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No. 2
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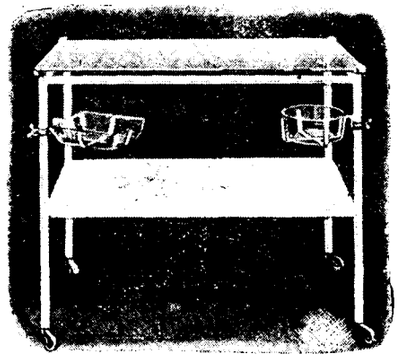

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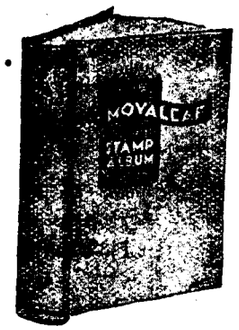

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N. S. Narasimhan, L.R.C.P. (Lond.), F.R.C.S. (Eng. & Ire.)

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TO LET

TO LET

C. A. S. Oct. '40.

"The Pledge of the Fellows of the American College of Surgeons"

"I pledge myself to pursue the practice of Surgery with thorough Self-restraint and to place the welfare of my patients above all-else: to advance constantly in knowledge by the study of surgical literature, the instruction of eminent teachers, interchange of opinion amongst associates and attendance on the important societies and clinics: to regard scrupulously the interests of my professional brothers and seek their counsel when in doubt of my own judgment; to render willing help to my colleagues and to give freely my services to the needy. Moreover, I pledge myself, as far as I am able, to avoid the sins of selfishness, to shun unwarranted publicity, dishonest money-seeking, and commercialism as disgraceful to our profession; to refuse utterly all money trades with consultants, practitioners or others, to teach the patient his financial duty to the physician, to expect the practitioner to obtain his compensation directly from the patient; to make my fees commensurate with the service rendered and with the patient's rights and to avoid discrediting my associates by taking unwarranted compensation."

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Renal Tuberculosis.

BY

DR. N. M. RAO, M.B., F.R.C.S.,E.,

Surgeon, Government General Hospital, Madras.

Renal tuberculosis is found in 1 per cent of all autopsies and 5-8 per cent of autopsies in patients dying of tuberculosis (Kapsammer). Kocher found pulmonary foci in 80 per cent of autopsies on cases of genito-urinary tuberculosis.

It may form part of a generalized miliary tuberculosis when both kidneys are found affected. This type is more common in children in whom it is bilateral in 53 per cent of cases.

It also occurs as a localized infection limited to one kidney with no other active tuberculous focus elsewhere in the body in 94-95 per cent and only 5-6 per cent show an active lesion. This type is known as Surgical Renal Tuberculosis. It is however never primary as 60 per cent of cases show a healed or latent focus in the lungs, mediastinal glands, etc. This type is seen in the third and fourth decades of life. It is unilateral in 85 to 90 per cent at the beginning. It forms 30 per cent of surgical renal lesions and is twice as common as renal tumours (Young).

In men, kidneys and genital organs may be affected in equal proportions. In women genital tuberculosis is rare and kidney is more often affected.

Renal infection shows a slightly higher incidence in men than in women as kidney may be affected from genital tuberculosis also.

Either kidney may be affected equally in men but in women the right kidney is more often affected.

Etiology.—Infection of kidney occurs through the blood stream from a focus elsewhere in the body, usually in the lungs, mediastinal glands, etc. In men it may also be infected from foci in the prostate and seminal vesicles either by the blood stream or by an ascending peri ureteral lymphangitis.

Clinical types of Renal Tuberculosis.—1. Miliary type. It is bilateral

forming part of a generalized miliary tuberculosis. It is more common in children.

2. *Surgical types.*—Ulcero-Caseous or Ulcero-Cavernous type. Thisis the commonest of the surgical types where nephrectomy is so useful.

3. *Massive Caseous type.*—This is a more chronic form. In this one or more caseous areas form in the cortico-medullary junction surrounded by tuberculous granulation tissue. This type often extends towards the calyces and ulcerates into the renal pelvis. Some may become encapsulated and later on become infiltrated with lime salts. The kidney is often enlarged, lobulated and palpable.

4. *Tuberculous Nephritis.*—A subacute nephritis with heavy infection may be sometimes seen in young people, albumin, blood and casts being present in the urine. It usually settles down to one of the previous types 1 or 2.

5. *Tuberculous Pyonephrosis.*—Partial obstruction of ureter leads to dilatation of pelvis and calyces into a sac filled with urine and tuberculous debri.

6. *Chronic Fibrotic type.*—It is rare and often missed. It is found occasionally in grown up adults. The kidney is shrunken by marked fibrosis with a few scattered tubercles, the only symptom being the presence of albumin in the urine.

Closed Renal Tuberculosis.—This may occur in the ulcero-caseous type by occulsion of the ureter. It also occurs occasionally in the massive caseous type.

Pathology.—The bacilli are deposited in the region of arcuate arteries. 75 per cent of lesions occur in the cortex and show a tendency to heal, 13 per cent occur in the cortico-medullary region and 11 per cent in the medullary area. The latter two are often progressive ones.

No healthy kidney ever passes tubercle bacilli in the urine without being infected. Microscopic lesions occur in both kidneys of phthisical patients, but in a large number these heal. Medler was able to demonstrate such lesions in over 60 per cent of cases dying of pulmonary tuberculosis. Where general resistance is good but the local resistance of a kidney has been lowered by trauma or disease, the lesions may persist and progress. The medullary and cortico-medullary lesions show a tendency to progress and ultimately involve the whole kidney.

The earliest naked eye lesion is a small yellow tubercle at one of the poles in the cortico-medullary region or near the apex of a renal papilla. As the cortico-medullary type spreads the kidney becomes enlarged and bossing and tubercles appear on the surface followed by adhesions to the peri-renal fat. At this time the tubules may be filled with bacilli and leucocytes which are now and then discharged in the urine without any actual ulceration into the calyces. In many cases the infection extends towards the renal pelvis and finally ulcerates into a calyx and the caseous debri is passed in large amounts in the urine attended with renal colic. In a few cases the caseous areas may become encapsulated and later on infiltrated with calcium salts.

