forms of A, an-A complex. We drew the attention of Dr. Aykroyd to this fact, three years ago, and he is investigating this. In the meantime we find mention made in the literature of this division into vitamin A₁ and A₂.

Dryness of the skin, loss of lustre in the face, the absence of shining appearance, perhaps, are the early symptoms of this deficiency. Sometimes, there appears this shining at the time of a girl's puberty, but it rapidly disappears after the first child birth. The sebaceous and sweat glands, hair and nails are affected—brittle and keratotic papules—goose or frog-skin—

appear, over the thighs and arms more in adults than in children. The dry scaly shrivelled appearance of the skin in infants often taken to be congenital syphilis is a symptom of deficiency; or congenital syphilis has produced the deficiency. In many specific diseases as syphilis, gonorrhoea, tuberculosis, pneumonia, typhoid etc., specific treatment supplemented with the treatment of deficiency is productive of better results.

Effect of vitamin A on epithelial tissues.—As mentioned before, vitamin A is very essential for the normal function of the epithelial tissue and detailed investigation by various specialists has led to the conclusion that in the absence of vitamin A the epithelial structure, practically all over the body, is affected, the earliest consequence being an atrophy. These are replaced by 'stratified keratinising epithelium'. As this atrophy progresses, while keeping up its normal appearance, its physiologic function and the function of covering the cell membranes are impaired. An attempt at repairing this atrophied epithelium ends in formation of the 'stratified keratinizing epithelium'—which explains the cause of several affections of the mucous membranes and diseases mentioned in this paper.

When vitamin A is supplied and absorbed by the system in adequate quantities, the cells very rapidly recover and recovery is sometimes dramatic. In keratomalacia, if Prepalin, a vitamin A concentrate, is administered subcutaneously, immediately the cornea begins to melt like wax, the result is dramatic. The progress is arrested and very often the cornea is restored within three or four days.

Epithelium being the first line of defence against bacterial invasion in this new keratinized form due to lack of vitamin A, organisms easily find an entry. This deficiency has already lowered the defensive power of other lines of defence as lymph glands etc., with the result that the body is easily overcome by invading organisms. Under such changed conditions organisms like Morax-Axenfeld, Koch-Week's, coma and dysentery bacilli multiply easily. Diseases caused by these organisms are more prevalent among the rice-eating races of India and China, rice being so low in nutritional value and through milling there is practically no vitamin A left.

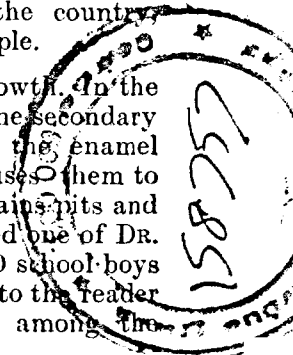
Sunlight with its abundant supply of ultra-violet rays—the precursor of vitamin D, a necessary factor in the absorption of vitamin A and calcium, is plentiful in this country. At the same time, it kills bacteria so easily. In spite of these, people in the Eastern countries are prone to all kinds of infections and the span of life is only half that in the Western countries. In addition to ill ventilated, dirty, over-crowded houses, with our present knowledge of vitamins, one could easily know that this is a very important cause. Of course, economic condition is also another factor. Hence, in India, more emphasis has to be laid on preventive work. At present, every medical man has to tackle hundreds of cases a day, and so he has neither the time nor energy to attend to details in the treatment of diseases. If sufficient time and attention is paid by national health forces to tackle nutritional problems, that can reduce half the attendance of our out-patient clinics. Each doctor will get fewer but real types of diseases, he will be able to treat them more efficiently and he too will become more efficient.

Among infants and children, we find symptoms of avitaminosis A, associated with or before xerophthalmia, and before keratomalacia sets in, thin mucopurulent discharge from the ear and running of the nose which

do not respond to any local treatment. Next are the changes in the mucous membrane of the respiratory tract, with a peculiar low cry, which once heard can always be remembered. Hence the frequency, severity and persistence of pneumonia. When pneumonia sets in, it always ends fatally. In these cases, there is always thin watery diarrhoea. This diarrhoea, appearing in all advanced cases of avitaminosis A, is controlled by administering vitamin A and astringents like Bismuth etc. But if, after once controlling, it recurs, like pneumonia, it ends fatally.

In the gastro-intestinal tract, this keratinizing process reduces the mucous secretion, exposes the lining of the gastro-intestinal tract to injuries caused by friction with coarse particles of food. In South India, in spite of the fact that there are at least seven varieties of cereal grains, each superior to rice in most respects and also grown easily as dry crop, rice-eaters, because they do not get enough of vitamins in their food, develop a digestive organ which cannot stand the harder grains. So, they go in for more and more polished rice, the stomach gets more and more delicate, and less of vitamins with the resultant vicious circle. An understanding of this condition has changed my attitude towards tackling these cases. I was a chronic dyspeptic for over 20 years with high acidity of the stomach. I managed to break this vicious circle with striking changes in my health. My dyspepsia has completely disappeared, and while I used to get a severe attack once in two to six months, the symptoms have disappeared for the past two years. I was not able to tolerate any rice at nights and instead, lived on bread, butter and milk. If I partook of a dinner, I could not sleep at night. Now, I stand any coarse food, all cereal grains and even chillies. At first, I introduced vitamins in the form of concentrates which did not have marked effect. Then, with a determination, I changed the food to unpolished rice and Kambu (*Pennisetum typhoideum*) bajra, sajjulu, the cereal richest in vitamin A, fat and iron, superior even to wheat. At first, it upset the digestion of the whole family. Then, gradually, in the course of six months, we introduced a complete change in the family diet with very good results. Several of my friends who had followed, among whom are several doctors, all have shown a marked improvement in their health. Some doubting Thomases remarked that it was a psychological factor. Whether the result was psychological or not, we have derived benefit. In the light of the above experience it is worth investigating whether most of the gastric ulcers, intractable, idiopathic, chronic affections of the gastrointestinal tract, which lead on to gastric surgery are all due to several causes attributed to them hitherto, or whether they are not simple or complicated nutritional disorders. Once a strong opponent of chillies, tamarind, betel leaves and chunam etc., now I advise people to use them freely because in the present economic condition of the country they are the major source of nutrition to most of the poor people.

As mentioned in the beginning, it is essential to normal growth. In the deficiency of vitamin A, interruption of growth seems to be the secondary result of a disturbance of the epithelial tissues. In the teeth, the enamel forming organs being of epithelial origin, avitaminosis causes them to atrophy. In less severe cases, the enamel is defective and contains pits and fissures which leads the latter to decay. Two years ago, I helped one of Dr. AYKROYD's assistants to investigate the dental condition of 500 school-boys of over 15 years. The finding is very significant and I leave it to the reader to make his own interpretation. My observation was that among the



poorer classes of the boys while the gum was swollen and easily bleeding, with plenty of tartar, indicating deficiency of vitamin C more than A; very few of them showed any tenderness of the dentine, or pitting. Among the middle and upper class of boys the gums looked normal and teeth clean; but a large percentage of them showed a pitting and some early stage of caries; early symptom of vitamin A deficiency due to polished rice but, in the case of the former, the diet was coarse and had more of vitamin A in food, some of them taking other grains as cholam and ragi. A few of the Anglo-Indian boys examined from the European school all showed, without exception, pitting. Several of them had advanced caries of one or more teeth and some had already had artificial teeth; accustomed more to artificial and tinned foods and foods in the course of tinning lose all vitamin A.

Most investigators believe that vitamin A deficiency is one of the several causes of uro-lithiasis. Most probably, avitaminosis A causes loss of power of the mucosa to prevent invasion by micro-organisms which are normally present about the urethral orifice but which, in health, are unable to thrive on a mucous surface.

Its effect on the nervous system is still under investigation. MELLANBY supposes that xerophthalmia is a degenerative change in the nerve supply of the cornea and that pyorrhoea and dental caries may be an expression of neurotropic control. Though nervous symptoms in such diseases as beri-beri and pellagra are due to water-soluble, heat-resisting vitamin B complex, lack of vitamin A aggravates the condition.

Thus our present knowledge of nutrition and vitamin is daily increasing and in the course of another decade we would have made very much progress. But this war is going to make the problem of proper nutrition a serious one.

In this paper, I shall not attempt to deal with the treatment of the condition which is another wide subject. I would only like to emphasise that we should not always depend upon specific vitamins put in the market. As Insulin, these are like handles which lift a sinking man. But our source of vitamin has to come from natural foods. We do not know yet what more latent life there is in the natural food and I strongly advocate a reform of the present diet and introduce natural food rich in all essentials of nutrition. A study of this has to be made by each individual and I cannot think of a better pamphlet than Health Bulletin No. 23, written by DR. AYKROYD and published by Government and sold at 2 As. per copy. With that could be added also another Bulletin No. 28 on Rice.

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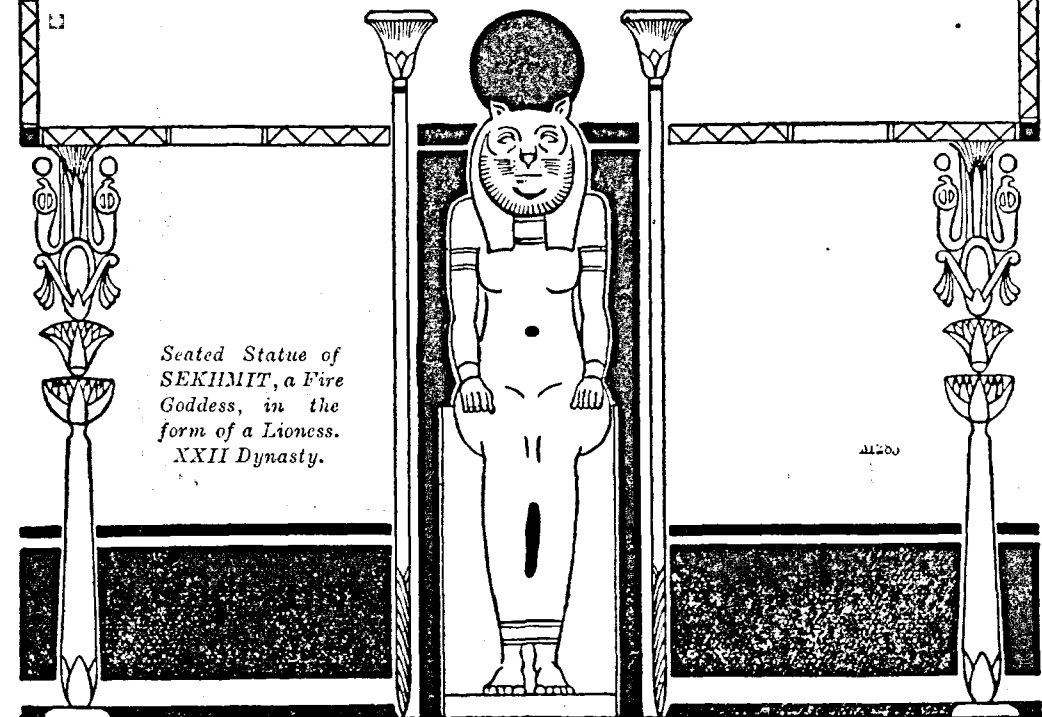
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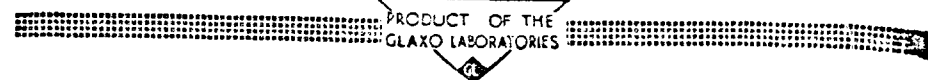
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Cases and Comments

A Case of Cerebral Malaria

BY

J. N. BHATTACHARJEE L.T.M.

R.M.O., Gopalpur T.E.

A TEA-GARDEN Paharia coolie, aged about 32 years, was admitted in a state of complete unconsciousness on the early morning of 4th Apr. '41.

Previous history.—He was a new resident of Duars. He came only 6 months back to this garden. On 1st April, he was attacked with a slight rise of temperature and with this temperature he went on his duty about 1½ miles away from his hut. Next day, he returned to his hut. I saw the case at about 10 A.M. on that day (2nd April 1941) and he looked very ill though the temperature was not very high. I found temperature only 99°F. I administered 4 doses of Mist. Cinchonæ containing Cinchonæ feb. gr. 5 in each dose to be taken thrice daily.

On the 3rd April, I got the news that the temperature was not very high and accordingly the medicine was continued.

On the early morning of 4th April, I was informed by the patient's relatives about the seriousness of his illness and I went at once to attend the case.

On examination, the patient was found in a state of complete unconsciousness with pupils equal on both sides. Temperature 97°F. Pulse 100. Resp. 30 per minute. The spleen and liver were not enlarged.

At 6 A.M., an injection of Quinine bi. Hydrochlor, gr. 10 in 2 c.c. was given intramuscularly. A blood film was taken after the injection. The blood film showed heavy infection of malaria parasites with the following particulars:—

- (1) Trophozoite B. T. parasites + ;
- (2) Trophozoite M.T. parasites + ;
- (3) M. T. Schizonts ++.

At 10-30 A.M., temperature 99.8°F. Pulse 112. Resp. 34 per minute. At 11 A.M., an injection of Quinine bi Hydrochlor. gr. 5 in 25% 20 c.c. Glucose solution was given very slowly intravenously.

At 3 P.M., temperature 100.8°F. Pulse 118 and Resp. 36 per minute. The breathing was stertorous. At 6 P.M., another injection of Quinine gr. 10 in 2 c.c. was given intramuscularly but no improvement was noticed. None of these measures had any effect on the patient, who died at about 10 P.M.

Points to note.—(1) Absence of fever in the beginning though M.P. were found in the blood film. (2) The patient was found unconscious when there was no fever. (3) No high rise of temperature in a case of cerebral malaria.

I am grateful to DR. T. M. GHOSE B.Sc., M.B., Chief Medical Officer, for his suggestion and kind permission to publish this note. My thanks are also due to DR. S. K. GUHA, L.M.F., for his kind help in this case.

Tit-Bits of Medical Jurisprudence

BY

HARENDRA NATH BAGCHI, M.B.,
Superintendent, Alipur Police Case Hospital, Calcutta.

I. Death from Electric Shock.—Accidental cases of injury of death from electric shocks occur frequently in cities like Calcutta, where electricity is used for lighting and motor purposes.

The effects produced on the body by the passage through it of an electric discharge may be :—

(1) Local—Burns, blisters, wounds or ecchymosed streaks, spots or patches.

(2) Constitutional.

(a) Immediate death from shock, or the individual may fall down insensible and die after an interval.

(b) Mental shock followed by severe neuræsthenia, insanity or paralysis of general nerves of motion and sensation.

CAUSE OF DEATH.—P. M. signs nothing characteristic. Blood is fluid. R. M. and decomposition are hastened. Brain and its membranes are congested.

(1) Heart failure—Fibrillation of the ventricles of the heart (P.M.—Left ventricle, empty—Right auricle and ventricle, full of blood).

(2) Very soon afterwards

(a) Hæmorrhage in brain, lungs etc.

(b) Shock to central nervous system :

(c) Burns, thrombosis.

SYMPTOMS.—(1) If the current is of slight intensity—tingling, pain and numbness (galvanic) or sudden severe contraction of muscles (faradic).

(2) If the current is severe but non-fatal—

(a) A tremendous pressure sensation across the chest, severe shock may be followed by paralysis, delirium, or convulsion.

(b) Vomiting, breathing—deep, slow, and interrupted; pulse—slow and unconsciousness with severe convulsions ending in death.

(c) Local injuries—burns with singed hairs, blisters punctured, contused or lacerated wounds, fractures, fissures, tree-like ecchymoses (arborizations).

(d) Mental shock followed by neuræsthenia, insanity or paralysis of general nerves of motion and sensation.

(e) As the result of lightning stroke, blind men have been known to recover sight, the deaf—their power of hearing and women—their sexual functions.

(3) Fatal current—instantaneous death with or without marks of severe injuries and tree-like markings on the skin.

CASE 1.—R. B., Hindu, male, aged 30, a maistry, while working with an electric drilling machine, was electrocuted and died instantly.

The body was well nourished. Rigor mortis was present everywhere. Pupils were moderately dilated and equal, and there was abrasions found on his right zygoma, right side of neck, right chest, right elbow, right knee, right leg, right thigh, right and left side of back.

Brain and its membranes—congested, larynx, trachea and lungs—congested. Heart—right ventricle and auricle: with dark fluid blood and left ventricle: empty. Stomach—congested, submucous hæmorrhages in patches contained 1 oz. of thick brownish fluid with semidigested muri and some blood clots. Intestines—congested, opinion reserved pending receipt of report of viscera from C. E. No poison detected by Chemical Examiner on the viscera (stomach and its contents, portions of liver and kidneys).

Opinion:—P. M. signs consistent with death from electrocution. The abrasions were probably caused by the fall.

CASE 2. Khattari, H. M. 26, at about 4-30 p. m. on 8-10-39, the deceased caught an electric wire, which was suspending from a light bucket, and was electrocuted. He was taken to a neighbouring dispensary and it was ascertained that his life was extinct.

Body—well nourished. R. M.—present on lower extremities. Pupils—moderately dilated and equal. Conjunctiva—congested. Marked cyanosis of lips and finger tips.

Several ecchymoses with tree-like markings on left upper extremities varying from 2" x $\frac{1}{2}$ " to $\frac{1}{2}$ " x $\frac{1}{2}$ ".

Brain and membranes—congested. Pleuræ—healthy. Larynx and trachea—congested and contain froth. Lungs—congested. Tardieu's spots on the surface of the lung along hilus. Pericardium—healthy and contains serous fluid. Heart—Rt. chamber: dark fluid blood; Lt. chamber: empty. Muscle etc.—healthy. Weight 9 $\frac{1}{2}$ oz. Stomach—petechial hæmorrhages in patches and contains 9 lbs. of semi-digested rice, dal and vegetables. Intestines—congested, (semi solid) fæces. Liver—4 oz., highly congested; Kidneys—9 oz., highly congested. Spleen—7 oz. perisplenitis. Bladder—congested, full, reddish turbid urine. Viscera including stomach and its contents, portion of liver and one kidney, and his urine were sent to the Chemical Examiner but no poison detected in the viscera by the C. E.

Opinion:—P. M. findings and C. E.'s report consistent with death from electrocution.

It was difficult to say how death was caused by electricity—whether it was through the heart or the lungs.

It was quite obvious from the P. M. appearance that it would be impossible to tell by medical evidence whether a person has been wilfully killed by electricity.

