

The Antiseptic

Vol-43

NO-4-

APR 1 1946

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LM24, NO4AN
N46.43.4.
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The Antiseptic

Estd. 1904

A Monthly Journal of Medicine & Surgery

Published on the 18th of every Month

Edited by: U. Rama Rau, & U. Krishna Rau, M.B., B.S.

Editorial & Publishing Offices: 328-24, Thambu Chetty St., G.T., Madras.

London Office: 34-40, Ludgate Hill, E. C. 4.

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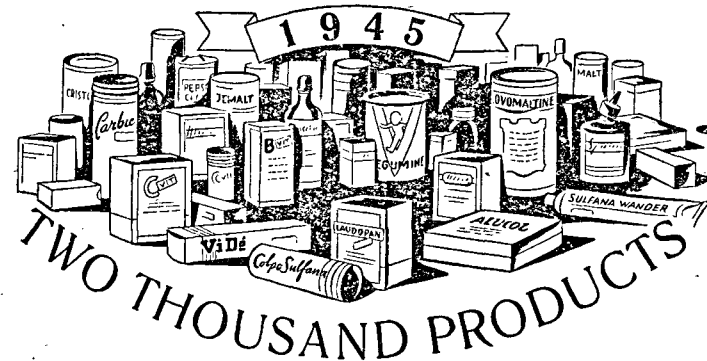
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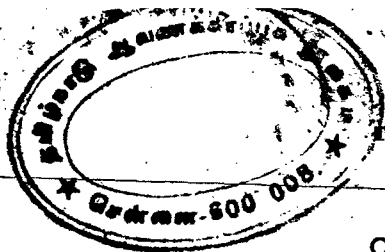
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CONTENTS

ORIGINAL ARTICLES:

	PAGE		PAGE
Filariasis in Bundelkhand.—Major Khan Bahadur Ahmed Baksh, O.B.E., I.S.O., Nowgong, C.I. ...	225	Penicillin in Chronic Arthritis Knee.—K. D. Lahiri, M.B., B.S., M.O., Bihar. ...	277
Teratoma of Testis.—D. B. Desai, M.B., B.S., R.M.O., Rampura, Surat. ...	229	Ascariasis.—Dr. M. S. Kundu, L.M.F., Gourganj, Dacca. ...	278
Nervous Complications of Diphtheria.—P. N. Laha, M.D., Medical College, Agra. ...	232	Stanno Sulphazide and Chaulmoogra Oil in Chronic Skin Ulcerations.—P. R. Chibber, L.S.M.F. & L.O., M.O., Ammunition Depot, Nowshera Cantonment, N.W.F.P. ...	279
Infantile Hepatic Cirrhosis. A. V. S. Sarma, M.B., B.S., (Mad.), D.C.H., R.C.P. & S. (Eng.), Madras.—(Concluded) ...	233	EDITORIAL:	
Auto-Intoxication.—Vallabhdas N. Mehta, L.M. & S., Morvi. ...	273	The Pharmacy Bill. ...	281
CASES and COMMENTS:		GLEANINGS from MEDICAL PRESS:	
A Case of Maggots in the Nasal Cavity. Bon Behari Saha Dalal, M.B. (Cal.), M.O., Chandanbaisa, Bogra, (Bengal). ...	276	Medicine & Therapeutics ...	284
Efficacy of Nicotinic Acid in Ear Troubles. D. N. Debsarma, A.M.O., Assam. ...	276	Surgery ...	293
		Obstetrics & Gynaecology ...	298
		Ophthalmology ...	299
		Book Reviews ...	299
		Correspondence ...	300
		Indian Nurses for Training Abroad. ...	300



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INDEX TO ADVERTISERS

	PAGE		PAGE
Albert David Ltd.	... 40	Jai Singh & Co., Dr.	... 71
Alembic Chemical Works Co., Ltd.	49, 57	James Wright Ltd.	... 53
Alkaloids Research Laboratories Ltd.	... 54	Jammi Venkataramanayya & Sons	... 46
Allarsin Pharmaceuticals (India)	... 65	John Wyeth & Brother Ltd.	42, 43, 61
Allied Drug Industries Ltd.	... 26	Jubilee Trading Company	... 2
Angier Chemical Co., Ltd.	... 13	Khatau Makanji Spinning & Weaving Co. Ltd.	17
Anglo-French Drug Co., (Estn.) Ltd.	... 19	Kothari Book Depot	... 55
Antool & Co. Ltd., S.	... 70	Kulkarni's Laboratories, Dr.	... 29
Aryan Pharmaceutical Works Ltd.	... 70	Lister Antiseptics	... 73
Associated Drugs & Chemical Concern Ltd.	25	Liverites Co.	... 37
Bengal Chemical & Pharmaceutical Works		Maharashtra Chemicals Ltd.	... 52
Inside of Back Cover		Mandoss & Co., Ltd.	... 67
Bengal Immunity Co., Ltd.	... 45	Marmite Food Extract Co. Ltd.	... 10
Bengal Modern Drug House Ltd.	... 69	May & Baker (India) Ltd. Front Cover & 3,	32
Bhattacharyya & Co. S.	... 47	Meelec Nutriment & Pharmaceuticals Ltd.	16
Biddle Sawyer & Co., (India) Ltd.	... 59	Medical Research Laboratory	... 53
Birla Laboratories	... 72	Medical Supply Concern Ltd.	... 47
Bombay Surgico Medical Agency	... 58	Metal Box Co. of India Ltd.	... 74
Boots Pure Drug Co., Ltd.	... 44	Morison Son & Jones, J. L. (India) Ltd.	... 52
British Drug Houses Ltd.	... 39	Mukerji & Co., Ltd., H.	... 55
Brother, B. A. &	... 47	Navaratna Pharmaceutical Labs.	... 30
Brothers, V. M.	... 51	New Surgical Trading Co., The	... 50
Burroughs Wellcome & Co.	... 31	Nonglay Modern Virtues	... 70
Butterworth & Co. (India) Ltd.	... 60	Oriental Research & Chemical Lab. Ltd.	12
Calcutta Chemical Co. Ltd.	... 54	Organon Laboratories	... 23
Calcutta Clinical Research	... 46	Orthopaedic Appliances Stores	... 11
Chemo-Therapeutics (India) Ltd	... 12	Parekh Trading Co.	56, 60
Ciba (India) Ltd.	... 27	Parke Davis & Co. Outside of Back Cover	
Cipla Laboratories	... 33	Pearl Chemical Industries	... 69
Continental Drug Co.	... 14	Pharmaceutical Laboratories of India Ltd.	8
Corn Products Co., (India) Ltd.	... 21	Phonix Drug House	... 46
Cossor & Son, A. C., Ltd.	... 11	Polyclinical Laboratory	... 63
Das Engineering Co.	... 48	Premier Drug Co.	... 69
Dhur & Sons, Ltd., U.N.	... 69	Primeo Ltd.	... 48
Deodhar Bros.	14, 54	Purohit's Pharmacy, Dr.	... 16
Eastend Laboratories Ltd.	... 52	Ranbaxy & Co.	... 56
Eli Lilly & Co. Inside of Front Cover		Roy & Co., S.C.	... 52
Fairdeal Corporation Ltd.	... 9	Scientific Indian Glass Co., Ltd.	... 51
Glaxo Laboratories Ltd.	... 34	Shree Shakti Syringe Works	... 68
Globe Research Institute Ltd.	... 66	Smith Stanistreet & Co. Ltd.	9, 76
Gluconate Ltd.	... 66	Squibb & Sons, E. R.	36, 41
Godrej Soaps Ltd.	... 50	Standard Pharmaceutical Works Ltd.	... 67
Gracely Drug Co., S.A.	... 48	Sterling Agency House	... 68
Hanovia Ltd.	... 6	Tablets Limited	... 64
Hering & Kent	... 15	Travancore Sugars & Chemicals Ltd.	... 10
Hewlet & Son, Ltd., C. J.	... 22	Union Drug Co., Ltd. Front Cover &	62
Himalaya Drug Co.	... 55	Unique Trading Corporation	... 48
Hind Agencies	... 51	Unique Drug House Ltd.	... 63
Herbal Healing Co.	... 73	Universal Drug House	... 65
Horlicks Malted Milk Co.	... 75	Victory Chemical & Pharma. Works Ltd.	68
Imperial Chemical Industries (India) Ltd.	18	Victor X-Ray Corporation (India) Ltd.	... 35
Indian Surgical Industries	... 73	Volkart Bros.	... 38
Indian Health Institute & Lab. Ltd.	... 55	Wander (India) Ltd., Dr. A.	... 1, 5
Indian Scientific Trading Corporation	... 46	William R. Warner & Co., Ltd.	13, 24
India Syringe Works	... 15	Zandu Pharmaceutical Works Ltd.	... 20
Indo-Continental Trading Co.	... 51	Zill & Co.	... 7
Indo-Pharma Phar. Works	28, 37	Zone Chemical Co.	... 48



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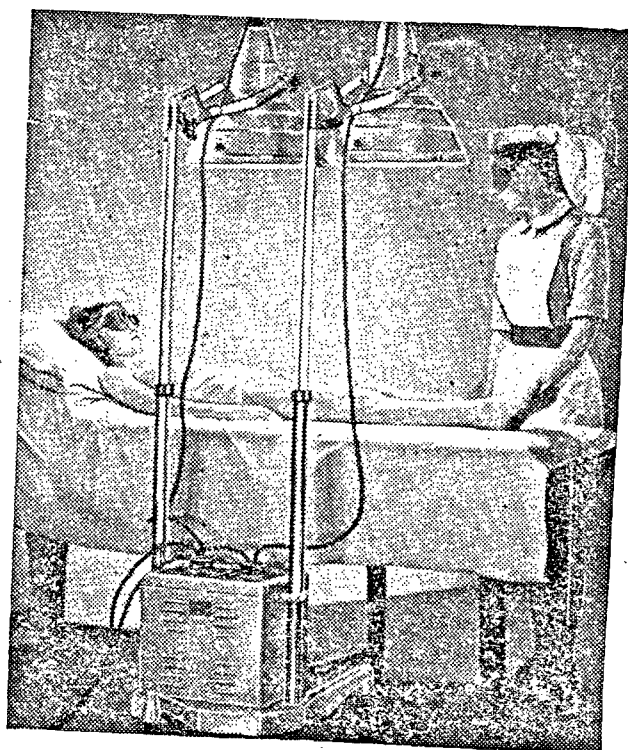


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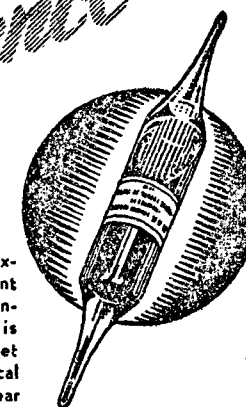
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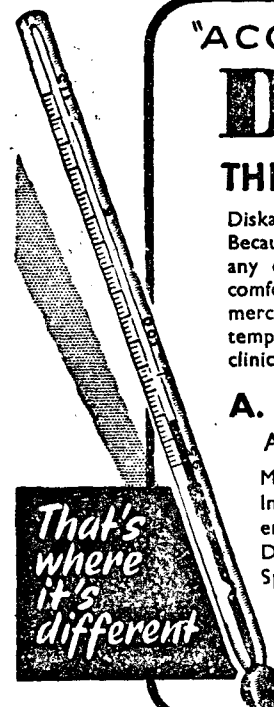
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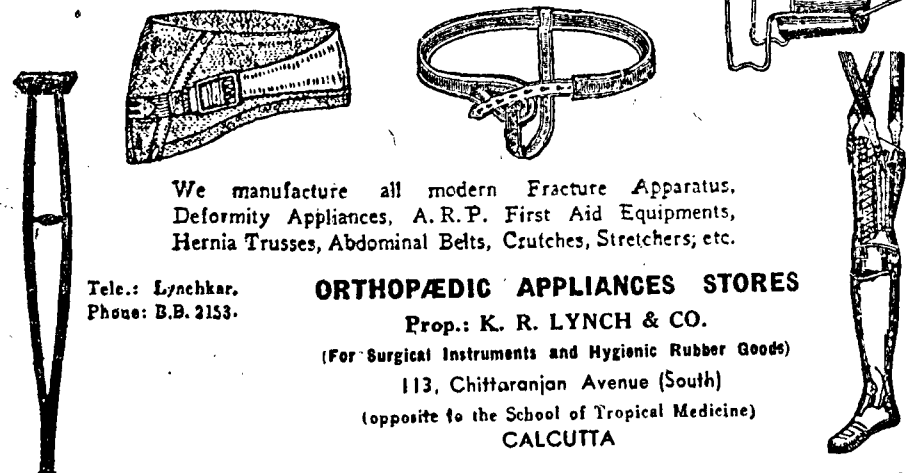
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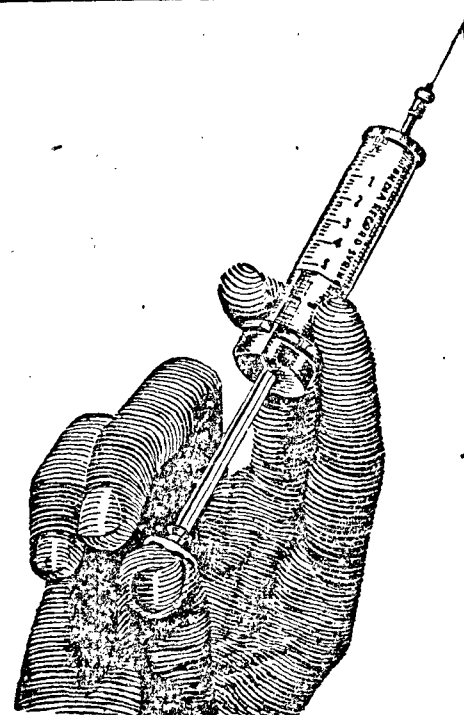
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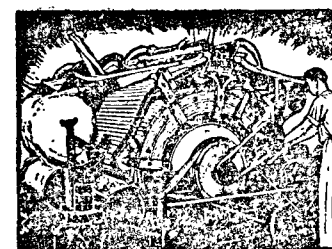
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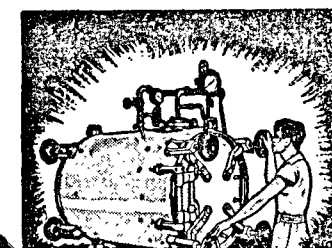
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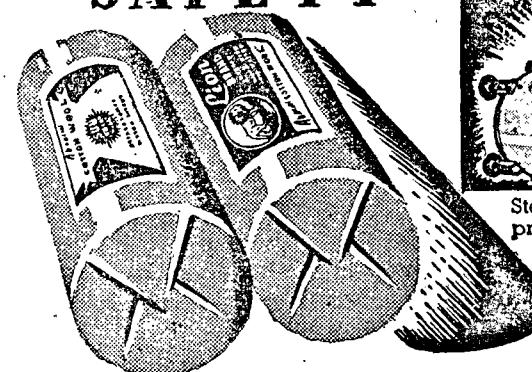
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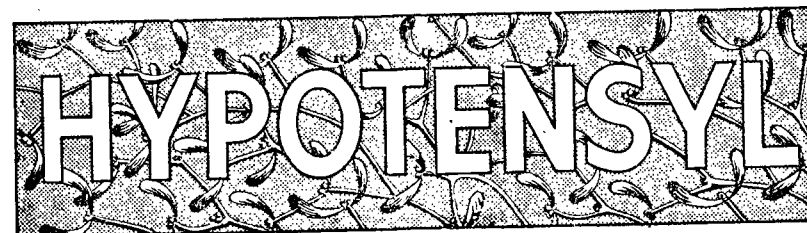
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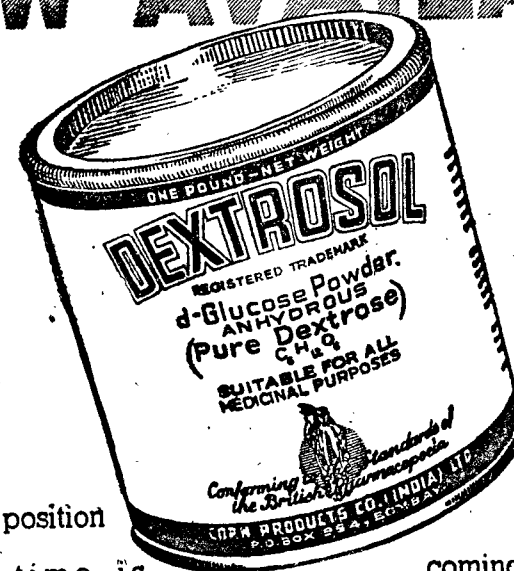
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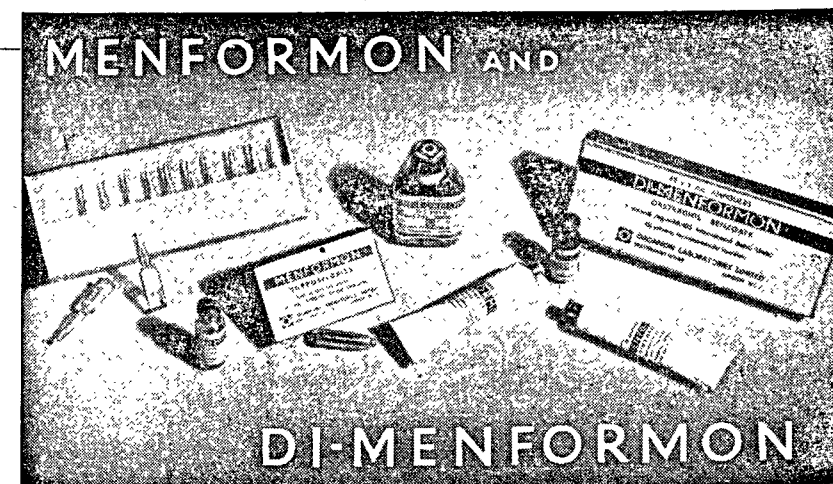
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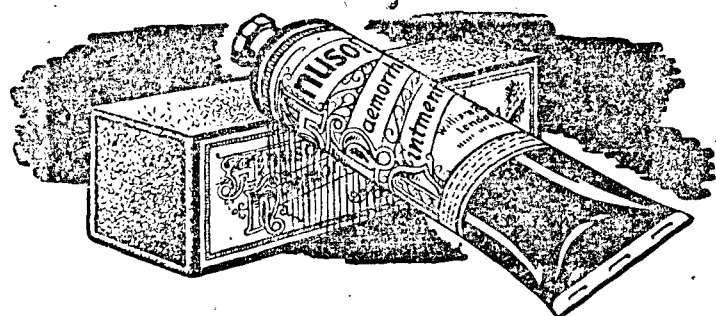
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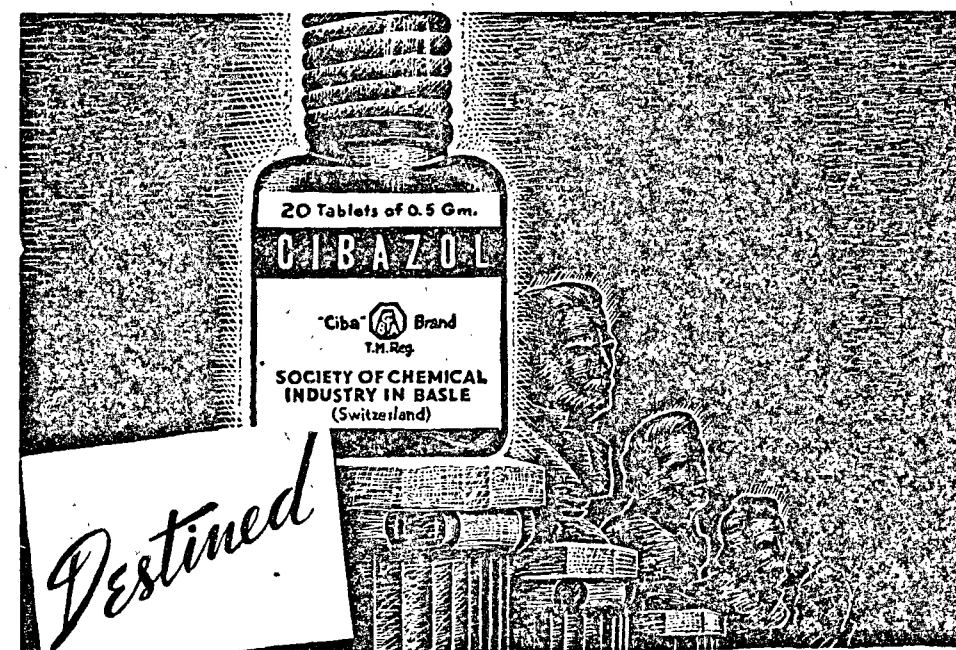
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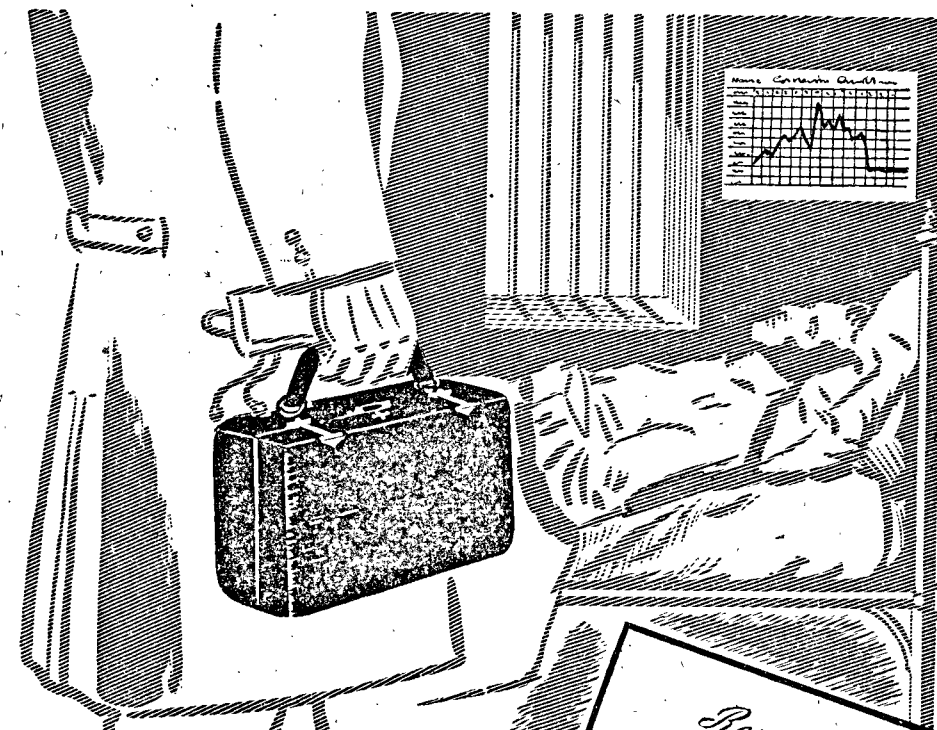
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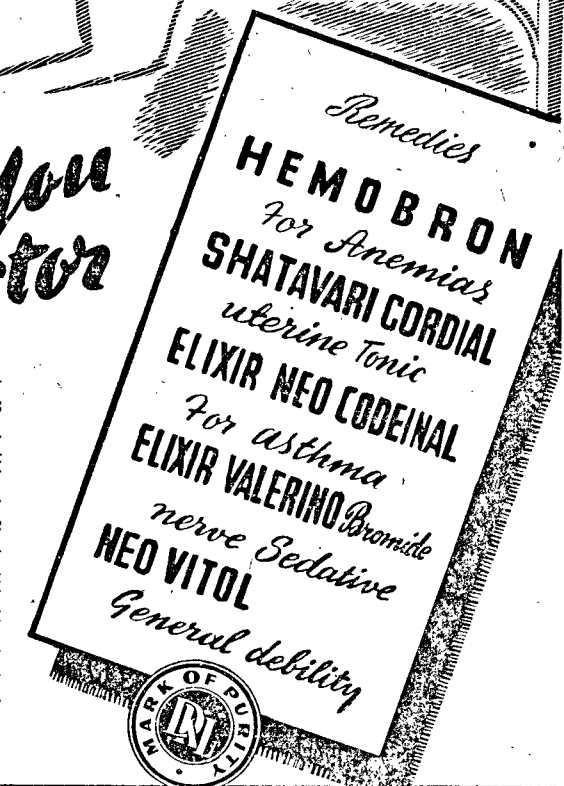


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Vol. 43.

APRIL, 1946

No. 4.

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MAJOR KHAN BAHADUR AHMED BAKSH, O.B.E., I.S.O.;

Agency Surgeon in Bundelkhand, Nowgong, C.I.,

Though this disease is common in Southern India and parts of Bengal it is no less common in parts of Bundelkhand and Baghelkhand in Central India and the nucleus of the disease appears to be in Panna State which is famous for diamonds and Districts and States bordering it as Rath, Mahoba, Charkhari, Ajaigarh and Teother Tehsil in Baghelkhand wherefrom cases come in here for treatment. It has been prevalent here for a very long time and these places abound in mosquitoes and specially culex and anopheles owing to the marshy condition of the soil due to innumerable tanks and pools of water in this part of the country. Common affection which is noticed due to the infestation of the host *i.e.*, filaria, as periodical filarial fever which is often confounded with malaria; glandular fever often mistaken for tuberculosis; lymphangitis and lymphadenitis, often mistaken for lymphatic and other glandular disease, lymph œdema for Bright's disease; and elephantiasis of the legs, thighs, arms, breasts, vulva scrotum with or without chylocele, chyluria, chylous ascites.

Filarial lymphangiectasis of the spermatic cord is not an uncommon condition and is often mistaken for varicocele. Superficial lymphatic varix is noticed in elephantiasis of scrotum, legs, thighs, breast, vulva, and arms etc., these lymphatic varix present whitish appearance.

(1) When this varix involves the integument of the scrotum the result is lymph scrotum;

(2) When in groin it is mistaken for varicocele, groin glands which are often mistaken for inguinal or femoral hernia;

(3) When the lymphatics of the bladder and kidneys are affected and rupture due to over distention, then chyluria is the result;

(4) When lymphatics of the tunica vaginalis rupture it results in chylocele;

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[225]

(5) When they capture in peritoneum they lead to chylous ascitis. The contents of the lymphangiectiasis of the spermatic cord may be milky or chylous or straw coloured. Lymphocele may also develop often after excision of glands. Chylous dropsy of tunica vaginalis does not transmit light and is associated possibly with lymph scrotum, varicose groin glands, with chyluria or chylous dropsy of the tunica vaginalis. Our results here in the search for micro filaria have all been negative. In all these cases bloods and chylous fluids were examined. What has led me to mention about this fell disease in Bundelkhand is that in my short tenure of office here, I have come across with almost all types of cases and more so an interesting case of lymphocele and chylocele which was mistaken by me for varicocele and hydrocele respectively and which is described below:—

On 27th December 1945 I operated another case of hydrocele which turned out to be a case of chylocele and a third case, aged about 20, of lymph scrotum with chylocele and lymphocele and spermatocele has again been admitted.

In addition to this, hydrocele of the tunica vaginalis also is very common in Panna and in my opinion it is due to filaria and very tight bandaging of the penis and scrotum leading to a so-called mutilated condition of the testicle and penis, when this bandage, the so-called *Langot* (Suspensory Bandage) is removed. Any one who is not experienced about its tightness would be horrified to see the condition of the penis and scrotum when such a bandage is released. In my opinion this tight strapping leads to trauma of the testicle which in turn retards the absorption power of the tunica vaginalis due to inflammation which is of a sub acute or chronic type and thus leads to the formation of common or vaginal hydrocele of the tunica vaginalis. In most of these cases there is no specific history or of trauma, and inspite of a hydrocele the patient is tightly strapping his genitals with a bandage. On operation I find testicle inflamed, slightly enlarged in some cases due to chronic inflammation and both the layers of the tunica vaginalis are congested, red and inflamed. These conditions vary in intensity according to the amount of the trauma sustained by the part due to tight bandaging. Here filaria may be the predisposing factor.

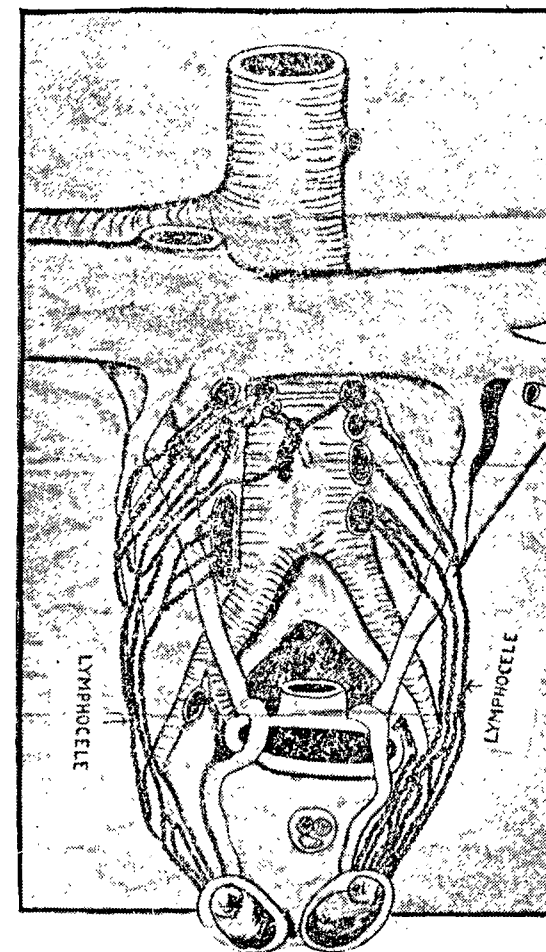
I will now describe the interesting case which has led me to report on Filariasis in Bundelkhand.

A young man, aged 28 years, came in for a swelling in the scrotum which he has got for the last one year. He is well built and strong with strenuous habits and comes from Ajaigarh State where filaria affection is said to be common. Other systems normal. Right scrotum presented all symptoms of a hydrocele of the tunica vaginalis except that the transparency test was not marked. The swelling was as big as a cocoanut. On the left side there was fulness in the loin with impulse on coughing, the cords present the appearance of a varicocele and the two fingers *i.e.*, index and middle could easily be inserted into the left inguinal ring. The testicle on this side is also enlarged longitudinally and is as big as a small mango. As he was very anxious for operation I decided to operate on him first for small left inguinal hernia. After the skin and subcutaneous incision, the aponeurosis of the external oblique was divided, when to my surprise 5 or 6 strands of vessels filled with opaque fluid were seen running along the cord from the abdomen to the epididymis, the aperture of the external abdominal ring, through which these emerged, was large and could easily accommodate 2

fingers as mentioned before. At first I thought these white strands which contained milky fluid, were veins of the pampeniform plexus which were empty, but on puncturing them with a needle whitish fluid oozed out from them—they looked beaded in appearance as the fluid was confined between valves as these white strands presented the appearance of a varicocele. I performed on them the operation of a varicocele *i.e.*, isolated them from the cord and the ductus deference, excised about 2" in length after ligaturing, and tied the two ends together. After this I finished the radicle operation of hernia on the left side by approximating the conjoined tendon to the inguinal ligament, the cord was superimposed on this, the external oblique aponeurosis was stitched and the wound was closed. There was no sack of a hernia in this and the bulging in the loin was due to lymphocele and not varicocele or hernia.

After a fortnight I operated on the right testicle for hydrocele when on opening the tunica vaginalis chylous fluid came out instead of pale straw coloured fluid. The cord on this side did not present the appearance of a lymphocele as on the left side. The operation was completed after excision of the sac and the wound was closed leaving a glove drain—the discharge through this glove drain was ordinary serum mixed with some blood but not chylous fluid. The patient made an uneventful recovery and was discharged cured. Important points about this case are: (1) no symptoms or signs of filariasis any where in the body, either the legs, arms or the scrotum. No history

of filarial fever and it presented the appearance of hydrocele on the right side and varicocele with a bubonocoele on the left. It was on operation only I could make out that it was a case of lymphocele on the left side and a chylocele on the right side. The latter confirmed the diagnosis of a lymphocele on the left. Lymphocele also will present the same signs and symptoms as varicocele from which it cannot be differentiated unless some other filarial conditions are present as lymph scrotum or elephantiasis of the leg, arms etc. Second point is to be solved how the



filarial condition led to this affection i.e., chylocele on the right and lymphocele on the left as both were affected simultaneously. Here the obstruction by the filaria would be somewhere lower down in the pelvic lymphatics leading to this condition. Lymphatic vessels themselves are in reality modified veins, hence their affection is often confounded with those of the veins accompanying them. The attached illustration shows the condition of lymphocele.