Medullary Type.—The focus usually on or near a papilla extends towards a calyx and ulcerates into it. The disease also spreads towards the bases of the pyramids resulting in a

cavity lined by ragged walls of necrotic tissue which may show a zone of grey tubercles. Outside this, areas of isolated tubercles may be seen dotting the renal tissue or arranged as streaks radiating towards the cortex. Other pyramids become similarly converted into caseating cavities with ragged edges surrounded by yellow streaks of tubercles leading to the cortex. In more advanced cases the renal pelvis is much dilated, the papillæ destroyed, and calyces deeply excavated and the ureter is found thickened and irregularly dilated. Partial occulsion of the ureter by tuberculous strictures may lead to tuberculous pyonephrosis in which the kidney is converted into a multilocular sac filled with urine and necrotic material. With complete occulsion the pus may become inspissated and later impregnated with lime salts. Fibrosis at the neck of a major calyx or at the pelvi-ureteral junction may lead to a partial or complete pyonephrosis. Where no obstruction develops, the whole pelvis and calyces become ulcerated. The fatty areolar tissue round the pelvis and the kidney becomes thickened, sclerosed and densely adherent to the kidney and surrounding structures. The fat round the hilum, renal pedicle and along the inferior vena cava also becomes sclerosed and adherent. Glands along the inferior vena cava may become enlarged and adherent.

In some young people the entire kidney becomes involved simultaneously resulting in an acute tuberculous nephritis. The kidney becomes engorged, œdematous and enlarged. The urine may be smoky and contain albumin, blood and casts. But it soon settles down into one of the more chronic types.

Changes in the ureter.—Passage of tubercular debri through the ureter causes infiltration, ulceration of the mucosa and fibrosis usually at or near the anatomical constrictions and leads to dilatation of the intervening portions of the ureter between the constrictions. There is also a descending periureteral lymphatic infiltration. The ureter becomes thickened and rigid. All gradations, from a few patches of ulceration to a most deformed ureter, may be seen. The ureter becomes adherent to the peritoneum and surrounding tissues. In long standing cases irregular infiltration of ureter with lime salts may occur.

Bladder changes.—Bladder becomes infected by the tubercular debri brought down by the urine, and also by descending lymphatic infiltration. In the early stages the ureteral opening is swollen and red and a halo of hyperaemia is seen in the mucosa where the impact of efflux occurs. Next round cell infiltration occurs in the submucosa near the ureteral opening. Then grey tubercles form. These caseate and break down into small superficial ulcers with sharply cut margins. These ulcers run together to form larger ones with irregular undermined edges and a greyish yellow or hæmorrhagic base. Yellow tubercles are seen dotted around the ulcers on a slightly congested mucosa.

Infiltration of the ureter causes its opening to be rigid, rounded and wide-open "Golf Hole ureteral opening". It is also pulled upwards and outwards into an asymmetrical position.

In the absence of secondary infection the lesions are discrete with healthy mucosa intervening. In the presence of secondary infection bullous oedema may hide the ureteral openings. If extensive ulceration is present irregular exuberant masses of

granulations may sprout up especially in the presence of secondary infection and are called Tuberculomas. With progress of the disease, wide-spread infiltration of the bladder wall and subsequent fibrosis leads to contraction of the bladder whose capacity may be reduced in late stages, to a couple of ounces when it is known as "Thimble Bladder". Pericystitis occurs followed by fibrosis of perivesical fat and the bladder may become adherent to surrounding structures as intestines, rectum, uterus, etc. Pelvic glands may enlarge but abscess formation is rare.

Opposite kidney.—It may become enlarged from compensatory hypertrophy. It may also suffer from toxic nephritis. Later it may become infected through the blood stream or by retrograde lymphatic infiltration from advanced tuberculous cystitis.

Clinical features.—I shall confine myself to 'Surgical Renal Tuberculosis in which clinically only one kidney is affected and where nephrectomy is so useful.

This type is usually seen in young adults, 38 per cent in the third decade and 32 per cent in the fourth decade. It is practically symptomless till ulceration into a calyx has taken place. At first the symptoms are entirely referred to the bladder and cases are diagnosed as cystitis and treated with drugs and lavage.

Frequency of micturition occurs in 75 per cent. Increased frequency is the first symptom. It may be slight and variable depending on the weather, diet and climate. It is at first diurnal and as it increases it becomes also nocturnal. It is not affected by rest. A burning sensation accompanies it. This increases to scalding and pain shooting along the urethra. At this stage centrifuged deposit shows a few pus cells and red blood corpuscles. Later frequency becomes so marked that the patient has to pass urine every twenty to thirty minutes and scalding is severe enough to cause tenesmus. There is now an urgency in micturition and in heavy sleepers incontinence may occur.

Polyurea is an early sign and is due to interstitial fibrosis and it increases the frequency. Urine is pale, opalescent, faintly acid or even neutral in reaction and often hazy from a small amount of well-mixed pus. Presence of albumin is an early sign and may occur independently of polyurea and hæmaturea.

Loss of weight.—A continuous slight loss of weight is noticed in the early stages. In the advanced stages loss of weight is more rapid from want of sleep, mental and physical rest and toxaemia.

Hæmaturea.—Usually it is microscopic and occurs in 40 per cent of cases. The deposit shows a greater proportion of pus cells to red cells. Visible hæmaturea may occur in about 5 per cent, especially in the acute type but it rarely persists.

Pyurea is fairly constant in the open types and occurs occasionally in the closed type unless lesions are encapsulated. The amount of pus is variable and often in small amounts. It occurs often with hæmaturea especially after ulceration of bladder.