For electrocution, three contacts of 1485 volts with interval between them of 20 seconds would suffice. Death may result from contact with the ordinary lightening circuit if the resistance of the skin is lowered by wetting.

II. Carbon Monoxide Poisoning.—It is a powerful narcotic poison much more powerful than Carbon Dioxide. Death from inhalation of the

products of combustion *e.g.*, the fumes of burning charcoal, is probably due to Carbon Monoxide poisoning. After death from poisoning by CO, the blood is found bright red in colour. This is held to be due to Carbon Monoxide forming with the hæmoglobin of the blood, a compound of a red colour (Carbonic Oxide hæmoglobin).

CASE 1.—Monsur Dhobi, Hindu—Male—35, was found on 25/8 at 6 P. M. by Ram Charittor Dhobi, a fellow boarder, lying unconscious and a hearth full of coal burning in flames and an iron for ironing clothes was kept by the side of the hearth. The doors of the room where the deceased lay were also closed and had to be forcibly opened. The man soon died.

P. M. EXAMINATION FINDINGS.—Body—well nourished. R.M.—present everywhere. Conjunctiva—congested. Bleeding from mouth. No external marks of injury on the body. Brain and its membranes—congested. Excess of C.S. fluid (weight of brain 48 oz.). Larynx and Trachea—congested and contains frothy mucus. Lungs—congested and œdematous; petechial hæmorrhage on base of lungs. Pharynx and œsophagus—congested and contains stomach contents. Stomach—congested and petechial and submucous hæmorrhages in patches; contents—1 lb.; semidigested bread and vegetables—weight 8½ oz. Small and large Intestines—congested and contain thin faeces. Heart—both chambers full of bright red fluid blood. Muscles and valves—healthy, weight 8½ oz. Liver—bright red colour (weight, 55 oz.) Spleen—bright red colour, enlarged, weight 9½ oz. Kidneys (Rt.—3½ oz. Lt.—3½ oz.)—congested. Bladder—healthy and empty.

Opinion reserved pending receipt from the Chemical Examiner.

Materials sent for analysis :—1. Blood from the heart. Report received with a parcel consisting of a glass test tube which contained a quantity of blood of Monsur Dhobi. Spectroscopic examination of the blood reveals the presence of Carbon Monoxide Hæmoglobin.

2. Materials sent—(a) Stomach and its contents—Report of the C. E.—could not detect any poison in them.

(b) Portions of liver and kidneys—Report of the C. E.—Could not detect any poison in them.

Opinion of M. O. as to the cause of death :—Death was due to Carbon Monoxide poisoning.

CASE 2. HARI DAS, H. F. 45, was a maid servant at Russa Road. On the night of 21-12-39 at about 11 P.M., she slept in the kitchen when water was boiling in an ovan. The door and window of the room were closed and the fire was burning in the room. At about 4 A.M., her master found her lying unconscious and called in a doctor who certified that death was due to heat-failure as she was asphyxiated.

P. M. FINDINGS:—Body was well nourished: Pupils—moderate and equal; conjunctiva—congested. Cyanosis of lips and finger tips. No external mark of injury was visible on the body. Larynx etc.—contain mucus and froth: cherry red in colour. Lungs—congested: (cherry red in colour; Heart—Right chamber: fluid blood, bright red in colour; Left chamber: empty. Muscles and valves—healthy; weight of heart—8 oz. Stomach—P.M. staining—1 lb. semi digested rice, dal and vegetables weight of stomach—5 oz.; Intestines—Healthy, contained semi-solid faeces; weight

of liver—37 oz.; cherry red in colour and congested; weight of spleen—2½ oz.; healthy; weight of kidneys—7 oz.; cherry red in colour and congested. Uterus and ovaries—healthy. Brain and membranes—cherry red in colour and congested. Blood from the heart shows Carbon Monoxide hæmoglobin. No poison has been detected in the viscera of the deceased.

Opinion :—“Death was due to Carbon Monoxide poisoning”

III. **Cresol Poisoning.**—Unidentified H.M., aged 30, was found dead by a H.C. in the Curzon garden, at about 5-30 A.M. on 17-8-38.

IMPORTANT POST MORTEM FINDINGS.—Body well nourished. R.M.—present every where. Cyanosis of lips, finger tips—present. Pupils—moderately dilated. Dried froth marks on sides of mouth. A mark by some corrosive acid stained black was found on the right angle of the mouth and goes obliquely to chin.

Brain and its membranes—Congested (Brain weighing 48 oz.). Mouth—grayish, whitening and softening of the lining membrane of the mouth. Tongue—sides eroded, hardened and grayish in colour. Stomach—eroded, hardened and stained grayish white. Contents—12 oz., grayish yellow fluid smelling of phenol containing partly digested rice; mucous blood clots. Weight 11 oz. Intestines—eroded, hardened and grayish white throughout. Blood was present in the duodenum. Liver—45 oz.—congested. Spleen—2 oz. congested. Kidneys Rt. 3½ oz. Lt. 4½ oz.—congested. Larynx and Trachea—congested and contains mucus. Lungs—congested. Heart—both chambers full of dark fluid blood; muscles and valves healthy—wt. 7½ oz. Bladder—congested—8 oz. of olive green urine.

Opinion reserved pending receipt of the report from the C.E.

Materials sent.—(a) Stomach and its contents—Report: C.E. detected Cresol in them;

(b) Portions of liver and kidneys—Report: C. E. detected Cresol in them;

(c) Urine—Report: C.E. detected Cresol in the urine.

Opinion by M.O. as to the cause of death—Death was due to Cresol poisoning. Cresol, Creolin, Cylin, Lysol and Izal are similar in action to Carbolic acid but less toxic. Death has occurred after taking 2 teaspoonfuls of Lysol.

IV. **Nitric Acid Poisoning.**—Gopal Nandi, H.M. 23, entered a room on 7-8-38 and bolted the doors of the room. He then took Nitric acid from a phial in the presence of his mother. He was removed to Medical College and died on 15-8-38.

POST MORTEM FINDINGS.—Burns by some corrosive acid and stained black on the right side of the neck, the right shoulder and the right arm.

Brain—wt. 48 oz. and its membranes congested. Mouth, pharynx and œsophagus.—Lips—eroded and stained yellow. Tongue—eroded in patches. Pharynx and œsophagus—eroded and stained greenish yellow. Stomach—charred, hardened and stained yellowish black—contents 2 oz. bloody fluid and some blood clots. Intestines—eroded, hardened and stained yellowish black. They contained blood all through. Larynx and

trachea—congested and partly eroded and stained yellow. Lungs congested. Heart—both chambers contain clotted and fluid blood. Muscles and valves—healthy; wt. $7\frac{1}{2}$ oz.. Liver—congested: 41 oz. Spleen— $1\frac{1}{2}$ oz., congested. Kidneys—Rt. $3\frac{1}{2}$ oz., Lt. 2 oz.—congested. Bladder—congested and empty.

Chemical Examiner's Report.—C.E. detected salts of Nitric acid in:

(a) The stomach and its contents;

(b) Portions of liver and kidneys.

Death was due to Nitric acid poisoning.—Nitric acid is very largely employed in the arts and manufactures. It is used for separating gold from other metals. It is also used for the preparation of gun-cotton, Nitro-glycerine, Picric acid, Sulphuric acid and colouring matters. Diluted with water, it is also used in medicine:

(a) $\text{HNO}_3 + \text{HCl} = \text{Aqua Regia}$, which dissolves gold.

(b) $\text{HNO}_3 + \text{H}_2\text{SO}_4 + \text{Glycerine} = \text{explosive compound}$.

Criminal uses—To sweat coins; to open padlocks; or to burn people (Vitriol throwing). Fatal dose—2 drams. Fatal period—12 to 24 hours.

My thanks are due to Lt. Col. K. S. Thakur I.M.S., Civil Surgeon, 24 Parganas, for his encouragement in publishing the reports of these cases.

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- | | |
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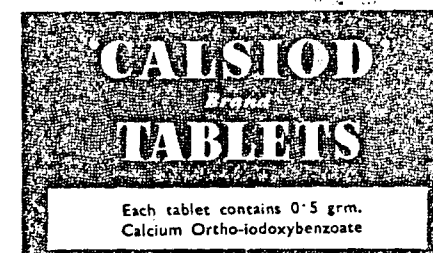
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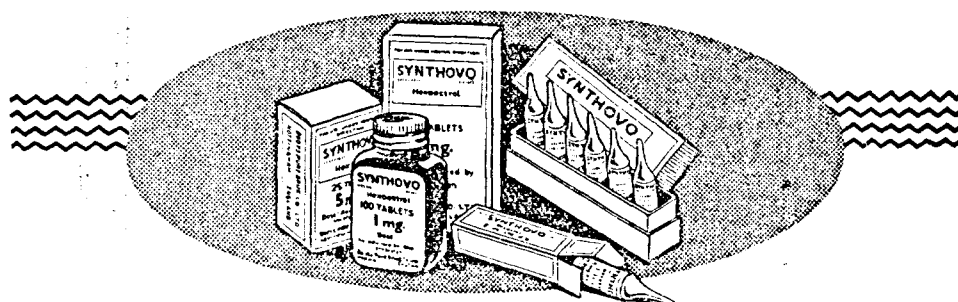
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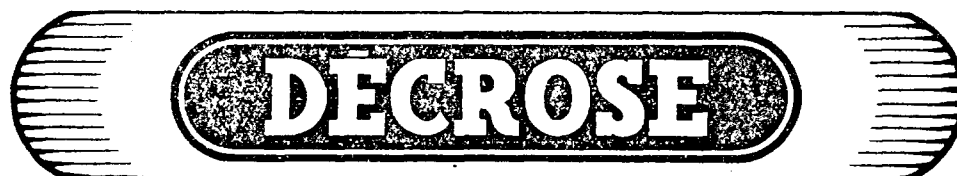
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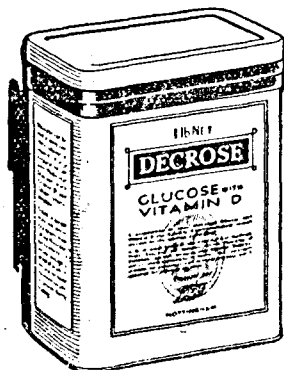
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Editorial

Manufacture of Chemicals in India— Government of India's Step-motherly Attitude

WE are in receipt of two Press Communiques from The Indian Chemical Manufacturers' Association, published elsewhere in this issue, which may be included among the evidences to show that the Government of India is more keen on safeguarding foreign interests rather than on defending the legitimate interests of India.

It may be noted here that The Indian Chemical Manufacturers' Association was established in July 1938 under the Presidentship of Acharya SRI P. C. RAY, with the object of promoting and protecting the indigenous trade, commerce and industries of India, connected with chemical and pharmaceutical and other allied products. The Association was soon recognised by the Government of India and by most of the Provincial Governments. The Association, in its 2nd annual general body meeting, passed twelve resolutions, of which, the communiques referred to above are directly concerned with the fifth and ninth resolutions respectively. In the fifth resolution, the Association strongly urged the Railway Board to change the classification of raw drugs and manufactured medicines for the purpose of lowering the freight rates and also to introduce special concessional rates of freight for certain raw drugs. In the ninth, the Association urged the Government of India to take full advantage of the opportunity, created by the present war, for stabilising and further developing the chemical and pharmaceutical industry in India, and to place all government orders with the Indian manufacturers, who are in a position to supply not only the civil but also the military requirements of the country, and that the preparations available in India should not be imported from abroad.

But, we learn from the Communiques, that the Government of India, acting on behalf of the Provincial Governments, have ordered for three years' stock of medical supplies from England. The Chemical Manufacturers' Association also asked for a list of items for which orders have been placed in England, but the India Government refused to supply this list. The reason cannot be that the list, if published, would hinder war efforts. Nor is

the explanation accompanying the refusal convincing. They said that the list covered items not available in India. That probably is all the more reason why the Manufacturers' Association should know it. For, it would give the Indian Chemical Manufacturer an opportunity to explore all the possibilities of preparing those drugs in India. That they are not available in India may be a cause of not placing the orders with Indian Manufacturers, but, certainly, it is no reason why the list should not be published.

The next thing that sounds as much unreasonable as the refusal to publish the list, is the long period for which stocks of drugs have been ordered. Such long periods of contracts would adversely affect the chemical manufacturing trade in India, as they will effectively kill any incentive for new enterprises in the manufacturing field. It is reported that the Government have assured the Association to place orders with Indian Companies alone, but vague assurances, generally qualified by equivocal terms and hypothetical clauses, are always worse than flat refusals. Is it not ironical that, when the Association was just requesting the Government to assure them protection against the probable dumping of foreign drugs, after the termination of the war, thus enabling them to undertake the manufacture of new lines for which they have facilities, the Government should, even during the war itself, be encouraging foreign in preference to Indian manufacturers by placing orders for large stocks with the manufacturers outside this country. It would appear to a casual observer that foreign competition is not, after all, an after-war matter, but it is already there and that too, on the special invitation of the Government of India! We hope that the HON'BLE SIR A. RAMASWAMI MUDALIAR, Commerce Member with the Government of India, will, even now, protect the rights of the Indian Chemical Manufacturers, and still use his weight in their favour, as promised by him, when he was met by the Committee of the Association on 26-11-'39.

Regarding the ninth resolution, relating to lowering of railway freight rates on manufactured drugs, the matter is reported to have been under correspondence since then, and the net result is that the Railway Board refused to make any general reduction in the classified rates for drugs and medicines. This seems to be a corollary of the India Government's attitude regarding foreign competition. The Railway Companies, incorporated in England as they are, it is not a surprise to us that they are not giving any encouragement and help to the development of Indian industries. We, therefore, do not see that much benefit could be derived in following the Railway Board's advice that the Association may approach individual Railways for quoting special rates in respect of such items of drugs and medicines for which the rates are considered very high.

We trust, however, that the Indian Manufacturers will, in spite of all these obstacles and discouragements, go ahead with their industries, and we only hope that, in the near future, when the political deadlock in this country has ended and National Governments begin to function, they will then have adequate protection completely assured to them. Till then, as is the case with all developing industries, their path is a difficult one. We wish them all success in their endeavours.

Recruitment to the I. M. S.

As if in response to the recent unanimous and loud protests of the Indian medical public against racial discrimination in the Indian Medical Service, the Conference of medical men which met at Simla and concluded its sittings on 22-7-41, came to the following decisions amongst a few others. The Conference was of opinion that greater publicity should be given to (a) the existing terms and conditions for Emergency Commissioned Officers of the I. M. S. so as to bring out the fact that they involve no discrimination between the European and Indian entrants in regard to rank, emoluments, promotion and other conditions of service, and (b) that for the period of the present war, no new permanent appointments would be made to the Indian Medical Service.

Major-General H. STOTT, I.M.S., Surgeon-General with the Government of Madras, recently addressed a large meeting of doctors and medical students in the course of which he made a few statements with regard to the recruitment to the Emergency Commissions in the Indian Medical Service. A report of his address is published elsewhere in this issue.

Neither the proceedings of the Conference nor the address of the Surgeon-General give us any clue whatsoever as to the new terms and conditions under which the Indian medical men will now be recruited. The public are not concerned with the language of the proceedings or of the address but are more intent and directly concerned only with regard to the actual steps if any, taken towards dispelling the suspicion from the minds of the Indian Medical graduates. The Surgeon-General announced that the additional concessions resulting from the Conference would be published shortly, but beyond this laconic statement, he was not prepared to give any actual facts and figures. He appealed to medical men to enlist in large numbers and assured them that all benefits would be given with retrospective effect.

We should have expected that the Government would prove the sincerity of their statement by immediately granting some permanent Commissions to Indians. But what the Conference of medical men which assembled at Simla decided in its wisdom was that no permanent recruitment should be made at all during the period of war! We do not know the reason why this must be so, nor are any reasons given in the Communique. We do not believe that providing a few Indians with permanent Commissions will in any way hinder the war effort. In fact, it will have a rousing effect on all classes of medical men and there will certainly be a good response to such broadminded offers.

With regard to the future entrants into these Emergency Commissions, the Surgeon-General was very clear. He gave hopes that these officers, after the war, would however, be eligible for the Provincial Medical Service. He gave no assurance whatsoever as to the number that would be made permanent in the I.M.S. after the war. It would have been very much better, and the response certainly more encouraging, if the Conference had suggested just the opposite, viz.—that all the appointments made during the war should be treated as though on a permanent basis. There would be no difficulty in giving this promise; for, the future army in India which is going to be a much greater standing army will require many medical officers and, there will be the necessity of continuing all these appointments on a permanent basis.

The proposals under consideration to make the conditions of service more attractive, especially for medical practitioners of some years' standing, are still "being considered".

With regard to the appointment of specialists, the Surgeon-General was very explicit. He made it very clear that the number of appointments as Majors was limited and therefore warned all entrants not to be certain of these specialist jobs, but assured the audience that those who possessed the necessary qualifications or the necessary teaching experience would have their claims thoroughly considered when these appointments were being made. He was, however, silent on the question whether medical practitioners who have put in a large number of years of practice would be given priority of rank over those who joined recently.

The Surgeon-General then referred to the grant of educational facilities for the children of those joining the military service and spoke about the grant of study leave for those who had rendered military service. He briefly referred to the giving of gratuities on retirement to those who were not on the official medical service. Beyond mentioning that these were engaging the attention of the Government, the Surgeon-General gave no tangible clue whatsoever as to the possible action the Government proposed to take.

With regard to the other very important matter which has been the subject of much discussion—we refer to the abolition of the Civil side of the I. M. S.—the Conference summarily dismissed it as impinging on the constitutional field and unsuitable for consideration during the war. It would have been much better if they had expressed their view on this matter instead of brushing it aside so peremptorily. The suggestion that nothing could be done or considered during the war does not, to us, convey much reason. We are perfectly satisfied that the abolition of the Civil side of the I. M. S. does not and will not affect the efficient prosecution of the war. We are also not prepared to admit that there are not enough medical men to take over these responsible jobs now held by the members of the I. M. S. On the other hand, we go a step further and we are of opinion that there are enough men, capable and efficient, who can discharge those duties as well, if not better. Strictly speaking, war is the time when extra-ordinary decisions and undertakings are necessitated and the abolition of the Civil side is nothing when compared with some of the other undertakings of the Government in other fields.