III CASE: A patient, aged 25 years, resident of Charkhari, came in complaining of a superficial abrasion on the scrotum with a constant milky discharge which is very annoying and for which he puts on a dressing which he has to change every 3 or 4 hours as it is soaked with the fluid. Duration of the disease is 8 years while that of the discharge is 2 years.

History: At first he was having fever preceded by shivering and rigor, pain in the glands and lymphatics of the groin and thigh. The fever lasting for 3 or 4 days and was subsiding with sweating. With the fever both the testicles enlarged and on its subsidence the swelling of the scrotum and testicle also subside. For the first 4 years or so he was having 2 or 3 such attacks but for the last 2 years the attacks of filarial fever are frequent coming on every week or 15 days with pain etc., in the groin, thighs and scrotum and subside in 24 or 48 hours.

Present condition: A healthy young man having all femoral and inguinal glands on both sides markedly enlarged, hard, discrete, movable. The lymphatic trunks in them felt like fine threads inserted under the skin. The skin over this area is coarse and rough the skin of the penis and scrotum is assuming an elephantiasis appearance and there is an abrasion, size of a 4 anna piece, through which whitish fluid is oozing and can be squeezed out. This was examined for micro-filaria but the result has been negative.

IV. Another case of a large elephantiasis of the scrotum weighing about 10 lbs. is awaiting here for surgical intervention as it has rendered him unable for even ordinary avocations.

Reference:

Raduag Maingot: "Monson's Tropical Diseases and Post Graduate Surgery."



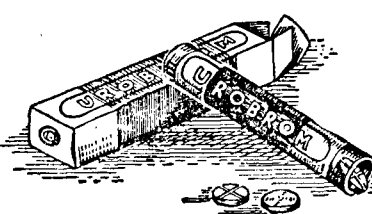
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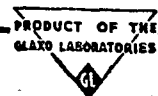
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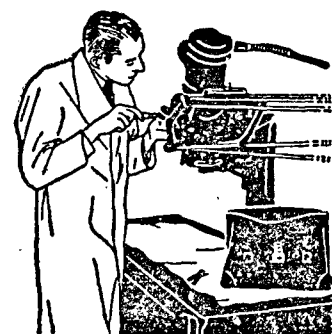
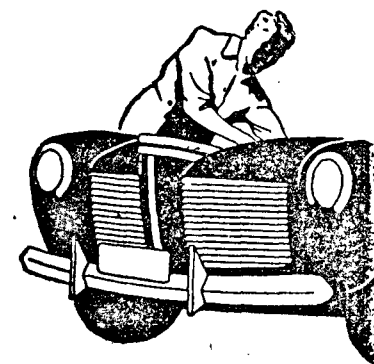
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Teratoma of Testis*

D. B. DESAI, M.B., B.S.,

R.M.O., L. W. Ashaktashram Hospital, Rampura, Surat.

It is no doubt an innocent tumour of the testis in the first instance but considering the various elements of which it is composed, it is very likely that any one of these elements may start undergoing a malignant change and hence for all practical purposes this tumour is to be treated as a malignant one. It may remain stationary for years but eventually takes on a rapid growth. It generally affects persons round about the age of 30 years.

Pathological considerations.—As regards the pathology of this tumour, there is still a difference of opinion, but that practically all the tumours of the testis fall into two great groups and the varied appearance presented by the tumour such as chondroma, carcinoma, fibro-cystic disease, etc., is due to the preponderance of particular cells of the primitive layers.

1. The first group is of a tumour arising from the cells lining the seminiferous tubules—the so-called spermatocytomas or seminomas: the growth is slow and the tumour is solid and the cut surface is fleshy and homogenous and traversed by fibrous septa which give rise to lobulation.

2. The other group is of a true teratoma or embryoma arising from the germinal blastomere—the progenitor of both ova and sperms and thus these cells are totipotent so that they produce a teratoid tumour which contains structures derived from all three of the primitive layers and hence the varied appearance of the tumour depending on the preponderance of particular type of cells and thus the name fibro-cystic disease of the testis because of the cysts found therein. The cut surface of an embryoma resembles that of a coarse sponge or it may be honey-combed and the metastatic tissue also looks gelatinous.

Symptoms.—In the early stages, there is neither pain nor discomfort but there is only a steady increase in the size of the testis. The whole scrotum feels very heavy and hard—a distinguishing feature from the simple hydrocele. Metastasis occurs in the inguinal and lumbar glands. A mass is felt in the abdomen on the right or left side depending on which side the testis is affected and it may react as high as the costal arch and near the middle line or beyond it. This lump is at times so painful and prominent compared to the testicular swelling that the abdominal lump only attracts our attention and for which only the patient is brought to us and thus the primary growth in the testis being so small and painless is likely to be missed if the practice of palpating the testicles is not remembered in case of any abdominal tumour.

Differential diagnosis.—1. *Hæmatocele*:—May have a history of injury and subsequent increase in size with pain but at times it is very difficult without exploration.

2. *Epididymo-orchitis*:—Generally an acute condition with a history of gonorrhœa and fever and tender scrotum on palpation; the palpation will also reveal the enlarged epididymis. Moreover some two or three

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injections of Calcium Chloride 10% 10 c.c. intravenously every alternate day will bring about a rapid change.

3. *Syphilitic orchitis*:—Absence of pain or tenderness. History of syphilis and anti-syphilitic treatment has a marked effect.

4. *Tuberculous testis*:—Nodules are being felt over the epididymis which are firm and hard and with passage of time, caseation occurs in these nodules with formation of tuberculous fistula.

5. *Simple hydrocele*:—Fluctuation all over and not difficult to diagnose where the skin is not filarial. When the metastatic lump is present in the abdomen, the diagnosis offers no difficulty otherwise. It is very difficult to differentiate it from an old hæmatocele.

Treatment.—In the early stages orchidectomy excising the cord high up near the internal abdominal ring if possible and then submitting the case to deep X-ray therapy later on.

It will be interesting to the readers to go through the following case notes. I will first make it clear that no specimen was sent for biopsy as it was inconvenient and the last described case was put down as an old case of hæmatocele by minute examination of the cut surface only.

1. A patient was admitted in the hospital for severe pain in the left side of the abdomen without vomiting but the pain was so severe that the patient was restless and tossing in the bed. On examination, a hard painful mass was felt in the left lumbar region but the cause could not be made out and hence many doctors examined him giving out various suggestions but not one was satisfactory. Then one intelligent fellow started palpating the left testicle and found out a small hard irregular mass in the testis which was not enough to increase the size of the scrotum and draw our attention there; but when palpated the patient complained of some pain. This was a case of teratoma of the left testicle with metastatic lump in the abdomen.

The point to be stressed is that for a metastatic growth in the abdomen, a testicular growth need not be a bigger one; it may be so small that we can easily miss it if we forget to examine the scrotum as in this particular case the patient only came for abdominal pain.

2. Another one, with an established diagnosis of the teratoma of the testis, was admitted for a big inguino-scrotal swelling of the left side. The scrotum was hard, painful and of a cocoanut size. There was also a thick rope-like swelling in the inguinal region continuing from the root of the scrotal swelling and the inguinal swelling can be followed right up into the left abdominal cavity and then a mass very hard reaching up to the left costal arch and extending up to the middle line. The patient was markedly emaciated and complained of severe colicky pains in the abdomen and severe constipation. For academic interest, we did orchidectomy and exposed the prolongation of the growth in the abdomen. It was going intra-peritoneally and was very friable and of gelatinous appearance. The patient died after some fifteen days or so.

3. The third was a very interesting case. This patient was admitted for a strangulated hernia-like swelling which was very painful on touch and vomiting. The difficulty in this particular case was to follow the

language of the patient or his relatives. When we asked about the duration of the swelling they said something which we could not understand and the cursory glance at the swelling suggested strangulated hernia and so the conservative treatment for the reduction of strangulated hernia was followed. The palpation of the abdomen revealed another painful lump in the abdomen and that put us off the diagnosis of strangulated hernia. Again a thorough interrogation of the patient and his relatives was undertaken and that brought out the fact that the swelling of the testis was already there for a pretty long time but that it had rapidly increased within a period of one and half months and that his bowels moved regularly without any difficulty. All these points suggested that it was a case of a teratoma of the testis with secondary metastasis in the abdomen.

4. Another patient was admitted for a left-sided scrotal swelling with nothing in the inguinal canal but a small painful metastatic mass in the left iliac fossa. We did left-sided orchidectomy and surprisingly the abdominal growth began to increase so rapidly that within a period of a fortnight the whole abdomen, except for a small space in the right iliac fossa, was full of the growth and the pain was excruciating and the constipation a marked feature of it.

The unbearable pain was relieved by a dig or two of Inj. Morphia gr. $\frac{1}{4}$.

He survived for a week more and died of complete inanition because of the constant vomiting.

It is an interesting fact to note that the patients cited as case Nos. 2, 3 and 4 came from one particular caste—a labour class peculiar to the district only. I cannot explain the reason of it.

5. A fifth case was admitted as hydrocele for radical cure. During operation, it looked like an old hæmatocele and orchidectomy was performed. After the wound had healed, the patient always complained of pain in the region of the excised cord but there was no swelling except for a little induration and hence I gave him some 2 to 3 inj. Calcium Gluconate 10% 10 c.c. intravenously every fourth day and discharged him one month after the discharge from the hospital, he noticed a small painful swelling at the root of the scrotum which slowly went on increasing. After some six months, he again came for a lobulated painful scrotal swelling and a few enlarged inguinal glands. It was now thought to be the teratoma of the testis and hence we advised the patient that the growth will be excised but then he will have to go for the deep X-ray therapy at Bombay. He again went away but came a week after with a marked increase in the size of the growth. The growth was excised and the patient was directed to the Tata Memorial Hospital for the deep X-ray therapy. I do not know as to what happened then.

6. Another was admitted for a chronic scrotal swelling with a discharging sinus at the bottom and an unhealthy granulation tissue round about the mouth of the fistulous opening. The case was diagnosed as teratoma of the testis. Orchidectomy was undertaken and the enlarged inguinal glands were removed. The cut-surface of the testicular swelling revealed a healthy testis but it was a very thick sac with old blood clot inside. The scrotal swelling was being tapped before and the sac got infected leaving behind a sinus discharging pus. This was then only an old case of hæmatocele infected by tapping which also was responsible for a sinus

and such funny appearance of the scrotum that it can easily be mistaken for a growth of the testis. The enlarged inguinal glands were because of the chronic sepsis in the scrotum.

Because of the filarial scrotums preponderating in this district, it is very difficult to clinically differentiate teratomas from the old hæmatoceles and the hæmatoceles are also only detected on the operation table when so-called hydrocele cases are being taken for radical cure because of the thick skin of hydrocele swellings.

My thanks are due to our C.M.O. Dr. G. M. SUBHEDAR, F.R.C.S., for allowing me to publish the Case Notes.

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Nervous Complications of Diphtheria*

P. N. LAHA, M.D.,

Lecturer in Diseases of Children, Medical College, Agra.

Introductory.—Nervous symptoms and complications of diphtheria occur with sufficient frequency to make them clinically important. They are peculiar in type.

Pathology.—Their pathology and pathogenesis are not yet perhaps completely understood. There is still some doubt as to the primary and essential feature of the pathology. In summary, we may say that (a) motoneurons and sensory protoneurons are damaged by a neurotoxin acting directly on parenchyma, and that while, in general, nerve fibres suffer first and most, other cases are distinguished by primary attack on either cells or motor end-organs (muscle): (b) aside therefrom, any part of the neurones connected will exhibit secondary reactions according to the rules of diphtheria (WILSON, 1941).

Different complications.—Paralysis: In the opening paper at a discussion on the nervous complications of acute fevers ROLLESTON (1933-'34) noted that his own observations based on a study of 2,300 cases of diphtheria showed that 20 per cent developed some form of paralysis. Local paralysis, viz., palatal, pharyngeal, superior laryngeal, inferior laryngeal are frequently met with. Local palsies of face, tongue and masseters are rare. There may be multiple neuritis. Paralysis of accommodation of subjective nature also occurs, and it is a specific paralysis in diphtheria. This paralysis occurs irrespective of the site of infection and hypermetropes suffer great inconvenience. SCHIRWINDT (1944) records his observations on 205 cases of toxic diphtheria in children. Paralysis developed in 24.4 per cent, its most frequent time being the end of the second week.

Encephalitis.—It is a very rare complication. QUERIDO (1928) who reported a case of post-diphtheritic encephalitis, remarked that parenchymatous inflammation of the central nervous system is the rarest sequelæ of diphtheria. The patient, who was a nurse, survived. HALL (1932) reported a fatal case of acute toxic encephalitis in a female aged 18 months, who became suddenly ill with vomiting, increasing drowsiness, high temperature,

and convulsions. In addition to membrane in the pharynx from which diphtheria bacilli were cultivated, the necropsy showed distension of the cerebral vessels, with linear hæmorrhages throughout the white matter. LAHA (1946) described a fatal case of diphtheritic encephalomyelitis in a Sikh male aged 15 months. 122,000 units of anti-diphtheritic serum, Cibazol, repeated lumbar puncture and Coramine by mouth could not check the fatal march of the disease.

Meningitis.—CARLSON and MORGAN (1937) recorded a case of diphtheritic meningitis, of which they could find no previous example in English literature, though four cases were recorded by German observers. Post mortem diffuse suppurative meningitis of the brain and spinal cord was mastoiditis.

Hemiplegia.—KENNEDY (1937) recorded a case of right hemiplegia in a girl aged 7 years, following severe faucial diphtheria on the twenty-first day of the disease. He thought that the hemiplegia was due to a primary cerebral thrombosis rather to embolism.

Prognosis.—It is good in paralysis. Encephalitis and meningitis are usually fatal. Hemiplegia is usually more or less permanent. A guarded opinion should always be pronounced.

Treatment.—In paralysis and hemiplegia, serum has doubtful value. For paralysis administration of vitamin B₁ in liberal doses is said to expedite recovery. In encephalitis and meningitis large doses of serum should be given coupled with other symptomatic treatment.

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* Specially contributed to THE ANTISEPTIC

Infantile Hepatic Cirrhosis

(Conditions described as infantile biliary cirrhosis; sub-acute toxic cirrhosis; and infantile liver.)

A. V. S. SARMA, M.B., B.S. (Mad.), D.C.H., R.C.P. & S. (Eng.),
Formerly Honorary Assistant Physician, Stanley Hospital, Madras, South India.

(Continued from page 114 of February '46 issue).

Curative treatment.—The curative treatment is tackled under the following heads:—(1) Hygiene; (2) Diet; (3) Drug treatment; and (4) Special treatment.

1. *Hygiene.*—The environment should be healthy, and particularly free from tuberculosis, malaria, kala-azar, intestinal infections, and eruptive fevers (particularly influenza, whooping cough, diphtheria, measles, small-pox etc.). The water used for drinking purposes should be pure and clean. Very hot areas are trying. Changes in the weather, particularly those from the hot to the cold rainy season, are badly tolerated by these little patients.

In all active phases of the condition rest in bed is essential. Sponge baths are preferred to ordinary leisurely baths. Oil baths are discontinued.

2. *Diet.*—The aim of diet-building is the formation of a balanced and vitaminous diet with lowered fat content and raised carbohydrate and protein moieties. Carbohydrate is preferred as cane sugar or pure dextrose or as karo syrup and not as pure starchy products. For infants below the ninth month breast milk or artificial feeding preferably acidified milks—is the routine recommended by the writer. For older children increased carbohydrate is necessary and soft bread and honey or congee or parboiled rice will form the increased carbohydrate. Buttermilk has been extensively and safely used as such or as medium for boiled rice. "Skimmed milk or buttermilk or skimmed lactic acid milk must not contain more than 1.5% of fat, and protein and carbohydrate must be approximately 3 and 7 per cent. The acidity of buttermilk must not be over certain grade. To be exact 25 c.c. of buttermilk must neutralise 4, 3 c.c. of of N/2 NaOH" (K. C. CHAUDHURI 1944). Juice of oranges, tomatoes, pine apple, aqueous extract of wood apple, are useful sources of vitamin C. The use of Amalaka (*Phyllanthus Emblica*) either as pulp or extract in water deserves more popular use in this connection. Articles of diet, vegetarian or non-vegetarian, producing intestinal allergy must be forbidden. The danger of hyper-sensitivity has to be borne in mind, particularly when a child is improving. Avoidance of tubers, sweets, hard, irritating and fibrous foods is essential. Excess of fat (ghee, milky puddings, cream) is to be strictly avoided.

Recent suggestions for treatment of hepatitis are: "Diets should be low in fat and high in protein and carbohydrate. Vegetable protein, which is easier to secure in war time than animal protein has been regarded as particularly suitable for disease of the liver. It is wiser to administer concentrates of fat soluble vitamins A and D and vitamin K, if there is evidence of prothrombin deficiency. Hypo-proteinæmia should be corrected by transfusion of plasma. Baker's yeast should be prescribed, and increased if tolerated from 3 teaspoonfuls to 1 to 3 oz. a day. No information is available about the use of Methionine in man but favourable results have been reported from the administration of choline chloride in

a dose of 1.g daily in cirrhosis of the liver, together with a diet like that outlined above." (*British Medical Journal*, 12-12-1942).

3. *Drug treatment:*—All these little patients with hepatic cirrhosis receive a prescription on the following lines:—

(1) Pepsin gr. 6 and acid hydrochloric dil minims 60 in six doses. given before feeds thrice a day, or papain before feeds.

(2) Sodii salicylas, Calcium Gluconate, Diuretin, Ext. Kalmegh. liq., Ext. Punarnava liq., Ext. Gulvel liq. Ol. Anise and syrup of Glucose. A suitable recipe given $\frac{1}{2}$ hour after feed thrice a day.

"TARR used 10 c.c. 10% Magnesium Sulphate intravenously and 20 c.c. 10% Mag. Sulph intraperitoneally in certain types of cirrhosis of the liver with success. In one case TARR gave 46 injections once daily. It is given 3 or 4 times a week. He reported satisfactory results." (K. C. CHAUDHURI).

4. *Special treatment:*—In a few cases the writer had favourable results with splenic extract. Raw spleen given in diet may be tried.

Adrenal cortex given orally or by injection has given very useful results in cases with allergic basis (case 38). The writer feels that adrenal cortex is beneficial particularly before ascites is pronounced. Fat intolerance is controlled by adrenal cortex and vitamins of B group (Aneurin and Nicotinic Acid). The use of adrenal cortex has not only theoretical justification but is beset with practical utility. W. A. Hislop gave one minim of adrenal cortex extract per two lbs., weight per day to marasmic babies with great benefit. Studying hay fever BLOOM has recently alluded to allergy being an electrolyte disturbance associated with adrenal dysfunction. W. N. KEMP has shown that adrenal cortex is useful as a catalyst generally for tissue function and particularly for hepatic activity. Toxæmias of pregnancy are reported to be beneficially affected by oral use of adrenal cortex.

"Insulin (3.5 units administered with glucose in some of these cases has produced accelerated improvement and in some of the recent cases treated by me, Zinc-protamine insulin (in doses of about 8 units) was substituted in place of ordinary insulin with very satisfactory result. Blood sugar being normal in these cases, Insulin helps by increasing glycogen store of the liver and thus facilitates repair of degenerative changes" (M. B. PRABHU 1940). "On the whole blood sugar is low, and this is particularly so at the terminal stages of the disease" (P. KRISHNA RAO 1941). The writer has no experience in this line of treatment but is afraid of giving Insulin to little babies with hepatic cirrhosis in view of the variations in the level of blood sugar in these cases.

Torantil has been used in two cases with considerable temporary improvement.

Vitamins C, B₁ and Nicotinic Acid as also concentrates of vitamins A and B, are indicated from the beginning of treatment. The writer's report on the beneficial effect of vitamin C in infantile hepatic cirrhosis has been confirmed by U. S. RAO (1940).

Auto-vaccines made from the germs present in the septic foci and particularly from the throat and tonsils, given in small doses and spaced without producing reactions, are always attendant with good results and

probably serve to prevent febrile attacks, diminish chances of ascending cholangitis, pyelitis etc. Sulphonamide preparations are known to affect the liver adversely and hence are better avoided or used only with caution in treating a complicating condition like pneumonia or some other coecal infection.

"Vaccines prepared out of B. Coli from infantile cirrhosis and inoculated in successive increasing doses over a period of 6 to 7 weeks has cured the disease. (This fact indirectly proves B. coli to be the infective agent in the production of the disease)" P. KRISHNA RAO (1941). The writer made extensive use of B. Coli vaccines as preventive and curative in 1939, 1940 and 1941 and had disappointing results (cases 42 and 79).

Infective diarrhoeas are given specific treatment whenever possible. 10 c.c. of mother's blood injected intramuscularly daily during acute phases of atypical diarrhoeas appeared to do immense good in a large number of cases.

The allergic crises seen in some cases with respiratory distress, abdominal distension, and constipation and rise in temperature are best treated with vitamin C. Calcium and Tinct. Hyoscyamus orally. A small dose of Adrenalin or Pituitrin by injection gives quick relief.

Alleged secret specifics on the market used by the public for infantile hepatic cirrhosis are believed to contain zinc in some form (Zinc Oxide) and therefore zinc therapy of the disease is coming to the forefront. Chemical analysis of the powders advertised as specifics reveals 99% of it to be zinc oxide.

Discussing the use of zinc oxide in infantile hepatic cirrhosis, M. B. PRABHU writes: "Zinc oxide in daily dose of 1 to 3 grains has given relief more than other drugs, and it seems to retard the rate of progress of the disease; even in fairly advanced disease (I will not use the word "cure") satisfactory results have been obtained. This drug, if considered necessary, can be prescribed with other suitable cholagogues. About the nature of action of zinc on liver one cannot say anything. Could zinc in some form have a specific action on the type of disease as arsenic in syphilis or antimony in kala-azar?" Commenting on the above, U. S. RAO (1940) writes "In his (PRABHU's) hands, it has proved quite harmless and seemed to retard the progress of the disease even in advanced cases. No body is able to explain the action of zinc on the liver. I have attempted to modify the evolution of toxic cirrhosis due to carbon tetrachloride in experimental animals, like albino rats, by simultaneous oral administration of vitamins A and C, and zinc oxide, suspended in olive oil, and the last mentioned drug has definitely delayed the induction of cirrhosis and produced a milder type of fibrosis of the liver in animals." The writer has also used zinc oxide in this disease and earned the impression that its action may be of the nature (non-specific) common to all heavy metals. It might have a slight astringent action and decrease toxic absorption from the bowel. The writer feels that zinc acts probably as a stimulant of the reticulo-endothelial system and that it might not always ultimately benefit the system. The writer saw fatal termination in many cases treated by traders in these secret specifics which are believed to contain zinc oxide and concluded that if success attended to treatment by these alleged experts outside the profession, it might not always be due to the zinc. Apart from

zinc the specifics advertised might also have other ingredients evasive to chemical analysis. The dietetic regimen advised by the trader-experts appears to be sound. How much of success then is due to the diet adjustment?

U. S. RAO (1940) reported beneficial results from the use of acetyl choline in the disease. It is to prevent vaso-spasm and counteract sympatheticotonia. The writer used nicotinic acid with the same aim and feels it is quite a useful adjunct to treatment. Nicotinic acid acts like nicotine against sympatheticotonia and is an acknowledged anti-allergic. Nicotinic acid is given by mouth, and above all is a safe dietetic supplement. Daily 5 to 10 milligrams are given with marked benefit. Post-mortem of the liver has been found to be cirrhotic in cases of pellagra which has a specific response to nicotinic acid therapy.

Symptomatic treatment.—1. *Fever*:—During the pyrexial stages, diet is reduced (especially fat) and more bland liquids like barley water and fruit juices with increased glucose are given. The fever is treated with Sodii Salicylas and Ammonium Benzoas which are usually effective. Hydrotherapy is resorted to whenever convenient.

Sometimes the fever is very persistent and then often a latent pyelitis (B. Coli) is present or the rise of temperature appears to be in some way related to the worms present in the bowel. Pyelitis is treated with alkalies and auto-vaccines of B. Coli. Sulphonamide preparations are used with caution.

2. *Cough*:—The heart and the lungs suffer a toxic damage in this malady as the liver is getting progressively cirrhotic. The cough which is present in the earlier stages and which seems to be due to the constant presence of upper respiratory infections (vitamin A deficiency? chronic mild hypoadrenia?) is best treated by raising the general resistance of the patient. The cough that is present in advanced cases is due to pulmonary damage and is best attacked by liberal use of calcium and vitamin.

3. *Helminthiasis*:—Worm infestation is tackled by Quinine or Bismuth Salicylas. Worm enemata could be given to older children for thread worm infestation; or Butolan may be used.

4. *Insomnia*:—Hydrotherapy or a dose of Ammonium bromide does well. Barbiturates and opiates are undesirable.

5. *Constipation*:—Reformation of diet with a liberal supply of fluids (barley water, Mellin's food, fruit juices, Glucose and water) in between foods is the first step. In the case of older infants increase in the bulk of food by way of soft part of bread and honey, apple, plantain pulp, boiled rice (when tolerated) is essential to have satisfactory bowel action. The "roughage" diet should not be advised to infants.

Vitamins of B group are freely given:—Drugs should be resorted to in the end. Olive oil, milk of magnesia, rhubarb, cascara sagrada are useful. Castor oil and Magnesium Sulphate are avoided and the first is not only a sensitising drug but also suspected to be a hepatic poison (TIRUMURTHI and NARAYANAMURTHI) and the second invites a pyelitis.

6. *Diarrhoea*:—Constipation and diarrhoea alternate with each other and deserve early tackling. Administration of Aneurin and Nicotinic acid is useful.

Rancid hard faecal concretions call for reduction of fat in the diet, and, when available, the use of adrenal cortex, vitamins of B group. Green diarrhoea with air bubbles in the stools calls for reduction in carbohydrate. Protein leathery curd summons decrease of protein in diet. Parenteral diarrhoea demands rational treatment.

Diarrhoea of enteric and bacterial origin has been treated by the writer with 10 c.c. of mother's blood intramuscularly. Bismuth Salicylas is also tried.

A rise of temperature (toxic absorption) follows complete arrest of motions and for such an undesirable state of affairs an emulsion of olive oil and Bismuth Salicylas or Kayalanol is used. Combination of Yeast and tannic acid (Eldoform, Tannyeast) is sometimes very effective.

Apple jelly or Kappectate (Upjohn), weak tea, and diluted Horlick's foods are useful. Thin arrow root water or buttermilk is very useful in some cases.

After recovery from diarrhoea, acidified milks, or foods with low fat content, are used. Diet is slowly built up with the addition of vitamins.

7. *Anæmia*:—Liver extract with the addition of vitamins of B group and vitamin C are given with remarkable benefit. Iron and arsenic are not tolerated well by infants. A combination of marrow and hæmoglobin is good.

8. *Hæmorrhagic tendency*:—Calcium and vitamin C combat bleeding tendency. Post-hæmorrhagic anæmia is treated with liver, B factors, C and ferrous iron (ferri-et-ammonii citras).

In urgent and severe cases of gastro-intestinal hæmorrhage hæmoplastic serum is tried.

Vitamin K is exhibited in cases with hæmorrhages after jaundice has set in. Tomato juice orally supplies some amount of vitamins C and K.

9. *Dropsy*:—(a) Generalised œdema; and (b) Ascites.

(a) *Generalised Oedema*:—A generalised œdema appears suddenly in some infants. Calci-diuretin and vitamin B₁ and vitamin C are definitely indicated and given preferably by injection.

Concentrated and salt-free diet rich in carbohydrate and protein may be instituted. Fat is cut down.

(b) *Ascites*:—Ascites when present is better treated without paracentesis which may be resorted to for relief of intra-abdominal tension to such an extent as to afford respiratory relief and lessen pressure on renal veins.

Thinking of the use of parenteral diuretics, though Theophylline is ideal, it is sometimes a failure. One is forced to resort to mercurial diuretic like Mersalyl (B.D.H.) or Esidrone or Salyrgan. Rapid elimination of fluids either by diuresis or by tapping produces very often a rise in temperature which further exhausts the baby. Such a rise in temperature demands the postponement or a diminution of the quantity of the next diuretic injection and temporary increase in fluid intake. Sudden deaths have occurred for no apparent reason after rapid diuresis. Additional use of Aneurin and ascorbic acid preferably by injection is particularly indicated in the ascitic stage.

Purgation as a means of dehydration seems very undesirable in infants. Vigorous purgation by means of salts is sure to pre-dispose to B. Coli pyelitis.

Diet is as for generalised dropsy.

10. *Jaundice*:—Jaundice generally follows ascites and means poor prospect of recovery. Decoholin and Chionia are indicated now.

When an infant is being treated a watch should be kept for the incidence of jaundice—sometimes a terminal cholangitis and sometimes an early manifestation—and in the advancing stages of the disease, an auto-vaccine made from the bacterial flora of the bowel is used (generally B. Coli vaccine). The idea is to mitigate the incidence of an ascending infection of the biliary tract. The procedure has had practical utility in the writer's hands but surely cures were not obtained. Vitamin K and bile salt therapy is to be instituted at this stage as a prophylactic against hæmorrhage.

11. *Arrest of growth and the recovering stage*:—POYNTON and WYLLIE (1926) showed that cirrhosis in infants is frequently associated with infantilism. The little patients that recovered under the writer's care developed a rickets-like state particularly during recovering stages; and the response

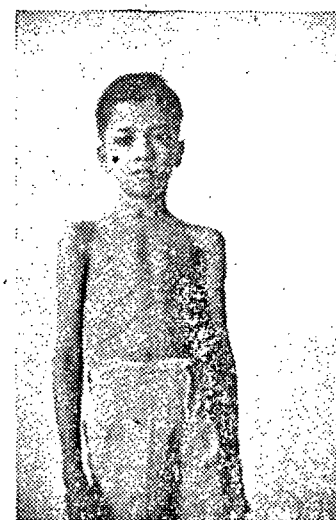


FIG. 18.

Case No. 3. Clinical photograph of a boy aged 6 years (cured).

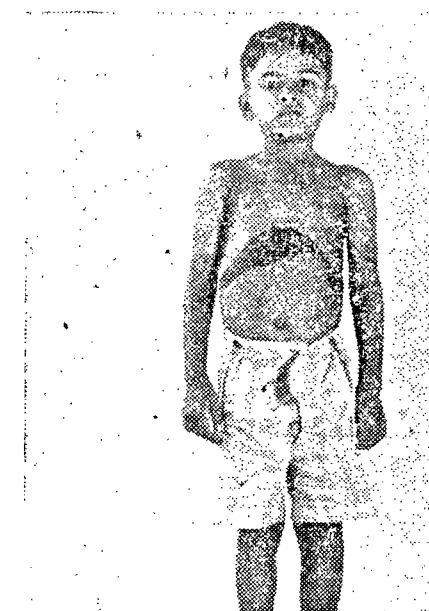


FIG. 19.

Clinical photograph of a boy at schooling age (cured).

to calcium and vitamin D has been uniformly good. Those that recover completely, appear quite normal later. Some children (case numbers 3 and 21) that were followed up by the writer to the schooling age showed nothing untoward clinically, except a persistently large and hard liver and occasionally palpably spleen. An applicable percentage of these recovered children have developed asthma in later life.

Appendix of

Serial No. Year of observation.	Name, age, sex, social status, caste, dietary habits	Complaint Duration	Family history	Previous his- tory, feeding	History of present illness
1934 1	Venkatasubbu, female 4 years, (Nellore), Hindu Brahmin. Rich class. Vegetarian.	Constipation fever, protu- berant abdo- men—2 years.	Mother had eclampsia fit during deli- very. Labour protracted, and delivery aided by for- ceps. Child delivered as- phyxiated.	Fed by mother and on cow's milk.	As a medical student the writer saw the child from date of birth. Treated for 'liver enlarge- ment' outside the profession. Started with constipation and fever.
2	Balasubramanian, male 3½ years (Madras), Middle class, Hindu Brahmin. Vegetarian.	Fever, yel- low eyes and bloated abdo- men—1½ years	Father a vic- tim of migrai- ne and diabe- tic since 5 years, mothers of 1 and 2 are sisters.	'Brest-fed and on cow's milk.	Summer diar- rhœa at 1 year.
3	Prakasam, male, 2½ years, brother of 2 (Madras).	Fever, yel- low urine and protuberant abdomen.	As in 2	As in 2.	Summer diar- rhœa in early infancy.
1935 4	A little sister to 2 and 3. 1 year (Madras).	Yellow urine and protuber- ant abdomen —3 months.	As in 2 and 3.	As in 2 and 3.	Severe diar- rhœa 3 months before death.
5	Baby c/o Mr. Gopal Raju (Madras), male 3 years, Hindu, Non-Brah- min, vegetarian, mid- dle class.	Chronic constipation since birth.	Mother has rheumatic car- ditis.	...	...
1936 6	Baby c/o Mr. T. Ramiah (Lakshmi- puram, Mylapore, Madras) female, 2 years, Hindu, Brahmin, vege- tarian. Middle class.	Diarrhœa and fever—1 month.	Mother asth- matic.	...	...
7	Baby c/o Mr. R. P. (T'Nagar, Madras), male, 4 years, Hindu, Brah- min vegetarian, middle class.	Chronic constipation and irregular fever—1 year.	Father asth- matic. Pater- nal uncle gets urticarial rash. Another pater- nal uncle dia- betic.	...	...
8	Prabhavathi, female, 3 years (Gudur, Nellore district), Hindu, Brah- min, middle class, vege- tarian.	Chronic constipation since birth.	Mother asth- matic.	Fed by mother and cow's milk.	Chronically constipated and sensitive to castor oil which always upset the child.