Pain.—It is present in about 37 per cent of cases. Renal pain as a dull ache in the renal angle may be slight or absent. Renal colic occurs during passage of clots and tubercular debri. Vesical pain occurs with ulceration of trigone and radiates along the urethra or the perineum.

No. I. Excretion pyelography—Note the irregular areas of calcification in both kidneys from a case of bilateral renal tuberculosis. (Female.)

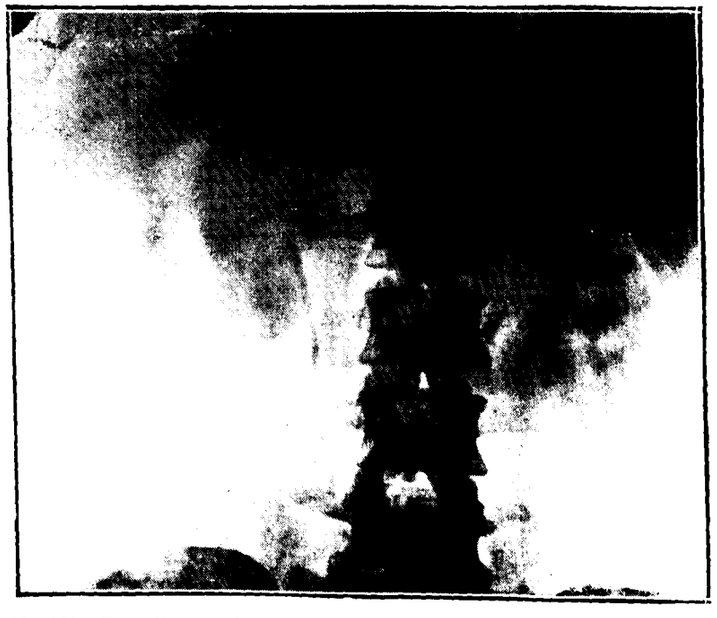
Lt. Rt.

No. II. Excretion pyelography in a case of bilateral renal tuberculosis. Note the dilation and fluffy outline of minor calyces. Note the dilatation of ureters. Bacilli were isolated from urine. (Male.)

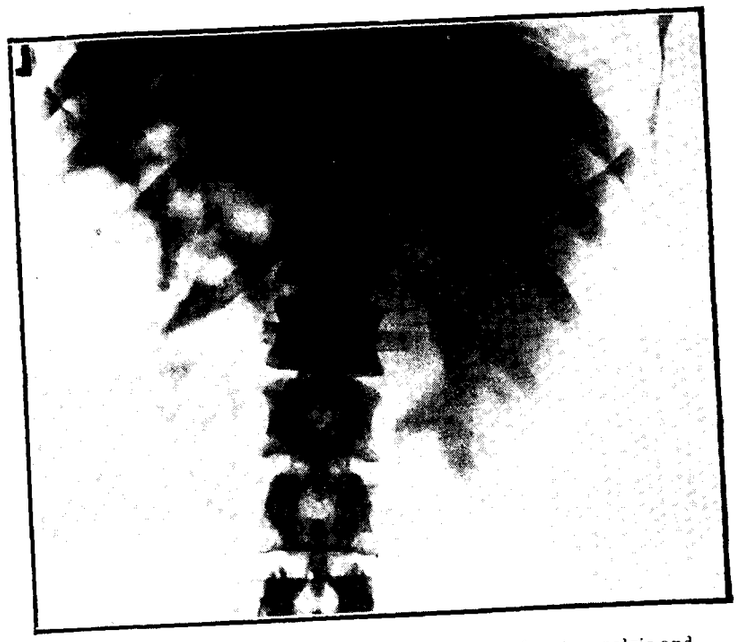
Lt. Rt.

No. III. Excretion pyelography—Note absence of excretion of dye on the left side. An enlarged kidney showing irregular opacities due to calcium deposit is noticed on the left side. The right ureter and pelvis dilated from back pressure due to "thimble bladder." Nephrectomy was done two years ago and patient is doing well. (Female.)

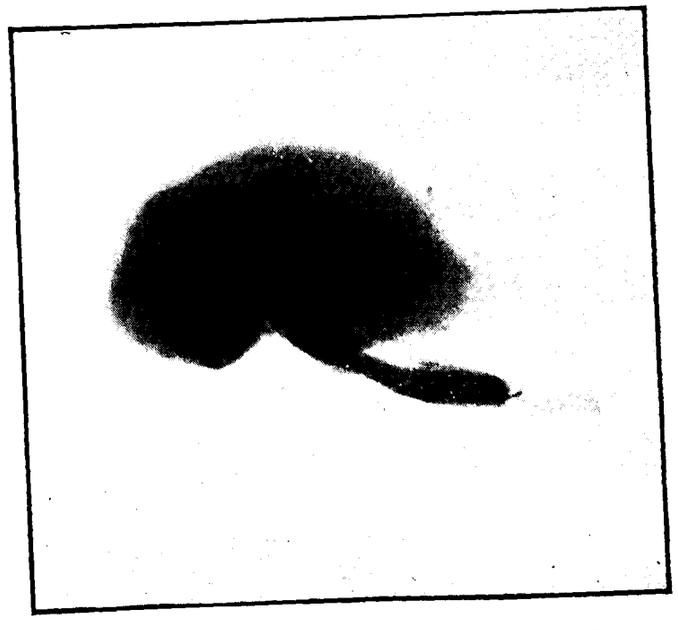
Lt. Rt.



No. IV. Excretion pyelography—Note the dilatation of ureter and calyces on the right side. Bacilli isolated. Nephrectomy right side was done two years ago. Patient is doing well and has joined his work. (Male patient.)



V. Excretion pyelography—Note the dilation of ureter, pelvis and calyces on the right. Note the irregular borders of calyces. Bacilli isolated. Right nephrectomy performed last year. Male patient. Doing well.