Considering the relative simplicity of the task, we feel that the Government have lost a golden opportunity to satisfy Indian aspirations. We are, however, waiting to see the actual concessions granted. The advertised attractiveness of the conditions will be lost unless these concessions are published widely at an early date. We, however, hope that these new proposals will include, among other things, at least a pension equivalent in amount to the average income of the practitioner recruited to the Indian Medical Service, to be given to him till the end of his life after the war. It will be attractive and will be treated as more or less tantamount to the granting of permanent Commissions now. Since the actual proposals are not made public, our comments on them are reserved for the future. But, for the time-being, we regret to say that the Simla Conference has not done much.

Gleanings From Medical Press

Obstetrics and Gynaecology

Sacral Anæsthesia in Obstetrics—In spite of many recent advances in analgesia and anæsthesia for obstetric practice the search for perfection still continues. W. H. Poole has reported the results he obtained when using sacral nerve block. This method of anæsthesia is by no means new in obstetrics, for both S. P. Oldham in America and St. J. Wilson in this country commented favourably on its use in selected cases. It is not a synonym for a low spinal anæsthetic; the spinal cord terminates at the level of the lower end of the first lumbar vertebra and the dura mater is prolonged as an ensheathing membrane to the cauda equina as far as the third piece of the sacrum. The dura is firmly attached anteriorly to the vertebral bodies but posteriorly is surrounded by loose areolar tissue filling the epidural space. This potential space extends to the level of the first coccygeal vertebra and varies in size; its capacity may be 25-50 c.cm. or more. The contents of the space are fat, areolar tissue, a venous plexus, and the obliquely directed nerve roots of the cauda equina, the terminal 1-1½ in. of which are devoid of dural envelope where they traverse the space; they are therefore accessible in this part of their course to the effects of any drug injected into the epidural tissues. The needle can be introduced in the hiatus between the last sacral spine and the first piece of the coccyx; the hiatus is ½ in. long by ½ in. wide and is guarded by the posterior sacrococcygeal membrane. A fine lumbar-puncture needle is used and the bony landmarks carefully defined, especially in an obese patient. The subject lies in the left lateral position and the tip of the coccyx is marked; 1½-2 in. above this the transverse processes of the first coccygeal vertebra are felt and superior and lateral to these the more prominent sacral cornua. Just below these lies a diamond-shaped depression like a miniature fontanelle marking the sacral hiatus. The posterior sacrococcygeal membrane lies 3-12 mm. below the surface. Having pierced the membrane the advancing needle impinges on the bony anterior wall of the canal; it is withdrawn and guided about 1½ in. up the canal. Neither blood nor cerebro-spinal fluid should escape from the needle, and no resistance should be encountered to the injection of the anæsthetic. Poole made several experiments to determine the best solution for injection and after a trial of 2% Procaine with 3% Quinine and Urethane, 2% Procaine only, and 1 in 1000 Percaine in aqueous solution, he rejected them all because of the brevity of the anæsthesia. Instead he used 1 in 1000 Percaine in normal saline in dosage of 25 c.cm.; this gives anæsthesia lasting at least 4 hours and often longer, of sufficient depth to allow the application of forceps in the low operation and the repair of the perineal tears. Any obstetric anæsthetic is required for two main critical periods of pain; that accompanying the later stages of cervical dilatation and that

produced by the final stretching of the soft parts of the pelvic floor. If carefully timed, sacral anæsthesia will successfully relieve both these painful crises, but it must not be employed too late for the first nor too early for the second. In primiparae the optimal time for injection is towards the end of the first stage of labour, in multiparae naturally at an earlier stage. The immediate results of a successful injection will be a decrease or even cessation of uterine contractions; this is a transient action lasting about half an hour, and provided the pains were previously strong and regular, they will return with equal vigour. Inefficient uterine action, as in primary inertia, is an absolute contra-indication to this method of anæsthesia. Poole analyses his results critically in 32 cases in which he has used sacral nerve block. He assesses his failures at 10 cases with the following causes: faulty technique in 3, too early administration in 2, and inefficiency of uterine action after the injection in 5. All these failures with attention to technique and careful selection of cases could have been eliminated. No ill effect in the mothers or the babies could be blamed on the anæsthetic. Sacral nerve block will no doubt have its critics; the English surgeon is averse from using regional in preference to general anæsthesia, but is to be hoped that more obstetricians will try for themselves a method that has obvious advantages at least from the fetal point of view, because of the avoidance of cardio-respiratory depressants in the circulation. Should the occasion arise for a major obstetric procedure, it is a simple and safe matter to supplement the regional with a general anæsthetic, the total dosage of which will be proportionately smaller and consequently less toxic—*Lancet*.

Dangers of Posterior Pituitary Extract in Labour.

—A drug whose power was not fully appreciated became available when the action of extracts of the posterior lobe of the pituitary on uterine muscle was discovered. There were many who heralded the discovery as marking a new era in obstetrics, and in some clinics the available preparations were spoken of as being "physiological forceps." The physiologist has shown that the uterine response to pituitary stimulation is not so simple as was at first thought, and over twenty-five years of clinical experience have revealed dangers which were unsuspected. The use of pituitary extracts in labour was reviewed recently at the ninety-first Annual Session of the American Medical Association in New York, when papers were read before the Section of Obstetrics and Gynaecology by Drs. Sharkey, Pendleton, and DeLee. In spite of the wide use of the hormone during the first and second stages of labour—a practice which was deplored by all three speakers—it is to be noted that in the discussion following the papers no speaker rose to defend this. DeLee referred to the

increasing demand among American women for "streamlined labour," and stated characteristically that such a labour could be as safe as a streamlined parachute. Reynolds has shown that posterior pituitary preparations produce tetanic contractions of the uterus, and stress was laid on the dangers to the mother and the foetus when this happens during labour. Cerebral damage with stillbirth and neonatal death, maternal lacerations, uterine rupture, and damage to the pelvic floor with resultant prolapse are the most common complications. DeLee stated that the number of cases of uterine rupture due to the administration of pituitary extract would never be known, for either the cases were not reported or the patient was buried and the death recorded as being due to shock, hæmorrhage, or embolism. Attention was drawn to the important fact that even doses as small as 3 minims (0.2 c.cm.) can be dangerous to the mother and the child, and it was claimed that there was no evidence to suggest that compound preparations such as Thymophysin are any safer than Pituitrin or Pitocin. In an analysis of the records of St. Vincent's Hospital, Kansas City, Dr. Pendleton found that when pituitary preparations were given during labour operative intervention was increased and the stillbirth and neonatal death rates were more than doubled. The speakers agreed that the use of pituitary preparations during labour placed a grave responsibility on the obstetrician, a responsibility which he is wise to avoid. While remembering the dangers associated with the abuse of these preparations, it is well to acknowledge the service they can safely render when employed with care. Pituitrin helps the uterus to expel its contents during the process of abortion, and in those cases in which evacuation of the uterus is necessary an initial injection of pituitrin into the cervix reduces hæmorrhage to a minimum and greatly lessens the risk of perforation. Pituitrin is also of value in the induction of labour, but should be administered in small doses—for example, 0.25 c.cm.—which may be repeated at half-hourly intervals but must be discontinued if pains begin. It must not be given if a placenta prævia is suspected. During labour the hormone should not be used, and in a prolonged second stage an episiotomy or an instrumental delivery is safer than the "physiological forceps." In post-partum hæmorrhage the drug is most valuable but has no advantage over ergometrine, and has the great disadvantage that on rare occasions it gives rise to severe shock.—B.M.J.

Pelvic Abscesses: Diagnosis and Treatment.—Harold Dodd, Ch. M., F.R.C.S. writes:—There are few operations more satisfying to patient and surgeon alike than that of draining a pelvic abscess through the rectum. Collections of pus in the pouch of Douglas in the female and in the recto-vesical pouch in the male occur after any septic abdominal operation, but especially after a perforated appendix: less often they follow a

perforated peptic ulcer, salpingitis, or suppurative diverticulitis. In a general surgical practice I meet about four cases a year; three will be caused by suppurative appendicitis with peritonitis, whilst the fourth will follow something less common. A recent instance developed after a partial gastrectomy for a huge gastric ulcer which was resected from the pancreas. An undetected pelvic abscess is one reason for a septic peritonitis not responding to Sulphapyridine.

Diagnosis.—The diagnosis of a pelvic abscess is straightforward. It is based mainly on the physical signs, for the symptoms as such seldom direct attention to the rectum. The story is approximately as follows:—

After an abdominal operation the chart shows a raised pulse and temperature for about a week, then after two or three days of normal readings there is a return of the elevated pulse and of pyrexia to around 100° F. with a tendency to swing. The respirations throughout are unaffected, which negates the possibility of a subphrenic abscess or respiratory sepsis. In a few days frequency of the bowels is noticed—perhaps four to six times a day. This is regarded as satisfactory at first, for it has often been preceded by constipation. Less often the bowels remain inactive. The stools consist mainly of mucus and a little fluid faeces. The patient is toxic, with colour pale or slightly grey, and the initial progress is obviously slowed down. The hand will be found coolish, the nails white instead of pink, and the pulse is small. The tongue is moist, smooth or shiny, and bright red, and the papillae have been shed; there is an exfoliative glossitis. It is interesting to watch this tongue condition recover when the pus has been drained: the whole of it may be affected, or occasionally only the tip or sides.

The abdomen is inclined to be tight, and to be full in the lower half, and the respiratory excursion is absent or even reversed—i.e., it is indrawn on inspiration instead of moving outwards. There is seldom abdominal tenderness or rigidity. In a thin patient visible peristalsis may be observed, raising the possibility of subacute intestinal obstruction; rarely a patient vomits, further strengthening this suspicion, but if there is the possibility of "pus somewhere" it must be sought out before considering intestinal obstruction, apart from an inflammatory paralysis of the bowel.

The diagnosis is clinched by inserting a finger into the rectum. According to the stage of development of the pelvic abscess when the examination is made, one of three states will be found. They are:

1. *The Early and Suspect Stage.*—The rectum will be found empty, dilated, and warm, and the mucous membrane is smoother than usual. The rectal wall at the tip of the finger is hard and unyielding, with induration. There is no fluctuation, and the impression formed is that the rectum is "set in felt."

2. *The Abscess Declared.*—A day or two later than the above, the findings will be the same except for a significant addition: at the tip of the finger, on the anterior wall, a dimpling spot will be discernible. This is the first sign of the rectal wall softening and the abscess pointing through it. The sensation to the pulp of the finger is almost identical with that experienced on palpating the tip of the nose—i.e., there is a soft spot between the two cartilages. It will be remembered that the reflection of the peritoneum is fully a finger's length away from the anus, so that in the early stages it is necessary to press up firmly to reach high enough, and that the finger will have to pass round the first fold of mucous membrane in the rectal wall; thus the examination is a painful one for the patient. I prefer to make it with the subject in the Fowler position and the knees wide apart. The dimpling spot having been detected, the time for operation has arrived, and this is the place through which to drain the abscess.

3. *The Abscess Obvious.*—The further development is when the rectal mucous membrane becomes oedematous and hot, the first valve of the rectum is obliterated, and there is an obvious bulging protrusion into the lumen. The tense bulge is so definite that fluid pointing into the bowel is undoubted.

The Abscess Ruptures.—A fourth stage, which should rarely occur, is that of a spontaneous evacuation of pus into the rectum, and this is passed into the bed-pan as a fluid stool. The abscess should be detected clinically before this.

An "Unusual" Presentation.—When the abscess points into the rectum from the peritoneal cavity it must traverse the pelvic cellular tissue. This usually becomes so indurated that spread along this plane is automatically limited. Rarely, however, in a debilitated patient the localization is not complete, and pus may break outwards and downwards through the sacro-sciatic foramen and point as an abscess on either buttock. The process is a quick one, and constant vigilance should be maintained to detect such a collection in a patient who is not progressing and who obviously has "pus somewhere." Another possibility is the abscess pointing in the groin at the pubic spine, having tracked along the round ligament or the vas deferens.

Vaginal Abscesses.—Pelvic abscesses also discharge through the vaginal fornices, but are rare in my experience, having seen only two in eighteen years of general surgical practice.

OPENING A PELVIC ABSCESS.—It is inadvisable to attempt the operation in bed. The patient is taken to the theatre and placed in the lithotomy position. The buttocks are elevated on a sandbag and protrude over the end of the operating table. This position is carefully arranged before the anaesthetic is given. Some surgeons perform the operation without an anaesthetic, with the patient in the knee-elbow position. Since the patient

is already weak and toxic, as little anaesthetic as possible is given. A whiff of gas-and-oxygen or 3 to 6 c.cm. of 5% Pentothal intravenously meets the need adequately.

The operation.—The anus is dilated to admit two fingers, using plenty of lubricant. Tearing of the mucous membrane must be avoided: such fissures would be infected. A Sims duckbill speculum is inserted into the rectum, and the lumen is thoroughly cleansed of faeces and wiped out with antiseptic swabs. The dimpling or fluctuating spot on the anterior wall is located with the tip of the left index finger, and with this as a guide a long wide bore needle attached to a 20-c.cm. syringe is confidently thrust through the suspicious area in the rectal wall. A hesitant penetration will fail to enter the abscess cavity. Aspiration will reveal the pus. If pus is not found, carefully check that the needle has been inserted through the softening place and that it has really penetrated the rectal wall. If no pus appears then the operation is not proceeded with, and observation on alternate days is maintained. When pus is found the syringe and needle are carefully held in position. A pair of uterine dressing forceps whose points are grooved on the inside are slid along the needle through the rectal wall and into the abscess cavity. Pus immediately emerges, often surprisingly fetid and in large quantities. As the flow slows, the syringe and needles are withdrawn and the forceps blades are opened in a north-and-south and then an east-and-west direction, thus making the opening in the rectal wall generous. In the early cases I used a large pair of Mayo scissors to open these abscesses. In one case a hæmorrhage began which exanguinated the patient and made a second operation necessary two hours later, followed by transfusion. It was surmised that the rectal wall, with a large vessel, was cut during the process of closing the scissors whilst turning them from north and south to east and west. Since then the uterine dressing forceps have always been used. They are more suitable, and no complication has been encountered.

The Drainage Tube.—The drainage tube is inserted through the anus and into the opening in the rectal wall; a stitch through the perianal skin anchors it for forty-eight hours, when it is removed and not replaced. A compress of Perchloride of Mercury 1 in 1,000 is applied externally and changed morning and evening.

AFTER-TREATMENT AND PROGRESS.—The after-treatment is simple. The patient is nursed in the full Fowler position. On the day of operation only water is given, by mouth; the next day fruit drinks; citrated milk is added the day afterwards, and full milk and all fluids after this. At least six pints must be taken daily. So often a fluid diet is synonymous with starvation, and it is important to feed these patients adequately. The diet is steadily built up through semi-solids to one of fish and chicken.

The subsidence of the pulse, temperature, fullness of the abdomen, and general malaise

is prompt and progressive. In only one patient have I seen a failure of resolution, when a pelvic collection of pus required draining three times. On each occasion the temperature and pulse fell for five or six days and then began to swing again.

No deaths have been seen after a drained pelvic abscess, but missed ones have been found at necropsy.—(B. M. J.)

Veratrum in Eclampsia.—The older textbooks of Obstetrics often advocate the use of Veratrum or American hellebore in the treatment of eclampsia, but modern writers either do not mention it at all or condemn it. Bryant and Fleming, in the J. A. M. A. however, sponsor the drug with enthusiasm. Their cases number 120 and cover a period of 15 years, so that their conclusions, though contrary to the trend of modern opinion, merit close examination. They look upon eclampsia as a syndrome of which the essential pathological feature is an abnormal spasm of the entire arteriolar tree. They accept the orthodox view that the underlying cause of the spasm is some undiscovered toxin. Veratrum has long been known as a powerful vasodilator, and Bryant and Fleming claim that in therapeutic doses it is entirely safe—a claim vindicated by their low fatality of 1.7%. They aim at complete control of the eclampsia, and only when this has been achieved do they consider the secondary problem of terminating the pregnancy; in this they adhere strictly to the best doctrines of conservatism. The preparation used is not mentioned in this paper, but Bryant has previously reported that he uses Veratrone (P. D. and Co.), a standardised non-alcoholic solution of the active principles of veratrum roughly equivalent in alkaloid content to the B. P. C. tincture. An initial dose of 0.6 c.cm. is given hypodermically, and this is repeated every 10–15 minutes until the pulse-rate falls below 60 or the systolic blood-pressure below 120; thereafter, until the patient is conscious, 0.2–0.6 c.cm. is given again if the pulse-rate rises to 80 or the systolic blood-pressure to 150. Once the patient is conscious a dose of 0.2–0.6 c.cm. is given if there is nausea, severe headache, visual disturbance, epigastric pain or convulsions.

As an adjuvant, 500 c.cm. of 20% dextrose is given intravenously at the beginning of treatment and repeated every 6–10 hours until consciousness returns; 10 c.cm. of 50% magnesium sulphate is also given at the start by deep injection and afterwards 5 c.cm. every 6 hours for four doses and 5 c.cm.

every 12 hours for four doses. The patient is catheterised and given a soap and water enema at the beginning of the treatment. The pulse and blood-pressure are checked hourly in the acute stages and at short intervals after the patient has recovered consciousness. Fluids are pressed to the point of complete hydration, 5 litres a day being given as soon as possible, and a salt-free diet is instituted as soon as food can be tolerated. Fluid intake and urinary output are checked and the urine examined daily for albumin. No sedatives are given except in cases of extreme restlessness or if the patient goes into labour; they claim that the three commonest causes of restlessness in these semicomatose patients are a full bladder, a full rectum and the pains of labour. The results of veratrum may be startling enough to alarm the uninitiated by a sudden fall of blood-pressure, with extreme slowing of pulse and respirations (for all of which atropine is an effective antidote if required), and may be followed by a copious vomitus of green or black fluid. The usual nursing regime for eclamptic patients must be strictly enforced to ensure proper postural drainage of the mouth and pharynx.