Cases

Symptoms	Clinical data : investigations	Additional notes
Fever, consti- pation and distended ab- domen.	Thin girl. Anæmic, abdomen distended with pro- minent veins over anterior abdominal wall. Ascites present. Slightly icteric sclerotics and skin. Liver hard and 3" enlarged. Spleen 3". Heart sounds weak. Urine: redness, Benedict's qualitative solution. Albu- men a trace present. Wasserman of child's blood negative. Treated outside the profession and recorded.	October 1944: patient was seen as a grown-up girl. Periods present. Abdomen tumid. Liver hard and 1" enlarged. Spleen 1½" enlarged. Active and doing appa- rently well. A victim of asthma during last six months.
Fever, jaun- dice, protube- rant abdomen.	A jaundiced male child. Generalised œdema pre- sent. Abdomen distended. Ascites and prominent veins present. Liver hard and 3". Spleen hard and 1". Right lobe of the liver very large. Kahn and Was- serman reaction of parent's blood negative on two different occasions.	Patient died.
Fever, tym- panitis and icteric sclero- tics present.	A boy irritable and having febrile bouts. Abdomen distended. Ascites and prominent veins present. Liver hard (right lobe large) and 3" enlarged. Spleen 1". Icteric sclerotics. A soft systolic murmur audible at apex. Urine: a trace of albumen present. Reduces qualitative Benedict's solution. Treated outside the profession and by scientific medicine.	Recovered. 20-6-1939- Height 4' 2", weight 46 lbs. A soft systolic murmur audible internal to apex-Liver hard and palpable. 28-9-1944.—Height 5' 3", weight 78 lbs. Liver just palpable. Is liable to allergic bronchitis (asthma).
Yellow body, and urine; and bloated abdo- men.	A very irritable baby. Abdomen distended. Jaun- dice intense and ascites slight. Fever present. Treated outside the profession.	Death occurred within three months after the diarrhœa was con- trolled.
Low fever and constipation 2 years.	A dull baby. Abdomen distended, liver hard and 2". Spleen 1".	Reported to have died.
Fever, loose motions (6 or 7 per day) whitish and foul.	An irritable girl. Abdomen distended. Liver hard and 3". Spleen 1". Ascites present. Slightly icteric skin and eyes. Treated outside profession.	Died of a complica- ting pneumonia.
Fever, consti- pation and bloated abdo- men.	An irritable little boy. Ascites and prominent veins present. Liver hard (left lobe layer) and 3" spleen. Slight icteric present. 2. The patient's abdomen was tapped 4 times and immediately after the last tapping, the child died in a comatose condition. Treated outside the profes- sion except for paracentesis.	Died.
Fever, jaun- dice and con- stipation pre- sent.	Thin baby. Bloated abdomen. Jaundiced. Ascites also present. Liver hard and 2" (left lobe layer) spleen 1". Treated. Showed improvement at Madras. Kahn of mother's blood negative.	Reported to have died at Gudur.

Appendix of

Serial No. Year of observation	Name, age, sex, social status, caste, dietary habits	Complaint duration	Family history	Previous his- tory, feeding	History of present illness
9	Balakumar, male, 5 years (Nungambakkam, Madras), Hindu, Brahmin, vegetarian, middle class.	Chronic constipation since birth.	Mother, asthmatic only child in the family.	Fed on cow's milk.	Chronically constipated sensitive to castor oil.
10	Balakrishna Tawker c/o Mr. S. R. Tawker, (Madras) male, 2½ years, Gujerati Brahmin, vegetarian.	Chronic constipation since early infancy.	Father asthmatic, maternal aunt asthmatic. Patient is the third in a group of 4 children.	Fed on cow's milk with slight breast feeding. Weaned at 9th month with rice. Vaccinated at 9th month.	The child is susceptible to frequent colds and fever.
1936 11	Alimelu c/o Mr. Srinivasan (Madras), female, 4 years, Hindu, Brahmin, middle class.	Constipation and irregular fever since infancy.	Father has chronic eczema, mother asthmatic.	Fed on cow's milk.	Child irritable since 2 years. Diagnosed by the writer as a probable case of biliary cirrhosis in 1934. Treated outside the profession.
1937 12	Devadoss, male, 2 years, c/o Mr. Doraisami Naidu (Tirutani) Hindu, Non-Brahmin, rich class.	Chronic constipation since birth.	Mother died of eclampsia after delivering the patient. A previous child died of hepatic cirrhosis.	Fed on cow's milk.	Chronic constipation and fever were frequent troubles almost since birth.
13	Mani c/o Mr. B. A. (Gopalapuram, Madras), male, 3 years, Hindu Brahmin, vegetarian, rich class.	Peevishness and irritability—1 year.	Father asthmatic, mother had toxic albuminuria during pregnancy. First child is the patient. 1-1-45—mother seen by the writer for jaundice. Liver dullness slightly diminished, spleen palpable, has now 3 daughters; the last 2 months old. Urine of mother: albumen a trace present. Bile salts absent. Bile pigments present and urobilin in excess. Motion whitish and shows no ova. Blood: Kahn & Wasserman negative. Total WBC 5,800. Bleeding time 0:55" Coagulation time 1:55"	Brestfed and fed on cow's milk.	June 1937—seen by writer for peevisshness. Liver was felt 2½" below ribs and rather hard. April 1937—Diarrhoea and irregular fever. October 1937—The child had typical picture.

Cases—Contd.

Symptoms	Clinical data, investigations	Additional notes
Fever and constipation present.	Thin baby, anæmic. Abdomen tumid, liver hard and 2". Spleen just palpable. Widal negative. Blood smear shows no parasite. Kahn of father's blood negative. (Seen as a possible liver cirrhosis in 1934).	Recovered. 5-9-1939 A boy of 7½ years of age, 3' 10". Weight 37 lbs. Liver hard and just palpable. Patient is asthmatic.
Fever, bronchitis and bloated abdomen.	An irritable boy. Abdomen tumid. Liver hard and 1". Spleen palpable. Lungs: Bronchitis present. Kahn and Wasserman of father's blood negative.	Treated outside. Recovered. 22-11-1939.—Patient came with a short fever. Height 3' 4". Weight 32 lbs. Chest 20" 19½". Tonsils enlarged. The patient has typical asthma.
Fever, recurrent bronchitis and bloated abdomen.	A thin girl. Abdomen distended, liver hard and ½" below costal margin, spleen palpable. Lungs: Breath sounds harsh with sonorous and sibilant rhonchi all over—asthmatic. November 1937 X-rays lungs: Hilar shadows dense with marked streaking upward and downward. Mother's blood: Kahn negative, 1934. Father's blood Kahn negative, 1934.	March 1939. Patient apparently well. December 1944: Treated by the writer for asthma.
Fever, generalised oedema jaundice and protuberant abdomen.	24-8-1937. Marasmic child. Generalised dropsy and slight icterus present. Ascites present. Febrile. Liver hard and 2½" and spleen 1½". Interscapular area of chest: dull to percussion. Heart: both sounds rapid and weak.	Treated "Cured" January 1938: Patient much better and moves about in the house.
Fever and distended abdomen since April 1937.	October 1937. Irritable child. Febrile. Abdomen distended. Liver hard, painless and 3 inches enlarged; and spleen 1" enlarged. Heart: Rapid Ec.g revealed auricular flutter. Soon developed generalised oedema and jaundice. Urine: Reduces Benedict's solution. No albumen.	Treated. Died in January 1938.

Appendix of

Serial No. Year of observation.	Name, age, sex, social status, caste, dietary habits	Complaints, duration	Family history	Previous history feeding	History of present illness
14	Gopal, male, 3 years 4 months (19, Pedariar Koil Street, Madras), Hindu, Brahmin, poor class, vegetarian.	Irregular fever and cough since January 1936.	Mother had hyperemesis gravidarum during pregnancy. Father susceptible to migraine. Patient is the 6th of 7 children.	Child born on 4th September 1934. Fed by mother and on cow's milk.	Treated for Kala Azar with 4 Neostibosan to no purpose.
15	Raman c/o Mr. Ramachandrarajah (G. T. Madras), male, 1 year 7 months, Hindu, Brahmin, vegetarian, middle class.	Constipation alternating with diarrhoea — 1 year.	Three other children died of liver enlargement. Father died of pernicious anaemia. Wasserman and Kahn of father negative.	...	...
16	Vijayalakshmi, female, 2 years 8 months old. (Perambur, Madras), Hindu, Brahmin, vegetarian, middle class.	Constipation or diarrhoea — 1½ years.	Patient is the 2nd child. Two others are alive and well, mother epileptic? Maternal grandmother asthmatic.	Fed on cow's milk.	Fever in July 1937.
17	Rajam, male 4, years (Bombay), Hindu, Brahmin, middle class, vegetarian.	Chronic constipation—3 years.	Mother and maternal grandmother asthmatics. Father asthmatic.	Fed on cow's milk.	Pneumonia in October 1937. Very liable to colds in chest and diarrhoea which alternates constipation.
18	Rajagopal c/o Mr. S. R. (Madras), male, 1 year, Hindu, Brahmin, middle class.	Chronic constipation since birth.	Mother asthmatic, father has filarial scrotum.	Breastfed and on cow's milk.	...
19	Ramakrishna Rao, male, 3 years and 10 months, (Madras), Hindu, Brahmin, middle class, vegetarian.	Protuberant abdomen since March 1934.	Paternal grandfather diabetic. Patient is first child, another baby 1 year old.	Fed on cow's milk.	Diarrhoea in March 1934 preceded the trouble.
20	Narasimharajendra Kumar, male, 3 years and 1 month old (Guntur) Hindu, Vysia, vegetarian, rich class.	Chronic constipation.	Maternal grandmother asthmatic.	Do.	...
21	Baby c/o Mr. A. K. P., female, 1½ years old (Madras), Hindu, Brahmin, lower middle class.	Chronic constipation since early days.	Mother asthmatic. An elder-sister to patient died of Bantis disease in 1939.	Fed by mother and on cow's milk.	...

Cases—Contd.

Symptoms]	Clinical date, investigation	Additional notes
Fever and cough and distended abdomen.	25-10-1937. Liver 3½" enlarged and hard. Spleen first palpable. Slightly anæmic. Ascites present. Subicteric tinge of conjunctivæ seen. A soft systolic murmur at apex heard. Dullness over interscapular zone elicited. Right supraclavicular lymph glands enlarged.	Treated from 24-10-1937 to 31-12-1937. Child improved. Later reported to have died in March 1939.
Fever and diarrhoea, and tumid abdomen.	Liver 2" enlarged, hard and painless. (Right lobe larger). Bronchitis present.	Not followed.
Fever present.	July 1937. Weak baby. Abdomen distended. Slight ascites present. Slight icteric tinge of skin and eyes present. Liver 2½" enlarged and hard. Spleen 1½" enlarged. August 1937. Diarrhoea supervened.	Treated from July 1937 to 8-1-1938. Child improved. Later reported to have died.
Constipation and distended abdomen and respiratory distress.	Thin boy. Abdomen distended. Liver hard and 1½" below costal margin. Spleen just palpable. Lungs: Breath sounds asthmatic.	Not treated. January 1942 patient alive and well.
Constipation and abdominal disfunction.	Thin baby. Peevish. Tumid abdomen. Liver 1½" enlarged and rather hard. Spleen ½" enlarged. Wasserman and Kahn of father's blood both negative.	Not treated. December 1941. Child alive and well.
Protuberant abdomen and foul motions—3 years.	Ascites present. No jaundice. Liver hard and 2" (right lobe larger), spleen hard and 1". Heart sounds weak.	Not followed.
Constipation and bronchitis.	26th September 1937. An irritable baby. Abdomen tumid. Liver hard, painless and 2½". Spleen very hard and 2" Lungs: Breath sounds harsh and rhonchi heard.	..
Constipation and fever.	19-10-1937. Thin and slightly jaundiced baby. Abdomen distended with slight free fluid. Liver hard and 1½" below costal margin. Spleen 1" below costal margin. Kahn and Wasserman of mother's blood negative.	19-10-1937 to 1-12-1937 treated. Patient died in intense jaundice.

Appendix of

Serial No. Year of observation.	Name, age, sex, social status, caste, dietary habits	Complaints, duration	Family history	Previous history, feeding	History of present illness
22	Narasimha Reddi, male, 2 years and 8 months, (Maduranta- kam, Chingleput Dis- trict), Hindu, Kapu, rich class, mixed diet.	Constipated generally. Coughs and colds—2 years.	...	Fed by mother till 3rd month and later on cow's milk.	Addicted to castor oil which upsets the baby.
23	Rajasekar, c/o Mr. A. T. C., male, 9 months old, Hindu, Vysia, rich class, vegetarian.	Chronic constipation since early days.	Mother asth- matic.	Fed by mother and on cow's milk.	...
24	Vedadri c/o Mr. N. R. C. (Madras), male, 1 year, Hindu Vysia, mixed diet, rich clas.	Chronic con- stipation since early days.	Father asth- matic, mother diabetic, elder sister asth- matic.	Fed on cow's milk.	Fever and constipation and diarrhoea are usual inci- dences.
25	Sreepathi c/o Mr. P. G. Naidu, male 3 years (Madras) Hindu, Non- Brahmin, middle class, mixed diet.	Fever and diarrhoea—2 months.	...	Whooping cough was treated with a patent con- taining chloral derivative and chloroform for a long time, 3 months.	Cough, fever and diarrhoea started after pertussis.
26	Bhakthavatsalu c/o Mr. Venkatanarayana Chetti, male, 1 year 1 month (Madras), Hindu, Vysia, rich class, vegetarian.	"Cyclic diar- rhoea" (whi- tish diarrhoea) and bronchitis 6 months.	Mother and sisters asth- matic.	Frequent whitish diar- rhoea.	...
27	Dinakar c/o Mr. Jeeva Rathnam, male, 2 years (Madras), Hindu, Non- Brahmin middle class, mixed diet.	Frequent fevers and— colds—6 months	Father asth- matic mother had eclampsia at the time of labour deliver- ed the patient with prostra- ted labour. Patient is first child.	Fed by mother and on cow's milk.	...
28	Pattabhiraman (Perambur, Madras) male, 2 years, Hindu, Brahmin vegetarian, middle class.	Fever and diarrhoea—15 days.	Father asth- matic, mother had genera- lised oedema during preg- nancy.	Fed on cow's milk.	Vaccinated at 1½ year of age; and later irregular fever started with diarrhoea.
1938 29	Ramachandran, male, 1½ years (Chingleput) Hindu, Non-Brahmin, mixed diet, rich class.	Constipated generally (pas- sed hard whi- tish lumps, 1 year.	Father is a victim of hyperchlor- hydria, mother migrainous.	Fed on cow's milk only.	...

Cases—Contd.

Symptoms	Clinical date, investigation	Additional notes
Constipation upset by cas- tor oil.	27-10-1937. Thin baby. Liver hard and nearly 2" enlarged. Lungs: Signs of chronic bronchitis. X-rays chest. (2-11-1937): slight infiltrative changes in right cardiophrenic angle. E. C. G. (4-12-1937): Simple tachy-cardia.	Not followed.
Constipation.	5-11-1937. An irritable baby. Abdomen distended. Liver rather hard and 2" enlarged. Spleen just palpable. December 1937: Had severe diarrhoea and liver got bigger. Treated.	January 1945. Child alive and well.
16-9-1937. Fever and diarrhoea.	16-9-1937. Thin baby. Abdomen tumid. Liver hard and 2" enlarged. Spleen just palpable. Lungs: Bronchitic signs present. (Father's blood: Kahn and Wasserman negative): Treated with success.	December 1944. Child alive and well. Asth-matic now.
Fever and diarrhoea.	15-11-1937. Abdomen tumid. Liver hard and 2" enlarged. Spleen just palpable. Treated with pertussis Vaccine and full doses of Vitamins (especially C) with good result.	31st December 1937. Child better and appears normal.
Diarrhoea and bronchi- tis.	20-11-1937. Abdomen distended. Liver rather hard and 2" enlarged. Spleen palpable. Lungs: Bronchitis present. Treated.	December 1944. Child alive and well.
Frequent colds and coughs and fever.	2-12-1937. Child has a protuberant abdomen. Liver 2" below costal margin—left lobe lager. Lungs: Signs of bronchitis present. Treated.	December 1944. Child alive and well.
Diarrhoea and fever.	6-12-1937. Ill-nourished baby. Chest rachitic. Abdo-men distended. Liver 3" enlarged and rather hard—(right lobe larger), slight ascites present. Icteric tinge of skin and eyes seen.	March 1945. Child is cured. Now asthmatic.
Distended abdomen and constipation.	January 1938. Fairly healthy child. Tumid abdo-men. Liver hard and 1½" below costal margin. Spleen just palpable. Advised treatment. 25-9-1944. Wasser-man and Kahn of parents' blood negative.	25-9-1944. Height 3' 9," weight 42 lbs. Liver hard and ½" below costal margin. Lung: asth-matic breathing. The boy is healthy but for asthma. Gel and Chopra: Negative. W.B.C. Total 6,300 per c.m.m. Diffe-rentially, p. 52 per cent. Lymphocytes 31 per cent. Monas 2 per cent. Eosinophiles 15 per cent.

Appendix of

Serial No. Year of observation.	Name, age, sex, social status, caste, dietary habits	Complaint- Duration	Family History	Previous History Feeding	History of present illness
30	Baby c/o Mr. Achicha male, 2½ years (Madras), Hindu, Brahmin, vege- tarian, poor class.	Distended abdomen — 6 months.	Maternal side asthmatic	...	...
31	Baby c/o Mr. V. R. (Madras) male, 9 months, Brahmin, vegetarian, middle class.	"Cyclic diar- rhœa."	Father sus- ceptible to migraine in early years and now a diabetic.	Fed on Allen- bury's and by mother.	Does not tolerate cow's milk and cas- tor oil. Easily gets a diarr- rhœa.
32	Baby c/o O. K. Appa Row, male, 2 years (Chin- gleput) Hindu, Non- Brahmin, middle class, Non-vegetarian.	Distended abdomen 1½ years.	Maternal side asth- matic.	...	...
33	Baby referred by Mr. B. B. J., male, 2 years (Madras) Gujarati Brah- min, vegetarian, middle class.	"Cake in abdomen — 1 year.	Mother asth- matic.	...	...
34	Lakshmiprasannam (Nellore district), female, 9 months, Hindu, Brah- min, middle class, vegeta- rian.	Bloated ab- domen, breath- lessness and irritability one month. Wast- ing—2 months.	Mother mig- rainous and is subject to fits (epilep- tic?)	Mother preg- nancy normal, labour pro- tracted. Child was breastfed for one month. Condensed milk was given during 2, 3, 4, months. Later cow's milk given and this resulted in pale motions, and at this juncture wet nursing start- ed and this improved the child slightly. Used castor oil	The use of condensed milk seems to have star- ted the disease
35	Muthukrishnan, male, 1 year and 9 months (Perambur Madras), Brahmin, middle class, vegetarian.	Protuberant abdomen and not thriving—9 months.	Father has filariasis mother asth- matic. Patient is 7th child. A little sister of this patient has developed cirrhosis liver.	Addicted to castor oil. Vaccinated in 6th month. Breastfed and on Horlick's milk.	Vomiting started in the 10th month. Since then going down.

Cases—Contd.

Symptoms	Clinical Data, Investigations	Additional Notes
Dyspnœa, distended ab- domen and cake in abdo- men.	A jaundiced baby. Dyspnotic. Ascites present. Liver and spleen both enlarged 2½" and both hard.	Not followed.
Pallor and whitish diar- rhœa.	An infant, slightly anæmic. Abdomen tumid. Liver enlarged 2" below costal margin. Treated with balanced diet and plenty of vitamins. (Kahn and Wasserman of father's blood negative, 1939).	19-1-1945. Saw the boy. Liver hard and ½" below costal margin, is healthy.
Distended abdomen and dyspnœa— fever.	Abdomen distended. Ascites present. No jaundice. Liver hard and 2." Spleen 2." Not treated.	Not followed.
...	A dull little boy. Abdomen distended. Liver hard and 2," spleen ½." No detectable ascites. No jaundice.	Reported to have died two months later.
Bloated ab- domen and dyspnœa. Fever and con- stipation.	21-7-1939. Weak and marasmic. Anterior fonta- nelle not closed. Not anæmic and not jaundiced. Abdomen protuberant and prominent, veins seen in anterior abdominal wall. Liver hard and 1½". Spleen 1½". Ascites present. Rachitic chest. Lungs show basal congestion. 23-7-1939. Kahn of child's blood negative.	Reported to have died later.
Loss of ap- petite and low fever.	Weak. Not anæmic. Coryza present. Tonsils dirty. Tonsillar glands enlarged. Chest rachitic. A few rhonchi heard over back of chest. No jaundice. Abdomen tumid. No ascites. Liver 1½" enlarged. Spleen not palpable. (Treated cured). Weights: 12-8-1939—21 lbs. and 2-9-1939—23 lbs. 18-8-1939. Kahn and Wasserman negative.	2-12-1939. Weight 24 lbs. 26-1-1940. Liver not palpable. 5-10-1944. height 3' 10", weight 42 lbs.

Appendix of

Serial No. Year of observation	Name, age, sex, social status caste, dietary habits	Complaint duration	Family history	Previous history, feeding	History of present illness
36	Balakrishnan, male, 3 years and one month (Madras), Hindu, Brahmin, vegetarian, middle class.	Bloated abdomen and thinned limbs—20 days.	Maternal uncle is asthmatic. Patient is the 5th child; the others alive and healthy.	Breast-fed and on sago congee and milk after third month. Not vaccinated. Frequently the child suffered from abdominal colic. Diarrhoea would be followed by constipation which was treated by castor oil. Child had eczema in 6th month.	After the completion of 3rd year the child was becoming peevish loosing flesh, and developing a protuberant abdomen.
37	Sankaran, male, 1½ years, (Madura), Hindu, non-Brahmin, vegetarian, rich class.	Fever, cough and vomiting—2 months. Constipation and sleeplessness—1 month. Swelling of feet—3 days.	Father susceptible to migraine. Mother asthmatic. Patient is the 4th of 5 children. Others alive and well.	Entirely fed on cow's milk. Weaned with rice in 12th month. Given castor oil.	Pertussis 2 months ago. Since then child is going down.
38	Krishnan, male, 1 year and 2 months (Erode), Hindu, Brahmin, vegetarian, middle class.	Whitish motions and distended abdomen—1 month.	Father of the child formerly asthmatic and now migraine. Patient is the 5th child; others are alive. The 3rd child (a boy of 3) died of liver enlargement. 4th child (a boy of 4 years) had eczema at 1½ years of age.	Breast fed and cow's milk. Given castor oil. Weaned in the 10th month with rice and rice combinations.	Vaccinated in the 7th month and about a week later developed generalised eruptions. Healed area obvious left thigh (9 photograph.)
39	Pattabhi, male, 2 years, (Madras), Hindu, non-Brahmin, mixed diet.	Constipation since early days and particularly after weaning.	Mother and father and maternal grandfather asthmatic.	Breast fed supplemented with Cow and Gate, and cow's milk. Weaned in 6th month.	(Child became definitely ill after vaccination 21-1-1940.)
40	Raju, male, 1 year 9 months (Madras), Hindu, Brahmin, vegetarian, middle class.	Recurrent whitish diarrhoea—1½ years.	Paternal aunts and uncle asthmatics. Father developed eczema in 1938.	Fed on cow's milk, castor oil given.	...

Cases—Contd.

Symptoms	Clinical data: investigations	Additional notes
Bloated abdomen and fever.	24-8-1939. Peevish and restless baby stays on bids present. Eczematous stamps discernible on the cheeks. Ascites present. Thin extremities. Feet dropsical. Not jaundiced. Axillary Temp. 100.2°F. Sleep disturbed. Bowels constipated, weight 24 lbs. Abdominal circumference (umbilical hernia present) 20". 1-9-1939. Weight 21 lbs. Abdominal circumference 17". Pulse 130. Axillary temp. normal. Drowsiness present. Liver and spleen not felt. Liver felt hardened by dip method. 2-9-1939. Malena noticed. 10-30 a.m. Seen in drowsiness and muscular twitching. 6 p.m. child expired.	Treated. Child died.
Fever and swelling of body and cough.	12-9-1939. Child is pale with generalised oedema. Pulse 120. Temp. 101° (axillary). Abdomen bloated with an everted umbilicus. Abdominal circumference 20". Ascites present. No jaundice. Liver 1½" enlarged. Spleen 1" enlarged.	Not followed.
Distended abdomen—cold.	23-9-1939. Abdomen protuberant. Nasal discharge present. Tongue clean. Teeth and bones normal. No jaundice. Abdomen: No ascites. Liver 2" enlarged uniformly. Spleen not palpable. Lungs: Rt inter scapular area dull. Treated with Adrenal cortex. 2-10-1939. Weight 17 lbs. 11-12-1939: X Rays chest taken for recurrent cough. Hilar shadows dense. 15-8-1939. Weight 19 lbs. Treated with colloidal calcium and vit D by injection. 16-4-1940. Weight 24 lbs. Liver ½" below costal margin. 17-4-1940 X Rays Lungs: left lung clear. Right lung field shows irregular opacities now much smaller than before.	This is post-vaccinal hepatomegaly—early cirrhosis. This case had to be studied along with another which developed cirrhosis of liver after vaccination and died of it. (Case No. 39 of 1939).
Cough, fever and constipation.	16-11-1939. Eczema present on cheeks. Abdomen: Liver 1½" Spleen not enlarged. Lungs: Breath sounds harsh with plenty of rhonchi. Father's blood-Kahn and Wasserman negative. 6-2-40. (vaccinated 14 days ago). Liver 1½" enlarged. The child did not improve on treatment outside and died.	Died.
Wasting and whitish motion.	24-9-1939. Chest rachitic. Abdomen: Protuberant. Liver 1" enlarged and rather hard. 25-1-1940. Liver 3½" and spleen 2". Right lobe of liver very much larger. No ascites or jaundice. Gums bleeding. A soft systolic murmur audible at apex. 3-2-1940. Abdominal circumference 20", weight 24 lbs. Pulse 120. Axillary Temp. 96.5°F. Jaundice very intense (tumeric yellow tinge all over the body and in the eyes) ascites present. Urine: No sugar. No albumen. Bile pigments present. Bile salts absent. 9-2-1940. Weight 25 lbs. Abdominal circumference 20½" chest 18½", liver 2½" below costal margin. Spleen 2" below rib margin. Ascites: less; but jaundice is intense. 14-2-1940. Abdominal circumference 21", liver 2½", Spleen 2", pulse 120; weight 24 lbs. Everted umbilicus (ascites present) jaundice very deep. 16-2-1940 cholæmia set in. 17-2-1940 Respiratory distress set in. 17-2-1940. 8 p.m. child expired.	Treated. Child died.

Appendix of Cases—Contd.

Serial No. Year of observation	Name, age, sex, social status, caste, dietary habits	Complaint duration	Family history	Previous his- tory, feeding	History of present illness	Symptoms	Clinical data : investigations	Additional notes
41	Veeraraghavan, male, 1 year 10 months old; (Saidapet), Hindu, non- Brahmin, mixed diet, poor class.	Fever and constipation —1 month.	Father mi- grainous, ma- ternal grand- mother asth- matic. Patient is the 3rd child in the family.	Brought up on breast milk and on cow's milk. Weaned at end of 1st year with rice and dall. Cas- tor oil given. Not vaccinated.	...	Yellow eyes. Protuberant abdomen.	22-10-1939. Chest rachitic. Yellow sclerosis, weight 18 lbs, pulse 100, Temp. 99°F (axillary). Abdomen: Abdominal circumference 17," Umbilicus everted. Ascites present. A systolic murmur heard at apex.	Not followed.
42	Subrahmaniam, male, 3 years. (Mylapore, Mad- ras), Hindu, Brahmin, vegetarian, poor class.	Cake in ab- domen : 1 year Dropsy—1 month.	Patient is the 4th child. 2nd child in the family died of liver enlarge- ment. Father migrainous and paternal uncle asthma- tic.	Brought up on breast till 3rd month. Later fed on conden- sed milk and cow's milk and Horlick's. "Virol" given during last 6 months; and Adexolin du- ring last 2 months. Wea- ned at the end of 1st year with rice.	The child took ill after pertussis in 11th month. Diarrhoea and fever one year ago. Vaccina- ted at end of 1st year.	Dropsy and distended ab- domen.	27-10-1939. Downy growth of hair over spine. Weak and dropsical over face and feet. Weight 20 lbs. Not anæmic cutting of finger and toe nails present. Abdo- men distended. Abdominal circumference 18." Liver 1½. Ascites present. Teeth well developed. Lungs: Diffuse bronchitis present. Heart: Nil particular. Temp: 100°F in evenings. 27-10-1939. Urine: no sugar. No albumen. No bile salts or pigments. Trea- ted with B. Coli vaccine and vitamins. 7-11-1939— weight 14 lbs. Dropsy decreased. Abdominal circum- ference 18". Liver 1½" enlarged. 19-11-1939 Weight 16 lbs. 25-11-1939. Low temperature and drowsiness seen.	Died.
Not counted.	Ramamurthi, male, 2 years, 3 months, c/o Mr. Cuniah Chetti, Hindu, Vysia, vegetarian, Rich class (Madras).	Constipation (chronic) and frequent fe- ver 1 year.	Mother died of typhoid fever at 25. Father asth- matic. Patient is the 5th child in the family.	Fed on cow's milk. Addic- ted to castor oil. Adexolin given. Wea- ned at 1½ years age with rice.	...	Constipation, cough and fever.	2-4-1939. Downy growth of hair present over spine. Abdomen protuberant. No ascites or jaundice. Liver 1" enlarged hard, and is uniform. Spleen hard and 1". Lungs: Sonorous and sibilant rhonchi heard. Kahn and Wasserman of father's blood strong positive. Treated with Grey Powder and "spinoid" (Bayer). The child took a course of typical infantile hepatic cirrhosis and died after developing ascites and jaundice in first week of December 1939.	This case suggests co- existence of hepatic cirrhosis and congenital syphilis.
43	Diwakaran, male, 11 months. (Royapettah, Madras), Hindu, non- Brahmin, middle class. (Patient is brother of No. 27 under 1937).	Fever, whit- ish diarrhoea, and sticky- eyelids—14 days.	Father asth- matic.	Breast fed and on cow's milk. Given Glaxo Bi- cuits. Weaned at 10th month with rice and dall. Vaccinated in 6th month.	Fever started with diarrhoea which is ema- ciating the baby.	Fever, whit- ish motions and wasting.	16-11-1939. Temp. (axillary) 102.5°F. Pulse 120 per min. Tongue coated. Patient dull and apathetic. Lids are sticky. Sclera icteric. Abdomen: Liver 2½" enlarged and uniformly hard. Spleen hard and 1" enlarged. No ascites.	Died in 1941 of a com- plicating pneumonia.
44	Jagannathan, male, 1 year 4 months old (Al- warpet, Madras), Hindu, Brahmin, vegetarian, rich class.	Diarrhoea—20 days.	Mother mi- grainous and maternal grand fathers asthmatics. Patient is the 3rd child.	Mother's la- bour preven- ture by 1 month. Breast fed and on Cow and Gate till 14 months. Weaned later with rice and dall. Weight at 10 months 7 lbs.	The child started this severe diar- rhoea after treatment for liver enlarge- ment by pow- ders.	Diarrhoea and wasting.	18-11-1939. Weight 6 lbs. Temp. 77.4°F (axillary) pulse 120 per min. Abdomen: Liver hard and 2½" en- larged. Kahn and Wasserman of mother's blood nega- tive. X Rays chest: Dense shadows at hilar. Heart large.	Died on 20-11-1939 pre- maturely. Probably pre- disposes to hepatic cirr- hosis.
45	Subrahmanian, male, 1 year 11 months. (Golla- wari Agraharam, Ton- diarpet, Madras), Hindu, Brahmin, middle class.	Thinning of extremities—1 month.	Father mi- grainous, first and second child died of liver enlarge- ment. Pa- tient is 3rd child.	Breast fed. Fed also on Cow & Gate 1½ cream till 6th month; and then with full cream till 1 year. Later cow's milk. Given castor oil. Not vacci- nated. Rice given at one and half years. Thumb sucking present.	Diarrhoea is frequent.	...	28-11-1939. Abdomen protuberant. Liver hard and 1½" enlarged. No ascites or jaundice.	Not followed.