VI. X-ray-Picture of removed kidney with sodium iodide injected. Note the stricture at pelvi-ureteric junction and the ulceration of upper calyces.

With onset of vesical ulceration and contraction of bladder, frequency rapidly increases and is accompanied by scalding and pain along the urethra. With ulceration of trigone, urgency, tenesmus or strangury and terminal hæmaturea appear. Pain in the bladder is constant and made worse by voiding urine. Frequency is so great that the patient voids urine every few minutes, sleep and rest become impossible. Nocturnal incontinence and wetting of clothes may occur from urgency. Patient avoids society, ceases work, spends sleepless nights, rapidly loses weight and leads a miserable existence. Incontinence is more marked in women owing to their shorter and wider urethra.

Tumour formation.–Tuberculous kidney is seldom palpable unless pyonephrosis is present. In the diffuse massive caseous type the kidney may be palpable as an irregular, firm and slightly lobulated tumour. But more often it is fixed high up by adhesions to be easily palpable.

Thickened ureters may be palpable in the fornices in women and sometimes per rectum in men. In a few thin patients a thickened ureter may be palpated at the brim of the pelvis.

Fever is usually absent in the earlier stages. In some temperature may rise to 99° to 100° F. Higher temperatures indicate a secondary infection, active pulmonary or generalised tuberculosis.

Prognosis.—The onset and progress of uncomplicated cases is slow. Fluctuations in the symptoms are frequent. They abate and patient improves to relapse again. This may be due to change of climate, diet and the tendency for the disease to be walled off by fibrosis. In some pus and bacilli may disappear from the urine for months to reappear suddenly after a strain or injury, etc. Spontaneous arrest of the disease is rare. When secondary infection is superadded, progress is more rapid accompanied by fever and pyurea. In untreated cases opposite kidney becomes affected sooner or later. Death usually occurs from phthisis, uræmia or sometimes from miliary tuberculosis.

Diagnosis.—Frequency with burning unaffected by rest occurring in young adults, with acid urine, sterile to ordinary cultural methods and containing a greater proportion of white cells to red blood corpuscles in the deposit, is very suspicious of renal tuberculosis. A diligent search for tubercle bacilli should be made. A twenty-four hours specimen should be collected, centrifuged and deposit stained for tubercle bacilli. Heavy deposits should be treated with antiformin for 6 to 12 hours before this. If urine has a high specific gravity, addition of 50 per cent alcohol helps in centrifuging the deposit quickly. The centrifuged sediment is agitated with 15 per cent sulphuric acid for twenty minutes and again centrifuged for 5 minutes. Slides are made from this. A loopful of this is inoculated into the chosen medium (Lowenstein) for culture. In 80 per cent of positive cases tubercle bacilli are obtained. A guinea pig inoculation from a fresh specimen of urine taken under aseptic precaution would give in 3 to 6 weeks a positive result in another 15 per cent. In the remaining 5 per cent which are of the closed type, diagnosis depends on clinical evidence.

X-ray pictures.—A plain rontgenogram may show an enlarged kidney with uneven density and areas of calcium deposit. Excretion pyelograms may show a moth-eaten appearance of one or more calyces, dilatation of pelvis and calyces

and irregular constrictions and dilatations of the ureter. They will show the presence of the opposite kidney and its functional capacity. Sometimes the affected kidney may show no excretion of the substance even in delayed pictures, being either destroyed or excluded in the closed type. 73 per cent of cases show suggestive changes.

Cystoscopy.—This should be done with absolute asepsis under a low spinal or caudal anæsthesia. Bladder should be gradually distended to hold at least 4 ounces of fluid. If ureteral openings are seen catheters are passed up and urine collected from each kidney for bacteriological, microscopic and biochemical examinations. Pathological changes as ulcers, tubercles and in the openings of ureters are also noted. Where ureteral openings are not seen an intravenous injection of 8-10 ccs. of 0.1 per cent. indigocarmine would demonstrate them colouring the efflux and give an idea about the functional capacities of each kidney by the time taken for colouring the efflux. A retrograde pyelogram should be avoided, but where diagnosis is uncertain it may be done on the affected side by using a 20 per cent solution of uroselectan-B. 86 per cent of cases can be confirmed by cystoscopic examination.

Treatment.—For unilateral renal tuberculosis with no other contra-indication nephrectomy gives good results. The ureter should be sectioned of as low down as possible. Where the whole ureter is infected the lower portion should be also removed by a separate incision. This is required in 10 per cent of cases. Into the distal portion a few drops of pure phenol are injected prior to ligaturing and sectioning. Where other active lesions are present, Sanatorium treatment is carried out first. Presence of genital tuberculosis is no contra-indication. Both can be dealt with.

The patient should have sanatorium treatment subsequently. A nourishing diet rich in vitamins A & D is given. Cod liver oil, malt with iron iodide is prescribed. Heliotherapy or U. V. R. therapy to the wound or sinuses should be given. I have found weekly injections of tuberculin T. R. useful in renal and genital tuberculosis, starting with 1/10 cc. of 1 in. 100 solution rising gradually till 1 c.c. is reached. Injections of sodium cacodylate grs. ½-1 or sodium methylarsenate gr. ½ are also useful.

Tuberculous cystitis usually clears up in about six months after its cause —renal tuberculosis—is removed. If ulceration is advanced instillations of 5 to 20 per cent gomenal are useful. Any ulcers left after six months should be treated by diathermy sparking.

Mortality of operation is 8 per cent. 70 to 80 per cent of women recover. In men owing to genital tubercle results are good only in 40 to 50 per cent.

Prognosis in children is poor as bilateral cases are more common. All bilateral cases should be treated in Sanatoria.

Late Results.—50 to 60 per cent become free, 15 to 20 per cent improve, 15 to 20 per cent end fatally. Average duration of life after nephrectomy is 10 to 12 years. Some remain free from trouble for 15 to 22 years.

Tuberculosis of Genital System.