According to Bryant and Fleming, in eclampsia a vaso-constricting toxin produces hypertension which may be complicated by cardiac failure and cerebral and retinal hæmorrhage; and this vaso-constriction causes local anoxæmia and retention of catabolites with resultant focal necrosis in the liver and kidney. It is feasible to correlate this clinical picture of a full-blown eclamptic with the pathology demonstrable at autopsy. Dilatation of the arteriolar tree with veratrum breaks the established vicious cycle, and by making a normal blood-supply available to the tissues, enhances the action of such therapeutic agents as dextrose and magnesium sulphate. The primary function of this drug is, therefore, to improve arterial circulation throughout the body and not merely to reduce the effect of hypertension. More extensive investigation of the action of veratrum is needed to determine whether we have not prematurely discarded a powerful therapeutic weapon. The maternal mortality of 2 cases in 120 is especially remarkable in that both patients died of puerperal sepsis, long after control of the eclampsia and delivery. The gross fetal mortality was 28.8%, the greatest number of deaths being in the antepartum eclamptics. It must not be forgotten, however, that Bryant and Fleming also used hypertonic dextrose and magnesium sulphate, both of which are potent agents in the reduction of a high blood-pressure.—(Lancet).

Medicine and Therapeutics

The Treatment of Interdigital Epidermophytosis.—A simple and effective treatment for epidermophytosis of the feet is given by Du Bois (*Schweizerische medizinische Wochenschrift*, December 14, 1940, 70, 1223). The feet are given a warm, prolonged bath twice daily in a solution of 1 in 1,000

potassium permanganate to which 20 grammes per 1000 c.cm. cooking salt is added. Weak lysol may be used, but there is the risk of individual intolerance. After the bath the scabs are removed with forceps, the blisters and bullæ are opened, and then energetic friction is applied with gauze soaked in equal

parts of ether and benzene. The cleansed surfaces are then daubed with a solution of iodized acetone:—

Acetone	...	0.20
Iodine in 60 per cent. alcohol	...	2.00

The burning sensation from the solution is bearable and passes quickly. Finally, the feet are generously powdered with a mixture of equal parts of talc, zinc oxide, and subnitrate of bismuth, and fine gauze is placed between the toes. The results are rapid—in a few days healing and cure seem to have occurred, but it is wise to continue with a daily bath and the powdering for three weeks at least.—*Practitioner*.

Chlorine in the Treatment of Nitric Acid Burns.—In chemical burns the destruction of tissue continues until the corrosive is absorbed, washed out, or neutralized, in contradistinction to burns in which the process ceases on the removal of contact. The inflammatory reaction is also delayed in corrosive burns, and it is usually not until a week after the accident that necrosis becomes evident. Healing is slow and may result in contraction or, in some cases, the destruction of a nerve involved. An interesting case is reported by H. M. Rovers (*British Medical Journal*, March 8, 1941, 1, 361) of a workman who sustained burns of the face by the splashing of a combination of nitric acid 23.5 per cent., sulphuric acid 68.8 per cent., and water 7.7 per cent. The forehead, the left cheek, the nose, the chin and neck were affected, the eyes being protected by goggles. The affected areas were swabbed with eusol (eupad shaken up with warm water) and decolorization took place immediately. Brisk friction was used and repeated after short pauses until a pale lemon colour resulted, then the burnt areas were painted with a very thin coat of tannic acid solution (5 per cent.) and dried by a hot-air drier. The smarting and tingling of the burnt areas disappeared during the application of the eusol solution, no discomfort was suffered, and sleep and appetite were not interfered with. Two weeks after the accident the patient could be shaved and returned to work in under three weeks showing no obvious signs of injury. In this case apparently the rapid removal of the nitro-compound from the skin prevented corrosive destruction of the tissues.—*Practitioner*.

Treatment of Acute Bacillary Dysentery with Sulphapyridine.—At the Government Isolation Hospital, Haifa, R. Reitler and K. Marberg have found sulphapyridine highly efficient in the treatment of acute bacillary dysentery. They treated 20 patients (dysentery Shiga, 4; Flexner-Y, 13; Strong, 1; Sonne-E, 1; Schmitz, 1) with results uniformly good even in the severe cases, but they point out that the drug seems to have no effect in amoebic dysentery.

All the patients were adults suffering from severe or moderately severe bacillary dysentery. Mild cases were not treated. The

treatment was begun on the second day after admission, the first day being used for observation so as to compare the patient's condition before and after administration. One gramme (two tablets) of sulphapyridine was given by mouth (with sodium bicarbonate to prevent nausea) 3 or 4 times daily for 2 to 4 days, according to the time taken in recovery. As a rule the drug was administered until one day after the motions became formed. No other treatment was given.

Usually 24 hours after beginning the treatment the temperature dropped and the diarrhoea was greatly improved as to the frequency of motions and the appearance of the stools. After 48 hours' treatment the motions were back to normal in nearly all cases, irrespective of the previous duration of the disease and the type of the dysentery bacillus. In 3 cases it took 3 days and in one case 4 days to achieve this result. Tenesmus and vague sensations of uneasiness in the abdomen were felt for one or two days more by some patients. General toxic symptoms, if present, improved rapidly. The stools were found bacteriologically negative as soon as they became formed, and relapses were not observed.—*Clinical Journal*.

Local Treatment of Tuberculous Lesions with Cod-Liver Oil.—From the Muirdale Sanatorium, Wisconsin, U. S. A., A. L. Banyai reports good results from the local treatment of tuberculous lesions with cod-liver oil. In this use he was encouraged by the work of Loehr, who used the oil with success in a large number of non-tuberculous conditions. He found that it exerted an inhibitory effect on the bacterial flora of wounds. Favourable results from the use of cod-liver oil and tomato juice in intestinal tuberculosis—a treatment recommended by McConkey in 1930—were obtained by Banyai, who suggests that they might be due to the direct contact of vitamin A with the ulcers or to its increase in the blood.

Banyai has treated tuberculous pharyngitis and laryngitis by spraying the throat thrice daily with cod-liver oil by means of an ordinary atomiser. Sterilization of the oil is not necessary, as it is free from bacteria. Active pulmonary tuberculosis was present in all the cases. An analysis of 91 in which the treatment was continued from 2 to 18 months shows that 24 remained unimproved (of whom 8 died during the treatment), 42 improved subjectively and objectively, and healing took place in 25. The following is an example:

A woman, aged 20, had advanced pulmonary tuberculosis and tuberculosis of the tonsils and larynx. Treatment consisted in cleansing the throat with Dobell's solution and spraying it with cod-liver oil thrice daily. In three weeks the ulcers disappeared, except on the posterior part of the right tonsillar fossa, which was normal 4 weeks later. The soreness of the throat and dysphagia disappeared.

In the patients who responded favourably

relief was prompt. In several cases the use of Cocaine spray, Anaesthesine or Euphagine could be discontinued. The gradual disappearance of hoarseness was impressive and made the patients more optimistic. The change in the laryngeal findings was striking. Ulcers showed rapid epithelialization and healing provided the treatment was well tolerated. But the utter failure of the treatment in some cases was discouraging. Cases with marked laryngeal oedema were rather resistant to the treatment. Two patients with tuberculous ulceration of the tongue were successfully treated by topical application of cod-liver oil.

Tuberculous empyema in the absence of bronchopleural fistula responded favourably to aspiration of the pus and replacement by cod-liver oil. The fistula does not contra-indicate the treatment, but renders the results less favourable. Suppurating tuberculous glands, when seen early, healed well under repeated aspiration followed by injection of cod-liver oil. Cod-liver oil drops induced more rapid recoveries in phlyctenular conjunctivitis and keratitis than other methods. But in tuberculous keratitis the results were unsatisfactory. Sinuses from tuberculous epididymitis healed promptly under cod-liver oil injections. Tuberculosis of the bladder was disappointing, though there was some improvement in the dysuria. In the great majority ischio-rectal fistulae responded satisfactorily. Lupus vulgaris and serofuloderma gave good results.—*Clinical Journal*.

Convulsions in Children.—Charles F. Mc Kham writes an article in *Digest of Treatment* on this subject.

During the neonatal period, convulsions may be associated with cerebral haemorrhage due to birth injury consequent to difficult labour. In these cases, spinal drainage with removal of bloody fluid is indicated. Cerebral haemorrhage due to haemorrhagic disease of the newborn as indicated by bleeding elsewhere and by the finding of low blood prothrombin, necessitates transfusion of whole or citrated blood or administration of vitamin K to stop bleeding, followed by spinal drainage to remove extravasated blood.

Subdural haemorrhage in infants as a result of birth injury or in older children from a blow on the head may result in chronic subdural haematoma. Diagnosed by subdural tap, it is best relieved by surgery.

Convulsions in older children may occur late in cases of intracranial tumours, cysts, or abscesses, and are preceded by other signs of increased intracranial pressure, such as vomiting, visual disturbances, and papilloedema. Intravenous injections of hypertonic sugar or salt solutions may be required to alleviate the symptoms until surgical treatment is possible. In infants opening of the sutures under increased intracranial pressure may mask expanding intracranial lesions.

Convulsions occur late in cases of purulent

meningitis, but are an early sign in tuberculous meningitis. Luetic central nervous system involvements in infants and children are not commonly causes of convulsions. Encephalitides of almost all types, including epidemic encephalitides, equine encephalomyelitis, postinfectious encephalitis such as that following measles, mumps, pertussis, influenza, or vaccination, or toxic encephalitis as in lead poisoning, are commonly characterized by convulsions. Death in these disorders often occurs after the patient has spent many hours in a recurrent or continuous convulsive state. Contrarily, convulsions are rarely associated with poliomyelitis.

The picture of tonic convulsions of tetanus or strychnine poisoning is usually characteristic enough to suggest the diagnosis.

Meningism, perhaps the most common cause of convulsions in children beyond the age of infancy, is the cerebral reaction to infection outside of the central nervous system, such as occurs at the onset of pneumonia, scarlet fever, otitis media, and other infections. The convulsions tend to subside when the underlying cause is well established.

Convulsions occurring in the course of acute haemorrhagic nephritis are accompanied by temporary hypertensive states, and may be relieved by Magnesium Sulphate injected intramuscularly followed by oral and rectal administration. Contrarily, only temporary benefit can be obtained in convulsions occurring in the uraemia of chronic nephritis.

Since the recognition of the association of vitamin D deficiency with infantile tetany, convulsions of this type are less frequent. Calcium chloride by mouth or calcium gluconate by injection usually gives prompt relief in infantile tetany. Gastric tetany associated with alkalosis secondary to continued vomiting may be relieved by large amounts of Sodium chloride by hypodermoclysis. Parathyroid tetany may require the use of Parathormone.

Hypoglycaemic convulsions occur infrequently in children but may be suspected with an overdose of insulin in diabetic patients, in newborn infants of diabetic mothers, and in otherwise normal children subjected to starvation. The response of these patients to glucose by mouth or by intravenous injection is dramatic.

Although an attempt must be made at once to stop a convulsive attack, symptomatic treatment is frequently only temporary. A diagnosis of the underlying cause may be necessary for control of the attack. Consequently, the relationship of age to the operation of the various causes assumes importance. In the early weeks of life cerebral haemorrhage associated with birth injury or haemorrhagic disease of the newborn is most important; infection ranks second, while tetany is uncommonly observed. Organising cerebral haemorrhages may cause trouble not only for weeks but for several months after birth. In infants between four months and two years of age, tetany becomes more

important as a cause of convulsions; meningitis also assumes prominence, and meningism begins to be observed. In children over two years of age, meningism is the most common cause of convulsions while meningitis, nephritis, and encephalitis are also prominent. The diagnosis of epilepsy is entertained only in children beyond the age of infancy in whom repeated attacks of convulsions have occurred in the absence of any of the demonstrable causes mentioned above.

When the underlying cause of convulsions cannot be identified or where no specific treatment is available for an ascertained cause, symptomatic treatment, primarily sedation to stop the attack, is indicated.

In an ascending order of effectiveness the procedures commonly employed to induce symptomatic relief are as follow:

- (1) Cold to head, heat to body, followed by colonic irrigation.
- (2) Chloral or Bromides by mouth or by rectum. Dosage from 5 to 15 grains, depending on body weight.
- (3) Ether. An immediate but often transient effect when inhaled.
- (4) Pentobarbital Sodium, Phenobarbital Sodium, or other Barbiturate orally or by injection, 1-3 gr. according to weight.
- (5) Avertin by rectum, according to body weight.
- (6) Paraldehyde by rectum, 2 to 6 cc., according to body weight.
- (7) Morphine. Tolerance to opium is marked in patients in convulsions, so that moderately large doses are indicated.

In very severe and prolonged convulsive states such as occur in tetanus or in certain types of encephalitis, heavy sedation with repeated doses of a single drug becomes less satisfactory than dependence on alternate use of several—e. g., Phenobarbital Avertin, Paraldehyde, and Morphine. Morphine is not recommended for patients with convulsive states in whom there is evidence of increased intracranial pressure, especially if there is already respiratory depression.—*General Practitioner*.

Antiemetic Properties of Magnesium Sulphate.—Repetto and Gamponavo (*Revista de la Asoc. Med. Argentina*, (Jan. 15, 1940) report satisfactory results from the administration of Magnesium sulphate in the treatment of vomiting. The authors carried out experiments on dogs and observations on twelve patients in a psychopathic institution. Patients and animals were given an intramuscular injection of from 5 to 10 cc. of a 25 per cent solution of Magnesium sulphate, five or ten minutes before or simultaneously with the administration of Apomorphine hydrochloride. The latter was administered subcutaneously to patients in doses of 0.005 gm. of Apomorphine and to the animals in doses of from 0.001 to 0.005 gm. of the drug by the intramuscular route. A group of patients was given Ipecac instead

of Apomorphine in three cachets, each containing 0.5 gm. at intervals of five minutes. The authors found that the effects of the drug are manifest in from ten to twenty minutes after its intramuscular administration.

The effective dose for man varies from 5 to 10 cc. of the 25 per cent solution of Magnesium sulphate in distilled water given intramuscularly. The dose can be repeated the same day, not to exceed the total daily dose of 20 cc.

The treatment is indicated in all cases of spontaneous vomiting and, that complicating various diseases, especially in the presence of pain, insomnia and neuromuscular excitability. Magnesium sulphate is the only substance capable of neutralizing the emetic effects of Apomorphine. It acts by diminishing the excitability of the bulbar centres of vomiting.

The authors did not observe a depressive effect of the drug on the respiratory centres. Theoretically an intravenous injection of calcium can be resorted to because of its antidotal action for Magnesium sulphate, along with the administration of drugs capable of stimulating the centres of respiration.—*General Practitioner*.

Cod-Liver oil as a Dressing.—The successful use of pure cod-liver oil in the treatment of burns and scalds, varicose ulcers, diabetic and senile gangrene, amputation stumps and carbuncles is recorded by J.P. Steel (*Journal of the Industrial Welfare Society*, March 1941, 23, 56). Before reporting the results obtained the author lays special emphasis on the need for the use of pure cod-liver oil, i.e. a medicinal oil "guaranteed to be pure cod-liver oil from which no constituent has been removed and to which nothing has been added." The results were checked by photograph and clinical observation and compared with control cases treated with tannic acid, Acriflavine and dyes. The method of applying the oil is easy; it is poured on the wound and a covering applied, the dressing being kept moist with oil without removing it for each application. Daily changing of the dressing was carried out and this was found to be less painful than with other dressings. The advantages claimed include (1) prompt relief of pain—this was marked in the treatment of burns—with consequent lessening of shock and a minimum of pain when re-dressing was carried out; (2) healthy granulation, and (3) reduction of scarring. The last was particularly marked in burns of the conjunctiva caused by acids. When the dressings are first removed what appears to be pure pus is noted; this quickly decreases, and below the healthy granulations will be seen. The use of cod-liver oil on frost-bitten feet resulted in immediate alleviation of the pain and in the treatment of carbuncle once the lesion is open the dressings have a soothing effect, help the discharge of pus, and lead to rapid healing of the wound. In conclusion it is stated that apart from the vitamin A

and D content of cod-liver oil, which has a specific action on epithelial tissue, some other agent must be present, as a combination of A and D in Arachis oil gave less satisfactory results, in spite of ultra-violet irradiation. It is of interest to note that latent sepsis was absent in the treated cases.—*The Practitioner*.

Is Alcohol a Stimulant? The belief that alcohol acts as a stimulant has its roots in centuries long past. "Mankind has grown old," said Arnold of Villanova, "and has become feeble; therefore God has given him this water of life that he may grow young again." Many other non-medical writers, usually poets, have recorded remarkably accurate observations showing how reasonable it is to suppose that alcohol is essentially a stimulant. Horace gave the classic description of its psychic effects: "What wonders does not wine! It discloses secrets; ratifies and confirms our hopes; thrusts the coward forth to battle; eases the anxious mind of its burden; instructs in arts. Whom has not a cheerful glass made eloquent! Whom not quite free and easy from pinching poverty!" At the end of the 19th century Binz and his pupils published a series of papers on the effects of alcohol which they thought could best be explained on the basis of stimulation followed by depression. Their views were probably the more acceptable to pharmacologists and toxicologists because the "pattern" exhibited by alcohol is often seen in the pharmacodynamics of other potent substances. Schmiedeberg opposed this view and contended that the action of alcohol is purely narcotic from the start, and that stimulation is only apparent, depending on paralysis of those cerebral functions which normally play a restraining part. It is nowadays accepted that the increased self-confidence and the assumption of a benevolent, communicative disposition are release-phenomena and are attributable to depression of the faculty of self-criticism.