Appendix cases—Contd.

Serial No. Year of observation	Name, age, sex, social status, caste, dietary habits	Complaint duration	Family history	Previous his- tory, feeding	History of present illness	Symptoms	Clinical data: investigations	Additional notes
46	Radhakrishnan, male, 2½ years, (Ekambares- warar Agraharam, Mad- ras), Hindu, Brahmin, poor class.	Diarrhoea 15 days.	Mother gets migraine.	Treated for liver enlarge- ment outside profession. Accustomed to castor oil.	...	Diarrhoea white and ul).	12-12-1939. Dull boy. Abdomen tumid. Liver hard and 2" enlarged. Spleen not palpable. (Right lobe of Liver larger.)	Not followed.
47	Kameswara Sarma, male, 3 years 3 months, (Nellore), Hindu, Brah- min, vegetarian, middle class.	Upset by cas- tor oil and oil baths. Diffi- culty in brea- thing at nights and cough—2 years.	Mother asth- matic.	Treated at Nellore for hepatic cirrho- sis during 2nd and 3rd year.	...	Constipation, roncho-spa- n and cough.	23-12-1939. Height 2 feet 11 inches. Chest 19"—18½". Weight 24 lbs. Abdomen: Tumid. Liver very hard and ½" below costal margin. Lungs: Diffuse bronchitic signs. X-Ray chest: Right hilar dense; Kahn's and Wasserman's of father's blood—Negative.	...
1940 48	Sivasundari Bai, fe- male, 1½ years old, (Jam- malamadugu), Hindu, non-Brahmin, poor class.	Bloated ab- domen and fe- ver—2 months.	Maternal grand mother asthmatic.	During preg- nancy mother had hypereme- sis gravidarum. Fed by mother and on cow's milk. Weaned with rice and pep- per water after 1st year. Rice used to be vomited. Given castor oil.	Vomiting and diarrhoea at 1½ years. Whoop- ing cough in December '39.	Bloated ab- domen and fever—2 mon- ths.	10-1-1940. Pale child. Dropsy of feet present. Abdomen protuberant. Liver enlarged 2" (right lobe layer). Spleen just palpable. Ascites present. No jaundice. Lungs: breath sounds harsh. 14-1-1940. Weight 20 lbs. Liver 2½". Abdominal circumference 19 inches. Temp. 98°F in morning. 102" in evening. Pulse 140 per min. 18-1-1940. Weight 21 lbs. Liver 2". Abdominal circumference 18½ inches. Temp. 101°F (axillary) in evening. 22-1-1940. Weight 18 lbs. 26-1-1940. Weight 20 lbs. Chest 18 inches. Abdo- men 19 inches. 28-1-1940. Weight 18 lbs. 2-2-1940. Weight 20 lbs. 8-2-1940. Persistent vomiting present 14-2-1940. Weight 20 lbs. 20-2-1940. Weight 20 lbs. 6-3-1940. Weight 20 lbs. Abdomen 19½ inches. Liver 2½ inches below costal margin. Spleen not palpable. 10-2-1940. Kahn and Gel of baby's blood negative.	Child died in 3rd week March 1940.
49	Venkatashubramaniam, male, 2½ years (c/o Mr. Venkatachala Iyer, Tin- nanur) Hindu, Brahmin vegetarian, middle class.	Bloated ab- domen and dropsical feet —4 months.	Mother asth- matic. Patient is the only child through second wife.	Mother's pregnancy complicated by hypereme- sis gravidarum and dropsy. Breastfed till 3rd month and later conden- sed milk used. Later cow's milk till 1 year. Weaned with rice after 1 year. Castor oil given.	The child was taken along with parents who went on long pilgrimage. Diet was bad- ly attended to.	Protuberant abdomen, and yellow eyes. Ascites pre- sent.	17-1-1940. A child in great misery, weight 20 lbs. Abdomen distended with free fluid, ascites present. Prominent veins on outer abdominal wall. Abdominal circumference 21." Liver hard and 3" below rib margin. Spleen 1½". Dropsy of feet present, jaun- diced sclerotics and skin not appreciably yellow. Pulse 120 per min. 23-1-1940. Weight 16 lbs.; 27-1-1940. weight 20 lbs.; 28-1-1940. Urine Benedict's reduced. No albumen. Bile pigments and salts absent; 31-1-40. Weight 18." Abdominal circumference 19." Temp. 100-5°F, pulse 140 min. 6-2-1940. Weight 19 lbs. 15-2-1940. Weight 19 lbs.; liver 1½" and spleen 1" enlarged. Pulse 120 per min. Child very drowsy and dropsical; 17-2-1940. Passed one round worm at 7 a.m. per os; 18-2-1940. Coma present, pulse 120; 6 a.m. vomited red blood. 19-2-1940. Very restless night. Respiration rattling, pulse 120; poor volume and fension, deep coma.	Died on 19-2-40.
50	Baby c/o Mr. A. Gopala Rao, (Guntur), male, 11 months, Hindu, Brahmin, rich class, vegetarian.	Whitish motions and distended abdomen—5 months.	Paternal uncle and cousins asth- matics.	Fed on Allen- bury's. Change to cow's milk produces whitish diarrhoea. Fat in- tolerance? Given rice and pepper water. Sensitive to castor oil, and citrus fruits. The writer saw this baby with ecze- ma and asthma in 7th month after giving cow's milk per- sistently.	The trouble is reported to have started after giving cow's milk in the 6th month which pro- duced whitish diarrhoea.	Diarrhoea, distended ab- domen.	30-1-1940. Abdomen distended. Liver 1½" below costal margin (rather hard) advised elixir lactated Pepsin and Vitamins and continued Allenburys; 13-3-1940. Liver 1½" has now whooping cough (treated by vaccine). 25-3-1940. Having rice in diet; 7-4-1940. Liver 1" below rib, weight 13 lbs. 22-4-1940. Liver just palpable, weight 17 lbs. 21-5-1940 Weight 16 lbs.; 1-1-1941. Cured and alright.	Treated (early stage) cured.

Appendix o Cases—Contd.

Serial No. Year of observation	Name, age, sex, social status, caste, dietary habits	Complaints, duration	Family history	Previous history, feeding	History of present illness	Symptoms	Clinical data: investigations	Additional notes
51	Satyavathi, female 2½ years (c/o Mr. P. V. R) Murthy, Hindu, Brahmin middle class, vegetarian.	Difficulty in breathing. Constipation.—2 months.	Father and mother susceptible to migraine. Father has gastric ulcer.	Mother's pregnancy complicated by anæmia. Labour premature by 1 month.	Dietetic irregularity—feeding with adults type of food—during convalescence from fever started trouble.	Distended abdomen and dyspnoea (asthmatic).	31-1-1940. Distended abdomen. Liver 1" enlarged. Chest: Diffuse bronchitis treated.	15-1-1945. Child doing well.
52	Raman, male, 2 years and 3 months (c/o Mr. M. B. Iyer G. T. Madras) Hindu, Brahmin, vegetarian, middle class.	Chronic constipation and diarrhoea—1 year.	Mother and sister are asthmatics. Six others previous to the baby died of hepatic cirrhosis.	Not breastfed. Brought up on Allenbury's foods till 9 months; later weaned with barley water and tapioca rice congee.	In April 1939, the child had virulent small-pox. In May and June 1939 careless feeding was indulged in; dosai, idli, vadai, etc. given and then liver was found, gradually enlarging. In early half of June 1939 no ascites or jaundice was present but was losing flesh.	Distended abdomen, scanty urination and constipation.	30-7-1939. Abdomen bloated. Ascites present. Abdominal veins present. No jaundice. Oedema of feet present. Constipated. Urine scanty. Liver 1" and hard. Spleen 1". 4-8-1939. Weight 22 lbs. Abdomen 20½"; 10-8-1939. Yellow sclerotics seen, weight 21 lbs. Abdomen 19½"; Axillary temp. 97.5°F. Pulse 120 p.m.; 11-8-1939. Liver and spleen each 1" enlarged and both hard.	Died.
53	Nagalakshmi, female 1 year (39, Ekambreswarar Agraharam, Madras) Hindu, Brahmin, middle class, vegetarian.	Chronic constipation—1 month.	Mother asthmatic.	Mother's pregnancy complicated by albumenuria. Eclampsia was feared as B.P. was remaining high. Labour normal. Birth weight of baby 6½ lbs. Breastfed till 8th month and also Allenbury's. Given castor oil pi.	Vaccinated in the 8th month and later vomiting started; this slowly led to chronic constipation and bloated abdomen and a state of chronic ill health.	Distended abdomen and chronic constipation.	31-1-1940. Weight 15 lbs. Abdomen tympanitic. Liver 2" and spleen ½" enlarged both hard. Not anæmic. Eyelids puffy. Lungs: nil particular; 22-4-1940. Liver very hard and 2½" enlarged. Spleen ½" enlarged; Distended veins seen in anterior abdominal wall—Ascites present. No jaundice. Constipation present.	Not followed later than 22-4-1940.
54	Dattatreya, male, 11 months, (G. T. Madras), Hindu, Non-Brahmin, vegetarian, rich class.	Whitish motions. Difficulty in breathing at nights—4 months.	Father asthmatic. A child older to the patient died of typical biliary cirrhosis.	...	...	Distended abdomen, and chronic constipation.	29-1-1940. Abdomen tympanitic. Liver 1½" enlarged, rather hard. Lungs: Sonorous and sibilant rhonchi heard. 1-3-1940. Vaccinated. Ten days later developed generalised vaccinia; 10-3-1940. Liver got more enlarged (3" below costal margin), treated, cured.	December 1944. Child alive and well. Now asthmatic.
55	Baya, female 2½ years old. (Perianaikaran street, G. T. Madras). Marwari, vegetarian rich, class.	Constipation alternating with diarrhoea—6 months.	Mother asthmatic.	Over feeding with cow's milk and fatty puddings present.	The child is generally constipated. Treated with castor oil which produced diarrhoea and bronchitis. Sensitive to castor oil?	Cough diarrhoea.	1-2-1940. Pale and weak baby. Abdomen tumid. Liver 2" enlarged and spleen ½". Lungs: Diffuse bronchitis present.	December 1940. Alive and well.

Appendix cases—Contd.

Serial No. of observation	Name, age, sex, social status, caste, dietary habits	Complaints, duration	Family history	Previous history, feeding	History of present illness	Symptoms	Clinical data, investigations	Additional notes
56	Viswanath Bhatt, male, 3 years, (c/o Mr. Khishnaji Bhatt, Sowcarpet, Madras) Gujarati Brahmin, vegetarian, middle class.	Whitish diarrhoea; Bloating abdomen — 2 months.	Both parents susceptible to migraine.	Mother's pregnancy complicated by vomiting. Labour normal. Breastfed till end of 1 year, then rice given.	Bloody dysentery with proctoprolapse and rectal prolapse. 2 months ago distension appeared to start the illness.	Chronic constipation and bloated abdomen.	25-2-1940. Weight 24 lbs. Abdomen tumid. Liver 2" enlarged. Spleen not palpable.	Not followed.
57	Seetha, female, 2 years 7 months (Ibrahim Sait Colony, Madras) Hindu, Brahmin, middle class, vegetarian.	Constipated after 1st year.	Mother asthmatic, maternal uncle asthmatic.	Mother anæmic during pregnancy. Breastfed till 3rd month; and later with Lactogen. Rice started at 1 year.	Constipation appeared after 1 year. Diagnosed as "rickets" at 1 year and 3 months and treated.	Constipation and bloated abdomen.	31-3-1940. Weight 29 lbs. Liver enlarged 1½"; and hard (left lobe larger). Chest rachitic.	Do.
58	Vasantha, female, 2 years and 1 month (referred by Mr. Krishna-swami Iyengar, Dadha & Co.) Hindu, Brahmin, vegetarian, middle class.	Cough and constipation — 15 months.	Mother migrainous.	Pregnancy complicated by anæmia. Breastfed till Feb. 1940. Cow's milk given. Sago congee in 6th month.	Vomiting in Dec. 1938. Dysentery in July 1939. Small-pox in Oct. 1939, in Nov. 1939 had fever and serious diarrhoea.	Distended abdomen. Dropsy of feet, cough.	25-3-1940. Emaciated baby, weight 14 lbs., anæmia present. Dropsy feet and ascites present. Liver 1½" enlarged and hard. Spleen 1." Lungs: rhonchi heard.	Do.
59	Sadasivasukkal, male, 3 years, Gujarati Brahmin, middle class, vegetarian, Madras.	Bloated abdomen and dropsy — 1 month.	Paternal grandfather asthmatic, mother migrainous.	Breastfed, also cow's milk till 1½ years. Weaned at 1½ years with rice. Vaccinated in 2nd year.	Fever of one month preceded the dropsical abdomen.	Dropsy, sore lips and angular stomatitis fever and cough.	18-4-1940. Ascites abdomen: Liver and spleen difficult to palpate. Angular stomatitis present.	Do.
60	Ramani Bai, female, 7 months (c/o Mr. Rajarathnam Mudaliar, Madras) Hindu, Non-Brahmin, mixed diet, middle class.	Constipation — 1 month.	Mother asthmatic. An elder baby died of cirrhosis of liver at 1½ years.	Accustomed to castor oil. Fever and diarrhoea are common.	...	May 1940: The child was watched as a prospective cirrhosis case. Advised milk of magnesia. Diet adjusted. Castor oil stopped.	2-1-1941. Ascites. Abdomen: Liver finely nodular and 2" enlarged. Spleen 1½" enlarged. No jaundice.	Do.
61	Baby c/o Mr. Doraisami Iyer, male 3, years, Madras.	...	...	...	The child took ill after vaccination and developed a protuberant abdomen.	...	December 1940. Ascites. Abdomen: Liver hard and 3" enlarged, spleen 2".	Do.
62	Baby, c/o Mr. A. K. Pandya (Kodambakkam — Chingleput), female, 1 year, Gujarati Brahmin, middle class, vegetarian.	Yellow body — 15 days.	Mother and brother of patient are migrainous.	Breastfed and on cow's milk.	...	...	December 1940. Intensely jaundiced baby. Ascites present as also puffy eyes and swollen feet. Liver hard and 1½" below costal margin. Spleen 1".	Died 2 days after observation.

Appendix of Cases—Contd.

Serial No. Year of observation	Name, age, sex, social status, caste, dietary habits	Complaints, duration	Family history	Previous history, feeding	History of present illness	Symptoms	Clinical data: investigations	Additional notes
63	Baby c/o Dr. Pancholi of Central India, male, 3 years and 1 month, vegetarian, middle class.	(Brought to writer as a case recover- ing from en- largement of liver, and spleen on 20-4-1940) com- plaint consti- pation and dyspnoea at nights-1 year.	Maternal grand mother asthmatic, only child. Another child after the patient born at full term and died 12 hours after birth; cause of death being oro-pharyn- geal isthmus.	Mother's pregnancy complicated by jaundice for some months, labour normal, child born on 13-3-1937, Birth wt. 4 lbs fed on Cow and Gate only. March to June 1937:—Fever	...	Protuberant abdomen and nocturnal dyspnoea.	20-4-1940. Child seen with bronchospasm at night. 21-4-1944. Abdomen protuberant. Liver rather hard and 1" below costal margin, spleen $\frac{3}{4}$ " enlarged. Lungs: bronchitis present, heart, nil particular; mother's blood—Wasserman negative before and after provocative dose. 29-5-1940. X-rays chest: nil parti- cular, X-rays wrist: only 2 corpal centres seen; blood: no parasite leucopenia present. Macrocytic anæmia present, Gel and Chopra negative. 30-5-1940. Wt. 20 lbs. 13-6-1940. Wt. 22 lbs. liver $\frac{1}{4}$ ", spleen palpable.	This case shows points of great interest. Aller- gic history in parents. Child's dietary was badly tackled. Cirrhosis treat- ed by scientific system right through and cured. Discharged "cured" from Madras on 13-6-'40.
64	Ramamurthi, male, 1 year (G. T. Madras). Hindu, Brahmin, middle class, vegetarian.	Distended abdomen — 6 months.	M o t h e r tuberculous. Father also re- covered from abdominal tuberculosis.	Diarrhoea in June 1940.	...	Abdominal distension.	13-8-1940. Child looks fairly well fed. Tongue clean, abdomen distended, no dilated veins, no detectable ascites, liver 2" and spleen 2" enlarged (Wasserman and Kahn of mother's blood negative); motion: nil particular; urine: no bile salts or bile pigments urobilin subnormal, later developed ascites; discharged from Stanley Hospital on 5-9-1940 in pre-cholemic stage.	Died.
1941 65	Babjee, female, 10 months old (Madras), Hindu, Brahmin, vege- tarian, middle class.	Bloated ab- domen — 2 months.	Nil parti- cular.	...	...	Distended abdomen and foul motions. Fever and jaundice pre- sent.	Admitted into Stanley Hospital on 22-1-1941 and discharged in a cholæmic stage on 29-1-1941. Abdomen distended, fever and jaundice present, ascites and dis- tended veins present, liver 2" enlarged and hard; spleen $1\frac{1}{2}$ " below costal margin; heart: rapid, a soft systolic murmur present at apex; lungs: basal con- gestion present; urine: acid, albumen present; bile salts and bile pigments present; faeces: whitish and pasty, (Kahn and Wasserman of father said to be negative when investigated for his deafness).	Died.
66	Gopalakrishna, male, 1 year, Hindu (Madras), middle class.	Bloated ab- domen — 6 months.	...	...	...	...	Admitted into Stanley Hospital on 1-1-1941: dis- charged on 8-1-1941; clinical: anæmia present, liver hard and enlarged 2", spleen $1\frac{1}{4}$ " enlarged.	Not followed.
67	Baby c/o Mr. Vepa, male, 2 years (Madras), Hindu, Brahmin, vege- tarian, rich class.	Bloated ab- domen — 6 months.	...	...	...	...	A boy in ascitic stage. Abdomen dropsical, ascites very tense, (consulted for paracentesis), icterus slight, liver felt hard and 2" below costal margin by dip-method; spleen not felt easily—about 1 inch below rib margin.	Do.
68	Lalitha, female 2½ years old, (Madras), Hindu, Brahmin, vege- tarian, middle class.	Fever and distended ab- domen—1 year.	Father asth- matic and sus- ceptible to urticaria very often, another child older to patient died of cirrhosis liver.	Breastfed and on Glaxo milk.	Frequent fevers set in and leave the child weaker.	Distended abdomen and dyspnoea.	A thin girl baby, ascites present, distended veins seen; liver hard and 2" below costal margin, spleen 1" enlarged; jaundice appeared 2 days before death; (Kahn and Wasserman of both parents negative on two different occasions).	Treated. Died. This child was observed by the writer from birth to death, cirrhosis was expected and carried the baby.

Appendix of cases—Contd.

Serial No., Year of observation	Name, age, sex, social status, caste, dietary habits	Complaint; Duration	Family History	Previous History, Feeding	History of present illness	Symptoms	Clinical data: investigations	Additional notes
69	A brother to 68, male, 1 year.	Yellow eyes and urine—1 week.	As in 68.	As in 68.	...	...	The baby was seen from birth to death by the writer. Seen in a condition of fever; bronchitis and distended abdomen on four occasions; abdomen dis- tended; liver hard and 1½" enlarged; spleen palpable, no detectable ascites, jaundice present; ascites set in as a terminal event just 4 days before death.	Treated and died.
70	Sitha, female, 3 years 4 months (Madras), Hindu Brahmin, middle class.	Whitish motions and distended abdomen—6 months.	Mother had eclampsia during first delivery. Second child is the patient.	Breastfed, also on Glaxo. Parboiled rice conjee also given.	...	Swollen ab- domen.	2-1-1941. Abdomen distended, ascites present, not jaundiced, liver hard 2½" below costal margin, spleen 2" below costal margin, no jaundice.	Not followed.
1942								
71	Bhargavi, female, 9 years old, Hindu, Brah- min, vegetarian, middle class.	Fever.	Mother a victim of mig- rairie and asthma. Patient is the 2nd out of 3 children.	...	...	...	Admitted into Stanley Hospital, on 26-8-1942; discharged on 18-9-42; 26-8-42 wt. 21 lbs., abdomen turgid, liver 1½" below costal margin, spleen not pal- pable. Blood: no parasite leucopenia present. Urine: no albumen or sugar, faeces: no ova. Blood widal: positive; 1 in 25; 3-9-42 liver 1" below costal margin and hard.	Not followed. (This is probably a febrile phase of early hepatic cirrho- sis).
72	Sundaram, male, 4½ years, Hindu, mixed diet, middle class (Madras).	Protuberant abdomen—15 days.	Mother epi- leptic, patient is first child.	Mother's pregnancy complicated by hypereme- sis gravidarum. Brought up on cow's milk. Rice started at 1 year.	Cyclic diar- rhoea (whitish diarrhoea periodically remaining) was present during last two years.	Distended abdomen and jaundice.	Admitted into Stanley Hospital 6-2-42, discharged 6-2-42. Skin and sclerotics yellow, abdomen distended with prominent veins on anterior abdominal wall, dropsy of lower limbs present, downy growth of hair present over spine, liver hard and 2½" below rib margin; heart: a soft systolic murmur audible inter- nal to apex.	Not followed.
73	Anglo-Indian, female child, 1½ year old, middle class (Madras).	Protuberant abdomen and whitish motion—1 month.	Father has a dyspepsia— hyperacidity?	Mother had toxic albumi- nuria during pregnancy.	...	...	A baby yellow in eyes and skin. Abdomen distended, ascites present, liver hard and 1½" below costal margin, spleen just palpable, (Kahn and Wasserman of father negative).	Do.
74	Balasubramaniam, male, 1½ year old, Hindu (Madras).	Protuberant abdomen—1 month.	Mother vic- tim to mig- rairie and asthma.	Dysentery 1 month previ- ous to the complaint.	...	Distended abdomen.	Admitted in the Stanley Hospital 20-10-42, dis- charged from 20-10-42, abdomen distended, liver 2½" enlarged and hard, spleen not palpable, no ascites. Heart: tic tac rhythm. Lungs: breath sounds puerile.	Early hepatic cirrho- sis, not followed.
1943								
75	Ramasundaram, male, 1 year, Hindu Non- Brahmin, vegetarian (Madras).	Bloated ab- domen—15 days	Patient is the only child.	Dysentery present, one month prior to complaint. Breastfed, given cow's milk.	...	Abdominal distension.	Admitted into Stanley Hospital 23-4-43, discharged on 4-5-43, wt. 14 lbs. anterior fontanelle open, abdomen: full, liver 3" enlarged, hard and not tender, spleen 3", ascites present, lungs: nil particular, urine: no bile salts or pigments, no albumen; blood: no para- site, lymphocytosis present, faeces: whitish nova or site, amobae, urine: 1-5-1943 no bile pigments, urine: 3-5-1944 bile pigments present, 4-5-1943 child is drop- sical, intensely jaundiced, skin and sclerotics intensely yellow.	Not followed.
76	Chittamma, female, 1½ years (Madras), Hindu, middle class, vegetarian.	Wasting.	Patient is the 7th of 8 children.	...	...	Abdominal distension and wasting.	5-9-1943. A female infant in poor nutrition, rachitic chest, cubbing of finger and toe nails present, puffy eyelids, a febrile wt. 13 lbs., abdomen: distended doughy ascites present, spleen 1" enlarged, liver hard 2" enlarged, prominent veins present over anterior abdominal wall (mother's blood: Kahn negative) urine: no albumen, no bile salts, bile pigments present, urobilin sub-normal.	Admitted in the Stan- ley Hospital on 5-9-43 and discharged on 16-9-43, not followed.

Appendix of

Cases—Contd.

Serial No. Year of observation	Name, age, sex, social status, caste, dietary habits	Complaints, duration	Family history	Previous history, feeding	History of present illness
77	Subramaniam, male, 2 years (Madras), Hindu, middle class.	Yellow urine and eyes. Fever 2 days, "cake inside" abdomen — Duration?	Patient is the fifth child in family.	...	...
78	Subbaroyan, male, 1½ years (Mangalore), Hindu Brahmin, rich class.	Yellow urine and whitish motions — 1 month.	Patient is fifth child, mother victim of migraine, maternal grand-mother asthmatic.	Breastfed, also given cow's milk.	Fever and diarrhoea common.
79	Suyata, female, 1 year (Nellore), Hindu, Non-Brahmin, rich class, mixed diet.	Distended abdomen — 6 months.	...	Fed on fresh unboiled cow's milk with broth in large quantities.	Whitish diarrhoea.
1944 80	Audisesha, male, 3 years (Nellore), Hindu Brahmin, middle class, vegetarian.	"Cyclic diarrhoea"—a couple of months.	Mother and patient's elder sister are asthmatic. Father migrainous, sister has eczema on the neck and upper chest, (clinical photographs reveal the condition).	...	...
81	Subbamma, female, 6 years (Madras), Hindu, Middle class.	"Jaundice."	...	...	...
82	Vasavambal, female, 4½ years (Madras), Hindu, non-vegetarian, poor class.	Yellow eyes and oedema—2 days.	Patient is 5th child.	...	...
83	Parvathi (alias Sivakamasundari), female, (Madras), Hindu, Brahmin, middle class (sister of Muthukrishnan No. 39 of 1939 series).	Bloated abdomen — 6 months.	...	Fed by mother, supplemented with cow's milk.	Child susceptible to frequent diarrhoea.
84	Nagendran, male, 1 year (Perambur, Madras), Hindu, Jain, vegetarian, middle class.	Bloated abdomen — 3 months.	Mother asthmatic.	Breastfed and on cow's milk.	Child had measles and broncho-pneumonia five months before observation.

Symptoms	Clinical data: investigation	Additional notes
Jaundice and distended abdomen.	Admitted into Stanley Hospital 13-7-'43. Discharged 15-7-43. Icterus present, abdomen distended with prominent veins on anterior abdominal wall. No ascites (detectable), liver hard and 3" below costal margin. Spleen 3," heart and lungs: nil particular.	Not followed.
Jaundice.	Admitted into Stanley Hospital 23-7-'43. Discharged on 24-7-43. Icterus conjunctiva and skin seen. Abdomen: distended, no detectable ascites, liver hard and 2" enlarged, spleen hard and 3" enlarged. Kahn of mother's blood negative). Urine: albumin and bile salts absent, bilirubin present, urobilin sub-normal. Blood: slight eosinophilia present.	Reported to have died.
Wasting and protuberant abdomen.	An infant very irritable recent examination: Abdomen: distended, liver very hard and 2" below costal margin, spleen just palpable, (treated with B. coli vaccine diet adjusted).	Not followed though seen 3 months afterwards with considerable improvement. Died.
Cough, fever and abdominal distension of a few days' duration at Madras.	2-10-1944. (Studied as out patient in Stanley Hospital). Thin baby, palpable glands present along the neck. Liver 2" and fairly hard, spleen palpable, X-rays wrists and tibia: nil particular (mothers blood Kahn negative, has marked eosinophilia).	Not followed.
...	Admitted into Stanley Hospital on 15-8-1944. Died the same day at 12-30 p.m. Intensely jaundiced girl. Ascites present, generalised oedema present.	Died.
...	Admitted into Stanley Hospital 8-9-1944. Discharged on 9-9-1944. Conjunctiva xerotic and jaundiced. Abdomen: distended with free fluid, prominent veins present in anterior abdominal wall, liver hard and ½" below costal margin (felt by dip method). Heart: tic tac rhythm. Lungs: breath sounds harsh, (Kahn of mother's blood negative).	...
Abdominal distension.	Observed as out-patient in Stanley Hospital. 6-10-1944. Child appears apparently healthy. Abdomen distended, liver hard and 3" below costal margin, spleen ½" below costal margin, no ascites or jaundice. Wt. 21 lbs., 4-1-1945 ascites slight, jaundiced, skin and eyes, (Kahn of child's blood negative). 20-1-1945 intensely jaundiced, ascites present, child slightly dyspnoeic. Urine: bile salts absent, bile pigments present, urobilin subnormal, Van Dan Bergh (Blow) direct biphasic.	Died on 10-1-1945.
Abdominal distension and whitish motions.	10-11-1944. An ill-nourished lad. Abdomen distended, liver hard and 2" below costal margin, spleen ½" below costal margin. Ascites and general oedema present, urine: no albumen, no bile salts or pigments, fed on albumen water and liberal use of vitamins, advised. 10-1-1945 child improved, liver still hard 2" below costal margin.	...

Appendix of

Cases—Contd.

Serial No. Year of observation	Name, age, sex, social status, caste, dietary habits	Complaint Duration	Family History	Previous his- tory, feeding	History of present illness	Symptoms	Clinical data: investigations	Additional notes
85	Baby c/o Kasyali Hotel, female, 1 year, Sowcarpet (Madras), Hindu, Brahmin, vegetarian, middle class.	Fever and abdominal distension—15 days.	Mother asthmatic.	Fed on cow's milk.	...	Abdominal distension.	20-9-1944. Child is peevish. Febrile. Abdomen distended, liver hard and 2" below umbilicus, spleen $\frac{1}{2}$ " enlarged, slight icteric tinge seen in sclerotics, prominent veins present over anterior abdominal wall—(Treated outside); 15-12-1944. Child has definite jaundice, liver and spleen still the same. Ascites not in evidence, (Kahn of mother's blood negative).	...
86	Baby c/o Mr. A. S. K. Murthy (Nellore), female 10 years, Hindu Brahmin, vegetarian, rich class.	Abdominal distension and white motions 1 month.	Mother and grandmother asthmatics.	Fed on undiluted cow's milk. The grandmother prided on the quantity taken by the baby.	Started with abdominal distension. Fever and with diarrhoea.	Abdominal distension and subicteric eyes.	Oct. 1944. An irritable baby, thin extremities. Abdomen: distended, liver very hard and up to umbilicus, (3" enlarged), spleen 1" enlarged, no ascites. Slightly jaundiced tinge of eyes seen.	Child died at Nellore—(Report).
87	Female, 2½ years (sister of No. 2, 3 and 4).	Pale motions—1 month.	...	...	...	...	November 1944. Baby apparently healthy. Abdomen, distended. Liver is soft and 1" below costal margin, spleen just palpable, (as previous children had cirrhosis of liver this child was kept under observation). Diet: fat cut down, vitamin advised, 15-1-1945. Child is well, liver the same.	Being followed up.
88	Baby c/o Mr. Gopinath Joshi (Hanumantharoyan St. P.T.), female 1½ years, Hindu Gujarati, Brahmin, rich class.	Yellow body and swollen abdomen—1 month.	Mother asthmatic. Only child.	...	...	...	The child was shown after all available treatment was futile. An intensely jaundiced baby, ascites present febrile. Liver very hard and 2" enlarged, spleen 1" below costal margin. (Developed broncho-pneumonia 15 days after first examination).	Not followed. A typical case of infantile biliary cirrhosis.
89	Baby c/o Mr. T. R. M. (12, Ekambareswarar Agrahram), female, 1 year, Hindu, Gujarati Brahmin, rich class.	Abdominal distension—3 months.	Mother and her sister asthmatic.	Suspected and treated for liver enlargement during last 6 months.	...	...	Child seen in moderate health. Abdomen tumid, liver hard and 1½" below costal margin, spleen just palpable. In a fortnight's time jaundice appeared and within a week became intense. Ascites was terminal. The treatment was of no avail and the disease pursued—a relentless course.	Child died. A typical case of infantile biliary cirrhosis.
90	Baby, male, 2 years, (Veerappan St. Madras), Gujarati Baniya, rich class.	...	...	...	...	...	Seen in the terminal stage of intense jaundice and cholæmia, malaena present. Abdomen ascitic, liver hard and upto umbilicus.	Died.
91	Baby c/o Mr. S. Hari-sankar Dummy, female, 1½ years, (Madras), Gujarati, Brahmin, rich class.	Abdominal distension and diarrhoea—3 months.	...	...	Frequent diarrhoea and short fevers are common.	...	Seen as in case No. 90 in a terminal stage. Jaundice very intense. Ascites present. Cholæmia and malaena present.	Died.
92	Baby c/o Mr. A. Seshagiri Rao, (Triplicane, Madras), male 1½ year, Hindu, Brahmin, middle class.	Abdominal distension—3 months.	Mother asthmatic.	...	Frequent short fevers and cough.	...	A boy in moderate nutrition. Tumid abdomen, liver hard and 2" enlarged. Spleen $\frac{1}{2}$ " enlarged. December 1944: Ascites present. No jaundice.	Not followed.
93	Baby c/o Mr. M. Satyanarayana, male, 2 years, (Madras, Hindu, Brahmin, vegetarian, middle class,	Abdominal distension—6 months.	Father asthmatic (eosinophilic lung).	...	Summer diarrhoea in April 1944.	Difficulty of breathing at nights and abdominal distension.	October 1944. A baby in moderate nutrition. Abdomen tumid, liver rather hard and 1" below costal margin. Spleen just palpable.	Being followed.
94	Baby c/o Sundaramma, male, 1 year old, (Chingleput), Hindu, Brahmin, vegetarian, poor class.	Abdominal distension—"cake in abdomen"—3 months.	...	...	...	...	July 1944. Abdomen distended. Liver hard and 1½" below costal margin. Spleen $\frac{1}{2}$ " below costal margin. October 1944. Ascites and jaundice set in, treated outside.	Died.
95	A male child (Poona), 1 year old, Hindu, Brahmin, middle class, vegetarian.	Abdominal distension and cough.	Mother migrainous.	Fed on cow's milk.	The child is slowly losing appetite and is upset by milk.	Cough at night and constipation.	An infant in apparently good health. Abdomen distended, liver hard and 1½" below costal margin, spleen $\frac{1}{2}$ " below costal margin.—Early case.	Not followed.