It is uncommon in children and in old age. The commonest age period is 20 to 40, the average age being 25 to 26 years.

Kocher found pulmonary foci in 80 per cent of autopsies on genito-urinary tuberculosis.

Compared to urinary tuberculosis, it is a milder infection with an insidious onset and slow progress and finally involves all the genital organs. The chief danger is spread to the urinary tract, kidney or bladder.

Genital system alone 50 per cent, genital system and urinary system 37 per cent, genital system with foci elsewhere 13 per cent.

Prostate and other genital organs—52 per cent. Prostate alone 14 per cent, Epididymis alone 32 per cent, Seminal Vesicles 2 per cent (Thomson-Walker).

Etiology.—There are two views held as to the primary genital site of infection. Young, Kenneth Walker, Keyes and others hold that the primary site is the prostate and seminal vesicles in the great majority of cases. This view is supported by the fact that in the majority of cases the epididymal focus is first noticed at the lower pole and on a rectal examination nodules are found in the prostate and homolateral seminal vesicle. The prostate is primarily affected through the blood stream and extension to the epididymitis is by retrograde lymphatic infiltration along the vas.

Tuberculosis of Prostate and Seminal Vesicles. Primary infection occurs in the prostate in the great majority and then extends to the seminal vesicles, sometimes both may be infected simultaneously. The process starts in the upper and outer parts of the gland and occasionally in the lower and outer angles. There may be a number of nodules or a single mass. In course of time the whole lobe becomes involved. Usually the homo-lateral seminal vesicle is found involved and caseous nodules are found on its inner side. Later as the whole of the seminal vesicle becomes involved, it is converted into a rigid nodular mass continuous with the nodular mass in the prostate. The nodules caseate and soften into abscesses intercommunicating with one another or into one large abscess. The abscess usually ruptures into the bladder by the side of the trigone or into the urethra by one or more openings Less frequently it may open into the rectum or into the perineum along the urethra. After the rupture the process becomes quiescent and the prostate becomes shrunken and hard. The tubercular process extends along the lymphatics of the vas to the lower pole of the epididymitis. It also involves the opposite seminal vesicle and later on the epididymitis of that side. The bladder may be infected directly or by extension from the infected prostatic urethra. In the later stages the prostate and the prostatic urethra are destroyed resulting in a cavity communicating with the bladder.

Symptoms.—When confined to the prostate in the earlier stages there may be no symptoms except sometimes a dull perineal discomfort. When the abscess opens into the prostatic urethra there may be a sudden urethral discharge and urine contains pus, prostatic threads and debri. The discharge may show tubercle bacilli but no gonococci. When the bladder neck is involved there is increasing frequency, difficulty, aching and burning in the perineum and anus. Where seminal vesicles are involved first, symptoms may be absent or patient may complain of sexual irritation such as frequent erections, painful and blood-stained emissions. Often symptoms may be absent and infection of prostate may be found on routine

rectal examination or a slightly painful nodule in the epididymitis induces one to examine the prostate. Secondary infection may occur after instrumentation or spontaneously. In late cases mixed infection is common and bladder is found to be often involved. Frequency, urgency, strangury and terminal hæmaturia, etc., are added. Homolateral kidney may become involved by an ascending lymphangitis. In course of time both the epididymitis become involved.

Diagnosis.—Rectal examination—In the early stages firm nodules at the upper and outer angles of the prostate or nodules in the inner part of seminal vesicles will be felt. These nodules are small and more prominent than in carcinoma. In the later stages nodules and boggy areas of softening or soft, tender fluctuant swellings in the prostate and a hard craggy elongated mass in the region of seminal vesicle on one or both sides may be felt. Where an abscess has burst into the rectum, a button like infiltration round the fistula may be felt.

Cysto-urethroscopy may show congested elevations or openings of abscesses into the prostatic urethra or an irregular cavity with tubercles around it may be seen along the side of trigone.

Treatment.—General constitutional treatment and nourishing diet, etc., are carried out. Tuberculin injections are very satisfactory in this type. T. R. 1/10 cc. of 1/100 solution is gradually increased avoiding a general reaction and given twice weekly till a dose of 1 cc. is reached. Prostatitic abscess should be opened by the perineal route, scraped and iodoform emulsion injected into it. Sinuses are excised and scraped.

Tuberculous Epididymitis.

It is most common during the age period of 20 to 30. Usually a family history of tuberculosis and often a history of injury to testis is given.

Pathology.—Most commonly it is due —an ascending infection from foci in the prostate and seminal vesicles *via* the lymphatics. Haematogenous infection is rare and when it occurs the testicle and epididymitis are infected simultaneously. Chronic form is most common. It usually affects the lower pole, occasionally the whole epididymitis may be involved as in the acute type or a nodule may form in the upper pole of epididymitis. Miliary tubercles form and gradually enlarge, caseate and give rise to an abscess. The abscess becomes adherent to the scrotal skin on posterior aspect and finally bursts on the surface leaving a sinus. Later the testis becomes involved and caseation occurs especially in the region of rete testis with scattered foci throughout the substance of the gland. These soften into an abscess which discharges on the anterior surface and the degenerating testis may protrude as 'Fungus Testis'.

Chronic Tuberculous Epididymitis.—A nodule usually develops in the lower pole and less often in the body or head of epididymitis. Attention of the patient may be accidentally drawn to its presence. The nodule is hard and not tender or slightly so and may be smooth or craggy in shape. The vas is thickened or beaded and can be separated from the other constituents of the cord. Later the whole of the epididymitis becomes an irregularly craggy mass lying vertically on the postero-lateral aspect of the testis. In about a third of the cases a small hydrocele is present containing either floculent or clear fluid. Adhesions in this are frequent. The nodules spread

and caseate into abscesses which finally discharge on posterior aspect of scrotum. After discharging it may heal after some months, to break down again. This may repeat several times. Progress is slow. In about 2 to 3 years the opposite epididymis becomes similarly involved. Bladder and kidney may become involved from the prostate.