The conception that alcohol might have a selective stimulating action on some parts of the nervous system while behaving as a depressant elsewhere is seen in Emil Kræpelin's work of fifty years ago. He held that alcohol is entirely depressant to sensory and intellectual processes, but that the generation of motor impulses is first stimulated and only later depressed; these are essentially Binz's conclusions restricted to the motor mechanisms of the nervous system. In 1923 a committee of British pharmacologists expressed the opposite view in a publication of the Medical Research Council. From experiment they concluded that the action of alcohol on the nervous system is essentially sedative; that it was not a cardiac stimulant; and that it had no noteworthy effect on respiration in moderate doses though in large doses it paralysed the respiratory centre. They pointed out that the reflex stimulation of the heart and respiration which follow irritation of the buccal and pharyngeal mucosa by alcoholic beverages must not be credited to the alcohol itself; in

wines, the bouquet due to higher esters may similarly confuse the issue. In order to eliminate the indirect results of psychical instability engendered by communicative companions the subject of the experiment, they insisted, must be segregated. The reader is left with the conviction that whatever may be the merits of alcohol as a therapeutic agent (and they are not to be underrated) it does not qualify for the description of "stimulant." But this does not mean that the last word has been said or that the work of Binz and his followers is discredited. On the contrary, recent experimental findings suggest that within certain limits of dosage the alcohol metabolised by the nervous system may produce stimulation after all. The uncertainty dates from a study made by J. D. Robertson and C. P. Stewart some ten years ago showing that the oxygen uptake of the brain is significantly increased during the first hour of alcoholic intoxication. Masserman, Beale and Sanders have recently been working with cats to determine the action of ethyl alcohol on cortico-hypothalamic function. Under Nembutal or light ether anaesthesia needle-electrodes are introduced into the cat's brain, and the effect of electrical stimulation is noted, first when the cat has had no alcohol and then when alcohol has been introduced through the needle electrodes. The method is elaborate and artificial, and thus open to serious criticism unless the data obtained are scrupulously controlled. A comprehensive preliminary study was undertaken by J. H. Masserman in order to meet these objections. He reports that simple electrical stimulation of the hypothalamus induced behaviour suggestive of rage and fear; neutral solutions injected through the needles produced no effect; weak alcohol (0.05–0.1 c.cm. of 0.1% alcohol in Ringer's solution) made the hypothalamic response to electrical stimulation more pronounced in a significant proportion of experiments. Intraperitoneal administration of alcohol (0.5–1 c.cm. per kg. body-weight) did not produce any material change. The responses of the posterior sigmoid cortex were not significantly affected by the direct injection of alcohol, but in some instances they were increased by the previous intraperitoneal administration of alcohol. It is well known that general anaesthetics and basal narcotics have actions on the nervous system which are predominantly selective for certain strata of the brain. Masserman's observations that the stimulating effect of alcohol on the hypothalamus was abolished by nembutal, and that its action on the cortex disappeared under light ether anaesthesia, are therefore consistent with accepted teaching regarding the mode of action of these anaesthetics; and this evidence supports his main thesis concerning the pharmacology of alcohol. The graphic records of the pulse-rate, blood-pressure and respiration included in Masserman's paper show that the injection of 0.1 c.cm. of 0.1% ethyl alcohol in Ringer's solution produced no direct effect, but the subsequent responses of the hypothalamus to 2.5 volts exceeded those elicited by twice that strength of current before the

injection. These experiments justify the tentative conclusion that alcohol facilitates certain reactive processes in the brain (especially in the lower diencephalic centres); and if this is so its action is analogous to that of Strychnine on the spinal and bulbar reflexes. It remains to determine the therapeutic usefulness, if any, of this transient stimulation. With the growth of neurosurgery the time seems ripe for the renewal of cooperation between pharmacologist and surgeon on the lines of the fruitful partnership that once existed between Clætta and Sauerbruch—*Lancet*.

Prostigmine with Morphine for Analgesia.—By Donald Slaughter, M.D., J. C. Parsons, M.D., and H. D. Munal, M.D., (Condensed from "The Journal of the American Medical Association, Dec. 14, 1940.")

Much work has been done on producing a chemical which might serve as a substitute for morphine. The present report considers the use of prostigmine methylsulphate in combination with morphine in a series of approximately 100 cases.

Prostigmine is the dimethylcarbamate ester of mxyphenyltrimethyl ammonium methylsulphate. Pharmacologically it is similar to physostigmine salicylate in that it is a stimulant of the parasympathetics and has the power of inhibiting the cholinesterase. It does have the advantage over physostigmine of showing little, if any, miotic or untoward cardiac effects and of having greater stimulating action on the small intestine. Recently it has been shown by Schweitzer, Stedman and Wright that prostigmine methylsulphate is the only therapeutic anticholinesterase drug which has a central depressant effect.

The use of prostigmine methylsulphate in myasthenia gravis is now well accepted. Recently it has been used with apparent success in the treatment of deafness. Further, its use to treat and prevent post-operative ileus and anuria is well established. The rationale in myasthenia gravis and post-operative use rests on the ability of this drug to protect acetylcholine against being destroyed by cholinesterase. The premise as regards deafness is not, at the moment, perfectly clear.

Since Slaughter and Munsell recently reported that prostigmine methylsulphate potentiates the action of morphine on pressure-pain in cats, it seemed plausible to try out such a combination on the human being. That prostigmine methylsulphate is a safe drug in itself has been shown by Schwert, Tietze and others.

RESULTS OF TREATMENT.—The patients in this study roughly fall into six groups: (1) trauma, (2) pre-operative sedation, (3) post-operative treatment, (4) coronary occlusion, (5) miscellaneous cases, and (6) addiction.

1. **Trauma.**—There were approximately fifty cases in this series. They consisted of trauma due to gunshot wounds, stabblings, fractures (simple and compound), and other types of extensive trauma in which the pain

made morphine mandatory. As a rule only 8 mg. of morphine with 1 c.c. of a 1:2,000 solution of prostigmine methylsulphate was given subcutaneously. In these patients apparent relief of pain came on in from two to ten minutes. In other accident cases of a similar nature in which only 16 mg. of morphine was administered as much relief from pain was not obtained and the onset of relief was longer, i.e., from six to twenty minutes. These observations were made from the time of the injection to the time the patient expressed real relief from pain and discomfort.

Only one minor, aged 15, received this treatment. He experienced excellent relief and there were no untoward effects.

2. **Pre-operative Sedation.**—There were twenty-five cases in this group. Most of them were gynaecologic but five appendectomies were included.

By trial, the administration of 5 mg. of morphine plus prostigmine methylsulphate four hours before surgical operation and repeated one hour before the operation proved unsatisfactory in producing sufficient sedation.

Further trials established the following routine as giving the most adequate pre-operative sedation:—

1. Phenobarbital 0.2 Gm. the night before operation.

2. Phenobarbital 0.2 Gm. four hours before operation.

3. Morphine 8 mg. plus prostigmine methylsulphate thirty minutes before operation.

This schedule appeared to give as good sedation as substituting 16 mg. of morphine instead of the 8 mg. plus prostigmine methylsulphate. At the time of operation this use of prostigmine methylsulphate did not interfere with any surgical procedure. All appendixes were removed in the gynaecologic cases, if present.

3. **Post-operative Relief from Pain.**—All these patients had orders for 8 mg. of morphine plus prostigmine methylsulphate on return from the operating room, to be repeated every three hours for three doses and then "as needed". There were five cases of moderate distention in this series following operation. These were relieved by the insertion of a rectal tube and the administration of 0.5 c.c. of a 1:2,000 solution of prostigmine methylsulphate every twenty minutes for two doses and then every two hours for four doses if needed. Only one patient required the use of a Wangensteen suction.

4. **Coronary Occlusion.**—Five patients with conditions diagnosed as coronary occlusion were treated in the emergency room with 8 mg. of morphine plus prostigmine methylsulphate. All received relief within ten minutes. They then were given this combination every three hours "as needed." In none of these cases was it necessary to order additional doses of morphine. Three

received this form of treatment until death, i.e., from eight to thirty-six hours. The other two were discharged after a few days of hospitalization on this treatment. Previously such patients had always received 16 mg. of morphine. We felt that the results of the combination were as good as followed the use of larger doses of morphine.

5. *Miscellaneous Cases.*—These included bronchiectasis, delirium tremens, kidney abscesses, nephritic pains, and the dressing of painful wounds. They required morphine. A typical case follows: A patient with nephritis suffered extreme lumbar pain which 16 mg. of morphine did not alleviate. The combination of 8 mg. of morphine plus prostigmine methylsulphate gave and maintained comfort.

6. *Addiction.*—Only one case of true addiction is presented. The patient was treated over a period of six weeks. He had urethral calculi with periurethral abscesses.

Six years previously he had gonorrhoea, followed in one year by stricture of the urethra. At this time he entered the hospital for a urethrotomy. Soon after this operation, periurethral abscesses and a urethral fistula developed which persisted until the present time. At intervals, urethral calculi complicated the picture. The patient was an ambulance driver who gave no history of any drug habits before the urethrotomy. After operation he was given 10 mg. of morphine for pain relief. It became necessary to increase this dose to 16 mg. of this drug. Shortly the patient became addicted and took as much as 30 mg. at intervals. Three years of this habit brought his admittance to a government hospital. After the hospital discharged him as cured of the drug habit, his urethral difficulties returned and a physician put him back on morphine for relief of pain. He continued to take 16 mg. of morphine. Six weeks prior to admission to the Robert B. Green Memorial Hospital, with the diagnosis of periurethral abscesses, urethral calculi and urethral fistula, he stated that he was taking 20 mg. of morphine every four hours.

A radical urethroplasty was successfully performed. Post-operatively he was placed on 30 mg. of morphine. Two days later 16 mg. of Morphine plus prostigmine Methylsulphate apparently satisfied him. Reduction of the dose of Morphine to 10 mg. gave excellent results for four days. Further reduction to 8 mg. did not suffice.

During this stage of the treatment he had not been told of the use of prostigmine methylsulphate but did comment that his bowel action had returned to normal. When he was told of this treatment, he co-operated well. The wound had healed after four weeks and the catheter had been withdrawn. Voluntary urination was instituted after two trials. At this time 10 mg. of morphine plus prostigmine methylsulphate was still being given and the patient did very well, complaining only of slight nervousness just before his dose. This procedure continued through the fifth post-operative week.

The sixth week 8 mg. of Morphine plus Prostigmine Methylsulphate was ordered. After the second day of this treatment the patient skipped every other dose without ill effects. He was then discharged to a government hospital for observation.

During the time Prostigmine Methylsulphate was used with the Morphine, no withdrawal symptoms were noted.

COMMENT.—This study indicates that one-half of the accepted dose of Morphine when combined with Prostigmine Methylsulphate in doses of 0.5 mg. gives as good relief as the larger dose of Morphine. There were no side reactions noted. An order of 0.5 mg. of Atrophine was always written, but it was never needed. No apparent tolerance to Morphine when used in this combination becomes evident.

There was one case of ruptured appendicitis complicated by peritonitis in which this combination gave satisfactory results. Sae-gesser has reported the use of Prostigmine Methylsulphate in a series of approximately thirty cases of peritonitis. He comments on the excellent results, and says that the untoward effects in this condition make this drug suitable for use as a prophylactic to lessen the danger of postoperative intestinal atony. In our series of cases there was none which showed ileus or anuria following operation.

In all but two cases the combination of Prostigmine Methylsulphate and 8 mg. of Morphine relieved pain. Twice in this series of 100 cases it was necessary to give 16 mg. of Morphine instead of the smaller dose; one was a case of extensive gunshot wounds and one of compound fracture.

While no untoward effects were observed following the use of this combination, it is felt that perhaps shock is a contraindication to use of prostigmine methylsulphate with morphine.

We feel that combinations of 1 c.c. of a 1:2,000 solution of prostigmine methylsulphate plus 8 mg. of morphine is an efficient pain reliever. Further, it is an excellent pre-operative sedative and post-operative analgesic. When used before operation, it definitely minimizes ileus, distention and anuria. Levis and Axelman believe that without prostigmine methylsulphate from 60 to 75 per cent. of patients will suffer from one or the other of these conditions. Used post-operatively, enemas and the use of catheterization are decreased.

The one case of addiction treated with prostigmine methylsulphate lends encouragement to the use of this drug in similar cases.

The results in this study would seem to substantiate the experimental work done on this problem. We believe that they bear out the findings of Slaughter and Munsell that morphine is a cholinergic drug as regards its action on pain. That it is cholinergic in other respects is evidenced by the reports of Slaughter and Gross on the dog's intestine, cat's blood pressure and toxicity in rats.

Migrainous and other Sick Headaches.—By A. H. Douthwaite, M.D., F.R.C.P. (Condensed from "Guy's Hospital Gazette", Nov. 15, 1940.

The layman, in complaining of sick headache, is found as a rule, to be suffering from one of three things—constipation, purgation, or migraine. The last is the commonest.

CONSTIPATION.—The peculiarity about the headache of constipation is that it is more apt to arise in persons who are only rarely constipated than in those in whom the disturbance is of chronic nature. A generalized, dull headache, furred tongue, loss of appetite and nausea are the symptoms here, and are seldom met with in dyschezia. The latter may be associated with headache, but not with sick headache. An example of the class to which I refer is given by the individual who, having gone without a night's sleep, sleeps long and deeply on the following night. He awakes unrefreshed and declares that he has the feeling that his inside has ceased to function. The rectum is found to be empty; the colon, on the other hand, is readily palpable on account of the contained faeces. Clear thought is often impossible; the mind shies at the prospect of work. Within half an hour of evacuation produced by an enema the furred tongue, lassitude depression, nausea and headache disappear.

"It isn't the day after a late night, but rather the following day that one feels so awful" is a *cliche* based on a background of the above signs. It seems that the sleep of extreme exhaustion must bring with it a profound retardation of digestive processes.

PURGATION.—The sick headache due to purging is a far commoner visitant, in this country at any rate. The responsibility for it rests upon earlier generations of doctors, and has now been eagerly assumed by unscrupulous vendors of purgatives. That most of the symptoms popularly attributed to constipation could be produced by purging was pointed out years ago by Hurst, but the knowledge is spreading very slowly. It seems that by hurrying the passage of intestinal contents the normal process of desiccation of faeces in the colon is prevented. This allows for profuse microbial growth, putrefaction and the absorption of various toxic substances into the portal circulation. Two years ago I selected six patients with sterile urine and gave them sufficient Cascara Sagrada nightly to produce liquid evacuation. Within three to five days five of these had *B. coli* in the urine. That purgatives must not be used with Sulphonamides is common knowledge. This emphasizes the point that purging increases microbial activity in the colon, H_2S is found in excess, and, being absorbed, leads to Sulphæmoglobinaemia.

The type of headache is very similar to that of constipation, but is more persistent. Two days' constipation will abolish the furred tongue, sallow complexion, fetor oris, lassitude, nausea and headache. Abdominal exercises, suitable diet and lack of panic about bowel action are all that are needed to restore to normal function the majority of those who purge themselves daily.

MIGRAINE.—Until recent years there was a tendency to diagnose migraine only on the strength of disturbance of vision followed by unilateral headache which was later accompanied by vomiting. This excluded a number of truly migrainous subjects whose symptoms were less clear-cut. Actually headache is the only manifestation which can be expected in nearly every attack. Another common fallacy is the belief that the advent of vomiting relieves the headache. In my series of 149 patients this occurred in eight (12 per cent.) only.

Migraine may be defined as a paroxysmal headache often preceded by disturbances of vision, and frequently coupled with nausea and vomiting. It usually starts unilaterally in the frontal or occipital region, but remains on one side of the head in approximately one-third of the cases only.

AETIOLOGY.—*Sex.* In this series, 102 were females 33 males. The proportion suggested is, however, probably false, for a review of greater numbers indicates that female preponderance is only slight.

Heredity.—From just over half the patients a history of bilious headaches or blind headaches affecting the mother or maternal grandmother was obtainable. In three there was a history of migraine in the father. It appears, therefore, that inheritance is of considerable importance.

Age of Onset.—The relationship of migraine to the female reproductive period is striking. Thus half of my female patients were first attacked at puberty, a few started in childhood, 12 per cent. at the menopause, and the remainder between 20 and 30 years of age. Thus the beginning and end of the reproductive period account for 62 per cent. of onsets. Note that the belief that one can promise relief with the menopause is not upheld. Of 13 females who had developed migraine years before the menopause and who consulted me some years after that event, eight had experienced partial relief; five were much worse up to a post-menopausal period of eight years, and five had had complete relief and were seen in consultation for some different reason.

PROVOCATIVES.—The various agents responsible for producing an attack of migraine may be grouped under four headings, namely; (1) Food; (2) Exhaustion; (3) Allergy; (4) Reflex. Ten per cent. of sufferers are sure to be attacked if they eat certain foods, especially those of fatty nature, rich cheese, eggs and chocolate. They resemble in this respect the gall-bladder sufferer.

Mental exhaustion and worry are potent provocatives. Comparatively few patients are attacked when on holiday, provided that the vacation is being spent with congenial companions.

Allergy tends to be dragged into the aetiology of so many obscure diseases that I mention it with some diffidence. In only ten of my patients did it seem fair to incriminate it. The foods blamed were chocolate, cocoa, pork, beef and coffee. An attack could usually be produced by the consumption of the suspected substance, and positive

skin tests were shown in six. On the other hand, not one of them was entirely free from migraine even if the food were rigidly avoided. The possible importance of allergy is heightened by enquiry into the earlier history and into that of associated diseases. Thus asthma had occurred in 14 per cent; urticaria in 13 per cent. In both it was striking that they never arose during an attack of migraine.

As children, over a third of the patients suffered from train or car sickness.

The provocation of an attack reflexly by an external stimulus is not very common, but is dependable on the particular individual. Thus in one the glare of head-lamps will at once initiate a migrainous paroxysm; in another the flashing light from the sea on a sunny day will cause prostration. It will probably surprise you to know that as recently as fifteen years ago it was being taught that migraine was the result of uncorrected errors of refraction. This theory had held its sway for many years in spite of the fact that migraine usually starts during, or immediately after, a night's sleep, and in spite of its peculiarly paroxysmal, periodic character. I have met a few patients whose attacks have diminished in frequency as the result of wearing suitable lenses, but cure must be rare. In short, eye-strain can be regarded only as a provocative.

SYMPTOMS.—Migraine may arise at any time of the day or night, but quite the commonest is the early morning. The patient may be awakened by the headache, and thus earlier visual disturbances will not have been noticed. This partly explains that only 58 per cent. admitted to having experienced visual phenomena.

Initial Disorders of Vision.—Of the 82 persons thus affected, 55 had major visual disturbances at the onset of an attack, e.g., complete blindness, loss of a large portion of the field of vision, a blind central circular area, blindness in one eye, or blindness in half the visual field of one eye. The well-known zig-zag lights, fortification figures and whirling balls of coloured light at the periphery of the blind area occurred in them. The remainder said that there was some blurring of objects, a running together of the lines of a book, black spots or the appearance of rain. Very rarely transposition of objects from a "black-out" visual field to another area would take place.

Visual disturbances usually last for a few minutes, rarely two hours. They subside as the headache appears—a fact of some importance in considering the mechanism of a paroxysm.

Headache.—Severe headache occurring in each attack was present in 80 per cent. of patients. In the remaining 20 per cent. headache was not always a part of the seizure. Thus on occasions an attack might consist of blurring of vision, nausea and lassitude, which would pass off in a few minutes. In others, typical migrainous attacks might be interspersed with abdominal migraine.

In character the headache commonly starts behind one eye or deep in the frontal region;

less often it is occipital. It spreads rapidly from this focus to involve the whole cranial area in two-thirds of cases, and is strictly unilateral in the remainder. In all patients it is usually most severe on one side than another, in which case it is invariably so on the side on which it began.