Appendix of

Serial No. Year of observation	Name, age, sex, social status, caste, dietary habits	Complaint, duration	Family history	Previous his- tory, feeding	History of present illness
1944 96	Narasimha, male, 1½ years, (Madras), Hindu, Brahmin, vegetarian, middle class.	Distended abdomen—for 6 months.	Father asth- matic.	Fed by mother and on cow's milk.	...
1945 97	Oliver, male, 2½ months old, Indian Christian, middle class.	Jaundice: yellow body— 1½ months.	Father heal- thy, mother had toxic albuminuria with high B.P. during preg- nancy.	Breastfed baby.	...
98	Alamelu, female child, 2½ years, Hindu, Brah- min, poor class, (Mad- ras).	Distended abdomen and yellow colour of skin—6 months and 1 month.	Mother asth- matic.	Breast fed Cow's milk given. Wean- ed at 1 year with rice.	...
99	Kadirvelu, male, 1 year old, (Madras), Hindu, Non-Brahmin, mixed diet, poor class.	Distended abdomen—6 months?	Father asth- matic.	Breast fed till date of ad- mission.	...
100	Baby c/o Mr. R. P. (Madras) female, 2 years, Hindu, Brahmin, vege- tarian, middle class.	Recurrent whitish mo- tion-cyclic diarrhoea—1 year. Disten- ded abdomen —3 months.	Mother (and paternal uncle and aunt) as- thmatic. Pa- tient is the 2nd of 3 child- ren. An elder- sister to the patient is said to have reco- vered from enlargement of liver.	Breast fed and on cow's milk. Mother's pregnancy complicated by hypereme- sis. Not given castor oil to which patient is sensitive. Milk of magne- sia is given for constipa- tion which alternates with bouts of diarrhoea.	...
...	Govindammal, female child, 5 years old, poor class, Hindu, non-Brah- min.	Swelling of the abdomen and feet—2 months.	...	...	Had whoop- ing cough 2 months ago.

Cases—Contd.

Symptoms	* Clinical data : investigation	Additional notes
...	Investigated as outpatient in Stanley Hospital (1-12-1944). An infant in moderate nutri- tion. Abdomen distended, liver hard and 3" below costal margin, spleen ½" below costal margin. No ascites or jaundice, prominent paraumbilical vein present; 30-12-1944. Slightly icteric tinge seen in sclerotics, slightly oedematous all over the body; urine: no albumen, Benedict's qualitative solution reduced. Bile pigments present. No bile salts, urobilin subnormally present, (Kahn of patient's blood negative); 16-1-1945. Admitted into Stanley Hospital in impending cholæmia. Intensely jaundiced, slight ascites present. A soft systolic murmur at apex. Observed as outpatient in Stanley Hospital (5-1-45). An intensely jaundiced infant. Abdomen distended, slight ascites present, liver hard and 2" below costal margin, spleen hard 1" below costal margin. Umbili- cus everted. Advised vitamin K and adexolin. Diet adjusted. Urine: bile pigments present. No bile salts, urobilin subnormal; 24-1-45. Child has im- proved in general health. No blood in motions. Urine: Bile pigments present. No bile salts, urobilin sub- normal. Kahn of mother's blood negative. (Wasser- man of father's blood before marriage negative. 20-1-1945. A slightly jaundiced baby. Gene- ralised oedema present. Ascites present. Abdomen: Distended. Liver hard and 2½" below costal margin. Spleen 1" below costal margin. Lungs: Breath sounds harsh (treated outside the profession).	Died in Stanley Hos- pital on 18-1-1945. A typical case of infantile biliary cirrhosis—des- cribed by pathologists as subacute toxic cirrhosis
Jaundice and protube- rant abdomen. Umbilical and left inguinal hernia pre- sent. (Passed blood in motions — 3 days.	...	Being followed. 31-1-1945. General condi- tion better. Skin and eyes yellow; urine: No bile salts or bile pig- ments. Urobilin sub- normal 10th March 1945: Urine bile pigments present.
Ascites, jaundice and distended ab- domen—Diar- rhœa 2 days.	...	Not followed.
Distended abdomen.	Admitted into Stanley Hospital. An ill nourish- ed child. Abdomen distended. Liver enlarged, hard, and painless; 2" below costal margin. Heart and Lungs: nil particular. (Kahn of mother's blood nega- tive.)	Early case.
Abdominal distension.	10-1-1945. A female child slightly pale. Coryza present. Abdomen distended. Liver hard and 1½" below costal margin. Spleen palpable. Lungs: Breath sounds harsh. Rhonchi heard all over. Heart: Nil particular. (Mother's blood: Kahn negative on two different occasions).	Early case.
Distended abdomen with prominent vein.	Admitted into Stanley Hospital 26-2-1945. Died in Stanley Hospital, 3-3-1945. An ill nourished baby. Cortex vascularised. Conjunctiva xerotic. Skin dry and pellagrous. Angular stomatitis present. Abdo- men: Distended. Ascites present. Liver ½" below costal margin. Spleen below costal margin. Promi- nent veins present over anterior abdominal wall. Heart: Nil particular. Lungs: A few basal rales heard Mother's blood: Kahn doubtful. Wasserman nega- tive. Urine. No sugar no albumen. Blood serum of patient: Van Den Bergh, direct and biphasic positive. (A portion of liver and spleen removed post mortem.)	...

"Work alone art thou entitled to and not to its fruit.
So never work for fruit, nor yet desist from work."—D. S. SARMA.

I am tempted to write this article on infantile hepatic cirrhosis which has interested me during the last eleven years.

A rational treatment of the disease is not appreciated by the practitioners in any detail. Improper treatment is rampant. Secret specifics are marketed by interested traders. It is my aim to make the profession interest themselves in a genuine study of the subject, and treat patients on the lines suggested in this article and help to dispel the present impression that no scientific or regular treatment is available for infantile hepatic cirrhosis.

I am indebted to many persons and institutions who directly or indirectly helped me in the completion of this article.

During the last five years I have worked in the Medical Wards (especially with the sick children) of the Stanley Hospital, Madras, under the aegis of Rao Bahadur Dr. G. R. DINKER RAO, M.D., Professor of Medicine and Principal, Stanley Medical College, and Physician, Stanley Hospital, Madras, who has always inspired and helped me in all academic endeavours and humble pursuit of knowledge; and therefore I record with great pleasure herein my feelings of extreme gratitude to him. My thanks are due to Rao Bahadur Dr. M. G. KINI, M.C., M.B., M.Ch. (Ortho.), F.R.C.S. (Edin.), F.R.S. (Edin.), Superintendent of Stanley Hospital, Madras; to Dr. P. ARUNACHALAM, M.D., M.R.C.P. (Lond.), Physician, Stanley Hospital; to Dr. V. SUNDARESH, M.D., Professor of Pathology, Stanley Medical College, Madras, and to Dr. M. D. ANANTHACHARI, M.D., my colleague, who have all given me valuable encouragement in the writing of this article. Dr. U. S. RAO, M.B., B.S., my friend and colleague, has helped me specially by supplying me precious information pertaining to the subject of infantile hepatic cirrhosis through personal communications.

My thanks are due to the editors of the following journals for the reproductions from their journals:—*The Antiseptic*; *The Madras Medical College Magazine*; *The Journal of the Stanley Medical College*; *The Journal of the Indian Medical Association*; *Indian Medical Journal*; and *The Indian Journal of Pediatrics*.

My thanks are due to Rao Bahadur Dr. B. T. KRISHNAN, B.A., M.B.C.M., M.Sc., Principal, Medical College, Madras, and Dr. D. G. REDDI, M.D., Professor of Pathology, Medical College, Madras, who have permitted me to utilise the specimens of liver and sections of the liver in infantile biliary cirrhosis for descriptive purposes.

Dr. G. S. VISWANATHAN, M.B., B.S., and Rao Sahib Dr. K. NARAYANA Nair, Department of Pathology, Stanley Medical College, Madras, helped me in the post-mortem study of the liver and spleen, and also in the preparation of the photographic pictures of the liver (macroscopic and microscopic) and herein I record my great indebtedness to them.

Every special department in the Stanley Hospital helped me in my work and, particularly, Dr. P. RATNASWAMY, M.D., Medical Registrar, Dr. M. R. NATARAJAN, M.B., B.S., Clinical Pathologist, and Dr. A. N. K. MENON, M.B., B.S., D.M.R., Radiologist. I will be failing in my duty if I did not record my thanks to all the members of the nursing staff in Willingdon Ward, Stanley Hospital, who co-operated with me in my studies.

My brother-in-law, Capt. D. K. MURTHY, I.M.S., now on active service, helped me in taking clinical photographs of my private patients, and my nephew, A. V. R. MURTHY, B.Sc., Medical Student, went through the manuscript with all care, and both richly deserve my thanks for the same.

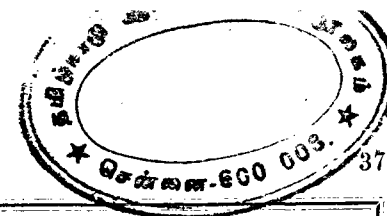
My grateful thanks are due to Drs. U. RAMA RAO and U. KRISHNA RAO, M.B., B.S., Editors, *THE ANTISEPTIC*, Madras, without whose willing co-operation, this little effort would not have attained its present shape.

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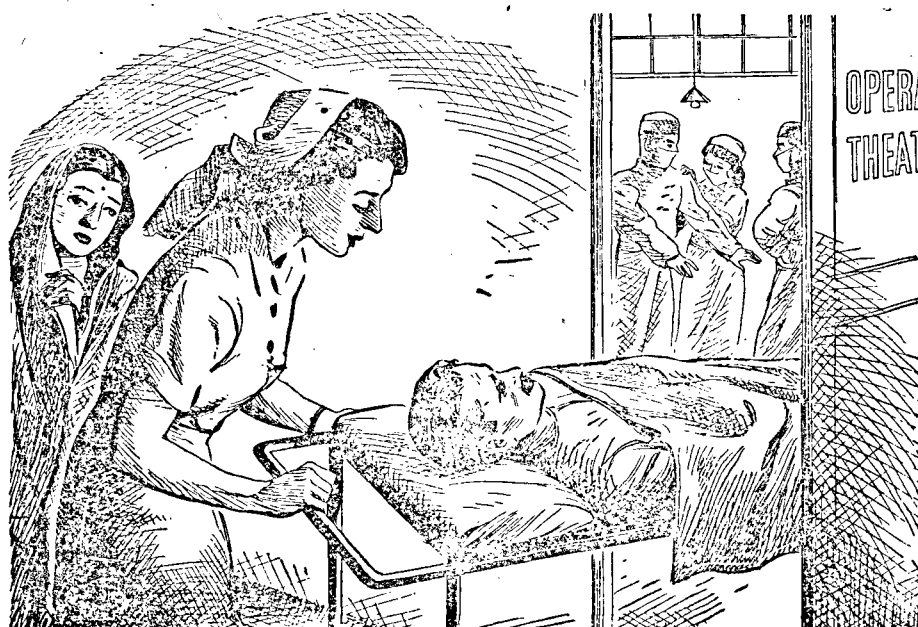
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Auto-Intoxication*

VALLABHDAS N. MEHTA, L.M.&S.,
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IN my practice of medicine extending over a period of thirty-five years, I have learned some things for myself that the books did not teach me, viz., a certain cause may produce a variety of symptoms so different in their manifestations as to give rise to the view that they are so many different diseases, when in reality they are the result of one general cause. For example, faulty elimination of effete matter from the organism may give rise to those symptoms that accompany rheumatism, neuritis, epilepsy, biliousness, headache, nephritis, eczema, urticaria and many others that are known to result from auto-intoxication.

The longer we practise medicine and the more we study this subject by careful clinical observation, the more we are convinced that the question of auto-intoxication is one of the most important subjects, now before the medical profession. There are few diseases, if any, that are so far-reaching in their possibilities, in their effect upon the animal economy. We are aware that there are some physicians that laugh at the term "auto-intoxication" and say that the thing is a myth and originated in the mind of some physician that wished to be different from the rank and file of the profession, but I want to say that auto-intoxication is no will-o'-the-wisp, or imaginary condition, but a real disease, a disease in which every organ and tissue of the body may be affected and that few people live to old age without suffering from it at some time or other during their life. It may be so mild that we are in doubt as to its existence, or it may be so severe as to cause death.

I will try to briefly state what intestinal toxæmia is. It is a poisoning of the system from within. The poisons and toxins that are generated within the system are reabsorbed into the blood stream and thus poison the individual producing a train of symptoms that are characteristic of the condition. In other words, it is a subtle poisoning of the general system from the absorption of bacteria and their toxins and of poisonous chemical compounds formed within the body. Little by little these poisons are absorbed into the blood stream in quantities beyond the power of the eliminative organs to deal with successfully.

The cause.—This condition may have its origin in the liver, kidneys spleen or intestinal tract or in a combination of these, but, at present, I will confine myself to treating only of the cause as it arises from the intestinal canal.

When auto-intoxication arises from the intestinal tract, it is the result of "intestinal stasis." This condition is an abnormal delay in the passage of the contents of the intestinal canal through a portion or several portions of its length. This may be gastro-enteric, gastro-entero-colonic, or the whole length of the canal may be sluggish, and thus the contents of every part may be delayed.

In whatever segment this retardation occurs, it not only allows but favours the rapid development of all kinds of bacteria, inhabiting that part of the alimentary canal.

* Specially contributed to THE ANTISEPTIC.

Hence bacteria, toxins, and various chemical poisons are absorbed into the blood stream and this process of absorption goes on so insidiously that the patient is not aware of its presence until the system is saturated and his health is badly undermined. Even then he has no idea of what has overtaken him.

The eliminative organs soon become crippled as it were and their normal functions impaired, hence they allow still more poison to accumulate. The lymphatic system also is soon poisoned and, in consequence, fails to guard the blood stream by not being able to convert poisons into innocuous substances as it does normally.

In intestinal torpidity or stasis many abnormal conditions occur, the contents become putrid and constitute a 'hotbed' favourable for the development of all kinds of organisms ordinarily present in the intestines, besides many new or extraneous strains of bacteria. These organisms and the toxins resulting therefrom, together with various chemical poisons, are absorbed and find their way along the ducts, leading from the alimentary canal and in due time reach the blood stream. By this they are carried to every part of the animal organism producing progressive degenerative changes in various organs, the cause of which is a mystery to those that know nothing about intestinal stasis and its relation to auto-intoxication.

In intestinal stasis, the pelvic colon and the rectum may become greatly elongated (perhaps to twice its normal length) sagging along the floor of true pelvis and capable of retaining the fecal matter for several days, even though a small section may be broken off, and evacuated daily, thus giving rise to a false impression of normal bowel movements.

The symptoms occurring in auto-intoxication are as numerous and as varied as the sands of the sea. We cannot now call to mind a disease in which the symptoms are so varied in different individuals as they occur in this condition.

In very young children, the picture is one of extreme prostration features drawn, extremities cold, eyes sunken, and fontanelle depressed, muscular twitching and even convulsion may be present. The mental condition is dulled, the patient often lying in a state of stupor. There may be either diarrhoea or persistent constipation. If an evacuation occurs, it usually is very offensive, green in color, and often contains quantities of mucus.

The majority of cases seen among adults are chronic in nature. The patient says he is not ill, yet is not well. He is languid, has no ambition, headache, and neuralgia are present nearly all the time. The back and limbs ache more or less day and night. Some patients complain of persistent pains between the shoulders, especially if they are lying down or sitting in one position. In some women, this pain under and between the shoulders and in the region of the short ribs is very trying, and persistent in others. The back, hips, and limbs are the special sites for the pains and aches. Indigestion invariably is present, in some form or degree, in some cases of intestinal toxæmia. In others, the digestion seemingly is normal, but the subjects cannot gain any strength, although they may be taking an abundance of food. Here we have faulty assimilation. In many patients suffering from this disease, the liver becomes very torpid. Their color is hard to describe, in fact some of them have jaundice in full development.

Thus we could go on and on enumerating symptoms and conditions that are complained of in this disease known as auto-intoxication or auto-infection, but I believe I have named sufficient, so that the reader may recognise the true condition when he is called upon to make a diagnosis.

The treatment is both prophylactic and curative. As to the prophylaxis, it is much easier to prevent this condition than to cure it after it has been acquired. Prevention should begin in childhood. Every child should be instructed in the habit of daily evacuating the bowels, the habit once thoroughly formed is not lightly to be neglected. Often the proper selection of food will overcome this condition and prevent auto-intoxication. In adults when constipation has become a fixed condition, there always takes place more or less absorption of toxic material. The patient is in a chronic state of auto-toxæmia, consequently a bad state of health obtains. This condition being present, it is only necessary for an extra amount of toxins to enter the blood stream, when a typical case of auto-intoxication confronts us.

To begin the medicinal treatment in this class of cases we know of no remedy more efficacious than the combination of Calomel, Podophyllin, and Bilein, repeated often until several movements have been secured, followed the next morning by full doses of a laxative Saline or of Magnesium Sulphate. This should be repeated every morning for sometime but in reduced dosage. Bilein should be given three times a day and a granule of Podophyllin and Aloin at bed time. When the physician has satisfied himself that the bowel has been freed from all offending accumulations he is ready to begin treatment that will build up the patient's general rundown condition. A weekly P.P. wash will also help in removing toxins from the intestinal canal. If the habits are faulty try to correct them—change of employment often works wonders for patients that live sedentary life. Look after all the eliminative functions. Patients having large or pendulous abdomens must be fitted with proper supports.

I have seen many people cured of this condition by spending a few months at some springs most people believe that the water is responsible for this happy change, but this is true only in parts. The drinking quantities of water is beneficial anywhere, but the change of food, and the manner in which it is cooked, the change of climate and scenery, all have their beneficial influence, while added to these is their getting away from oneself, living a broader life. All this combined contribute to the cure.

Cases and Comments

A Case of Maggots in the Nasal Cavity

BON BEHARI SAHA DALAL, M.B. (CAL.),
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Chandanbaisa, Bogra (Bengal).

THE following case report will be helpful to my mofussil fellow brothers in view of the fact that they sometimes may feel troublesome to treat such rare cases as there is no specific treatment in literature.

On the 12th February, 1946, I was called to attend a Hindu female aged 36 years suffering from nasal bleeding.

Past history:—The patient was of good physique and healthy with no other ailments but slight nasal catarrh. Two days ago while she was taking her meal all on a sudden one house-fly entered into her right nostril. To-day while she sneezed one dead fly and two maggots came out and since then blood was oozing from the nostril drop by drop.

On examination:—Found slight superficial oedema of the nose; no bleeding point could be ascertained, sero-sanguinous blood coming drop by drop. She had a slight rise of temperature. No abnormality in other systems.

Treatment:—With the suspicion that there is some septic process in the nasal cavity and there are still more maggots, I advised an alkaline nasal wash every 4 hours, nasal drops (Nostal) every 4 hours, local application of Spirit Turpentine twice and Adrenaline Chloride solution every 4 hours, Septanilum—1 tablet T.D.S.

On 13th February, 1946, the inflammation spread above upto the eye and below upto the upper gum. The bleeding did not stop. I guessed that the medicine used could not reach the superior nasal meatus where lie the maggots. So I advised the guardiam to shift the patient for a better treatment. Meanwhile I injected Collo. Calcium 1 c.c. and prescribed a steam inhalation of Spirit Turpentine and Tincture Benzoin Co. twice a day.

On 14th February, 1946, 3 dozens of maggots came out after inhalation, the inflammation subsided and bleeding stopped.

The inhalation once a day and Septanilum 1 tablet T. D. S. was continued for 3 days and the patient had an uneventful recovery.

Points of interest:—(1) Formation of maggots in such an awkward place without any previous septic focus.

(2) Helpless condition of mofussil practitioner.

(3) Failure of local application of Spirit Turpentine but rapid action of the vapour.

Efficacy of Nicotinic Acid in Ear Troubles

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The following case will illustrate how a small amount of Nicotinic Acid could bring about a rapid cure in a case of severe tinnitus aurium accompanied by some other trouble.

In January, 1946, a heavily-built tea-factory 'mistri' of over 50 years of age came to me for treatment of the following complaints of several

[276]

weeks' duration: (1) Tinnitus aurium (more distressing at night); (2) flashes of light before the eyes (occasionally); (3) insomnia; (4) sense of fullness in the head; and (5) gastro-intestinal disturbances (indigestion, irregular motion etc.)

Investigation:—Long ago he had an attack of severe tinnitus aurium, and was cured by some capsula-like things (he can't say exactly what he took) prescribed by a certain Calcutta doctor. Last year he had several attacks of malarial fever and took Quinine and Mepacrine Hydrochloride tablets for the fever. No definite history of otorrhoea or any other ailments was obtained. Mental depression was detected in him and partial deafness (bilateral) was also present. But examination of the ear, nose, and throat revealed nothing particular to account for his present ear-troubles.

Treatment:—Usual symptomatic treatment was given for the first few days. This did him no good. Then I tried Nicotinic Acid. This was suggested by his nervous, gastro-intestinal and circulatory disturbances (*Antiseptic*, Oct. '43, P. 503). I gave him only two tablets of Pelonin (100 milligrams) daily, and thus only 25 tabs. were administered, and the result was really marvellous. The patient felt much better on the very fourth day of nicotinic therapy and within a few days all the distressing symptoms disappeared. His tinnitus was gone, he could now hear well, sleep well, and eat well. No recurrence of any one of those disorders has occurred up to date. It should be noted here that no other medication was given along with the vitamin. I do not know how the drug acted in this case, and in fact, I did not expect such a rapid cure of the symptoms. In this case Nicotinic deficiency was probably associated with high B. P. although according to some authorities this is usually attended by low B. P. I shall be thankful for comments on this note from erudite readers.

Penicillin in Chronic Arthritis Knee

K. D. LAHIRI, M.B., B.S.,
Senior Hony. Medical Officer,
Skin and V.D., Department, Patna Medical College Hospital, Bihar.

Uday Lall, H.M., 24, was admitted in the indbor of V. D. skin with chronic arthritis of right knee joint on the 6th November, 1945.

History of no fever and trauma. Had urethritis one year back which got cured, but about 9 months back he developed pain and swelling of the right knee joint which went on increasing but he went about his work and for the last one month he is unable to walk and even squatting is impossible.

Examination showed swelling of the right knee joint and movement was painful but there was no tenderness.

Investigation:—(1) Prostatic smear examination on 7-11-'45, 13-11-'45 and again on 14-11-'45 showed—G. C.—nil, P. C.—present—many, and E.C.—few.

(2) Blood W. R.—negative—A/12103 of 23-11-'45.

(3) Aspirated fluid from the knee joint was sterile.

(4) Skiagram—X—ray regd. report no—4068 of 16-11-'45 showed right knee A.P. and lateral views—rarefaction of bones and diminution of joint space.

Treatment:—(1) Sulphonamide—0.5 gr. 4 hourly for 4 days and was repeated after 10 days without any result.

(2) Scott's dressing—which was of no use.

(3) Penicillin therapy—*intra-articular* injection—on 23-11-'45 had one lac and on 25-11-'46 one lac units dissolved in normal saline.

Progress:—There was marked improvement as regards pain and swelling within 24 hours after the first injection on 23-11-'45 and the pain totally disappeared and very little swelling was left 24 hours after the second dose of *intra-articular* Penicillin in normal saline. Patient came to the hospital in stretcher and went away walking on 27-11-'45 of his own accord.

Discussion:—The marked effect of Penicillin in chronic arthritis of Nisserian origin when used *intra-articularly* has been observed.

Acknowledgment:—I am thankful to Dr. S.M. NAWAB, Surgeon, for help and to the Superintendent for permission to publish the case report.

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- (1). Lahiri, K. D. (1946) Penicillin in V. D. of India.
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Ascariasis

Dr. M. S. KUNDU, L.M.F.
Gourganj, Dacca.

I. On the morning of April 2, 1941, I was called to attend a case of cholera who had been purging and vomiting for 2 hours.

On hastening to the patient's house I was told that the patient, a Mahommadan girl of 8, had so far 8 to 10 liquid motions and vomited as many times expelling two full-sized round worms. On examination the patient was found under collapse of moderate degree. In diagnosing the case I could easily rule out cholera by (1) stool not rice-watery; (2) vomit only contained frothy liquids; (3) temp. 100.6° and (4) degree of collapse not proportionate to the loss of fluid through both the channels. Though I at once decided that round worm infection was responsible for the whole trouble, yet I had to start treatment symptomatically by hypertonic Saline transfusion. I also prescribed Bi-choleraphage (B. I. Co., Ltd.) orally with the idea to prevent a complication of cholera which was then prevalent in the locality. In the afternoon I again watched the patient to see if her general outlook improved. She had a few more motions and eructations, each time discharging one or more round worms through either channel. In all she expelled as many as 10 worms by the mouth and about the same number by the rectum. After the evacuation of the worms her condition improved in all respects and after a routine course of Mist oil Ricini treatment she was given rice diet on the 4th day.

Discussion:—(1) Though diagnosis of round-worm infection was made Anthelmintics could not be administered due to collapse.

(2) Discharge of the worms was accelerated after the Hypertonic Saline transfusion.

(3) The number of worms discharged was rather heavy for the age of the child and recovery was sharp and uneventful after their expulsion.

II.—On 7-9-'45, I was called to see the same girl, now aged 12, who was running a high temperature for over 36 hours in an unconscious state of about 12 hours' duration.

On examination:—Temp. 104° F., Pulse 146 p.m., Resp. 40 p.m., Heart, lungs—normal; Liver and spleen—not enlarged. Patient unconscious. No Babynsky or Brudzinsky present. Pupil normal and re-acting to light. I did not think the condition due to malarial infection as I had treated her for Malaria with adequate quantity of Quinine both parentally and orally just a week ago. However I made two blood slides and sent them for examination. Next afternoon the blood report showed 88% poly and 12% small mono and no M.P., Another consulting doctor was called in and the patient was found in the same condition as last night excepting the addition of a few catarrhal rales in the chest and the patient was this time delirious. A provisional diagnosis of septicæmia (Strepto and pneumococcal) was made and the patient was treated with Dagenum Sodium 3 c.c. mixed with 50 c.c. of 25 % Glucose Soln. I. V. once a day and Sulfadiazine, Sulfapyridine and carbochal $\frac{1}{2}$ tab each made into powder orally alternated with Mist alkaly T. D. S.

On the 3rd day, *i.e.*, after the 2nd injection of Dagenum Sodium (M. & B. 693 soluble) she vomited out two round-worms and her condition began to improve. Consciousness returned, delirium gone, and temp. dropped to normal. Dagenum Sodium was then omitted from the treatment and the rest were continued for two days more. The patient was given rice diet on the 7th day and since then she has been maintaining good health.

Points of interest:—This time also her condition began to improve after the expulsion of the worms. Do the Sulpha drugs possess some anthelmintic property as is evidenced here by the expulsion of the worms after their administration?

Conclusion:—Though I have cited the case under the heading "ascariasis," yet I had to adopt symptomatic treatment only owing to the grave condition of the patient and after that no anthelmintic was given on either occasion. Hence the diagnosis has been based on clinical experience only. Without laboratory findings how can ascariasis be definitely diagnosed in this case?

My thanks are due to Dr. H. P. KUNDU, B.Sc., M.B., of Rohajang, Dacca, who helped me in treating the patient on both the occasions and specially by virtue of his clinical diagnosis of septicæmia the patient could be treated along the lines stated on the later occasion, for which all credit is due to him.

Stanno Sulphazide and Chaulmoogra Oil in Chronic Skin Ulcerations

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Hereunder I am giving details of two cases of chronic ulceration of the skin. The cases were treated with Tanno Sulphazide with Chaulmoogra oil locally and Stanno Sulphazide orally.

The results were not only encouraging but magical.

I must point out that I am not a skin specialist and hence my experience should be taken only as of an observatory nature specially as I did not go into the microscopical examination of the discharges etc., as the

poor patients could not bear the expenses. It depended solely on macroscopical appearances and a follow-up of the case for some time.

Case No. 1.—Laj, No, 2139, aged 3½ years, Hindu, girl, dated 14-2-'45. Had ulcers covering the entire scalp for the last one year and a half. Ulcers small sized with dried scabs covering them. Had been resisting all treatment. Scabs, when removed, revealed a pussy area giving the shape of a cavater.

TREATMENT.—Head was shaved with a machine and the treatment started. Scalp washed with Spirit soap (Soft soap 1 3 Spt. Methyl. 1 3).

The following cream was applied—(Stanno Sulphazide 3 tablets Chaulmoogra oil, 2 3, Eucerine ad, 1 3). Covered with lint cotton wool and bandaged. In a cases when it is not desirable to shave the head, prepare the following cream for convenience :—

Stanno Sulphazide	...	3 tablets
Chaulmoogra Oil	...	2 3
Cocconut Oil	...	3 3
Eucerine	...	3 3
Sandal oil	...	few drops

Rub into the scalp after drying the hair after a bath with soap and water. Orally the following mixture was administered :—

R Stanno Sulphazide	...	6 tablets
Nicotinic Acid	...	3 tablets
Gum Acacia	...	q. s.
Glucose	...	q. s.
Aqua Chloroform ad	...	8 3

Divide into 16 doses. 4 doses a day—one every third hour.

Along with the mixture the cream and spirit soap were given for changing the dressings at home, once daily.

Result:—After 4 days, to my surprise, I found the scalp in a perfectly healthy condition.

The mixture was repeated for another 4 days and the cream and soap used for 12 days in all. Till to-day she is alright.

Case No. 2.—Roshan Bibi, No. 2141, aged 10 years, Mohammadan girl, dated 25-2-'45. She had ulcerations on the left side of the face affecting left lateral half eyebrow, eyelids, entire cheek, left alæ nasi, upper lip left half and laterally affecting upto the front part of the ear, maxillary angle and finishing of at the chin in the centre. Raw bloody surface with greenish white crusts hither and thither and especially all round the ulcerated surface. No itching, no pain but, the part tender.

Duration 2 years. Resisted all treatment.

TREATMENT.—The ulcerated area was swabbed daily with lukewarm 2% soda bicarb lotion, crusts removed, part dried and the same cream applied on a piece of lint covered with cotton wool and bandaged and the following mixture was given orally.

R Stanno Sulphazide	...	24 tablets
Nicotinic acid	...	8 tablets
Gum Acacia	...	q. s.
Glucose	...	q. s.
Aqua Chloroform	...	16 3

Divide into 32 doses. 4 doses a day—one every 3rd hour.