Diagnosis.—A rectal examination would show nodules in the prostate and seminal vesicles. Gonorrheal epididymitis leaves behind a fibrous nodule which is tough, smooth and ill-defined. Filarial epididymitis is accompanied by fever, orchitis funiculitis, lymph scrotum or hydrocele, etc. Night blood shows microfilariæ. Syphilis usually affects the body of testis and testicular sensation is lost early and it is painless and W. R. would be positive.

Acute tuberculous epididymitis.—It occurs in younger people occasionally and is often mistaken for gonorrheal epididymitis. There is acute pain and tenderness with swelling of epididymitis which in couple of days becomes of large size. There may be a purulent discharge from the urethra due to prostatitis and vesiculitis and tubercle bacilli may be found in the discharge. Some weeks later an abscess forms and bursts. It then settles down into the chronic form.

Diagnosis.—A rectal examination would show nodules in the prostate and seminal vesicles. Gonorrheal epididymitis is more painful and does not form abscess. The discharge is negative for gonococci and tubercle bacili may be found. The sinuses are always on the posterior aspect of the scrotum.

Treatment.—General treatment of sanatorium type. A nutritious diet rich in vitamins A and D, haliverol, syrup ferri iodide, heliotherapy, U.V.R. etc. are given. Tuberculin injections of T. R. starting with 1/10 of 1/100 c.c. given bi-weekly till a dose of 1 c.c. is reached in the course of 3 months.

Operative.—If the lesion is limited to the epididymitis, *i.e.*, in the early stages, an epididymectomy with removal of vas as high as possible is done. But in advanced cases an orchidectomy is done. It is always advisable to reset an inch of the opposite epididymitis to prevent extension of the process to the healthy testicle.

"And what proof can you put forward in support of your contention that you were not speeding?" demanded the magistrate.

"I was on my way to visit my mother-in-law," said the motorist.

The girl entered the bus with a pair of skates under her arm. A man immediately got up and offered her his seat.

"Thank you very much," she said, "but I've been skating all the afternoon.)"

A Report on Three Cases of Infantile Hepatic Cirrhosis.

BY A. V. S. SARMA, M.B.B.S., D.Ch. (London).

First Case of Infantile Hepatic Cirrhosis.

BABY B.—

Male. Three years and one month old.

Admitted 24th August 1939.

Complaint.—Bloated abdomen and thinned extremities.

Duration—Twenty days.

History of present illness.—After the completion of the third year the child was becoming peevish, losing flesh and having a protuberent abdomen.

Previous history.—Mother's pregnancy and labour normal. Breast-fed and supplemented with sago-conjee and milk after the third month—note the early inclusion of starch in the diet. Not vaccinated. Umbilical hernia present from birth.

The child used to get attacks of abdominal colic and treatment for the same by enemata generally resulted in whitish and rancid motions. Occasionally "worms" were noticed in motions. The child was also having occasional attacks of diarrhœa—whitish and foul motions. Diarrhœa would be followed by constipation which was treated by castor oil. The parent complained that castor oil lost its earlier efficacy after some time of its use with the child. The child had eczema in the sixth month—cheeks, scalp and legs were specially affected—and the description reveals resemblance to allergic types.

Family history.—Maternal uncle is an asthmatic. The patient is the fifth child in the family and the others (four older and one younger than the patient) are alive and healthy.

Clinical.

24th August 1939.—Peevish and restless. Styes on lids present. Eczematous stamps discernible on the cheeks. Bloated abdomen (ascites present). Thin extremities with dropsical feet. No jaundice. Pyrexia present (axillary temperature: 100·2°F) Sleep disturbed. Bowels constipated. Urine scanty and coloured.

Nothing particular in other systems.

Weight of the child 24 lbs. Abdominal circumference at umbilicus 20".

Treatment.—Diet—Barley water and milk in equal parts with added Glucose D. Plenty of orange juice (Vitamin C thus afforded). No salt.

Half c.c. adrenal cortex extract (cortin) and Half c. c. salyrgan given intramuscularly. Also Calcio-iodo-diuretin ¾ tablet given per day. Photographs taken.

27th August 1939.—Passed urine thrice and had four motions. Temperature (axillary) normal in the morning and 99·5° F in the evening. Slept well in the night.

28th August 1939.—Weight of the child 21 lbs. Temperature (axillary) 97° F in the morning and 99° F in the evening. Pulse rate 120 per minute. Circumference at umbilicus 18½". Œdema of feet has decreased. Had three motions and passed urine four times.

Urine.—Reaction neutral. No sugar and no albumin.

Treatment.—Half c c. adrenal cortex extract and half c.c. salyrgan given intramuscularly.

29th August 1939.—Had one motion, and passed urine thrice. Temperature (axillary) morning and evening normal.

30th August 1939.—Weight of the child 20 lbs. Circumference of the abdomen at umbilicus is 18". Œdema of feet slightly present still. Temperature (axillary) normal morning and evening. Pulse rate 120 per minute. Had two motions and passed urine six times. Sleep is sound.

Treatment.—Half c.c. adrenal cortex extract and half c.c. salyrgan given intramuscularly.

31st August 1939.—Temperature (axillary) normal morning and evening. Passed urine five times. Had three blackish motions (Malæna?).

1st September 1939.—Weight of the child 21 lbs. Abdominal circumference at umbilicus 17". Temperature (axillary) normal in the morning. Pulse rate 130 per minute. The child is drowsy (Note the second picture).

Liver and spleen are not palpable. Liver felt hardened by dipping the fingers under the ribs.

Passed urine once for the day. Had one motion. The child became very irritiable by midday and bit his mother. Vomited blackish matter (Hæmatemesis?).

The child became very drowsy by the evening with smell of urine in the breath and had twitching of muscles.

Treatment.—Had half c.c. adrenal cortex extract and half c.c. salyrgan intramuscularly in the morning.