Sufferers describe the headache as blinding, devastating; prostrating, or of boring nature. It never gives a sensation of pressure from without, but often of bursting from within. In nearly every case the headache develops a throbbing character soon after its commencement.

Of the 47 persons who had unilateral headache, 22 experienced it always on the same side; in 25 it was on one or other side with a bias towards one, usually the left. If the headache always occurs on the one side of the head, the possibility of its being due to a leaking cerebral aneurysm should be considered.

The headache is aggravated by noise, light, mental effort, and by the strain of vomiting. It usually renders the victim incapable of work, and he seeks repose in a darkened room. When vomiting occurs it makes the headache worse in 88 per cent. and brings relief in the remainder.

The duration of the headache is usually a matter of hours, sometimes days. The common story is that an attack starting in the morning increases in severity until evening, when sleep comes to ease the exhausted victim. On the next day all traces of the attack have passed, and the patient may even feel better than he has done for the last few weeks.

Nausea and Vomiting.—Roughly one-third of migrainous patients does not suffer from nausea or vomiting. Vomiting occurs in 48 per cent., nausea only in 12 per cent., and prolonged retching not preceded by vomiting in 8 per cent. Those who vomit are to be pitied, for every outburst intensifies the headache, and when the stomach contents have been ejected bile is brought up. When the supply of this fails, unproductive retching adds to the agony.

UNUSUAL TYPES.—1. A fluctuating but persistent headache lasting for several days (*status hemicranicus*) is always associated with rapid loss of weight, and sometimes with numbness or hemiplegic distribution.

2. Phenomena suggesting focal lesions in the brain, e.g., aphasia, unequal pupils, hemianopia ophthalmoplegia, or even hemiplegia. These, rare accompaniments of a migrainous attack only arise after many years of the disease. The signs disappear with the subsidence of the attack, but with each subsequent seizure they are likely to recur. In the long run they may become permanent. This can only be explained on the grounds that an organic change has been superimposed on a disorder of function. Autopsy has revealed cerebral thromboses as the cause of this perpetuation of symptoms.

3. Migraine-epilepsy is a rare form. It is most likely to arise in persons with a family history of epilepsy. It is characterized by the advent of loss of consciousness when the migrainous headache has reached its height.

Convulsions may occur. With recovery of consciousness it is found that the headache has vanished.

4. Migrainous neuralgia.—The attack of migraine may be accompanied and/or followed by severe pain on one side of the face, neck, head or throat. Occasionally this pain arises without the headache.

5. Abdominal migraine.—The greatest care must be taken before committing oneself to this diagnosis, which I have seen oftenest misapplied to examples of appendicular colic. The outstanding features are prolonged vomiting, severe abdominal pain of colicky type, no rigidity, and no point of tenderness. On the other hand, tenderness along the liver margin is almost constant. It is necessary also to have a history of attacks of true migraine. Sometimes urticaria follows an attack.

Liver tenderness and slight transient enlargement is often noticeable after a typical migrainous attack, especially in those who incriminate foods as provocatives. The tenderness is not limited to the gall bladder area, and a cholecystogram has a normal appearance. Unfortunately it is not needless to point out that cholecystectomy never helps the patient. A poor gall-bladder shadow after oral administration of the dye usually indicates faulty absorption and not cholecystitis. Intravenous administration should in such cases be undertaken.

WARNINGS OF ATTACK.—In roughly half of these patients a warning the day before an attack of migraine could be recognized. These were drowsiness, furred tongue, nasty taste in mouth, pale stools, diarrhoea, constipation, anorexia, unusual hunger, or exceptionally deep sleep. For any one patient the type of warning was constant.

Onset and Closure.—Headache started in the early morning in 68 per cent. The end of the attack usually coincided with deep sleep, sometimes with polyuria, rarely with great hunger.

Spontaneous Relief.—Relief from migraine can be confidently expected only as the result of pregnancy or the advent of old age. The menopause will probably ease matters.

SPECIAL INVESTIGATIONS.—There is no constant biochemical abnormality to be found during or after an attack of migraine. It is true that the blood sugar in an attack is low, but this can be explained in the usual hour of onset, and the rejection of all food. Laevulose tolerance tests are normal. In an attack the blood uric acid has risen in the six out of nine cases examined for this. The inter-paroxysmal figures have been in the neighbourhood of 3 mgs. per 100 c.c., rising to 5 to 6 mgs. in the attack. The significance is not clear. The Van den Bergh test is usually negative except in the instances where slight icterus follows an attack. The urine after paroxysms often gives a positive ether-reaction, indicating the presence of the "proteose" of Ortel. Whether this observation is of any practical value has yet to be proved. It is of interest in that it also arises in asthma and urticaria.

The C.S.F. pressure in an attack is usually low. A migrainous girl whom I had in the

wards always had an attack after drinking cocoa. Her normal C.S.F. pressure was 150 mm. of fluid. She was given cocoa and had an attack that night. At 4 a.m. the C.S.F. pressure was 65 mm. The blood uric acid had risen from 3.5 to 5 mgs. per 100 c.c. The blood sugar was 0.08 per cent. as compared with an interparoxysmal figure of 0.105 per cent. at 8 a.m.

THE PROXIMATE CAUSE OF MIGRAINE.—Many ingenious but quite untenable hypotheses have been advanced to explain the migrainous headache. We are still wandering in the field of conjecture. There is, however, a lot to be said for the theory of vascular disturbances being responsible. Thus during the paroxysm the affected side, if unilateral, may be paler or redder than the other; the temporal artery may be contracted or dilated. The headache is throbbing in nature. It can be temporarily arrested in unilateral cases by compression of the common carotid only to return in greater severity when the pressure is released. Locally, the two drugs which are the most reliable for the relief of headache, namely ergotamine and ephedrine, act principally upon the arteries and arterioles. The transient paresis occasionally seen remind one of similar results in hypertensive cerebral attacks.

It is suggested then that the visual changes and the headache are due to alterations occurring in the calibre of the intracranial vessels, the first being possibly that of contraction, the second being that of dilatation.

TREATMENT.—(a) In attack. Ergotamine Tartrate injected intravenously or intramuscularly produces striking relief in 80 per cent. of cases. The intramuscular route is usually adequate, and the dose is 0.5 mg. The drug may produce vomiting, but this is shortlived, and can be prevented by the simultaneous administration of atropine. It often causes cramps in the legs for a day or two afterwards. An interesting side-effect is the shrinkage of varicose veins for some hours after its administration. If the drug given in this way is a success, then the effect of oral administration of 1 mg. may be tried. If the tablet be allowed to dissolve under the tongue before swallowing, enough is absorbed through the buccal mucosa to ensure earlier action. When Ergotamine fails, Ephedrine Hydrochloride (1 to 1½ grs.) may be tried.

Mild attacks may yield to such simple adalgesics as phenacetin or aspirin. In all severe paroxysms the patient is best kept in a dark room, recumbent, and guarded from solicitous friends. If the above drugs fail, the attack must be allowed to take its course, and any fussing of the victim only makes matters worse.

(b) Between attacks.—Our duty here is to search out the provocatives and to help the patient to avoid them. These I have already enumerated. The "liverish" group, whose members are upset by fats and who often have pale stools, undoubtedly, benefit from a diet free from cooked animal fats, cream, eggs, egg dishes, cocoa, chocolate, spiced food and coffee. Furthermore, the administration of the cholagogue dehydrocholic acid 7½ grs. twice daily on alternate days

and glucose one ounce at night is worthy of trial. The value of a sharp walk between dinner and bedtime, though scarcely to be

recommended at the moment, should be remembered for the days of peace.—*The General Practitioner*.

Surgery

The Treatment of Burns.—The following notes are based on the recommendations of the co-ordinating team on burns, appointed by the War Wounds Committee of the Medical Research Council. They are circulated for the guidance of Medical Officers in E.M.S. Hospitals and First Aid Posts.

1. *At the First Aid Posts:*—(a) Minor burns, which do not require admission to a hospital should, after cleansing with plenty of soap and water, be dressed as follows:

(i) On the face or hands, with gauze or lint impregnated with sterilized vaseline.

(ii) In other parts of the body with tannic acid jelly or gentian violet jelly, 1 per cent. If jellies are used no dressing should be applied, and clothing not be allowed to come into contact with the burn area until the coagulum has formed and is dry. The application of tannic acid or other coagulant jelly to a burn which has not been adequately cleansed is inadvisable:

(b) Serious burns requiring admission to hospital should be treated by applying sterile vaseline on gauze or lint to cover the exposed portions. Provided the patient is shortly to be transferred to a near hospital, no attempt should be made to remove clothing in order to expose the entire burn area. Serious burns always require injections of morphine for pain, in full doses ($\frac{1}{2}$ — $\frac{3}{4}$ gr. for adults).

2. *At the first hospital*, where the case can be fully treated, the following treatment is advised for extensive burns of whatever degree. Under gas and oxygen or pentothal anaesthesia, the burn areas should be thoroughly cleansed by the gentle but liberal use of soap and water, followed by saline and by removal of debris under strict aseptic precautions (soft soap should not be used; the best application is some form of soap flakes). No violent scrubbing or scraping should be used, nor antiseptic solutions or spirit. If the burn area is impregnated with oil or grease, ether soap is useful for cleansing. Extensive burns should never be fully exposed at one time, owing to the risk of increasing shock by loss of body heat; only a limited area should be exposed for treatment, which should be completed before the next area is dealt with. Loss of heat is especially marked when ether soap is used. After the cleansing, the burn surface and the skin around should be dried and the burn lightly dusted with sulphanilamide powder; a coagulant is then applied *except in the areas mentioned below* (paragraph 4).

3. *Coagulant applications.*—The following solutions are the best, as they act quickly and require no special apparatus, etc.:

- (1) Silver nitrate, 10 per cent.
- (2) Tannic acid, 10 per cent.
- (3) Silver nitrate, 10 per cent., and tannic acid, 5 per cent., used alternately.
- (4) "Triple dye" (one formula is gentian

violet, 1 in 400; brilliant green, 1 in 400 with neutral acriflavine, 1 in 1000. The solutions are mixed in equal proportions).

The application is best made with a large gauze mop; no dressings are required over the coagulated area, but it is advisable to cover the part with a sterile towel after the coagulum is dry. The edges of the area and any cracks that may develop on the surface should be dusted daily with sulphanilamide powder. In whatever way coagulation is effected the coagulum must cover the whole area and a good margin of unaffected skin around, and must be firm and dry or else the effect is lost, as the patient will still lose fluid from the burn area.

4. *Areas in which no coagulant of any kind should be used.*—The face. The hands and wrists. The feet.

Circumferential tanning of a limb should be avoided if possible, and if carried out the peripheral pulse must be carefully watched for the first 48 hours. Coagulants tend to contract on drying, and there is a risk of dangerous constriction of vessels or distortion of tissues, e. g., the fingers and eyelids, as this occurs.

Treatment of Burns in Areas which should not be Tanned.—After cleansing the burn in the manner described, the surface is dried and dusted with sulphanilamide powder. A tulle gras dressing is then applied, and this in turn is covered by 6-8 layers of gauze wrung out in warm normal saline. Tulle gras, originally a French preparation, may be prepared from curtain net, with a mesh of 2 mm., cut into pieces 9 cm. square. These are placed in a slightly larger tin box. The box is then filled with a mixture of soft paraffin, 96 gm., and balsam of peru, 2 gm., sufficient to impregnate and cover the material completely after sterilization by heat in an autoclave. The gauze must be kept moist by warm saline dripped on it from time to time, and must not be allowed to become dry. The tulle gras will enable the gauze dressings to be removed without causing pain or injury to the burn, provided they are kept moist; as a rule it is advisable for the first few days to change the gauze every 3 to 4 hours, the tulle gras being left undisturbed during this process. Twice a day at first, and subsequently, according to the process of the case, once a day, the tulle gras dressing is changed; where it is possible to immerse the affected part in a bath, the tulle gras dressing is allowed to float off, which it does readily. On the face or other parts for which a bath cannot be used a gentle stream of saline greatly assists in the removal, which should be painless and not associated with bleeding. Where baths are used the limb can remain immersed for half an hour or more, the saline being kept warm throughout. Before applying a fresh dressing the unburned surface of the limb is

dried, and the burn area dusted with sulphanilamide powder. Dressings must be carried out with full aseptic technique, and special care must be taken to ensure the surgical cleanliness of the arm and leg baths. An oil silk or jaconet bag or cover is of value in keeping the gauze moist on the arms and legs, both while the patient is in bed and during transport. Where burns of the face are treated by saline packs a jaconet bib assists in keeping the patient and the bed dry. All severe burns likely to be followed by loss of function or disfigurement, or to require extensive grafting, should be transferred to a burn centre as soon as they are fit to travel.—*Clinical Journal*.

Problems in Wound Healing.—The healing of wounds was considered at a recent meeting of the Medical Society of London, and an account of the discussion appears at page 568. The time is opportune for a brief survey of this important question. The surgeon's work rests upon the fundamental assumption that wounds will heal provided certain relatively simple conditions are fulfilled. This inherent tendency to healing and repair is so marked a characteristic of a damaged living tissue that it has led to a comparative neglect of many of the underlying and determining biological factors. The wounds may be accidental or surgical, clean or infected; they may be wounds of civil life or wounds of warfare. Yet in the absence of some overwhelming disaster such as serious shock, hemorrhage, or infection, Nature can be relied upon to make the most strenuous efforts to repair these breaches of her tissues however they have been inflicted. And if, in Pirogoff's words, we consider war as a "traumatic epidemic," and under modern conditions an epidemic that knows no frontiers, then a time of war must be a special one for the study of the problems of wound healing. Why then has the subject been so neglected? First, because, the whole of surgical technique assumes the potentialities of wound healing, as, in the absence of these all surgery would be impossible. Secondly, the more detailed study of wound healing has been dependent on a whole host of ancillary developments, all having some bearing upon the processes of tissue repair: on surgical technique, asepsis, bacteriology; on the effects of various suture and ligature materials in wounds; on biochemistry and the knowledge of specific growth stimulants; and finally on the ramifications of the more recent knowledge of nutrition. The advances in all these directions have done, and are still doing, much to elucidate the mysteries of wound healing. The words of Ambroise Pare are as true to-day as when he used them to conclude his case records, "I dressed him and God healed him." The greater our knowledge of the processes of wound healing, the greater the need for the surgeon to see the therapeutic implications of this knowledge. The patient must be made safe not only for the actual operation but also for the whole course of repair of his injury.

The subject may be approached by many ways, but all lead in the same direction. In war it is inevitable that the question of infection should be uppermost in our minds and therefore claim our first consideration. Infection is the greatest enemy of wound healing. But although we know that the elimination of bacterial infection is the first step in securing the healing of a wound, infected wounds do not always provide the best material for the study of those factors which promote healing. These wounds are such complicated battlegrounds between offence and defence, between destructive and reparative forces, that they make difficult any detailed analysis of the various elements in repair processes. On the other hand, the conclusions drawn from a study of experimental wounds in animals cannot always be directly applied to human beings. But in addition to these two types of wounds there are those produced by the surgeon in an attempt to rid the body of some disease process, and a study of these may go far to illuminate the processes of repair.

Much of the more recent work on wound healing has come from America, and much of it has been concerned with biochemical and nutritional aspects. At the same time much has been learnt from the failures of normal wound healing, from the cases in which for one reason or another the expected results have not been achieved. The conditions which lead to failure in wound healing may thus indirectly shed much light on the conditions which are the prerequisites of success. Whipple has recently reviewed the whole subject, especially from the standpoint of the lag or latent period in the development of healing. His work shows that wound healing is an orderly biological process which conforms in general to the laws of growth. In the individual wound the three most important processes are the amoeboid movement of cells, their mitotic proliferation, and the maturation of cells engaged in the fusion of wound surfaces. The local factors which determine the reparative processes depend upon the amount of the damaged tissue in the wound, the vascularity and the integrity of the blood supply to the damaged tissues, the nature of the exudate in the wound space and the adjacent tissues, the number and character of the local infecting organisms, and the number and character of the local foreign bodies to be encapsulated or extruded. But these purely local conditions are far from being the only ones of import. The more general factors include the age of the tissue (whether adolescent, adult, or senescent), the state of hydration, the state of nutrition (especially in relation to protein and to antecedent loss of weight), the vitamin balance, and the state of the general circulation and the blood. Orderly and normal wound healing is thus dependent upon a close interrelationship between local and systemic factors. Experiment has confirmed the view of experience that age is a factor of great significance. Experiment has demonstrated that departures from the normal electrolytic and protein balance delay the processes of healing. Vitamin C is now known

to play an important part in the formation of collagen fibres in wounds, and vitamin K in the production of prothrombin and so in the control of hæmorrhage. Circulatory efficiency and the state of the blood may determine not only the immediate outcome of the operation but also the speed of the repair processes.

The subject of wound healing may also be looked at in another way. What, for example, are the conditions that determine the breakdown or dehiscence of surgical wounds which might be expected to heal by first intention? Sokolov has reviewed and analysed the factors in the disruption of abdominal wounds in 732 cases. This phenomenon was twice as common in men as in women, and commoner in the first half of the year than in the second (this was thought to be due to a lowering of the vitamin C content of the tissues). It occurred especially between the ages of 40 and 60, in association with emaciation, in the presence of malignant disease, or in association with operations on the stomach or intestinal tract; and, finally, the wounds which broke down were usually not infected. The figures quoted by Sokolov revealed that wound disruption took place in 2 to 3% of all abdominal cases. Other workers have arrived at similar results and conclusions, and all have stressed the fact that failure of the healing process is most likely to arise in patients suffering from gastro-intestinal disease. In reporting the post-mortem findings in a series of cases after partial gastrectomy Payne stressed the fact that in a considerable proportion of these the fatal outcome was associated with an almost complete lack of response at the site of the anastomosis. Studley has also shown that the mortality in operations for peptic ulceration is directly proportional to the antecedent loss of weight. The general nutrition of the surgical patient is thus inseparably bound up not only with the immediate prognosis of the operation but also with the breakdown or dehiscence of the operative wounds, whether visceral or parietal.