Results.—In 8 days she was totally cured with no relapse of symptoms till to-day.

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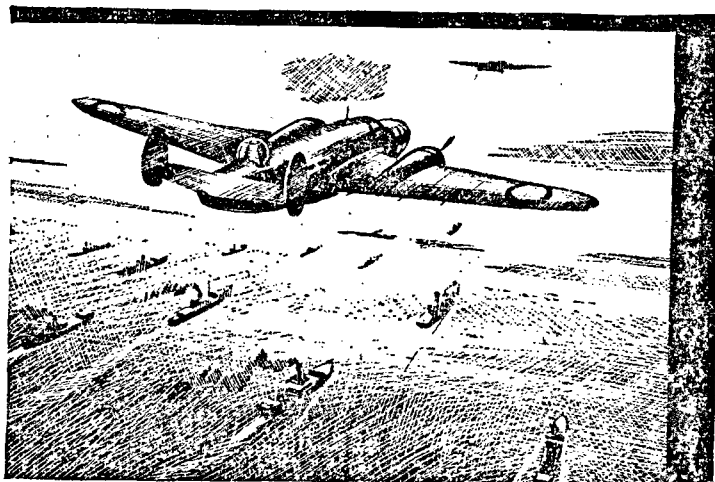
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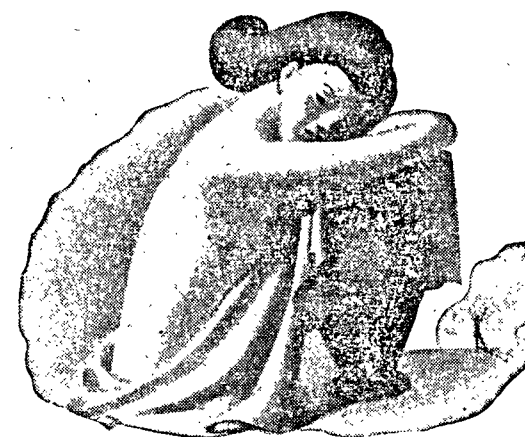
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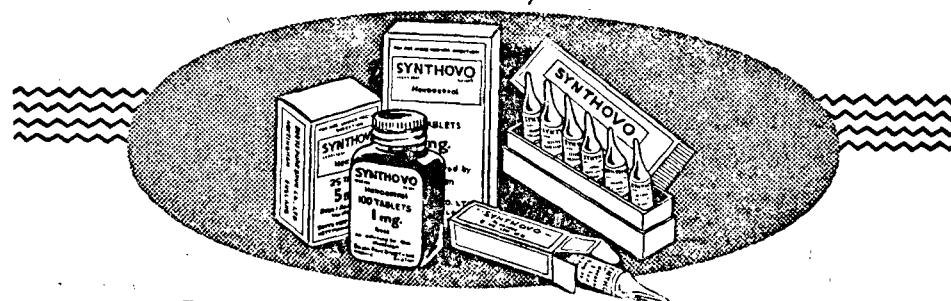
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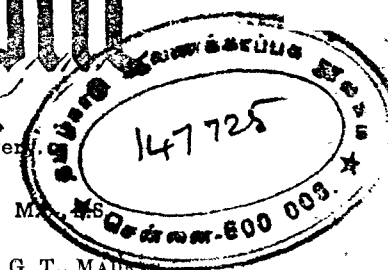
Published on the 15th of every month

Editors: U. RAMA RAU, & U. KRISHNA RAU, M.A.

Editorial & Publishing Office:

"RAMARAU BUILDINGS" 323-24, Thambu Chetty St., G. T., MADRAS.

Annual Subscription: Rs. 7-8-0; Foreign—15 Sh.—Post Paid.



Vol. 43.

APRIL, 1946

No. 4.

The Pharmacy Bill

"PHARMACY in India is in a most deplorable condition in contrast with Great Britain, the continent of Europe, the United States of America and nearly all civilised countries of the world," observed the Drugs Enquiry Committee in their report submitted to the Government of India in 1930-31. This was not the first hint that the Government had of the position as it existed in the country at the time. Long, long before that time the medical profession realised it and it had been dinning into the ears of the Government of India the urgent need for the improvement of the class of people who are connected with pharmacy. But, as is well known, the Government machine in this country moves but slowly, very slowly too, when the health and well-being of the people of this land is concerned. And more than two decades had been necessary for them to realise the truth that "compounding and dispensing of medicines by unqualified persons may endanger human life and that it is necessary to ensure that the persons practising the profession of pharmacy should attain a satisfactory standard of education and training." The Bill that has recently been published by the Government of India is a result of this realisation. "Better late than never."

In judging this measure we have to take into consideration the position as it exists to day, and the opinions expressed by men who are closely connected with pharmacy in India. The Bhoze Committee on Health and Rehabilitation which went into the matter had the advantage of the advice of a pharmaceutical sub-committee composed of eminent pharmacological and pharmaceutical authorities, which it had appointed. That sub-committee advised the establishment of All-India Pharmaceutical Council and Provincial Pharmaceutical Councils. Broadly speaking, their recommendations are to the effect that in addition to the All-India Pharmaceutical Council, Pharmacy Councils representative of the pharmaceutical trade, education and other pharmaceutical interests should be set up in each Province, their functions being—

- (1) to maintain a register of all registered pharmacists of the Province and to register the names of newly qualified pharmacists on payment of a prescribed fee;
- (2) to maintain the standard of education of students;

[281]

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- (3) to specify the drug establishments or hospitals where apprenticeship training may be undertaken;
 - (4) to conduct and maintain the standard of examination;
 - (5) to maintain such disciplinary control over the practice and profession of pharmacy as may from time to time be necessary;
 - (6) to advise the Ministry of Health in respect of any regulations for the distribution of drugs, including poisons, dangerous drugs and drugs of addiction; and
 - (7) to advise on any other legislative control that may be necessary in this connection.

The Bhoré Committee accepted the advice of the sub-committee and recommended that Central and Provincial Pharmaceutical Councils of the character recommended by it should be established by legal enactment. They further recommended that in disciplinary cases the decisions of the Central Pharmaceutical Council should be subject to the direction of the Federal Court to the same extent as that of the Privy Council under the Pharmacy and Poisons Act 1933 over similar cases in the United Kingdom. They also endorsed the opinion of the sub-committee that it is essential to raise the professional standing of those (other than medical practitioners) engaged in the handling of drugs and that to this end legislation designed to protect the public from incompetence should be enacted. The profession of pharmacy, they said, should be reserved for pharmacists and the latter should be restricted to their profession and should not be permitted to undertake functions such as prescription of medicine, administration of anaesthetics, etc. It would be the common knowledge of many that the compounders are often found, particularly in the rural areas, engaged in duties, e.g., that of a junior doctor, a hospital dresser, a laboratory and clinical assistant, anaesthetist, operation assistant, instrument clerk, etc., which their colleagues in western countries consider completely out of their purview.

Concerning pharmaceutical education, the sub-committee were of the opinion, in which the Bhoré Committee concurred, that the present standard of training of those engaged in the practice of pharmacy is unsatisfactory and that a revision of the educational system is necessary. The Bhoré Committee divided those engaged in the profession of Pharmacy into three classes: (1) the licentiate pharmacist; (2) the graduate pharmacist; and (3) the pharmaceutical technologist. The initial standard for the licentiate should be, they said, the matriculation or an equivalent examination with Physics and Chemistry as compulsory subjects of study. A two year course at an approved educational institution is suggested, and Chemistry, Biology with special emphasis on Botany, Elementary Physics, Pharmacy, Pharmacognosy, Elementary Pharmaceutical Chemistry, Elementary Physiology and Forensic Pharmacy are included in the syllabus of study. There should be, they said, two examinations held, one at the end of the first year, and the other at the end of the course. The standard of the examination should be controlled by the All-India Pharmaceutical Council and a uniform standard should be maintained throughout the country. Even after passing the examination they should undergo apprenticeship and then only should the title of pharmacist be conferred on them. For the graduate pharmacist, the entrance qualification, they recommended, should be the Intermediate Science with Physics, Chemistry and Botany as a compulsory course of study or its nearest equivalent. The duration of the course should be three years and should include Inorganic, Organic and

Physical Chemistry, Botany, Biology, Elementary Physiology, Pharmacology, Pharmacy, Pharmacognosy, Pharmaceutical Chemistry and Forensic Pharmacy. Only those who come out successful should be eligible for the pharmacists' diploma without further examination. But even to the graduate pharmacist an apprenticeship of one year has been prescribed. For the third class, i.e., the pharmaceutical technologist, they recommended that in addition to the graduate course in pharmacy, they should undergo an additional course of one year in chemical technology, design, equipment, etc. Of the pharmacists' register, they said, that it will of necessity on its inception include those persons who have been engaged in the profession of pharmacy, albeit without due qualification, for a stated number of years, but we consider that such persons *before being admitted to the register should be required to pass a practical examination. Subsequent admission to the register should be confined to persons who have acquired the status of graduate or licentiate pharmacist, and to bona fide registered students spairing after these qualifications* (Italics are ours).

It is now for us to compare the provisions contained in the Bill with the recommendations of the expert committee and see to what extent they fall short of their recommendations. The All-India and Provincial Pharmaceutical Councils are there, but we do not find any justification whatsoever for the power of nomination reserved for the Governments in constituting them. On the question of qualifications for registration the provisions contained in the Bill fall far short of the recommendations of the Bhoré Committee and the actual requirements of the situation. According to the Bill, any one who holds a Degree of a British or Indian University, other than a Degree in Pharmacy or Pharmaceutical Chemistry and has been engaged in the compounding of drugs in a hospital or dispensary or other place in which drugs are regularly dispensed on prescription of medical practitioners for a total period of not less than 3 years, or any one who has passed an examination recognised as adequate by the Provincial Government for compounders of dispensers, or any one who has been engaged in the compounding of drugs in a hospital or dispensary or other place in which drugs are regularly dispensed on prescription of medical practitioners for a total period of not less than 5 years, can have his name entered in the first register by applying within the prescribed time and by paying the prescribed fee. For subsequent registration, and before the Education Regulations are brought into force in any Province, any one who satisfies the conditions, prescribed can have his name entered. Only he should have passed the matriculation examination or an examination equivalent to it. Thus we find the practical examination test insisted upon by the Bhoré Committee in the case of the first entrants and those who seek admission before the Education Regulations are prescribed, and which is very necessary in the interests of the public has not been insisted upon. This we consider to be a very serious omission. Maybe the Government of India had not before them the Report of the Bhoré Committee when they drafted the Bill. But it is not too late yet. Such a provision must be incorporated when the Bill is finally brought before the Assembly or in the Select Committee stage.

Provision has been made in the Bill for the appointment of Inspectors to inspect any institution which provides an approved course of study, to attend at any approved examination, and to inspect any institution whose authorities have applied for the approval of the course of study or

examination and attend at any examination of such institution. This is as it should be. But Government should go further and take steps to prevent adulteration of drugs. It is here that the enforcement of the Drugs Control Act becomes necessary. This Bill is in a sense, complementary to that Act, and, if results are expected, that Act should also be brought into force forthwith. That is why the Bhole Committee have recommended that the Drugs Rules 1945 under the Drugs Act 1940 should be brought into force either before or concurrently with the pharmaceutical legislation.

The present Government have always been afraid of courts and they have in every legislation tried to oust their jurisdiction. We find a similar attempt made in this Bill also, in sections 36 and 38. This is against the spirit of the times, against the opinion of a Lord Chief Justice of England, and against the recommendations of the Bhole Committee. It is very necessary that the Federal Court should be given jurisdiction in disciplinary matters as recommended by the Bhole Committee.

While therefore this belated attempt of the Government is welcome, we consider that the Bill should be amended on the lines suggested above, if pharmacy in which the term is generally used in Western Countries is to become a fact in India. We trust that the National Government which is in the offing, or the present Government, if it is to continue owing to the unfortunate failure of the ministerial mission, will take note of these points and have the Bill suitably amended to make it really effective and beneficent.

Gleanings From Medical Press

MEDICINE and THERAPEUTICS

***Renal failure in acidosis and alkalosis.**—In persistent acidosis the buffering mechanisms of the blood eventually become inadequate and the accumulating acid radicles are combined with the main body base, sodium, the resulting salts being excreted in the urine and causing a drain of body sodium. Without an adequate supply of sodium the body cannot retain water, so dehydration occurs, comparable to the dehydration and renal failure in any other variety of sodium deficiency. The renal failure is due to a pre-renal cause, dehydration, there being insufficient circulating fluid to maintain an adequate flow of blood through the kidneys.

Alkalis tend to cause renal failure by damaging the renal tubular epithelium, particularly the distal convoluted tubules, in which there may be necrosis of the epithelium and subsequent deposition of calcium salts. It is not yet known why this damage occurs; but it has been pointed out that this part of the tubule is most active in regulating the reaction of the urine.—*B.M.J.*

Cirrhosis of liver.—The existence of a special hypertrophic type of cirrhosis, (Hanot) is not generally accepted nowadays.

Patients with chronic disease of the liver who have hepatic enlargement and persistent or recurrent jaundice usually prove to be suffering from sub-acute necrosis of the liver, and at necropsy they show a course or adenomatous type of cirrhosis with a variable amount of fatty infiltrations. In cirrhosis due to alcoholism the liver may be large as a result of an increased content of fat. Jaundice may occur in all forms of subacute necrosis and cirrhosis; it usually signifies an increased activity or recrudescence of the disease process. A biliary type of cirrhosis should not be diagnosed in the absence of signs of obstruction or infection of the biliary passages. In the absence of these signs the patient should be treated as for subacute necrosis or cirrhosis. Possible causative factors, such as toxic drugs or chemicals, should be looked for. The main treatment is by diet. The fat intake should be reduced to a minimum, the protein intake moderately increased, and the bulk of the calories provided by carbohydrate. The vitamin intake should be raised, A, D, and K to compensate for the fat restriction, and the B complex for its therapeutic effect. Choline chloride has been recommended in this type of case. It is expensive, and the treatment is still experimental. It would be wise to begin

with a dose of 4 g.t.d.s. and increase gradually up to, say, 4 g.q.d.s. preferably in solution after meals. If oedema or ascities appears the salt intake should be reduced and one of the mercurial diuretics given. If jaundice is present the most effective remedy is probably rest in bed.—*B.M.J.*

Cobra venom in the treatment of angina pectoris.—A. S. Freedberg and J. E. F. Riseman (*New England Journal of Medicine*, 233:462, Oct. 18, 1945) report the use of cobra venom in 12 patients with angina pectoris. Those whose angina pectoris was of coronary arteriosclerotic origin, had been under observation at a special clinic for two months to seven years, 9 patients for two years and over. During this period of observation, the frequency of attacks was recorded, the amount of work that could be performed was established by repeated standardized exercise-tolerance tests. Four of the 12 patients responded "strikingly" to many different forms of treatment, including nitroglycerin; these were classified as "marked reactors." Group 1. Three patients showed only moderate response to therapy, "moderate reactors." Group 2. Five patients failed to respond to any of the usual methods of treatment, "nonreactors." Group 3. Cobra venom was given by intramuscular injection (deltoid muscle) in doses of 10 to 20 mouse units (1 to 2 cc.) at least once daily for three to seven days. This resulted in a 25 to 75 per cent increase in exercise tolerance in the standardized test in 7 of the 12 patients, including 3 of the Group 3 patients. In 2 of the patients in Group 3, cobra venom was the only drug out of 57 tested that had any effect on exercise tolerance or effected any demonstrable clinical improvement. Electrocardiographic studies in 2 of the 7 patients showing improved exercise tolerance after treatment with cobra venom indicated that this therapy did not affect the electrocardiographic changes observed after exercise. It was found that the most effective dosage of cobra venom was three injections of 10 mouse units each on the first day, then one injection daily for seven days; to maintain the effect biweekly injections were subsequently necessary. Local pain occurred at the site of the injection, but was not severe enough to make it necessary to discontinue treatment. With the dosage recommended, there were no toxic symptoms; but if the dosage was increased to 20 mouse units, toxic symptoms developed.

If the drug was discontinued after the initial course of treatment, repetition of the treatment gave good results in several cases. In the patients in Group 1, who showed marked response to nitroglycerin, cobra venom was no more effective, and usually less so. It is of definite value, however, in cases who do not respond to other methods of treatment. Electrocardiographic studies indicate that cobra venom does not affect the underlying processes that are responsible for the angina, i.e., "the discrepancy between the demands of the myocardium and the supply of blood." Cobra venom

apparently acts by preventing the patient from feeling pain, and in this way is comparable to surgical procedures to interrupt sensory nervous pathways; and is best employed as a substitute for such surgery when it would otherwise be indicated.—*Med. Times.*

Penicillin.—MODE OF ACTION.—Penicillin appears to interfere with the bacteria at the stage of division, and by preventing multiplication of the organism it gives time for the body defences to come into play. As soon as the concentration of the drug falls sufficiently, the surviving bacteria are able to proliferate. Penicillin is rapidly excreted, particularly in the urine. A single intramuscular injection of 15,000 to 20,000 units produces an effective concentration in the blood in fifteen to twenty minutes and has, for all practical purposes, been totally excreted in four hours. Thus administration must be continuous or repeated at three-hourly intervals. Work is progressing at the moment in methods of delaying absorption of penicillin from local deposits in the muscle so that doses may be given at longer intervals, but the results are still in the experimental stage. Penicillin, in contradistinction to the sulphonamides, is not made ineffective by the presence of pus, lymph or tissue-breakdown products, but certain organisms, especially certain coliforms, by the production of penicillinase, may rapidly destroy penicillin in infected wounds.

ORGANISMS AFFECTED.—Table I classifies the more important organisms into three classes, sensitive, partly sensitive and insensitive. It must be realized, however, that in the sensitive class the sensitivity may vary from strain to strain and that some completely insensitive strains of a normally sensitive organism may be encountered. In every case the infecting organism should be isolated and tested for sensitivity in the laboratory. This is usually a simple matter and within the capabilities of even the smallest bacteriological laboratory.

TABLE I		
SENSITIVE	PARTLY SENSITIVE	INSENSITIVE
Gonococcus	Actinomyces bovis.	Bacterium coli.
Meningococcus.	Haemophilus influenzae.	Bacterium typhosum.
Streptococcus pyogenes.		Bacterium paratyphosum.
Streptococcus viridans.	Corynebacterium diphtheriae.	Bacterium proteus.
Streptococcus pneumoniae.		Bacterium dysenteriae.
Staphylococcus aureus.		Ps. pyocyanea
Clostridium welchii.		Streptococcus faecalis.
Bacillus anthracis.		Myc. tuberculosis.
Treponema pallidum.		Brucella abortus.
Treponema vincenti.		

DOSAGE AND METHODS OF ADMINISTRATION.—As penicillin is rapidly destroyed by acid, oral administration is unsatisfactory and wasteful, only at best 2 to 10 per cent. being absorbed, even if buffer mixtures are given at the same time.

The common and most useful method of administration for systemic infections is by intramuscular injection, either by means of the continuous drip or by intermittent three-hourly injections. Continuous intravenous drip can seldom be carried beyond two or three days, as local thrombophlebitis is common. For local application, penicillin may be used: (1) For skin lesions as a cream made up, usually with lanette wax base, 500 units or more per gramme, and applied every twenty-four hours. (2) For irrigation of cavities twelve-hourly, or as drops in the eye two-hourly, or as a spray for mucous membrane or skin infections, as a solution in distilled water or normal saline in a dosage of 250 to 1000 units per c.cm. (3) For injection into serous or joint cavities or into the subarachnoid space in a dosage of 20,000 to 60,000 units in one dose, in 1 to 3 c.cm. of pyrogen-free distilled water every twelve to twenty-four hours. (4) For lightly dusting open wounds every twenty-four hours, as a powder usually made with a sulphathiazole base and containing 1000 to 5000 units per gramme. (5) For Vincent's angina and other local infections of the mouth and throat, as pastilles containing 500 units each, one to be allowed to dissolve in the side of the mouth to the number of eight to ten each day.

The daily dosage of penicillin for general infections depends upon the sensitivity of the organism, the location of the infection and the clinical condition of the patient. The usual dosage is 15,000 to 20,000 units every three hours. This may be increased to 60,000 units three-hourly for relatively insensitive organisms, such as *actinomyces bovis*, or when the location of the disease is such that penetration of the penicillin is difficult, e.g., subacute bacterial endocarditis. The injections, if given three-hourly, should be continued day and night. The average blood penicillin level to be aimed at varies from 0.1 unit per c.cm. for streptococcal and gonococcal infections to 0.3 units, or even more, in severe *Staphylococcus aureus* or *Streptococcus viridans* infections.

The total dosage depends upon the infecting organism and the reaction of the patient. Thus, a short course, five injections of 60,000 units over twelve hours, will cure nearly all cases of gonorrhoea, whereas in subacute bacterial endocarditis a daily dosage of 480,000 units may need to be given over three weeks or more.

In most severe systemic infections 160,000 to 320,000 units should be given daily until one or two days after the temperature has returned to normal or the infecting organism cannot be found in the lesions. Good results may often be obtained with a smaller dosage than recommended above, but in cases of serious illness full dosage should be

given. It should be remembered that in any of the serious conditions mentioned above in which local instillations are made, e.g., meningitis, parenteral treatment should also be given.

Prophylactic use.—Penicillin locally and parenterally has been used prophylactically with success whenever an infected area containing sensitive organisms is being operated upon, e.g., prior to lobectomy or extraction of teeth.

Storage.—The fixed rule should be that penicillin products should be kept in the refrigerator. Solutions of penicillin have a short life at room temperatures but will maintain potency for at least three days, and usually more, at 5°C. Dry penicillin may keep its potency for several weeks at room temperature, as may certain penicillin pastilles, but this varies from batch to batch, so it is safer to keep them at a low temperature. Certain common contaminating organisms produce a penicillinase which rapidly destroys penicillin; thus all aseptic precautions must be taken in preparing solutions and such-like.

Toxic Reaction.—Penicillin is a non-toxic drug and most of the reactions are due to impurities in the product. Pyrogen-free distilled water, that is, freshly distilled water which has been immediately autoclaved in a sealed container must always be used for making up penicillin solutions or for normal saline.

Thrombophlebitis of the vein at the site of injection has already been mentioned; local pain in intramuscular injection varies from batch to batch of penicillin; urticaria, and other signs of the toxic hypersensitivity to penicillin have been reported in a few cases.

FAILURES IN PENICILLIN THERAPY.—As a general rule it may be stated that when a patient who is suffering from a systemic infection, due to an organism which has been proved susceptible in the laboratory, fails to show a satisfactory response, then the dosage should be increased and the question of an undrained abscess considered.—*Practitioner*.

The use of digitalis.—*Congestive heart failure*, whether accompanied by auricular fibrillation or normal rhythm, is the indication for the administration of digitalis. The dosage and the choice of preparation depend principally upon the severity of the failure.

In a case of *moderately severe heart failure* there is a choice of three B.P. preparations:—tinctura digitalis, digitalis pulveratum, and digoxinum. From the point of view of efficiency there is little to choose between these, but for the maintenance of equal digitalization the powdered leaf is the best and has the further advantage of being cheaper than the others. The dosage is 15 minims of the tincture, 1½ grains of the powdered leaf, 0.25 mgm. of digoxin, thrice daily. This dosage is maintained until the patient is well digitalized; subsequent dosage depends entirely

upon the individual reaction of the patient. In the case of patients with auricular fibrillation, the best clinical guide is to maintain a ventricular rate of 70 to 80 per minute. The maintenance dose in the vast majority of cases is 1 grain of the powdered leaf (or the equivalent of the other preparations) thrice daily, but in some patients this is excessive, and occasionally patients are encountered who require only half a grain of the powdered leaf (or its equivalent) daily. So long as an individual has auricular fibrillation, digitalization must be maintained and, as in the great majority of cases auricular fibrillation is permanent, this means that such patients have to continue taking digitalis permanently. In these patients, in the absence of complications or relapses, periodic examinations must be made (e.g. three-monthly) to ensure that digitalization is satisfactory.

In the case of *severe heart failure*, when it is considered essential to obtain rapid control of the ventricular rate, either digoxin or strophanthin may be given intravenously or massive doses of one of the three digitalis preparations already mentioned may be used. The type of case in which such therapy is indicated is the patient with severe congestive heart failure and auricular fibrillation with a ventricular rate of 140 per minute or more. In such a patient it is obviously essential to relieve the load on the failing myocardium, and one of the most effective methods of achieving this is by slowing the ventricular rate as quickly as possible. Before using either of these methods of rapid digitalization, however, it is essential to make certain that the patient has not recently been given digitalis.

In the massive dosage method, 60 minims of the tincture of digitalis are given six-hourly during the first twenty-four hours, and subsequently half this dose is given until the ventricular rate has slowed to 80 per minute, when the dose is reduced to that indicated in the paragraph dealing with moderate failure. This method, although still widely used, and based upon the belief that the amount of the drug required for digitalization in man can be reckoned in so many cat units per kgm. of body weight, is not recommended for the following reasons:

(1) There is no good evidence that the cat units is a reliable guide to the activity of digitalis in man.

(2) It is not possible to calculate the appropriate dose of digitalis from the patient's weight, apart altogether from the fact that in grossly oedematous patients it is extremely difficult to assess the actual body weight.

(3) When a potent drug such as digitalis is given in full doses by mouth, it is often difficult to decide whether lack of response of the heart is due to inadequate dosage, poor absorption, or to loss of the drug if vomiting has occurred, as is so liable to happen in severe congestive heart failure. The danger of giving an overdose is therefore considerable.

All these difficulties and dangers are avoided by using an intravenous preparation, and it is for this reason that it is strongly urged that such preparations should replace the massive dosage method. Strophanthin and digoxin are equally effective. The dosage with strophanthin is as follows:—1/120 grain is given intravenously; if, after six to eight hours, the ventricular rate is not definitely slowing, then this dose may be repeated, but if there is evidence of such slowing then 1/240 grain is given. This last amount can be repeated in another six to eight hours should the ventricular rate still be too rapid. As a rule, two or three such injections are required, but occasionally the initial dose is sufficient, so that, once again, it is essential to emphasize the necessity for deciding upon the dosage according to the response in each case. While strophanthin is being given intravenously, one of the three preparations of digitalis should be given by mouth, so that by the time the effect of the intravenous strophanthin begins to wear off, the heart is coming under the control of digitalis. If digoxin is used, the initial dose is 1 mgm., again accompanied by the oral administration of either digoxin, tincture of digitalis or the powdered leaf. Occasionally it may be necessary to give a further intravenous injection of digoxin (0.5 mgm.) eight hours after the initial dose.

Toxic Effects.—Like all potent drugs, digitalis is dangerous in excessive amounts. If signs of toxicity are carefully looked for and dealt with by reduction, or stoppage, of the dosage of digitalis, the danger to the patient is negligible. Nausea and vomiting due to digitalis are indications for immediately reducing the amount of digitalis the patient is receiving. Excessive slowing of the ventricular rate is another toxic manifestation and is dealt with as follows:—

If the ventricular rate falls below 70 per minute, the dose of digitalis is reduced; if it falls below 60 per minute, digitalis is immediately withdrawn, to be renewed again in a smaller dose when the rate has risen again to 80 per minute; if the rate falls to 50 per minute or lower, digitalis is immediately withdrawn and the patient is put to bed until the rate has risen to 80 per minute.

Coupling, which is due to each normal cardiac contraction being followed by a ventricular extrasystole, is also a danger signal, which is dealt with in the same way as slowing of the ventricular rate to 50 per minute. It is only if these signs of toxicity are ignored that the patient is exposed to the most dangerous complication of all—ventricular fibrillation—a fatal, but fortunately excessively rare, complication.

Two other conditions may be mentioned, in which digitalis is often used. In the treatment of *auricular flutter*, digitalis is the treatment of choice, when it is given in ordinary doses, e.g. 1½ grains of the powdered leaf thrice daily, until the flutter is converted to fibrillation. Digitalis is then withdrawn, and in a proportion of cases the fibrillation reverts to normal rhythm.

Occasionally toxic manifestations, such as nausea and vomiting, appear while flutter is still present, and in such cases the administration of digitalis must be stopped even though flutter is still present. Should normal rhythm not ensue an auricular fibrillation, then quinidine is given, unless there are any contraindications to its use.

In auricular fibrillation accompanying thyrotoxicosis, the response to digitalis is seldom satisfactory, although it is always worth while attempting to control the arrhythmia with ordinary (not massive) doses of digitalis. The only remedy for the auricular fibrillation in thyrotoxicosis is treatment of the thyrotoxicosis.—*Practitioner*.

Morphine and respiration.—Contrary to popular opinion morphine does not increase the depth of respiration, according to Dripps and Comroe of the University of Pennsylvania, who administered the drug to 49 normal persons ranging in age from 19 to 88 years, and from 42 to 99 kilograms in weight.

Following intravenous injection, morphine produced maximal depressant effects within 3 to 7 minutes in the majority of instances. Respiratory minute volume, rate and tidal exchange were diminished. The intensity of action of morphine on respiration is not significantly increased by intravenous administration.

The maximal respiratory depression following morphine is approximately the same whether the morphine is administered intravenously or intramuscularly.—DRIPPS, R.D., and COMROE, J. H.: Clinical studies on morphine. *Anesthesiology* 6: 462-468, September 1945.—*Na. Med. Bull.*

Treatment of a symptomatic congenital syphilis.—If a mentally defective child is found to have a persistently positive blood Wasserman's reaction and if, as a result, a diagnosis of a symptomatic congenital syphilis is made, the cerebrospinal fluid should be examined to exclude neurosyphilis. Assuming that the fluid is negative, treatment should consist of weekly injections of an arsenical compound-neoarsphenamine if intravenous therapy is practicable, and sulpharsphenamine if the child is very young or had difficult veins—and a bismuth compound. These injections should be given concurrently in courses of ten of each, with rest intervals between courses of four to six weeks at first, increasing later. Treatment should extend over two years, or even longer if the serum reactions are not reversed, and thereafter the patient should be kept under observation for at least three years; dosage will depend on the age of the child and its tolerance to the drugs. Penicillin might be worth trying, but should be employed in addition to, not instead of arsenic and bismuth. As yet there is little evidence available as to its value in such a condition. Clinical or serological evidence of syphilis in parents and/or siblings would serve to confirm the diagnosis, but would not affect the type or amount of treatment. *B.M.J.*

Globin insulin: a clinical study.—Analysis confirms the favorable reports on the value of globin insulin in moderately severe diabetics requiring a large dose of protamine zinc insulin alone for stabilization. However in the more severe cases needing separate injections of protamine and regular insulin, results with globin insulin were not so favorable. Only 2 of 6 such patients were controlled satisfactorily with a single dose of globin insulin. Close scrutiny of the literature on globin insulin reveals that there are few detailed clinical reports on any large series of cases which previously needed both protamine and regular insulin. In those papers, which do discuss this type of diabetic, the degree of regulation is judged by absence of glycosuria rather than by blood sugar tests. Since in treatment with protamine zinc insulin it is often considered not advisable to eliminate glycosuria because of the danger of nocturnal hypoglycemia, and further because of well-known wide individual variations in renal threshold for glycosuria, it is clear that there are many pitfalls in evaluating diabetic control by urinary sugars alone.—TRASOFF, A.; BORDEN, C.; and MINTZ, S. S.: Globin insulin; clinical study. *Am. J. Digest. Dis.* 12: 313-315, September 1945.—*Na. Med. Bul.*

Pleural reaction in primary tuberculosis.—A primary tuberculous pleurisy develops when primary tuberculosis is at its height. This disorder is to be differentiated from post-primary exudative pleurisy, including the exudative pleurisy which develops within three to six months subsequent to the primary tuberculous infection and from the pleural exudate which is frequently associated with phthisis and miliary tuberculosis. Physical examination fails to demonstrate the pleural exudate, and even a careful roentgenologic examination in the sitting position will fail to disclose comparatively large exudates, which gravitate into the phrenicocostal sinus. Laurell's method, modified by Muller and Lofstedt, facilitates the roentgenologic demonstration of minimal amounts of exudate in the pleural cavity. Investigation on 120 healthy persons revealed that the most suitable projections may be obtained with the rays directed horizontally and by rotating the patient while recumbent on the affected side 20 to 30 degrees backward around his longitudinal axis and by elevating his hip so that the spine forms an angle of about 20 degrees with the horizontal plane. By this method fluid in the pleural cavity was demonstrated in 15 of the 120 examined persons (12.5 per cent). Thoracentesis demonstrated that 3 to 5 cc. of fluid may be disclosed by this roentgenographic technic. In 9 out of 10 patients with primary tuberculosis a pleural exudate was demonstrated on the affected side. The amounts of exudate aspirated by thoracentesis varied between 7 and 175 cc. The largest exudates were found in persons who were examined in the course of the first days after the onset of clinical symptoms. As a rule the exudate was absorbed in two to three weeks; in some cases small residual

quantities persisted for a long period. These exudates have a high albumin content and contain a large number of polymorphonuclear cells. Guinea pig inoculation was positive in 2 cases. Demonstration of tubercle bacilli in the pleural exudate establishes the diagnosis of pulmonary tuberculosis even if tubercle bacilli cannot be recovered from sputum or gastric contents. Determination of the cell content of the pleural exudate is an aid to early and definite differential diagnosis from acute pneumonia, which cannot be made unless the patient is studied for a prolonged period.—*J.A.M.A.*

Sulfadiazine in treatment of pneumococcal pneumonia.—In a 20-month period, nearly 1,500 patients with pneumococcal pneumonia were treated at the Permanente Foundation Hospital in Oakland, California. In the first half of this period, 618 consecutive patients were treated with the usual doses of sulfadiazine, namely, 5 gm. initially and 2 gm. every 6 hours thereafter, maintaining an average blood level of 8 to 10 mg. per 100 c.c. In the second half of this period, 748 consecutive patients with pneumococcal pneumonia were treated with double doses of sulfadiazine. The majority of patients in the group receiving the double dose received 5 gm. of sodium sulfadiazine in 500 c.c. of sixth-molar sodium lactate solution (pH 7.0 to 7.2) intravenously and 5 gm. of sulfadiazine orally (total 10 gm.) initially followed by 4 gm. of sulfadiazine orally 6 hours thereafter, maintaining average blood concentrations of sulfadiazine from 12 to 20 mg.