2nd September 1939.—Passed urine twice and had blackish motions (Malaena?).

10-30 a.m. — Drowsiness marked (uræmic coma). Eyes rolling up and muscles twitching (uræmic fits). Thus clinically uræmia had set in. The child expired in the evening.

Observations.

1. Allergic disease present in the family, *i.e.*, asthma; and in the patient himself, *i.e.*, eczema in infancy.

2. The child was seen in a condition of ascites and obviously atrophied liver.

3. The child improved to a large extent in the early days of treatment and this is attributed by me to the use of adrenal cortex.

4. The child died of uræmia during a period of improvement. Uræmia might be primarily the result of damaged liver and secondarily to the kidneys activated to increased diuresis.

5. The case reported herein exemplifies how allergy probably forms the basis of a definite type of infantile hepatic cirrhosis.

Second Case of Infantile Hepatic Cirrhosis.

BABY LAKSHMIPRASANNAM.—

Female, 9 months old.

Admitted 21st July 1939.

Complaint and duration :

1. *Bloated abdomen* of one month's duration.

2. *Breathlessness* of one month's duration.

3. *Irritability and crying* of one month's duration.

4. *Emaciation* of two month's duration.

History of present illness.—

The child was observed to get ill definitely from the time condensed milk and cow's milk were given in place of breast milk after the first month.

Previous history.—

Mother's pregnancy was normal. Delivery full term but prolonged (by two days).

The child was breast-fed for only one month, because at that juncture the mother developed peurperal insanity. Condensed milk was given during the 2nd, 3rd and 4th months though it did not agree with the child. Later on cow's milk was given and this resulted in pale motions (five per day), and the child was losing flesh. At this critical point wet-nursing was resorted to and this improved the child slightly.

The child was given castor oil regularly and this produced loose evacuations.

Family History.—

Brahmin. Vegetarian. Middle class villager.

Mother migrainous and used to have "fits" (epilepsy ?). Father healthy. The mother had a miscarriage in the seventh month prior to the birth of the patient.

Clinical condition.—

Weak and marasmic child. Anterior fontanelle not closed. Not anæmic and not jaundiced. Abdomen protuberant with engorged veins at the navel. From the report ascites seem to have been present from the last week of June 1939. Fever and constipation present.

Abdomen.—Liver hard and 1½″ enlarged with a wavy anterior border. Spleen 1½″ enlarged. Signs of free fluid present in the abdomen.

Chest.—Rachitic contour. Lungs show basal congestion.

19th August 1939.—Jaundice set in.

23rd July 1939. Investigation done.

Blood.— 1. *Kahn*—negative.

2. *Gel test for kala azar*—negative.
3. *Calcium*—11·4 mgms. %
4. *Phosphorus* 6·7 mgms. %

24th July 1939.—X-ray study of bones at the wrist revealed no evidence of any definite lesion.

Observations.

1. Presence of allergic disease in the family. (Mother had migraine, fits (?) and even insanity.)

2. The dietetic factor as an important causative agent in infantile

I. Baby Balakrishnan, 3 years and one month; Note (1) ascites present with prominent veins around umbilicus; (2) the presence of umbilical hernia, 24—8—39.

II. Baby Balakrishnan
1—9—39.

III. Baby Lakshmiprasannam
25 7 39.

IV. Baby Sankaran
12—9—39.

hepatic cirrhosis is well exemplified in the case under study. Feeding with condensed milk is to be specially noted. Cow's milk produced pale diarrhœa. (intolerance?).

3. The figures for blood calcium and phosphorus are higher than approved normals, and this is probably so, because of renal insufficiency consequent on ascitic pressure. This case also shows no definite bony changes as studied from a skiagram of the wrist, in spite of the fact that the chest appears rachitic clinically and the anterior fontanelle has not closed (age 9 months). For the characteristic changes to occur in the long bones in a case of infantile hepatic cirrhosis, it appears that the patient must first survive the various stages of the malady and later start growing with inadequate and ill-balanced diet. A case has been reported by the writer wherein bony changes were detected.

Third Case of Infantile Cirrhosis

BABY SANKARAN.

Male. One and half years. (Admitted 12th September 1939.)

Complaint and duration:

(*i*) Fever, cough and vomiting.—Two months.

(*ii*) Constipation and improper sleep.—One month.

(*iii*). Swelling of feet.—Three days.

History of present illness:

The illness dates after the onset of pertussis two months ago.

Previous History.—

Mother's pregnancy and labour normal. Not breast-fed. Fed on cow's milk. No tonics were given. Weaned with once-boiled rice in the 12th month. Accustomed to medicated castor oil of Madura.

Vaccinated in the fifth month. Diarrhœa in the 12th month—bloody stools for 20 days. Pertussis in the 16th month.

Family History:

Father migrainous. Mother is an asthmatic.

Family of—

1. Girl of 11 years. Alive and healthy.

2. Boy of 5 years. Alive and healthy.

3. Boy of 3 years. Alive and healthy.

4. Patient.

5. Male child died of *Mandam* at 21 days' age.

Brahmin. Vegetarian.

Clinical: *12th September 1939.*—The child pale and plump. Dropsical feet. Swollen and puffed eye lids. Eczematous and impetiginous scalp. Discharging nose. Temperature (axillary) 101° F. Pulse rate 120 per minute.

Abdomen.—Bloated with an everted umbilicus. Abdominal circumference at umbilicus is 20″. Shifting dullness present. Liver 1½″ enlarged. Spleen 1″ enlarged.

Chest.—Basal congestion present.

Observations.

1. Presence of allergic diseases in parents.

2. The precipitation of the disease by PERTUSSIS. (The diarrhœa

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Abdomen.—Bloated with an everted umbilicus. Abdominal circumference at umbilicus is 20". Shifting dullness present. Liver 1½" enlarged. Spleen 1" enlarged.

Chest.—Basal congestion present.