During the recent years much work has been done on vitamin C in relation to the healing of wounds. Lauman and Ingalls demonstrated the importance of this in securing normal repair of experimental wounds in guinea-pigs, an observation that has now been confirmed and amplified in a recent paper by Hunt. These experimental studies show that collagen does not form in the healing wounds of guinea-pigs partially deficient in vitamin C: the proliferating mesodermal cells fail to mature, and the healing of the whole wound is disturbed. In a series of twenty-eight human cases investigated post mortem and reported by Hunt there was a comparable histological picture in eight, and in five there was disruption of the abdominal wound, and in three leakage at the gastro-intestinal suture line. It seems clear, therefore, that a subscorvy state leads to a gross disorganization of the whole orderly process of wound healing, and supports the view that the same deficiency may lead to failure of wound healing or even to wound disruption in human beings after surgical

operations. This work has recently been confirmed by Fox in a paper on experimental human scurvy.

The general nutritional state of a surgical patient is of course only one of the aspects of the problem of wound healing, but it is one which in this country has been largely neglected. Clinical and laboratory evidence, however, both show that in the presence of important nutritional defects the normal sequence of events in healing does not take place, that the precesses of repair may be seriously delayed or retarded, that the risks of complications from the non-healing of visceral surfaces are greatly increased, and that breakdown of a wound is likely to happen. And for every wound in which there is some serious departure from normal healing there are probably many more in which some minor disturbance occurs and may prolong the patient's stay in hospital and retard his ultimate recovery. Behind every wound there is the nutritional state of the individual patient against which all the reparative processes of Nature must operate. A nutritional disturbance may result from the patient's disease or be independent of it; it may be directly due to some gastro-intestinal lesion: or the lesion may reside in a background of poor wages, inadequate food, bad housing, or ignorance of personal hygiene. It is not difficult to remember that a surgical wound will not heal in the presence of a foreign body. But it is easy to overlook the fact that delays in healing, or even non-healing or disruption, may perhaps be due to years of poverty and malnutrition.

The surgeon, in fact, must look beyond the neat incision he makes in the patient's flesh to the social and economic position of the patient as a human being. He might well, too, look more closely into the question of hospital dietary. Tradition and the steward still so often combine to provide the patient with a diet that has little to recommend it apart from its caloric content. Every surgical patient presents an individual nutritional problem, and the help of the expert dietitian should be the rule and not the exception. A serious burn or a major abdominal or other operation is as much an acute nutritional problem. To heal the burn or to repair the damage after the surgical operation will require first-class building materials, without which local dressings and careful surgical technique will be of little avail.—*B. M. J.*

Acute Appendicitis in Hernial Sacs.—

By John Bruce, F.R.C.S. Ed., writes:—

The operation for hernia is very often common-place; yet every now and again an interesting situation arises or an unusual pathological event is discovered. The following two cases of acute appendicitis in the hernial sac, which occurred in the past year, are of interest as they gave rise to diagnostic difficulties.

CASE 1.—ACUTE APPENDICITIS IN A FEMORAL HERNIA.—A woman aged 35, attended hospital for a painful swelling at

the upper medial part of the right thigh. The pain had begun rather suddenly the previous day, and had been felt both in the groin and in the lower abdomen. She was sick twice after the pain and the power had been present for some hours. The swelling was first noticed the following morning, and was firm and very tender. Later, the skin became reddened, and the pain and tenderness increased appreciably.

On admission there was mild pyrexia and an increased pulse rate. The right hip was held in a position of slight flexion, and attempts to extend it caused considerable pain. The swelling was situated over the femoral canal, and there was a large area of redness around it. The skin was slightly oedematous, and the local temperature was increased. Palpation of the swelling revealed exquisite tenderness. There was no history of previous trouble, and no knowledge of a septic area on the thigh, leg, foot, or vulva. Acute inguinal adenitis was diagnosed, and rest in bed, combined with fomentations, was the initial treatment.

I saw the case on the following day, and was a little uncertain about the diagnosis. Instead of a simple incision being made into the inflamed area, therefore, a deliberate dissection was undertaken, and the swelling was demonstrated to be a femoral hernial sac. When this was opened, a quantity of very turbulent fluid was evacuated, and the only content found to be a gangrenous appendix. The base of the appendix was firmly held at the neck of the sac. In order to remove it the inguinal ligament had to be divided. Uneventful recovery followed.

Commentary.—The principal interest in the case lay in the diagnosis. The original diagnosis of a glandular inflammatory mass was reasonable enough, but looking back on the symptoms, they correspond very accurately with those of acute appendicitis, with the appendix in an unusual position. The acute onset, the early gastro-intestinal disturbances, the lower abdominal pain and the really extreme degree of tenderness were not in keeping with a diagnosis of acute adenitis, and it was these features which suggested the wisdom of a careful exploration of the swelling.

CASE 2.—ACUTE APPENDICITIS IN A SLIDING INGUINAL HERNIA.—A sergeant, aged 38, complained of acute pain in his groin at the site of a hernia which he had previously noticed. The pain radiated over the lower right quadrant of his abdomen, and was followed by sickness. The pain persisted and he was admitted to the hospital two days afterwards. The bowels had moved, and there had been no recurrence of the sickness, though the temperature and the pulse rate were both raised.

The hernia was quite apparent on examination. It was tender but not hard, and there were no obvious inflammatory signs in the overlying tissues. The bowel did not seem strangulated, but it was thought that a portion of omentum might have become imprisoned in the sac.

Operation was undertaken, and considerable difficulty was experienced in determining the anatomy of the swelling. A peritoneal sac was finally identified, and when it was opened, the cæcum and appendix were found in its posterior wall, which was in fact the peritonium covering these viscera. The appendix was acutely inflamed but not obstructed, and after being cut out of the sac, it was removed, and the stump invaginated. The cæcum was then pushed up into the abdomen, and a sound repair of the inguinal canal carried out.

Commentary.—This was a case of sliding hernia, which had been previously noticed, and the appendicular lesion was a somewhat unusual complication of the hernia. In this type of hernia, a viscus lying normally on the posterior abdominal wall, gravitates—or “slides”—gradually into the inguinal canal, dragging its covering peritonium after it in the form of a tube or sac. The usual cause is a general visceroptosis, and it must be borne in mind that the hernial sac is not a free one, as in the usual hernias, but has the protruded viscus—the cæcum in this case—its posterior wall. The only difficulty presented by the operation in this case was the identification of the hernia as a sliding one; the appendicectomy was rendered simple by the displacement and the free mobility of the cæcum.—*Clinical Journal.*

Proceedings of Associations

The Madras Medical College, Madras.

Major-General H. Stott, I.M.S., Surgeon-General with the Government of Madras, addressing a large meeting of medical men on Tuesday, 5th Aug. '41, at the Madras Medical College, advised them to volunteer for war service, thus helping in the war effort and incidentally benefiting themselves.

Before the end of this year, the Surgeon-General said, the Army would require not less than 1,000 additional doctors, Graduates and Licentiates, and 500 additional nurses. The response so far, he continued, had been certainly distinctly fair and in the Madras

Presidency alone, no fewer than 200 Graduates had received Commissions, a number larger than in any other province in India.

The Surgeon-General, proceeding, said that there were several points in favour of young men joining military service. The first was that they would be supporting the war effort. Secondly, as medical men, they would be providing succour and assistance to the afflicted, the wounded and the diseased. By doing war service they would be getting various advantages: for instance, they would get good food, live in healthy surroundings, have regular physical exercise, acquire discipline and gain wider experience of men and things. So far as the conditions

of service were concerned, they were satisfactory from every point of view. The question of granting certain additional concessions discussed at the recent Conference at Simla, which he attended, would shortly be announced. But those who liked to join military service need not wait till the announcement was made. They could accept the assurance that the terms were satisfactory and would be given retrospective effect. Those who entered service early would have the distinct advantage of seniority. One point which was particularly gratifying was that it had been decided to give gratuities on retirement to those who were not in the official medical profession, so that they might start private practice.

The Surgeon-General further said that there was a misconception in regard to the appointment of specialists. The number of appointments as Majors was limited; it was only about a hundred, and so various points would have to be considered in filling them, and one could not expect to get the appointment on the ground that he possessed the M. D. Degree or had had teaching experience.

Future of Entrants:—As regards the future of those entering war service now, the Surgeon-General said that all appointments for permanent posts in the Provincial Medical Service had now been stopped and the Government were at present only making temporary appointments and after the war every single one of those holding these temporary posts would be discharged and these appointments would be given to men who returned from war service. He hoped that municipalities and local bodies would also encourage young doctors to go to war service and be prepared to grant them on their return similar conditions. One or two other concessions like the grant of educational facilities for children of those joining military service and grant of study leave, were also under consideration.

In conclusion, the Surgeon-General said that what he required was the type of graduates and licentiates who were ambitious to serve and better their prospects and anxious to gain experience. He wanted particularly members of the House Staff, honorary and paid; and House Surgeons who were willing to join military service, now getting only an allowance of Rs. 25 a month, would get a starting salary of Rs. 450 besides other amenities. In regard to final year students, a proposal was under consideration to advance the date of their examination by three months and those who were willing to join the military service on passing their examination could go through the preliminary stages of enlistment. The matter was under correspondence with the University and a decision would be announced shortly. Other students could join the St. John's Ambulance Brigade and the Red Cross.

Rao Bahadur Dr. A. Lakshmanaswami Mudaliar, in proposing the vote of thanks, said that the question of advancing the date of the examination for final year students was now under correspondence with the

University and a decision would soon be announced.—*The Hindu*.

The Kistna District Branch of the Indian Medical Association, Masulipatam.

A monthly meeting was held on Sunday, the 27th July 1941. Dr. C. B. Ethirajulu, M.B.B.S., Asst. Dist. Medical Officer, Kistna, occupying the Chair, owing to the absence of the President and Vice-President.

Among other businesses transacted, it was resolved that all the members of the Kistna Dt. Branch of the Indian Medical Association shall constitute the reception Committee for the ensuing Andhra Provincial Conference to be held at Masulipatam, during Dasara and members were elected to be the office-bearers of the same. At the Clinical Meeting which followed:—

1. Dr. P. Seshagiri Rao, L.M.P. Gudivada, demonstrated an interesting case of a boy of about 8 years with the right hip joint in a flexed position and which could not be extended even under anaesthesia, and discussed the differential diagnosis.

2. Dr. J. Venkataramasarma, M.B.B.S., and L. O., Masulipatam, demonstrated a rare case of "Spring Catarrh" and explained in detail about the treatment of the case.

3. Dr. A. Narayana Rao, M.B.B.S., Gudivada, demonstrated two cases of (1) Ulcer Tongue (2) Multiple Gummata in Muscle which yielded to treatment with injections of Neosalvarsan and administration of Potassium Iodide. He also demonstrated a case of Gumma Sternoclavicular joint.

4. Dr. K. V. Subba Rao, L.M. & S., Masulipatam, demonstrated a case of Perinephric Abscess in a child aged about 2 years and discussed the differential diagnosis of the case.

The President in his concluding remarks, summarised the salient points of the above papers and clinical cases read. The meeting was concluded at 7 p.m. with a vote of thanks by the joint Secretary.

At a meeting of the above association held at Masulipatam, on 28-6-41, Dr. N. Rama Rau, L.M.P., spoke on Patent Medicines as follows:—

Prescribing a patent to a patient is one of the easiest means of dispensing with patients and a time-saving remedy too: It indeed relieves a lot of worry and waste of time that is generally involved in writing out a prescription and balancing it properly. Now-a-days, this practice is resorted to by many and it is growing in direct proportion to the daily increasing number of patents that are flooding the medical field; and there seems to be no end to the list of manufacturers or their medicines. The moment they are manufactured and bottled, they automatically find their places on the doctor's table by way of advertisements, blotters, literature etc. besides free samples for clinical trial.

Owing to the enormous increase of these patents to-day, compared to the very few that were existing some decades ago,

consistent with the extremely rapid strides with which medical science is progressing from day to day, one can easily imagine how the present-day doctor is confronted with the problem of keeping himself abreast of the rapidly advancing medical science when compared to the little or no worry on the part of the doctor of olden days.

Now, it has become almost a fashion with many to treat their patients with only patents and nothing else, though there are reasonable number that do not advocate their use as much as others. It is very doubtful if there is at least one member of our profession, especially from among the old and grey-haired with vast and ripe experiences, that could manage without taking recourse to the use of proprietary drug during his practice. Even such people could never have managed without prescribing at least the old time-honoured medicines like Kaufmann's Sulphur Bitters, Hewlett's mixture etc.

Let us put to ourselves one pertinent question: "Is it an absolute necessity? How far is it essential that we should have some knowledge, if not a thorough and complete one, regarding the proprietary medicines?" My humble reply to this would be in the affirmative. Some unforeseen circumstances which are not rare in our daily practice, may force us to prescribe and treat our patients with mere patents. You may be called to a distant and out-of-the-way place for consultation, when you feel you will not be justified in writing out a long list of medicines from our British Pharmacopœa to be got compounded from a chemist and that too repeatedly, especially when you know that there are no chemists close by. A single patent is sure to solve the problem; A rich and well-to-do person may insist on a pleasant tonic to his tastes and you naturally would be losing his confidence in you if you do not do so, or you may come across a queer and delicate lady patient, who can never be convinced to swallow your bitter mixtures unless they be sweet and palatable syrups. Further, it may often happen that some one may show you a prescription containing the name of a patent, which is either illegibly written or which you have never come across and ask your opinion as to the advisability or otherwise of taking it. Any delay and hesitation on your part in expressing your view, is sure to make your position awkward and gives the patient an easy scope for a hasty conclusion that you are not as up-to-date as the other. These are some of the peculiar and probable circumstances which really may necessitate us to keep at least an average knowledge of these patents. Besides, some members of our profession who have only consulting practice have only to depend on their use in majority of cases. Similar is the case with those holding high positions in service who have only a limited time at their disposal for private practice and an extremely busy general practitioner may find in this an easy means of obliging as many patients as possible.

Coming next to the study and the knowledge one should possess about these

proprietary drugs, it is as essential as it is difficult. It involves a special study as it were, containing several thousands of names exhausting the whole alphabet from A to Z, with their various ingredients, therapeutic uses besides the vast literature behind them. It has happened on more occasions than one, that when I was asked to prescribe a patent medicine, I could describe all the details about packing of the bottle, the contents, its taste and other characteristics, except the very name itself and I had no other go to glide over that awkward moment and to cover my failing memory except by convincing the patient that I could get the correct address, its present cost, and all other details the next day, as I was busy that particular day, or by saying that I could bestow my thought leisurely over his complaint and prescribe a nice medicine to suit his pocket. The difficulty and confusion experienced in mastering this subject is easily comparable to that we once upon a time bothered ourselves with—the study of Anatomy in our student career. Sir, the names of the patents are too many to remember in the first place, secondly some are peculiar and curiously named, so much so, they are hard to remember and harder to pronounce, while others are jarring to the ears. There is much irregularity and indiscrimination in the nomenclature of these medicines by the manufacturers, who have the sole right in this respect and so appear to faithfully follow the rule of permutations and combinations of the entire alphabet. One can understand easily and remember them without much strain on our brains if their names are consistent either with the ingredients they are composed of, or with the therapeutic purpose they are intended for, or their action on the human system, or the disease in which they are supposed to be indicated.

For instance—Heporal, Hepatex—Hepanol—Leveron—Livedex—Hepaxol—Hepatosan etc. clearly indicate that their main ingredient is Liver Extract. Kurchisol—Kurchibine—Kurchibide—Kurchinetum etc. indicate Kurchi as their ingredient.

Ergimine—Ergotine—Ergotoxin, Pituitrin, Pitressin, Pituitobin—Hydeotin Creasotone, Stannoxyd—Tinoxyd etc. are also self explanatory.

Hypotensyl—Anaesthesin—Analgesin—Amæbiarson—Spasmodyl etc. denote their actions on the system, while,

Thisin, Thisol, Pertussin, Otalgin, Dysmeno, Nephritin, Rheumin—Trachomin Catractol, Opacitin etc. definitely show their relationship with the diseases they are used for. In some, the name of the drug along with the name of the disease is combined like Iodobesin—Amebetin—Rheumatoidin.

In a way, the following names are not very difficult to remember though they are not as clear-cut as the above—Codopyrin—Colitique—Stipticin—Mammogen—Eumensin—Eumenol—Ovacoids—Testacoids—and Emetin.—Rhythmin (to maintain the regular action of bowels). Tamolon—Mascolon—Juvenol—Respirogen (Asthma Vaccine and Pepton.) Fevrol and Alfever, (both indicated in all varieties of fevers).

As already said, some names do not properly sound well to the ears or are difficult to pronounce besides involving the difficulty as to their relationship with their ingredients or therapeutic uses or the diseases they are used for—Cafatron—Thisotron—Barbatron—Tophotron—Tussispect—Uroclassio—Sanostol—Aktisol—Neoket—Ammoket—Utrostip etc.

Again, a few drugs are actually misleading in their very names. For instance:—

1. Ergol which apparently denotes some Ergot preparation actually is a preparation of supra renal extract.

2. Genelin has nothing to do with genital organs or genital diseases but is a stomach extract.

3. Bivatolis not a vitamin drug but an injectable Bismuth.

4. Gonodol is liable to be linked with similar pronouncing names like Gonokin—Gonocure—Gonoson—Gonosol, Gonorgin, all clearly indicating their use in gonorrhoea but, it is an aphrodisiac. Similar is the case with Gonon which is a gonadotropic hormone of urine of pregnant women.

5. Vibrona does not contain any vibernum but is a tonic wine.

6. Examen is likely to be mistaken for an eczema remedy but is a liver extract.

7. Calsod contains Sodium Acetate and Sodium Chloride but not a Calcium preparation.

8. While Cafiaspirin—Enaspirin—Genaspirin all mean Aspirin content in them, Alasphin is Neo-Arsphenamine.

9. Leucotropine has nothing to do with leucoma or Atropine but it is an injectable product for rheumatism.

10. Hexeton is a non-Hexamine product but is a cardiac stimulant.

11. One can only understand oenta as a galactagogue if he remembers that its chief ingredient is placenta.

12. Heparon is not a liver extract but is a cholagogue containing as it were Am. Chloride, Taraxicum—Cascara, Podophyll etc.,

13. Similarly, Hepatone is a cholagogue. Let me mention few more names from which one can make neither head nor tail as they bear no relation to anything. For instance, just imagine how the name 'Unden' has anything to do with follicular hormone and 'Oubain' to do with strophanthin, Sang-cre with liver extract or 'pabutole' with any of the many ingredients it is said to be composed of. The drug named 'Pen' consists of Salol—Urotropine, Sandalwood oil—Bromo camphor and is indicated in gleet. Orisol—Erwisol—Enisol are similarly confusing.