A comparative analysis of the various important factors influencing the mortality rate in pneumococcal pneumonia was made, with special reference to age, specific pneumococcal types, incidence of bacteremia, leukopenia, severity of associated diseases, extent of pneumococcal involvement, number of days of illness prior to hospitalization and incidence of patients admitted who died soon after. It was found that these two groups of patients had pneumococcal pneumonia of comparable severity. Adjuvant therapy, including the use of specific pneumococcus serum, was similar in both groups. The only significant difference in these two groups of patients was that one group was treated with approximately twice the usual dose of sulfadiazine. The treatment of patients with pneumococcal pneumonia with double doses of sulfadiazine resulted in (1) a decrease in the gross mortality from 10.7 to 6.2 per cent (corrected for patients dying within 6 hours after hospitalization, the mortality rate decreased from 10.2 to 5.5 per cent), (2) a decrease in the incidence of sterile pleural effusions from 5.2 to 2.7 per cent, and (3) a decrease in the average number of days in the hospital required for recovery (66 per cent required less than 7 days, as compared to 45 per cent). There was no greater incidence of drug toxicity in patients treated with double doses of sulfadiazine than in those treated with usual doses.

It was concluded that the use of higher

doses of sulfadiazine results in a great decrease in length of stay in the hospital, without any notable change in incidence of drug toxicity. For severely ill adult patients with pneumococcal pneumonia, sufficiently large doses of sulfadiazine should be administered to maintain blood concentrations of at least 15 mg. with the optimum blood levels maintained preferably at about 20 mg. of sulfadiazine per 100 c.c.—*U.S.J.S.*

Misuse of morphine.—Evidence is mounting that morphine has a single legitimate use; namely the relief of severe pain, a view that is widely accepted but too little put into practice, says Benry K. BEECHER, consultant in anesthesia and resuscitation, Mediterranean Theatre of Operations. Causes of morphine misuse or overdosage, according to him are:

1. Failure to appreciate that nearly the safe maximum analgesic effect of morphine can be produced by $\frac{1}{4}$ grain and that $\frac{1}{4}$ grain can produce serious depression in small persons or those whose tolerance may have been lessened by wounds. An otherwise modest dose may become an overdose in a given case.

2. Failure to realize that subcutaneous injections of morphine will be only poorly absorbed, if at all, when the peripheral circulation is sluggish or inactive, as a result of cold or low systemic blood pressure. Failure to get pain relief from the unabsorbed morphine leads to the administration of a second or third dose, all of which may be absorbed at one time when the peripheral circulation is re-established as a result of resuscitative measures.

3. The incorrect belief that extensive wounds are inevitably associated with severe pain often leads to needless use of morphine. At Anzio, only a quarter of the severely wounded patients said, in response to a direct question, that they were having enough pain to want pain relief medication.

4. Administration of morphine for conditions that will not respond satisfactorily to the agent, however large the dose, is a common error. Specifically, the use of morphine to treat the restlessness associated with bleeding, with anoxia, or with hysteria cannot be soundly defended. The use of morphine in shock, except for the treatment of severe pain, is contraindicated. Morphine is occasionally administered when other agents (barbiturate sedation, for example) are far better.—BEECHER, H. K.: Editorial: Misuse of morphine as therapeutic agent. *Surg. Gynec. and Obst.* 81: 461-462, October 1945.—*Na. Med. Bull.*

Epidemic hepatitis in a combat zone.—The icterus index is not a reliable guide in determining either the activity of hepatitis or the degree of hepatic functional impairment following an attack of epidemic hepatitis. That is, the degree of icterus does not always indicate the severity of hepatic damage. The icterus index is, however, useful in following the course of the disease. The hippuric acid test of hepatic

function frequently indicates residual hepatic functional impairment long after the icterus index has returned to normal. Also the sedimentation rate is useful in determining the residual activity of the hepatitis and frequently remains elevated for several weeks after the icterus index has returned to normal.

These conclusions were drawn from observations on 238 unselected patients with hepatitis and jaundice seen in an Army general hospital in Italy over a 6-month period. An additional 17 patients having hepatitis without jaundice were studied. All cases were verified by various clinical and laboratory methods. An icterus index of at least 16 was arbitrarily set as the lower limit indicating the presence of jaundice; milder forms of the disease were thus excluded. There was one death in the series.

The clinical characteristics of epidemic hepatitis with jaundice were found to be not essentially different from those of catarrhal jaundice. Clinically it is similar to post-vaccinal hepatitis, but differs in the frequent occurrence of chills, fever, and lymphadenopathy, and in the relative absence of dermatitis, anemia, and mental reactions.

Epidemic hepatitis is frequently a serious disease which by its seemingly benign course may mislead both the patient and clinician. A prolonged convalescent period and conservative management are important in treatment—FINKS, R. M., and BLUMBERG, R. W.: Epidemic hepatitis with and without jaundice; some clinical studies on 255 patients among troops in combat zone. Arch. Int. Med. 76: 102-113, August 1945.

Penicillin indications for its use and methods of administration.—The suitability of a case for treatment with penicillin depends not so much on the nature of the disease as on the susceptibility of the micro-organism causing it: some species are highly susceptible, and others far too resistant for this treatment to have any effect. It follows that in diseases which may be caused by any of several different bacteria (e.g. meningitis, peritonitis) a bacteriological diagnosis is usually necessary if treatment is to be undertaken with any assurance of success.

The chief organisms susceptible to penicillin are:—Gonococcus, Meningococcus, Staphylococcus aureus, Streptococcus pyogenes, Streptococcus viridans (most pathogenic strains), Pneumococcus, *C. Diphtheriae*, Clostridia of gas-gangrene, Spirochaetes of syphilis and relapsing fever; possibly of Well's disease, Actinomyces, *B. Anthracis*.

PENICILLIN IS INDICATED in the treatment of the following conditions when due to a susceptible organism unless the infection is likely to respond rapidly to sulphonamides:

- (a) Septicæmia (systemic administration),
- (b) Puerperal sepsis (systemic administration),
- (c) Acute cellulitis (systemic administration),
- (d) Acute osteomyelitis (systemic administration),
- (e) Cavernous and lateral sinus thrombosis (systemic administration),
- (f) Pneumonia (systemic administration),
- (g) Meningitis (intrathecal injection, often

combined with systemic administration),

- (h) Carbuncles and acute abscess (systemic or local administration),
- (i) Certain types of empyemata (intrapleural injection),
- (j) Suppurative arthritis (intra-articular injection, sometimes combined with systemic administration),
- (k) Anthrax (systemic administration),
- (l) Actinomycosis (systemic administration),
- (m) Gonorrhœa, acute and chronic (systemic administration),
- (n) Gas gangrene and "malignant œdema" (systemic administration), and
- (o) Other acute infections due to organisms sensitive to penicillin but resistant to sulphonamides.

PENICILLIN IS OFTEN OF VALUE in the following conditions:—

- (a) Burns (cream or spray),
- (b) Sycosis barbæ (cream or spray),
- (c) Impetigo (cream or spray),
- (d) Dermatitis with infection (cream or spray),
- (e) Conjunctivitis and infections of cornea (eye drops, cream or lamellæ),
- (f) Infected wounds (powder, cream, or in solution),
- (g) Encouraging results have recently been obtained in infections of the mouth and throat, using pastilles containing penicillin,
- (h) Penicillin may be used as a prophylactic agent in wounds exposed to infection,
- (i) Infections of the urinary tract due to sensitive organisms (systemic administration).

PENICILLIN MAY BE OF VALUE in the following diseases, but its place in relation to other forms of treatment has not yet been defined:—

- (a) Syphilis,
- (b) Subacute bacterial endocarditis,
- (c) Diphtheria,
- (d) Acute mastoiditis and otitis media,
- (e) Peritonitis,
- (f) Chronic osteomyelitis.

PENICILLIN IS OF DOUBTFUL VALUE OR OF NO VALUE in the following conditions, for which it should not be used:—

- (a) Tuberculosis,
- (b) Acute rheumatic fever,
- (c) Rheumatoid arthritis,
- (d) Ulcerative colitis,
- (e) Infections caused by viruses such as influenza, anterior poliomyelitis and encephalitis lethargica,
- (f) All Gram-negative bacillary infections such as typhoid fever, dysentery, undulant fever, and infections with *Bact. coli*, *H. influenzae*, *Proteus*, *Ps. plocyana* and *Bact. Friedlander*,
- (g) Glandular fever,
- (h) Pemphigus,
- (i) Hodgkin's disease and the reticuloses,
- (j) Leukæmia,
- (k) Malaria,
- (l) Cancer.

ADMINISTRATION:—Unless the following properties of penicillin are appreciated, unsatisfactory therapeutic results are likely to be obtained and wastage of this expensive drug is bound to occur:—

(a) Once penicillin has been removed from the ampoule or tube it is likely to deteriorate, particularly if exposed to moisture or heat. Solutions or other preparations of penicillin exposed to air and kept at room temperature will not deteriorate significantly in 24 hours, but should not be kept for more than 48 hours under these conditions.

(b) Penicillin is rapidly destroyed by boiling, by most antiseptics, and by enzymes of many of the common air bacteria.

(c) Penicillin should not be given by the mouth.

(d) Penicillin passes rapidly from the blood into vascular tissues, but the serous membranes and meninges present a barrier which penicillin does not readily penetrate.

(e) Penicillin is rapidly excreted in the urine: for this reason, it must be given at frequent intervals or continuously.

(f) The diffusion of penicillin into dead or avascular tissues is slow; sequestra, large sloughs, or collections of pus, are, therefore, likely to harbour bacteria out of reach of the drug, and these usually have to be removed if treatment is to be effective.

I. SYSTEMIC ADMINISTRATION.—Either the sodium or calcium salt may be used

(a) *Intramuscular injection.*—Fifteen thousand units dissolved in 2-3 cc of sterile water or saline are injected every three hours throughout the day and night. This method is simple and effective, but many patients find it difficult to tolerate, on account of the pain produced.

(b) *Continuous infusion* by "drip" of 100,000 to 120,000 units daily into a muscle or vein. The drawback of the intravenous route is the high incidence of thrombosis; otherwise this method is very satisfactory. The main drawback of the intramuscular route is discomfort, and the development of local œdema when large amounts of fluid are given. This does not occur if special equipment delivering 100 cc in 24 hours at a constant rate is used. The apparatus required is comparatively simple, and this method of administration is preferred by many.

(c) *The duration of treatment* varies considerably, according to the condition and response of the patient but should seldom be for less than five days or more than 12 days. Treatment is usually continued for about two days after a favourable response has been obtained.

(d) *The dosage* described above is recommended for all conditions requiring systemic treatment except the following:

(1) Syphilis: 240,000 units daily for 20 days has been recommended.

(2) Gonorrhœa: in uncomplicated cases a total of 100,000 units given in one day is usually effective.

(3) Bacterial endocarditis: a daily dose of 240,000 units has been recommended, but even prolonged treatment may be unsuccessful.

(4) Actinomycosis: the dosage varies considerably according to the sensitivity of the actinomyces.

(e) *Toxic reactions:*—Pure penicillin will probably prove to be virtually non-toxic, but penicillin as used at present contains impurities which occasionally give rise to minor reactions, such as fever, and urticarial rashes.

II. LOCAL APPLICATION: When applied locally, penicillin must be brought into contact with the infecting organism. Merely to apply penicillin to the surface of an infection of considerable depth, such as a boil or carbuncle, is to waste time and penicillin.

(a) *Solutions* in sterile water or saline of 250 units or more per c.c. are used for local

treatment. The solutions may be used for injection through tubes stitched into wounds after closure, or through a needle into abscess cavities after aspiration of pus. The injection should be made twice daily. Irrigation of an open wound is ineffective.

BURNS of the face may be sprayed but penicillin creams are usually preferred for burns in other areas.

In *meningitis* a strength of 1,000 units per c.c. is generally used, and from 5-10 c.c. are usually injected daily by lumbar or cisternal puncture. (The purer the penicillin, the less likely it is to produce meningeal irritation.) In *empyema* 25,000 to 75,000 units in 50-100 c.c. are injected every day, or on alternate days, into the pleural cavity.

For *infections of the eye*, drops of 500 to 2,000 units per cc. may be applied every half to two hours.

(b) *Powders:*—Penicillin (preferably the calcium salt) is mixed with sufficient sterile sulphanilamide or sulphathiazole powder to produce a final concentration of 2,000 to 5,000 units per gram. The powder can be blown with an insufflator on to raw surfaces or into wounds, and the application should be repeated once or twice daily.

(c) *Creams.*—Several types of cream have been recommended, but the following is satisfactory for most purposes:—

The base consists of 30 per cent. lanette wax SX in water. The requisite amounts of lanette wax and water are heated to 40°C. The water is slowly mixed into the wax in a mortar, and stirring is continued until the mixture is almost cold. This usually takes about ten minutes, when a smooth cream is formed. The emulsion is then autoclaved at 20 lbs. pressure for 20 minutes. When it has cooled to about 48°C., the correct amount of penicillin, dissolved in a few c.c. of sterile distilled water, is added and thoroughly mixed with a sterile rod or spatula.

The cream is usually applied daily.—*Tropical Dis. Bull.*

Primary atypical pneumonia.—By C. Kelman Robertson, M.D., D.P.H., F.R.C.P.E.

The term "pneumonia" has for long inferred either a clear-cut clinical entity recognised as pneumococcal lobar pneumonia or a grumbling toxic pulmonary change with diverse bacteriological findings known as broncho-pneumonia.

Within recent years, however, a "new" pneumonia has commanded a great deal of attention, and this has been variously named pneumonitis, viroid, viral or primary atypical pneumonia. Recent reviews relative to this modern type of pulmonary inflammation indicate that it is not a new disease *per se*, but one that has been in our midst for the best part of a century, at least, and that it has only now become more easily recognised because of greater facilities for X-ray examination and more frequent bacteriological testing.

No doubt much of the interest centred around this latest pulmonary arrival is

due to the sudden increase in the recognition of cases as a result of the congregation of large numbers of young people for military and national duties, bringing greater advantages for clinical observation and the use of ancillary aids to diagnosis than could ever have happened in the normal run of general practice.

The essential cause of so-called atypical pneumonia is not definitely known, but much work has been done which proves that viruses can be isolated from these cases. It is known, however, that an exactly similar pulmonary syndrome can arise as the result of infection with various agents such as rickettsia, fungi, some bacteria and certain well-recognised viruses, but it is uncertain whether or not one specific type of virus alone can be held as solely responsible for this malady.

That the spread of this condition is by air-borne infection has been proved by spraying the throats of human volunteers with infected sputum from known sufferers, thus inducing the disease.

The epidemiological characters of this form of pneumonia are protean, as it may not only bring about sharp explosive outbreaks in a community, but can also display endemic propensities when it causes isolated sporadic cases from time to time. Susceptibility to this illness is most pronounced in adolescence, although no age group can be considered entirely immune, as people of all ages may be affected when the disease assumes epidemic proportions. Like all other respiratory tract infections, the majority of cases appear during the colder winter months. Pathologically the steps in the advance of this malady seem to be fairly straightforward. The infecting agent (virus) causes a simple inflammation of the bronchial mucous membrane, and this gradually spreads outwards to the surrounding peribronchial tissues, which brings about swelling, oedema and cell infiltration of the collagenous reticulum of the strands of lung parenchyma. This, in turn, finally gives rise to increased pressure on adjacent air spaces, with resultant alveolar collapse.

The clinical features of this disease reveal its true atypical nature. The ailment is introduced by the general signs of febrile indisposition, malaise, with disinclination for food and aching pains on moving the eyes. Soon after this vague series of symptoms set in, attention is directed towards the respiratory system because the patient develops a dry rasping cough, troublesome as a result of its frequency and lack of accompanying sputum. Any expectoration that does appear is mucopurulent in character. The tracheal irritation responsible for this cough usually gives rise to a constant deep-seated substernal ache or gnaw. Rhinorrhoea and lachrymation are sometimes marked.

Beyond the obvious cough, then this train of clinical events could hardly be considered as pathognomic of any serious form of pulmonary upset.

Physical examination of the chest is also prone to be inconclusive, as only a few small

localised patches of dullness may be found, over which are highpitched or crackling rhonci with distant "post-tussic" crepitations. Obvious clinical pleural involvement is relatively uncommon.

Probably the most significant thing about this chest illness is the fact that much of it is essentially negative in character, as these patients very rarely look really ill; they are not flushed and poisoned-looking; they have no marked tachypnoea, no herpetic eruption, nor yet any of the other obvious appearances suggestive of a grossly toxic state.

No characteristic blood changes are found in primary atypical pneumonia. Blood culture is invariably negative, although, of course, there is usually a moderate leucocytosis with raised erythrocyte sedimentation rate.

X-ray examination of the lungs shows many variations, the common finding, however, being increased shadowing of the tracheo-bronchial glands with associated haziness or blurring of the pneumonitic sites and sometimes a generalised retiform appearance of the whole of the lung parenchyma.

Some other diseases merit consideration before atypical pneumonia can be asseverated, and of those the more important are the common cold, influenza, bacterial pneumonia, pulmonary tuberculosis and ornithosis. Primacy must always be given to the possibility of pulmonary tuberculosis, demanding, if necessary, repeated bacteriological and radiological investigation.

Although atypical pneumonia usually runs an uninterrupted course, the patient being in bed for some two weeks and thereafter convalescing for a similar period, setbacks due to complications are not unknown, and of these two big groups must be recognised. The first group includes local pleuro-pulmonary changes such as the reactivation of a latent tuberculosis focus, transient bronchial dilatation, serous pleural effusion or even rib fracture due to the strain of coughing. The second group affects the central nervous system when toxic psychotic phases of elation and depression appear. Meningismus can, of course, herald the onset of the malady.

Treatment in the beginning must be entirely along general and symptomatic lines because there is no known specific for this form of pneumonia. A sulpha drug, such as sulphadiazine, can, however, be advantageously administered to offset any secondary invading organisms. Steam inhalations of a soothing type are helpful in allaying symptoms due to tracheo-bronchial irritation. Expectoants such as ammonium carbonate or ammonium chloride definitely serve a useful purpose here, as they avoid the grosser bronchitis and atelectatic pneumonia of this disease. Diaphoretics have no real place in the treatment of atypical pneumonia because they only increase the patient's feeling of exhaustion and weakness by causing extra loss of salts and fluids from the bodily tissues. Adequate rest in bed with good general nursing must

form the basis of treatment. Ordinary diet is indicated.

Perhaps one of the best prognostic guides is the estimation of the patient's blood sedimentation rate. A good period of convalescence should always be advised and during this time a strychnine or quinine tonic can be given justifiably because of its stimulating effect on the nervous system.

—*Medical World.*

Penicillin with aluminum hydroxide.—A convenient practical and effective procedure of administering penicillin by mouth consists of mixing the drug with one table-

spoonful of aluminum hydroxide. Substantially higher blood levels are obtained following a 50,000- and a 100,000-unit dose of penicillin with this method than after administration of the drug dissolved in water, either with or without previous ingestion of a mild antacid, such as aluminum hydroxide. Absorption of penicillin on aluminum hydroxide appears to exert a local protective effect against that portion of gastric acid with which it comes into contact.—BARACH, A. L.; GARTHWAITE, B.; OPPENHEIMER, E. T.; FORMAN, J.; and OSBURG, H.: Oral administration of penicillin. *Science* 102: 247-250, September 7, 1945.—*Na. Med. Bull.*

SURGERY

The prevention of post-operative pulmonary embolism.—Post-operative pulmonary embolism is one of the dreaded complications of abdominal surgery. More than usual interest therefore attaches to a report from the Chelsea Hospital for Women (J. P. Erskine, and I. C. Shires; *Journal of Obstetrics and Gynaecology of the British Empire*, October 1945, 52, 480). In 1937 a massage department was established, the duties of which were to "instruct and supervise post-operative exercises, and to carry out massage in all cases after abdominal operations and operations for the repair of genital prolapse." The report is based upon a comparison of the findings for the seven years preceding the institution of the massage department (1930-36) and the seven years following its institution (1937-43). In the period 1930-36, there were 4,596 abdominal operations, with nineteen deaths from pulmonary embolism, i.e., the mortality from this cause was 0.415 per cent. In the period 1937-43, there were 3,436 abdominal operations, with nine deaths from pulmonary embolism; two of these deaths are excluded, as one of the patients had never had post-operative exercises or massage, and the other died during the operation. This gives a mortality rate of 0.203 per cent. from pulmonary embolism. In three of the years in this latter period there were no deaths from this cause. In other words, the incidence of fatal embolism following abdominal operations fell by more than 50 per cent. following the institution of regular exercises and massage. Some other interesting points emerge from the report. During the period 1930-43 a total of 21,066 operations was performed at this hospital, followed by 202 deaths, of which forty were due to pulmonary embolism. The maximum incidence of fatalities due to pulmonary embolism was in the sixty to sixty-nine years age-group, the average age being 50.5 years. The fact that 43.5 per cent. of the cases of fatal embolism occurred during the first week after operation indicates that exercises and massage should be instituted as soon as possible after operation. Temperature charts were only available in thirty-two of the cases of fatal embolism, but in twenty-five of these (i.e., 78.1 per cent.) a

rise in temperature occurred during the three days preceding the fatal embolism. Four of the fatal cases of embolism followed the insertion of radium.—*Practitioner.*

Grey Turner's sign in acute pancreatitis.

—Grey Turner's sign is the discoloration of the abdominal wall which occurs in certain cases of acute pancreatitis. In reporting a case showing this sign, H. T. Cox (*British Journal of Surgery*, October 1945, 33, 182) discusses its etiology.

The patient was a woman of sixty-five, who was admitted to hospital on account of upper abdominal pain of sixteen hours' duration. A diagnosis of acute pancreatitis was made. On the third day of the disease a discoloured area appeared in the skin around the umbilicus, followed on the seventh day by a similar, but smaller, area of discoloration in the left flank. These areas, consisting of an admixture of blue, green and yellow colours, resembled the colour changes in a fading ecchymosis. Subsequently a well-localized mass formed in the left hypochondrium; incision revealed permeation of the oblique and transversus muscles with dark red blood, and a cavity containing 3 ounces of blood clot which was reported as showing increased lipase activity. The patient made an uneventful recovery.

It is generally agreed that the discoloration in the flank is due to direct extraperitoneal spread of blood and pancreatic secretion, but the umbilical discoloration, which is rare, is more difficult to explain. This rarity may be due to a pathological variation in the disease or to an anatomical variation at the umbilicus. One suggested mechanism for its production is an extra-peritoneal spread, limited above and below the umbilicus by the suspensory ligament and the urachus, with a pooling in the potential sub-umbilical space where there is direct contact with the subcutaneous tissue as a result of absence of the fascia transversalis. Another explanation, which might account for its rarity, is to be found in the umbilical fascia (a thickening of the fascia transversalis), which as a rule completely or practically covers, and is firmly applied to,

the umbilical ring. Rarely, this fascia occurs as a band about the thickness of a finger, well above the umbilicus; in these cases there is direct continuity at the umbilicus between the extraperitoneal and the subcutaneous tissues. This anatomical variation in the presence of a retro-peritoneal spread might well be one of the deciding factors in the development of umbilical discoloration in acute pancreatitis.—*Practitioner*.

Traumatic shock.—Fine and his associates earlier presented evidence that in traumatic shock the capillaries outside areas of injury do not "leak" plasma; the essential pathologic condition is not a disturbed capillary permeability but a disturbed peripheral circulation in which the amount of blood in active circulation is deficient. The therapeutic problem is one of restoring normal velocity and volume flow through capillaries. If after loss of blood replacement is effected early the problem is solved, but if too much time elapses before replacement the damage caused by the loss becomes irreversible. Whether this damage consists in a loss of integrity inherent in the central or peripheral circulatory apparatus *per se* or is secondary to the failure of an extravascular controlling factor is not known. No explanation for the shock state other than the hemodynamic imbalance was necessary. Tourniquet shock differs from hemorrhagic shock, but the authors show that the increased hematocrit of tourniquet shock is a liability over and above that of volume deficiency and can account for the observed differences between tourniquet shock and hemorrhagic shock. Volume deficiency is by far the greater danger, and its restoration is a more urgent need than is that of normal viscosity. Utilizing hemorrhagic shock and eliminating as far as possible operative manipulations, anesthetics and other forms of trauma, which facilitate the development of irreversibility or otherwise complicate the shock state, the authors studied various agents in every instance only after the shock state was found to be unresponsive to the replacement of all lost blood. The course of events in hemorrhagic shock leading to the development of a state of irreversibility to transfusion is a function of the severity and duration of inadequate capillary flow, with a resulting cumulative adverse effect on the integrity of cellular function in general or on a basic biochemical function in a controlling vital organ such as the liver. In studies on the intermediary metabolism in shock the aim was to discover some point in the process of biochemical disintegration, *i.e.*, failure of completion of a normal biochemical reaction, which would identify the onset of irreversibility. It was found that metabolites, including the amino acids, are catabolized as readily in the irreversible as in the reversible phase of shock, almost as well as in the normal animal, and the increased concentration of these substances in the blood represents an adjustment of metabolic reactions at a new level. The effect of

viiperfusion of the liver on the prevention of the development of irreversibility is not due to the removal of toxins or to the provision of protective substances by the donor dog during the shock phase, because the same processes operate in the dog viiperfused via the jugular vein.—*J.A.M.A.*

Experiences with the Miller-Abbott tube.

—The Miller-Abbott tube has made the small intestine accessible for physiologic observation, diagnosis, and therapy. Used prophylactically before operation, the tube has been successful in decreasing the incidence and clinical severity of post-operative ileus associated with operations upon the small intestines, large hernias, resections of the colon, and in the presence of peritonitis.

In right colectomy its advantages have been so striking that its rather universal use has permitted a single-stage resection to displace the formerly considered more conservative method of two-stage resection, with enterostomy.

Whereas it has also been useful in deflating the small intestine following resections of the left colon and rectum, it has not been as effective in these cases in deflating the remaining colon, and thereby obviating the necessity of a proximal colostomy.

Post-operatively it has deflated the stomach, but it should not be used primarily for this purpose. Its greatest function is deflation of the small intestine.

This tube has been a means of preoperatively diagnosing the presence and site of small intestinal tumors, foreign bodies, adhesions, kinks, bands, bleeding sites, and malformations. At operation, it has served as a guide to the site of obstruction. It has also permitted the small intestine to be, so to speak, pleated on the tube, thereby facilitating the retraction of the small intestine from the operative area.—Smith, B. C.: Experiences with Miller-Abbott tube. *Ann. Surg.* 122: 253-259, August 1945.—*Na Med. Bull.*

Teratoma of the testis.—J. L. Barner (*American Journal of Roentgenology*, 54: 257, Sept. 1945) reports 65 cases of teratoma of the testis treated in an Army General Hospital; the average age of the patients in this series was twenty-eight years. Early diagnosis of teratoma of the testis is important; the most frequent symptom is painless swelling (observed in 45 patients in this series); pain was present in 14 cases. In the earlier stages, the testicle is smooth and freely movable; and is usually firm or hard, although cystic areas may be present. In the series reported, surgery followed by irradiation was employed. Orchiectomy was done with removal of the cord with accompanying structures high at the internal abdominal ring. This was followed by roentgen therapy, using daily treatments (except on Sunday) of 200 to 250r to each of two fields, in rotation, with a total dosage of 1,600 to 2,000r per field. A follow-up of these patients showed that 7 have died in the last thirty-three months; 7 are showing

symptoms of recurrence; but the majority are well and have returned to work. The author considers the combination of surgery and irradiation superior to either alone in the treatment of teratoma of the testis.—*Medical Times*.

Back pain.—In disease of the gastro-intestinal and accessory gastro-intestinal tract:—It has been suggested that back pain as one of the manifestations of gastro-intestinal disease can result from a summation of impulses originating in visceral walls and referred to the back along the sympathetic nerves which carry such impulses to the spinal cord.

The authors believe that the unilateral shifts of pain arising because of penetrating lesions involving these tracts can, and probably often do, travel to the cord along branches of the spinal nerves. They believe that this is a slightly different type of pain from that usually caused by impulses arising within visceral walls, being more acute, more localizable and better limited to definite segmental distributions. In the case of inflammatory disease—and occasionally of erosive neoplastic disease—involving retroperitoneal tissues such as acute pancreatitis, back pain is an important symptom. Lesions in this region could involve both spinal nerves and those using sympathetic routes. In such lesions pain is deep seated, frequently severe and fairly well localized. A characteristic feature of this pain and one which is of great diagnostic importance is that patients who have such lesions are usually found to lean forward to relieve their pain. There is still another in which back pain can be caused by disease originating in the tracts under discussion. Malignant lesions originating in the tracts there may metastasize to the spinal column. In such instances the pain is usually boring, deeply located, bilaterally felt and constant. In most instances it is well to remember that back pain is one of the manifestations of a disease of the digestive or accessory digestive organs, is a symptom which should be considered with great care because as a rule it indicates that the lesion causing it is a complicated one.

It is always well to suspect disease arising in the gastro-intestinal or accessory gastro-intestinal tract of patients complaining of dyspepsia as one of the symptoms associated with other back pain.—*J.A.M.A.*

Backache.—*Examination and differential diagnosis:*—Backache may be divided into following groups:

1. In most cases of acute backache, an accurate diagnosis should not be attempted on the first examination as the picture is masked by pain and muscle spasm.

2. A fairly large group of patients seek advice because of the fear that their backache may be due to some serious involvement of the spinal column or kidneys or some such parts.

3. Third group with definite organic lesion.

4. Group of patients with compensation backache.

5: Hopeless backache due to malignant conditions or other diseases for which nothing can be done.

HISTORY: Type of pain, onset and duration of the pain, the localisation and distribution or reference of the pain and many other features.

Pain may be: (1) Static relieved by rest and brought on by activity, as in traumatic spondylitis, spondylolisthesis, spondylolysis, old fractures of vertebrae, facets and pedicles;

(2) Morning: usually of inflammatory lesions.

(3) Nocturnal: usually associated with neurologic conditions *e.g.* tumours of the spinal cord.

(4) Constant: usually seen in malignant lesions, osteomyelitis, etc.

EXAMINATION: *Inspection:*—Posture, deformities, movements of the vertebral column, whether limited, should be noted.

Palpation and percussion:—Tender points should be noted. Localised area of superficial tenderness should be detected such as tenderness at the ligamentous and muscular attachment over the spinous processes, etc.

Spasm of the muscles of the back, wasting of the muscles, flabbiness of the muscles, etc., should be noted.

Special tests: Straight leg rising test, indicative of spasm of hamstring muscles caused by lumbosacral lesion. (Lasague's sign). Goldthwait's sign, Ely's sign and Patrick's test and other tests have been used in differential diagnosis. Another important sign is Ober's sign which means a tight iliotibial band. Antero-posterior and lateral roentgenograms are essential.