Still more complicating and confusing, though a little interesting, are the following which are almost unparalleled either in their pronunciation or spelling and belonging to different companies and possessing different therapeutic properties.

1. Betaxin may easily be confused with another similar name Beetoxis, the former as is familiar to one and all is a vit. B. product, the latter for rheumatic conditions. (2) Lixen

is a snake-bite remedy and Lixen is an elixir containing senna. (3) Lixol is for asthma and Laxol is a laxative. (4) Eumenol and Luminol have of course both sedative properties—one on the uterus and the other on the general system respectively. (5) Phyllo-calcine is a calcium product and Phenocalcine is an antipyretic. (6) Droserin is a whooping cough remedy while Drosylt is for high blood pressure. (7) Regula is a regulator of menses and Regulal regulates bowels. (8) Tyeol is a gargle and is liable to be confused with Thioeol. (9) Arcanol contains Atophan, Aspirin etc. while Arganol is a silver preparation for gonorrhoea. (10) Uleron is a chemotherapeutic product while Uleron contains adrenal cortex. (11) Unden is a follicular hormone while Undin is an eye washing instrument. (12) Virilin and Viriline are different drugs belonging to different manufacturers, but their ingredients differ though their indications are the same. (13) Robeline contains Strychnine, Quinine and Phosphorus, while Robeleine contains vit. A. B. C. and bone marrow, egg yolk and Lecithin. (14) Lobulina is for diabetes and Lobiline is Lobelia. (15) Opacitin is an eye ointment for corneal opacities whereas Opacin is an intravenous injection for x-ray visualisation of gall bladder (16) Mandecol is Calcium Mandelate while Mendico is for asthma and bronchitis. (17) Calsoid of M and J is for rheumatic arthritis and Calsod is an intravenous injection containing Sod-Acetate and Sodium Chloride. (18) Metagen of P. D. and Co., a vitamine product, is easily confused with Metazin of C. D. C. for rheumatism. (19) Esmodyl produces spasm while Spasmodyl and Spasmyl are antispasmodics. (20) Similarly, Fatalin and Obesin of C. D. C. are confusing as to which adds fat and which deprives fat. (21) Jeculin is a liver Extract and Jecocin is cod-liver oil. (22) Tensolin to relieve tension in high blood pressure is likely to be mistaken for a probable similar name Tonsolin which may, tomorrow, be the name of a drug which reduces tonsils. (23) Hepatogen is liver extract and Hepatagen is a cholagogue. (24) Heparol is liver extract and Hepol is codliveroil.

Friends, I have made an humble attempt to narrate only those instances that I could come across and many more might have similarly attracted your attention. I do not mean to condemn or deprecate the use of patents at all, as I think one cannot get on without them as already stated. All the same, one should admit that there is always the possibility of this sort of confusion prevailing for which there seems to be no remedy as matters stand at present because there is not any hard and fast rule to prevent these manufacturers from their choice of naming the medicines. As newer drugs come into the field day by day, this confusing factor also multiplies. However, the manufacturers make their living solely depending on our profession and I leave the problem to be solved by the medical profession at large.

Preparations and Inventions

"Opacitine" (Regd.)—Manufactured by S. Krishna Clinical Research Laboratories, Pasrur (Punjab), is composed of Phenyl-dimethyl-isopropyl, Urea Sulphazide, Dimethyl-acetyl dehide, pure honey and an alkaloid extract of a cornus. (Formula:— $C_{14}H_{24}O_3COODHa.$) and is indicated in any kind of Corneal Opacity (a Nebula, a Macula or even a Leucoma) of any size, shape or duration whatsoever. It is priced: Large Phial (Complete Course) Rs. 10/- Medium Phial: Rs. 5/- Small Phial: Rs. 2-8.

Sarpagandha.—Prepared by S. S. A. Pharmacy, Mangalore.—This preparation which is issued in the form of tablets has Rauwolfia Serpentina as its chief ingredient. From time immemorial this herb which is familiarly and popularly known as "Pagal-ka-Dawa", has the reputation of being a specific for the cure of insanity. Mahamahopadhyaya Kaviraj Dr. Gananath Sen Saraswathi and Dr. Kartick Chander Bose jointly reported favourably on the successful use of Rauwolfia Serpentina in case of high blood pressure. Their report was based on the results of researches made on modern scientific methods and appeared in the columns of Indian Medical World, July 1931. This was quickly followed by the result of researches made and published by Bt. Col. R. N. Chopra director, and others of the School of Tropical Medicine, Calcutta, with a detailed account

of the pharmacology of Rauwolfia Serpentina confirming its properties to reduce, blood pressure. But, the manufacturer's experiments and the careful watch over the results obtained, have shown that the single drug did not show prolonged effectivity and had bad after effects.

Their experiments of the herb showed that the addition of certain other drugs, issued now by them as Sarpagandha, increased its efficacy and gave more lasting benefits and has a curative effect upon insanity and the allied diseases, and also in neurasthenia, hysteria, insomnia, epilepsy, after-pain of delivery, dysmenorrhoea &c. It is issued in bottles of 50 tablets @ Rs. 3-8-0.

Galleno's Throat-Tabs—Manufactured by Galleno Pharmaceutical Works, Manufacturing Chemists, Succinol House, P.O. & T.O. Waini, Rly. St. Pusa Road, B.N.W. Ry., Dist. Darbhanga, Bihar, India.

These tablets are mainly composed of Mal. Acid, Benzoic Acid Menthol, Eucalyptus Saccharin and Iodine (a trace) combined in a neutral Glucoside base; and found to be useful in inflammation of the mucous membrane of mouth, nose and throat and especially cold coryza, cough, tonsillitis, urilitis, pharyngitis, bronchitis and rhinitis etc. These tablets are issued in phials each containing 35 tabs.—the price being Re. 1/- per phial.

Correspondence

The Indian Chemical Manufacturers' Association

The Indian Chemical Manufacturers' Association has sent the following Communiqués for publication.

COMMUNIQUE 1.—Apropos Press Reports regarding the Government of India's scheme to place orders for three years' stock of medical supplies from England on behalf of Provincial Governments, the Indian Chemical Manufacturers' Association has been in correspondence with the Government of India and the Provincial Governments in the course of which the Association requested for the list of items for which orders were placed in England. The Governments were also urged not to place contracts for so long a period abroad as that would adversely affect the Indigenous Industry and would take away the incentive for production of new items. While the Government of India in reply gave an assurance that they would be always prepared to purchase in India the medical supplies of Indian manufacture which were up to the prescribed standard, they refused to supply to the Association the list of stores ordered from England and stated that the list covered items not available in India.

The Association points out in a further communication that the information about

the items ordered is essential as even if the list consists of items not available in this country according to the Government, that would enable the Association to explore the possibilities of manufacture and extend the production of many drugs, which would not only benefit the Industry but also the Government and the country as a whole by making the supplies available locally and dispensing with the necessity of having to obtain them from abroad. As regards the list of imported drugs with peacetine consumption supplied to the Association, it is pointed out that the Government requirements having increased by more than hundred per cent. and many more items having been added in the requirements, the peacetine consumption figures would offer little assistance in planning production. The Association suggests that a stage has been reached when instead of repeating assurances in general terms the Government should consider with the Industry individual items and find out the possibilities of production in this country. As regards the contention of the Government that it is not possible for them to place orders against indigenous supplies which may not materialise, the Association points out that under the present circumstances there are more chances of supplies from England not materialising and Indian manufacturers may actually be in a

better position to supply whatever items they undertake to produce.

The Chemical Association understands that most of the Provincial Governments including the U.P., Assam and Sind, have agreed to participate in the scheme envisaged by the Government of India.

COMMUNIQUE 2:—The necessity of reducing railway freight and the classification of raw drugs has been stressed by the Indian Chemical Manufacturers' Association in course of a communication addressed to the Railway Board. This matter has been under correspondence between the Association and the Railway Board for the last two years. In reply to previous representation the Railway Board had regretted their inability to make a general reduction in the classified rates for drugs and medicines, although they desired the Association to approach individual railways for quoting special rates for particular items of raw drugs and medicines, in which the rates were considered very high. The Association points out that pharmaceutical concerns being located at different places in India and important raw drugs having to be brought from distant stations in the Himalayan Ranges, it is essential in cases of almost all the raw drugs to change the classification and thus a general reduction in the rates is required. Moreover, approaching different railways separately, the Association further states, for each item giving figures of traffic etc. would be an almost impossible task and it would only mean shelving the issue.

As regards medicines, the Association points out that rates for liquid and light powdered medicines, 90 per cent of which are used in hospitals and dispensaries located all over India, are particularly high as 65 per cent of the weight charged as medicine is only packing material and they involve long distance transport. The Association strongly feels that there was little justification in the Railway Board disregarding the recommendation of the Railway Rates Advisory Committee for reduction in rates of medicines and for quoting telescopic scale of rates for medicines.

Notice

The Sixth Maharashtra and Karnatak Provincial Medical Conference will be held at Poona, under the auspices of the Poona Branch of the Indian Medical Association on the 13th, 14th and 15th October, this year. The main subject for discussion is "Blood-Pressure in Health and Disease". Papers on

DR. SACHINDRANATH ROY, M.B. Gopalpur, Mymensingh, writes as follows:—

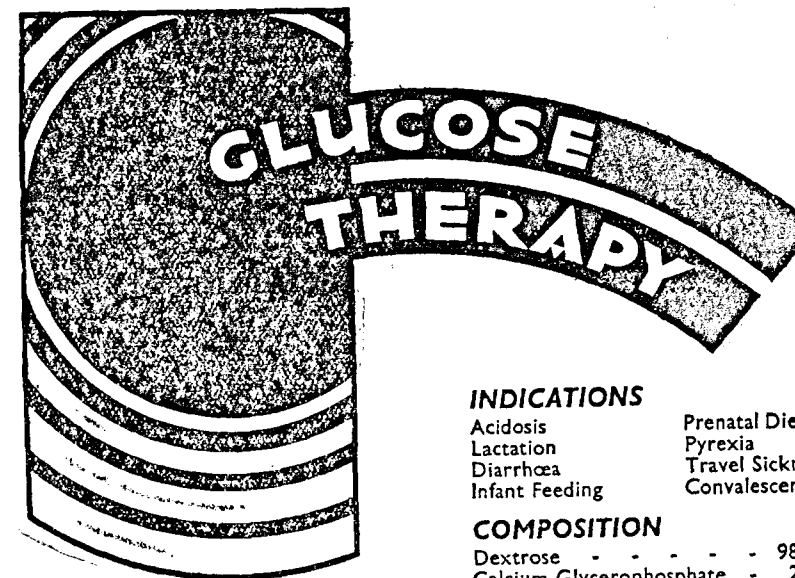
I was consulted for a case of chronic endometritis with daily rise of temperature up to 100°F. in an emaciated H. F. of about 25 years of age. I prescribed Lacto Protein B. I. 5 to 10 c.c. to be given intramuscularly every 4th day. But as the patient was much emaciated and the attending Physician thought that the I.M. injection would cause severe pain and abscess, he at his own responsibility gave the first injection of Lacto Protein 5 c.c. intravenously. There was no reaction of any type and curious it was that although during the last one month she was never afebrile for a single day, there was no rise of temperature on this day. But about 36 hours after the injection, the patient complained of severe and unbearable pain in the uterus which lasted for about three hours. The attending physician consulted me again on the next day when to my utter astonishment I heard that the injection was given intravenously and there was no reaction of any kind. I warned him for his action and subsequent injections were given intramuscularly. From the date of the pain in the uterus, the usual daily rise of temperature again became prominent.

I send this case note because I do not know if milk has ever been tried intravenously. I solicit criticisms and opinions about the intravenous injection of milk.

DR. K. K. CHATTERJEE, L.M.F., M.O., Garh Lakhanpur, Surguja State, E. S. A.—

A man was stung by a wild bee on his lower lip and a few minutes later he became restless with blood shot eyes and gradually became unconscious. At this moment I gave him a few whips of chloroform, an experiment I had read in an article in the Indian Medical Gazette, that was tried to eradicate the aftereffect of scorpion bite. To my utter surprise the patient regained his senses, became quiet and complained of no pain even due to the bite. Though to me this is a rare case, may I ask the brother medicos to communicate through your esteemed journal other such novel and interesting cases cured completely by the aforesaid course of treatment?

other Medical Subjects will also be read and discussed. An exhibition of medical products, surgical appliances and interesting specimens is being arranged. All medical practitioners in the Province are cordially invited to attend the Conference.



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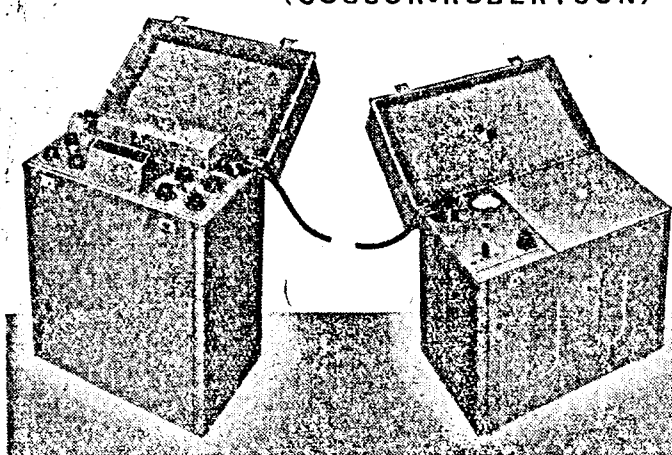
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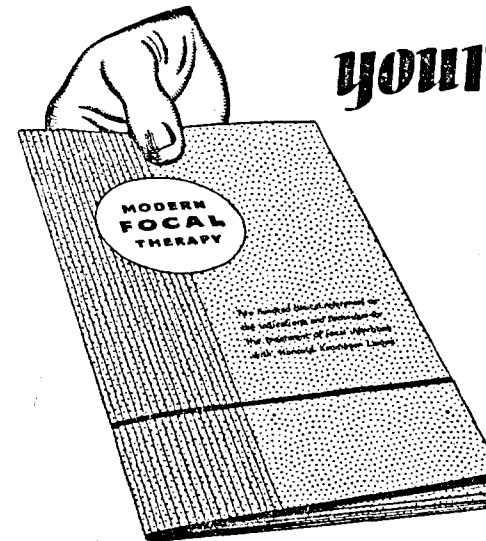
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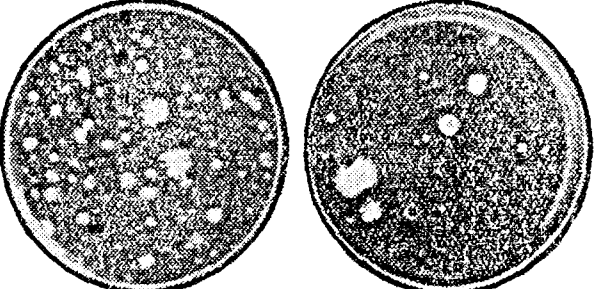
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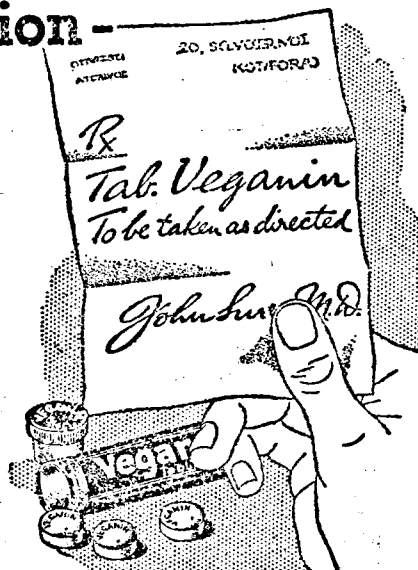
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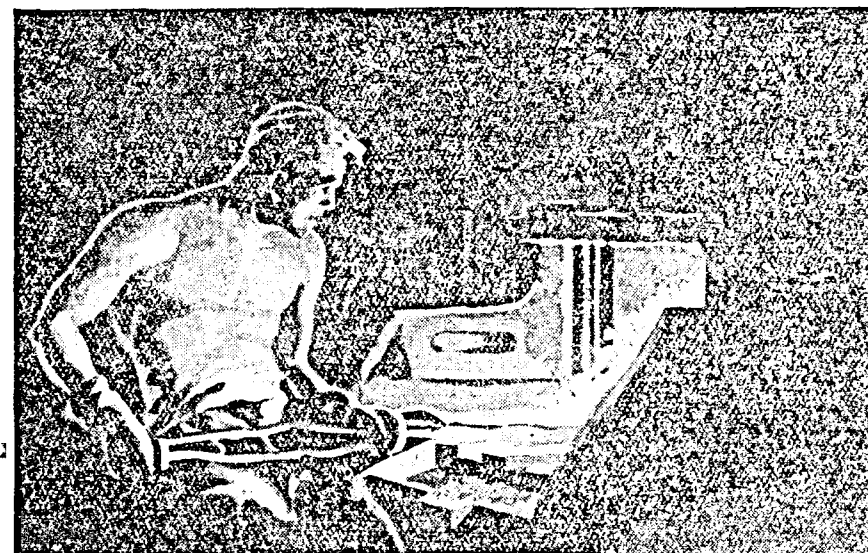
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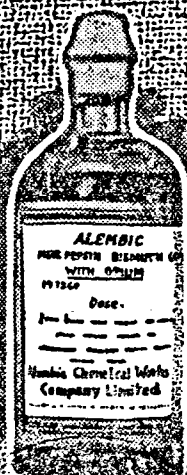
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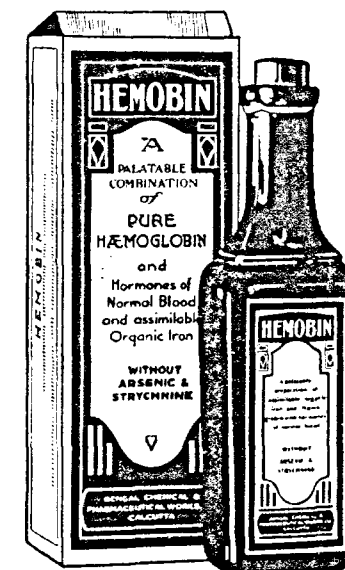
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