Laboratory tests:—Urine: routine. Blood: Hemoglobin test, leucocytosis, sedimentation rate, etc.

LESION WHICH OFTEN UNDERLIE: *Pain in lower part of the back:* 1. *Postural Strain:*—Abnormal lordosis, adolescent obesity, faulty body and foot mechanism.

2. *Static or post-traumatic:*—Spondylolisthesis, defective pedicles, old fractures, thin disks, protruded disks, congenital anomalies.

3. *Rheumatic:*—Infectious arthritis, spondylitis deformans, myositis.

4. *Infectious:*—Tuberculous—benign osteitis, osteitic deformans.

5. *Senescent:*—Senile osteoporosis, senescent and hypertrophic arthritis.

6. *Neoplastic:*—A. Malignant myeloma, metastatic malignant lesion. Primary sarcoma, Ewing's tumor.

B. Benign osteoma, and osteochondroma, Giant cell tumor, hemangioma.

The typical poker spine with calcification of the ligaments and rounded kyphotic deformity with limited expansion of the thorax and elevation of sedimentation rate = spondylitis deformans.

Pain and stiffness particularly if it occurs after sitting or rising in the morning, that is relieved to a large extent by activity and is not associated with evidence of skeletal

changes and very stubborn to treatment—myositis or fibrositis.

Moderately painful vertebral column with some localising tenderness on percussion, thinning of inter vertebral disk with evidence of tuberculosis elsewhere. Kyphos and abscess in late stage—probably tuberculous.

Woman of middle or advanced age with sharp pain in the thoracic or lumbar region and gradually increasing thoracic kyphos, roentgenograms showing extensive osteoporosis of vertebrae, compression fractures and ballooning of intervertebral disks—probably senile osteoporosis.

Vertebral pain which cannot be relieved by rest, heat, massage and mild sedative with primary lesion somewhere (breast in female and prostate in male) points to metastatic malignant lesion.—*J.A.M.A.*

The management of acute craniocerebral injuries.—A. Price Heusner, M.D. A practical clinical approach to the problems arising out of such injuries must be based upon:

(1) An acquaintance with the various lesions which may be excited by a blow to the head;

(2) An ability to distinguish between "operative" and "non-operative" lesions; and,

(3) A knowledge of the basic supportive or medical treatment essential for all such patients.

Table I sets forth the craniocerebral lesions commonly encountered in civilian practice.

TABLE I.—COMMON TRAUMATIC CRANIO-CEREBRAL LESIONS.

- A. Scalp:
 - (1) Abrasion.
 - (2) Hematoma.
 - (3) Laceration.
 - (4) Avulsion.
- B. Skull:
 - (1) Linear Fracture.
 - (2) Depressed Fracture.
 - (3) Compound Fracture.
 - (a) Of vault.
 - (b) Of air sinus.
 - (c) Through ear.
 - (d) Through nose.
- C. Brain:
 - (1) Concussion.
 - (2) Congestion and edema.
 - (3) Contusion and laceration.
- D. Meninges and Vessels:
 - (1) Extradural Hematoma.
 - (2) Subdural Hematoma.
- E. Cranial Nerves:
 - (1) Stretching and avulsion.
 - (2) Laceration.
 - (3) Compression.

The only acute "operative" lesions are those in which the injury has exposed:

(1) The intracranial contents to infection which antibiotic and chemotherapy cannot be expected to prevent (*i.e.*, some compound fractures); or,

(2) The brain to localized compression (*i.e.*, extra—or subdural hematoma).

The "operative" cases constitute a relatively small percentage of all head injuries. On a traumatic neurosurgical ward to which the more severely injured patients tend to gravitate, fewer than 33% of the cases require surgical intervention, and in any unselected group of craniocerebral injuries this percentage would be far smaller. Their importance lies in the fact that the devastating consequences which attend failure to recognize these lesions are, for the most part, avoidable.

Recognition of compound fractures is rarely difficult and may be achieved, in nearly every instance, during the initial physical examination. Those of the vault may be apparent by inspection alone and are always detectable by palpation through the scalp wound with a sterile gloved finger. X-ray examination is a less reliable method of diagnosing this lesion and entails unnecessary transportation of the patient. (On the other hand, X-ray examination is the only way of diagnosing the rare "internal" compound fracture involving the posterior wall of a frontal air sinus). Most of these fractures require surgical debridement if serious intracranial infections are to be prevented. It is otherwise, however, with the traumatic fistulae permitting the escape of brain and or cerebrospinal fluid through the nose or ear. Such fistulae are regularly detectable on the first examination and serve to classify the patient as a potentially operative case. If the patient is placed on sulfadiazine and penicillin it is safe to withhold surgery for several days or even longer, and one is usually rewarded by spontaneous closure of the fistulous tract.

Recognition of the second group of "operative" cases, the compressing clots, is sometimes possible at the time of the first examination, but more frequently their detection requires a period of clinical observation during which the patient's response to active supportive therapy is carefully noted. Occasionally the discovery of these lesions entails diagnostic trephination for direct inspection of the extra and subdural spaces. Much of the difficulty attending recognition of these clots may be overcome if it be borne in mind that they are "delayed" phenomena. The laceration of the vessel which gives rise to the clot occurs, to be sure, at the moment of the injury, but it requires the lapse of an appreciable amount of time for the hematoma to attain compressive proportions. The amount of time required for the clot to express itself clinically, the so-called latent period, is determined in the main by the vigor of the bleeding vessel. In the case of an extradural hemorrhage which regularly arises from a dural artery or a large dural sinus, this interval is usually a matter of a few hours or even less time. In the case of subdural hemorrhage, which usually arises from a small vein, the interval is longer and may be a matter of days. In any event, a compressing clot should be suspected and a surgical consultant summoned when following a blow to the head:

(1) The patient first improves (*i.e.*, exhibits a lucid or quasi-lucid interval) and then regresses; or,

(2) The patient fails to improve or fails to "hold his own" under a regime of adequate supportive therapy.

The early signs of regression include increasing headache, failing or waxing and waning of responsiveness, neurological evidence of hemiparesis and increasing intracranial pressure as indicated by lumbar puncture. Unless surgical intervention is undertaken before bradycardia, convulsions, coma, periodic respirations and a dilated pupil supervene it is not apt to prove beneficial.

It can hardly be overemphasized that the conventional signs of regression are not pathognomonic of compressing clot. They may arise singly or in any combination as the result of one or more of the common complications which are prone to develop if active supportive therapy of these acutely ill patients is neglected. These complications include persistent surgical shock from unsuspected extracranial lesions, hyperthermia, anoxia, hypercapnia, toxic dehydration, acute infections (especially pneumonia), and cerebral compression resulting from congestion and edema of the brain. Most of these complications can be avoided by the supportive regime presently to be outlined, but the matter of shocking extracranial lesions requires special consideration.

Surgical shock which does not promptly yield to the application of warmth and the transfusion of whole blood or any of its substitutes is a very uncommon consequence of head injury. When shock persists after such treatment, extracranial causes must be sought and in nearly every instance they can be discovered by bedside methods alone. The lesions most frequently found under these circumstances include fracture of a long bone, ruptured abdominal viscera, hemothorax, hemopneumothorax, cervical cord injury, and fracture of the pelvis with extravasation of urine. The importance of this understanding of surgical shock in patients with head injuries is evident. Without it, patients succumb to the shock of untreated extracranial lesions which have been masked by the coma of (otherwise) favourable craniocerebral injuries.

TABLE II.—INITIAL EVALUATION OF PATIENT IN ACUTE PHASE OF A CRANIO-CEREBRAL INJURY.

- A. Look for surgical shock:
 - (1) If present: (a) Start transfusion; (b) Seek extracranial cause. (2) If absent, proceed as below.
- B. Estimate state of responsiveness:
 - (1) Note responses to various stimuli
 - (2) Record in objective fashion.
- C. Examine head:
 - (1) Look for lacerations: palpate for fracture (2) Look for hematomata (3) Look for fluid from a cranial orifice (4) Look for intracranial bruit.
- D. Examine eyes:
 - (1) Note pupils: size and reactivity
 - (2) Note axes: are they parallel?

E. Estimate motor power and sensation:

- (1) Is there hemiparesis? (2) Is there hemihypalgesia?

F. Test speech.

G. Test reflexes.

H. Perform lumbar puncture (omit Queckenstedt).

I. Determine need for x-ray examination:

Treatment of the patient should follow immediately upon completion of the initial examination. If the patient is comatose the first consideration in every instance is that of so positioning him as to prevent the aspiration of vomitus or nasopharyngeal secretion (common forerunners of pneumonia) and insure an adequate respiratory exchange (the best safeguard against the development of cerebral congestion and edema). Such a position is shown in Figure 1. It will be noted that the patient's face is tilted downward thereby utilizing gravity as a means of keeping the flaccid tongue well forward of the hypopharyngeal air passage.

Further steps in therapy are outlined in Table III.

TABLE III.—TREATMENT OF PATIENT IN ACUTE PHASE OF A CRANIO-CEREBRAL INJURY:

- A. Position patient in bed.
- B. Administer complete antishock therapy.
- C. Insure adequacy of respiratory exchange:
 - (1) Proper position in bed (2) Install oral or nasal airway if necessary (3) Combat accumulation of secretions in air-passages by (a) Suction (b) Atropine (4) Combat irregularity of respiratory rhythm with aminophylline.
- D. If a simple scalp laceration exists:
 - (1) Debride and suture if general condition permits; otherwise (2) Shave surrounding hair, cleanse, and apply sulfa powder with D.S.D. locally.
- E. If a compound fracture exists:
 - (1) Start systematic chemotherapy
 - (2) Apply sulfanilamide and D.S.D. locally.
 - (3) Give antigas and antitetanus sera.
 - (4) Arrange for debridement.
- F. Control intracranial pressure:
 - (1) Lumbar drainage at suitable intervals, (2) Hypertonic sugar solutions.
- G. Attend to vegetative functions:
 - (1) Hydrate and nourish (a) Preferably by mouth or indwelling stomach tube; otherwise (b) By intravenous therapy, (2) Regulate temperature, (a) Avoid too many bed clothes, (b) Combat hyperthermia with rectal aspirin and tepid sponges with rubbing, (c) Keep bladder decompressed by indwelling (nupercaine lubricated) catheter if necessary. This reduces "restlessness" and bed sores in incontinent patients.
- H. Administer penicillin (antipneumonia measure):
 - (1) To the elderly (2) To all who remain long in coma.
- I. Prevent patient doing harm to himself:
 - (1) Restraints if necessary; *i.e.* bed-sides (do not restrain limbs). (2) Judicious use of sedation: (a) Keep to minimum (b) Paraldehyde by choice.

While this regime is in force there must be

repeated observations of the patient by both nurse and doctor. The nurse must make and record frequent estimations of the state of responsiveness, pulse, blood pressure, respiratory exchange, temperature, motion of the limbs, size of the pupils, and she should report a seizure at once. The doctor must supplement these observations with repeated testing of the state of responsiveness, speech, state of the pupils, reflex activity, and motor power of the limbs. Under such management the signs of regression will either be averted or detected sufficiently early to insure timely surgical attack upon compressing clots.

In conclusion it is well to emphasize that the problems of acute head injuries are chiefly medical rather than surgical. The choice of treatment no longer rests between surgery and "intelligent neglect." Active supportive measures are imperative if the profession is to obtain end-results of which it may be proud.

—*Bull. New. Eng. Med. Centre.*

Antitoxin in gas gangrene.—The prophylactic use of either serums of drugs, or both together may serve a valuable purpose in making both early and delayed surgery more secure, or in permitting primary closure of a wound which would otherwise be left open, and many surgeons now prefer to use both these adjuncts. But the use of serums or drugs can never be regarded as a substitute for adequate surgery.

The treatment of infected wounds under catastrophic conditions, as in war, earthquakes and train accidents, must necessarily be based upon the assumption that prompt

surgery is generally impossible. Under these conditions the injection of polyvalent, preferably pentavalent, gas gangrene antitoxin and tetanus antitoxin assumes a major prophylactic role, making possible and effective delayed surgery to save lives and limbs. It is necessary, of course, to guard against anaphylactic shock in asthmatics and others sensitive to horse serum.—*HALL, I. C.*: Value of antitoxin in prevention and treatment of malignant edema and gas gangrene: review of observations. *Ann. Surg.* 122: 197-209, August 1945.—*Na. Med. Bull.*

Paget's disease of the nipple.—The Paget cell develops from the normal epithelial cell. It has the same structure as the so-called vacuolated cells of carcinomatous ascites and pleural fluid and the cells of tar cancer. The changes with characterize the Paget cell are not the product of degeneration or of fixation, or a peculiar type of intracellular edema or a disturbance of glycogen metabolism or vacuoles. The Paget cell is not a misplaced embryonal germ cell or a nevus cell or a melanoblast. Whether the Paget cell is, according to L. Savatard (1935) and other authors, already a cancer cell is a question which cannot be answered satisfactorily, as cells of the type of Paget in the Paget cell is a normal response of the epithelial cell toward different kinds of animate and inanimate stimuli, among which are tar, x-rays, sunlight, viruses and the unknown causes of cancerous conditions, psoriasis and lichen planus.—*MEIROWSKY, E., and KEYS, S.*: Paget cell; its structure, occurrence and significance. *Proc. Roy. Soc. Med.* 38: 495-499, July 1945.—*Na. Med. Bull.*

OBSTETRICS and GYNÆCOLOGY

Menstrual migraine.—Cyclical headaches of this kind, sometimes described as menstrual migraine, may be of pituitary origin, but not necessarily. They are sometimes associated with high gonadotrophin and low oestrogen levels; gonadotrophic hormone therapy is therefore contra-indicated—it may make matters worse. Sometimes they are related to the retention of sodium and fluid in the tissues, and this in turn to a high oestrogen level in the blood. If, therefore, there is any sign of premenstrual oedema, distension, or increase in weight, oestrogens are also contraindicated. Many are the theories to account for the headache. Swelling of the pituitary gland, cerebral anaemia, vasodilatation of cranial and cerebral blood vessels, hormone imbalance, allergy, neurosis. It would be of interest, if nothing more, to have the skull X-rayed to see if the roof of the pituitary fossa is calcified. A complete investigation of the eyes, teeth, ears, sinuses, etc., as in any case of migraine, is also called for. Although oestrogens (including stilboestrol) and testosterone preparations administered premenstrually are reported to be helpful sometimes, in general the results are disappointing. Treatment on more simple lines, aimed at reducing

fluid retentions and intra cranial pressure is often the best. Advise the patient to have a salt free diet and to restrict her intake of fluid to 30 oz in 24 hours, for seven to ten days premenstrually. Over the same period give ammonium chloride gr. 15 t. d. s. by mouth, and also saline aperients if necessary. Avoid all sodium salts. Sometimes the intramuscular injection of a mercurial diuretic, one or two days before menstruation, is worth while in cases in which the above is insufficient.

If in spite of treatment the headache continues large doses of ergotamine tartrate by mouth (1 to 5 mg. initially, followed by 1 to 2 mg. every three or four hours) should be tried, keeping in mind its changes if continued too long. Inhalations of pure oxygen are also said to give symptomatic relief in some cases. It has been suggested that ergometrine works equally well. If the headaches still persist, the patient will probably sooner or later raise the question of having the menopause induced artificially, if she does it is contraindicated. As a last resort, however, and if the headache is very intense and associated with a calcified roof to the sellaturica, X-ray therapy to the pituitary gland might be considered; although it

often has untoward side-effects, such as the development of obesity.—*B.M.J.*

Treatment of anovular menstruation.—Assuming that the patient has a twenty-eight day bleeding cycle, begin treatment on the fifth day (counting from the first day of the periods giving 400 I. U. of serum gonadotrophin intramuscularly each day up to and including the fourteenth day (ten injections). From the fifteenth to twenty fourth days inclusive, give daily injections of 500 I. U. of chorionic gonadotrophin. Carry out endometrial biopsy just before, or at the beginning of, ensuing menstrual period to see if there is a secretory reaction. If there is

no sign of ovulation omit treatment during the next cycle: but during one following that repeat the course, doubling the doses of both preparations. If there is still no response there is little point in continuing the treatment, but a course of injections of chorionic gonadotrophin combined with pituitary synergic factor might be tried instead. Should ovulations occur as a result of the injection, repeat the treatment every second or third month, instructing the patient to try to conceive and omitting, of course the endometrial biopsy. Since these gonadotrophic are protein derivatives, it would be wise to carry out preliminary skin sensitivity tests before each course.—*B.M.J.*

OPHTHALMOLOGY

The prophylaxis, diagnosis, and treatment of sympathetic ophthalmia in the light of further observations.—*Friedman, S. J., Viestnik Oft., 1944, v. 23, pt. 5, p. 37.*

Without a clear concept of its genesis we have been handicapped in prevention and treatment of sympathetic ophthalmia. Until now we have been preoccupied with the mode of transmission from eye to eye and with the nature of the pathogenic agent. There is evidence in favor of a meningitic process mediating between the exciting eye and the sympathizing eye, and of the ocular affection not being limited to the eyes. The meningitic process is of the serious, basal type with meagre symptomatology, and the sympathetic ophthalmia responds to this same treatment to which basal meningitis responds, namely spinal punctures. These therefore are indicated in all uveitides, and in the prevention of sympathetic ophthalmia. Two cases additional to those previously observed are reported in detail. Further arguments in favor of the relation between the meningitic process and sympathetic ophthalmia are advanced. The 2 or 3 weeks interval between the appearance of the process in one eye and its appearance in the other is similar to that observed in development of uveitis secondary to a virus

in basal meningitis. The development of sympathetic ophthalmia after enucleation, sometimes 2 to 3 months later, and at times years later, argues for localization of the infectious process at the base of the brain. The occasional observation of optic neuritis in sympathetic ophthalmia is also in favor of a chiasmatic arachnoiditis. Both sympathetic ophthalmia and meningitis are more common in children, and the fact of sympathetic ophthalmia not being reproducible experimentally also speaks in favor of the belief. So far however no specificity of the spinal-fluid picture or specificity of the blood picture has been determined, nor has any marked difference in the blood picture after enucleation or evisceration been observed. But sufficient observations have been made to justify careful neurologic study of all sympathetic ophthalmia cases, and the addition to local therapy, in perforating injuries, of treatment directed to the meningitic process namely, repeated lumbar puncture, intravenous urotropine, and glucose and salt solutions. Evisceration should only be done in cases of panophthalmitis. Operations for traumatic cataract should not be done too early. Patients with perforating eye injuries should not be discharged as cured until there is normal spinal fluid and normal hematology.

Book Reviews

Dictionary of Indian and Foreign Medicine (With Home remedies)—Embodied and edited by Sri Swami Ekanand of Rishikesh—Pp. 506. Published by Sagar Medico Publico Co., Sole Representative—Seth Lilaram Ramchand, Bankers, 87, Bazar Road, G. T. Madras.

On account of several languages that are being spoken of in different parts of India, it was a wise decision on the part of the author to embody in one bookform the various names in the different languages of the drugs, medicinal plants etc., in this country. Food grains, vegetables, flowers, fruits, minerals, animal products, spices, oil seeds, herbs, plants etc., commonly used in Indian medicine are mentioned in this book together with a brief summary of their uses and the

parts played in Indian Medicine. There is a chapter on home remedies. With some of the treatments in this chapter we may agree as they are based mostly on common sense. But, with reference to the treatment of some of the more dangerous and infectious diseases such as cholera, diphtheria etc., it would be preferable to put oneself under the more modern allopathic treatment. The treatment suggested for the bite of a mad dog is not modern. To those who are interested in the system of Indian Medicine, this book will be found useful and handy.

Recent Advances in Therapeutics—Part I and II by J. R. GOVAL, M.B., B.S., 1945, pp. 345. Price Rs. 6-8-0.

Since the year of publication of the first edition of this book, many notable advances

have been made and the author has incorporated some of these in this new edition. Part I deals with many new Sulphanilamide compounds like Sulfapyridine, Sulfathiazole, Sulfamethylthiazole etc., Part II deals with Penicillin, Hormones and with some of the recent methods in the treatment of diseases such as shock treatment of Schizophrenia, Gold treatment in Rheumatoid Arthritis etc. It is a very useful book giving to the general practitioner information on the latest drugs and methods of treatment of diseases.

Intravenous Therapy—By K. V. THAKKAR, L.M. & S., Bombay University, Late Chief Medical Officer to the States of Palitana and Idar—1944, Second edition, pp. 349, with a coloured frontispiece and ten illustrations. Price Rs. 8-8-0.

This is a second edition of a very useful little handbook containing details of the methods of intravenous injections. A large number of drugs that have been found to be of use in modern therapeutics find a place in this book. Chapters on Saline Infusion and on blood transfusion have been revised. New chapters on Speed Shock, Intravenous Drip and Intravenous Anaes-

thesia have been added. It is well illustrated and is bound to be useful to the general practitioner.

Anatomy and Physiology for Students of Physiotherapy, Occupational Therapy and Gymnastics—By C. F. V. SMOUT, M.D., M.R.C.S., L.R.C.P., and R. J. S. MacDOWALL, M.D., D.Sc., F.R.C.P. (Edin.), 1944, Pp. 418 illustrated—(Edward Arnold & Co., London) Price Sh. 30/- nett.

This book on anatomy and physiology has been written for the student of Massage and Medical Gymnastics. It has been planned in strict accordance with the syllabus of the Chartered Society of Physiotherapists.

In the section on Anatomy all unnecessary details have been omitted and what is absolutely essential has been included. Prof. MacDowall has written the section of Physiology and has condensed into a very limited space, essential features of a wide and difficult subject, and has presented it in a form that could be easily assimilated by students.

The book is printed on art paper and is profusely illustrated. The general get-up is very good.

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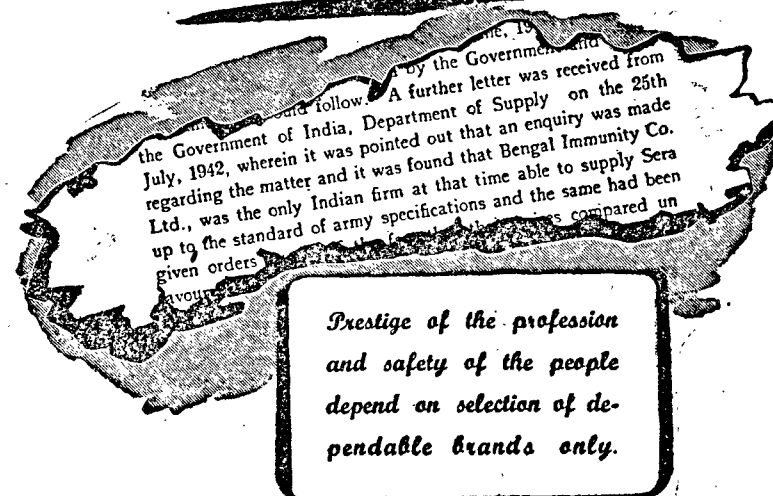
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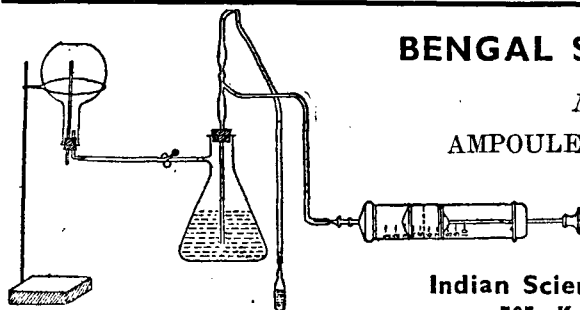
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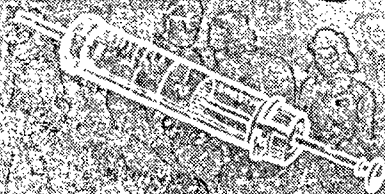
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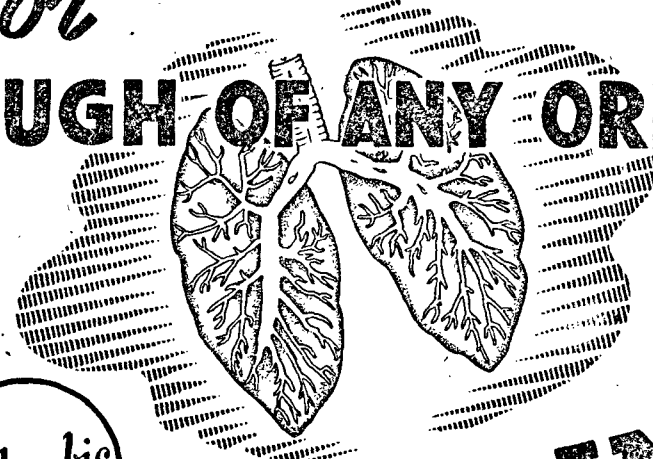
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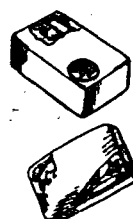
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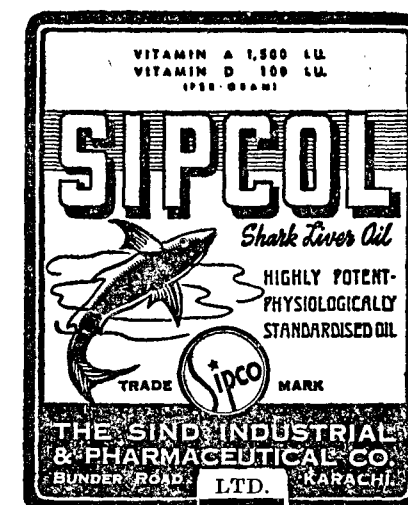
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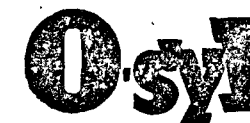
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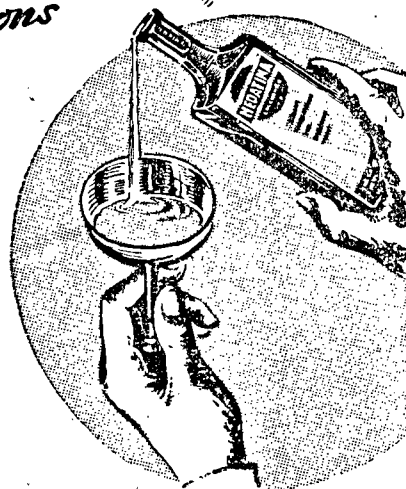
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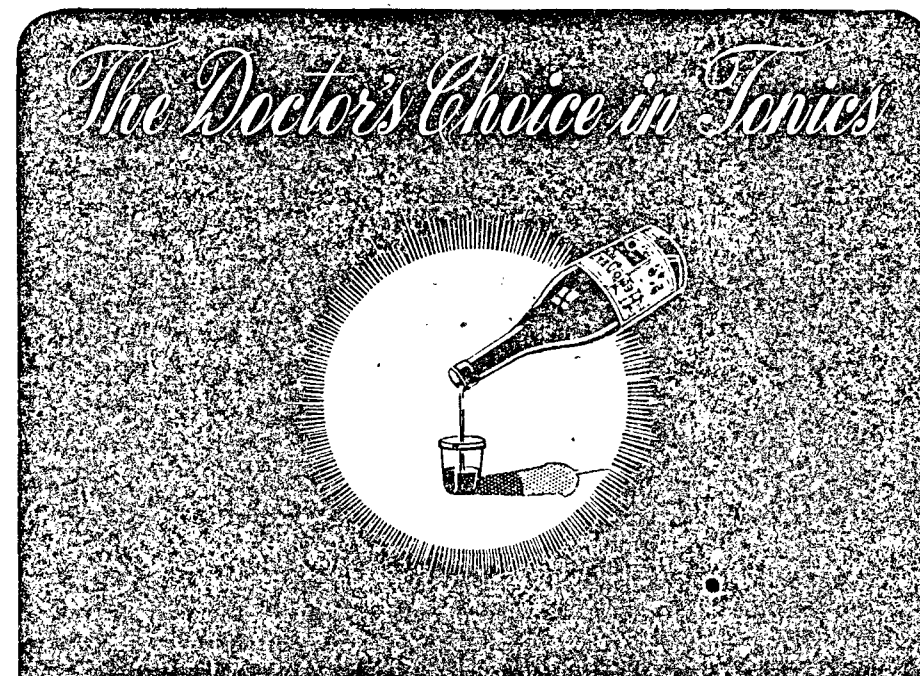
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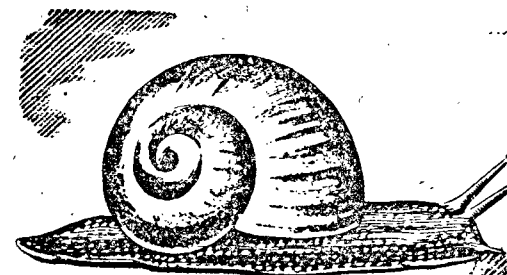
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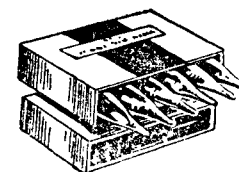
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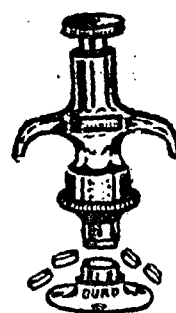
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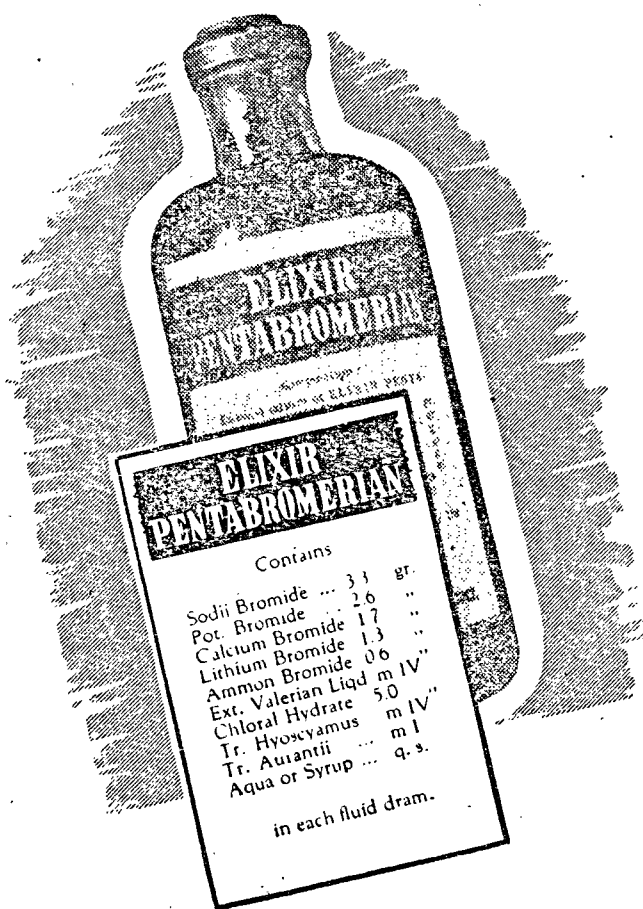
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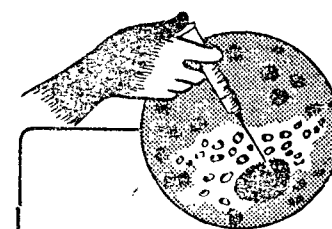
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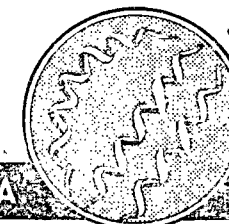
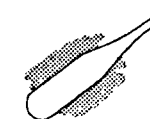
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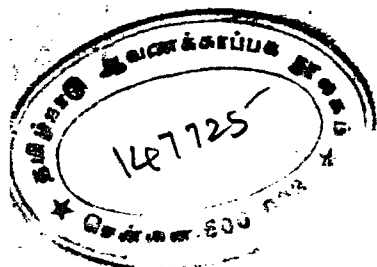
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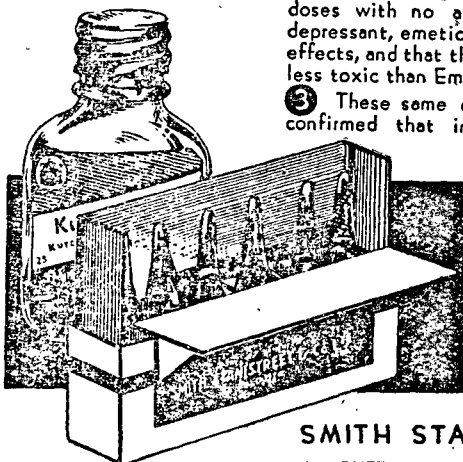
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