Observations.

1. Presence of allergic diseases in parents.

2. The precipitation of the disease by PERTUSSIS. (The diarrhœa

that visited the child earlier than pertussis is also important).

3. The child was treated with benefit by an extract of adrenal cortex (orally given) and very small doses of dissolved vaccine of B. Pertussis (given parenterally).

Condition of the patient on 19th September, 1939.—Abdominal circumference at umbilicus is 18½". Temperature—axillary—is 97° F. Pulse rate is 120 per minute. Œdema of feet has decreased. The child has regular bowel action from 13th September, 1939.

Unfortunately the patient could not be followed up.

An English Doctor received from the squire of a village some five miles away the following letter:

"Dear Doctor, will you come to see me in the morning and bring some stuff for the gout with you?" The next morning the medico rode over to the squire's and after examining his patient, said, 'why, it is not gout you have at all, it is rheumatism'. I know that, was the reply. 'Well, if you knew it was rheumatism' asked the astonished doctor, 'why did you ask me to bring medicine for the gout?" 'Well,' replied the unabashed squire, 'though we knew it was rheumatism, devil a sould was there in the house that could spell the word, and so we had to put gout.'

The manufacturers of a patent medicine recently received a letter from a grateful woman who had been a customer.

"Four weeks ago," she wrote, "I was so run down that I could not even spank the baby. After taking three bottles of your tonic I am able to thrash my husband in addition to my other housework. Thank you!"

A man 'blessed' with a very talkative wife had brought her to the Doctor's surgery and stood quietly by watching intently all the time the doctor was taking her temperature. Just as the medico was about to remove the instrument, the man who had never before known his wife to be silent for so long a period said, 'Doctor, what will you take for that thing?'

Notice in a doctor's surgery: "Ladies in the waiting-room will please not exchange symptoms. It gets the doctor hopelessly mixed."

If you don't spend money you are a grouch. If you spend you are a loafer. If you get it you are a grafter; If you cannot get it you are a fool. And just about the time when you are making both ends meet, somebody moves the ends.

The less we wear the longer we shall live says a doctor. There will be a lot of old women in this country in the next generation.

Paraplegia.

BY

CAPT. S. SHONE, I.M.S.

By paraplegia is meant a complete or partial paralysis of both lower extremities. The paralysis must ensue from disease of the central nervous system or of the muscles themselves. Inability to walk on account of bone or joint involvement is not covered by the term. Hence paraplegia will result from a lesion of the upper or lower motor neurons, of peripheral nerves, or of muscles. When the lesion involves the upper motor neuron, the condition is known as spastic paraplegia.

Paraplegia may be of recent onset in an adult, or it may be a legacy of some condition which occurred in childhood. It is therefore profitable to consider the outstanding conditions which may produce paraplegia in childhood.

There may be a failure of development of cortical neurons and this gives rise to the malady known as Little's disease. Here all four limbs are affected, but the lower usually to a much greater extent than upper. Associated with the motor defect is frequently some degree of mental maldevelopment, sometimes amounting to idiocy. Other grosser congenital abnormalities, such as porencephalus, meningocele, or spina bifida, may be responsible for paraplegia in the infant or child.

Injury at birth, especially from the faulty application of forceps, may produce paraplegia, and various inflammatory conditions inside the cranium may cause a similar result. For example, an encephalitis may occur, particularly after some specific fever, which may damage the cortical neurons. Meningitis, thrombosis of the superior longitudinal sinus, and polioencephalitis may be followed by permanent paralysis of the lower extremities.

Tuberculous caries of the spine in children may result in spastic paraplegia. The uncommon familial degenerative disease known as Friedreich's ataxia occurs in children. Here the degeneration is widespread in Clarke's columns, the spinocerebellar tracts, the lateral and posterior columns.

All the conditions mentioned above are examples of paraplegia caused by involvement of the upper motor neuron. As examples of lower motor neuron we may mention the important disease acute anterior poliomyelitis and the various types of peripheral neuritis. The so-called motor neuron disease does not commence in childhood, but the allied condition of the Charcot-Marie-Tooth peroneal type ot progressive muscular atrophy usually beigns in mid-childhood.

The muscular dystrophies are representative of paraplegias of primary muscular type. The common ones are the pseudo-hypertrophic variety and the infantile and juvenile forms.

When confronted with an adult who is suffering from paraplegia,

therefore, attention to the history of the case is essential, in order to eliminate some affection in childhood. When this has been done, or when the history indicates an illness of more recent origin, a rapid mental survey of the causes of paraplegia in adult life is required. The great majority will again be due to lesions either of the upper or of the lower motor neurons, but a few will fall outside these categories.

Consideration of the upper motor neuron lesions will lead us to investigate cerebral conditions first. Obviously, there must be a wide spread involvement of the cerebrum to produce weakness or paralysis of both legs. A diffuse condition, such as an encephalitis—and particularly encephalitis lethargica—is capable of producing the necessary result, but it must be stressed that this is rare. Bilateral cortical or subcortical hæmorrhages and bilateral cerebral thromboses are among the vascular conditions which may occasionally result in paraplegia. A cerebellar tumour or abscess has to be borne in mind and meningitis may produce the necessary lesion.

It is obvious, however, that the majority of paraplegias will ensue from lesions of the spinal cord, and a large number of the known diseases of the cord are associated with some degree of weakness of the lower extremities. First to be considered are the cases of transverse myelitis. This is produced when an acute inflammatory process affects the spinal cord and the possible causes are very numerous. It may occur in the course of many acute infectious diseases, such as influenza, measles, scarlet fever, and even pneumonia, tonsilitis and whooping cough. It may be the result of a thrombotic process, as in syphilis, which, as a matter of fact, is a very common cause of transverse myelitis in India. Trauma may be a possible factor and vague conditions, such as exposure to cold, are spoken of as precipitating cases of obscure origin. I once saw a dental student who developed a transverse myelitis after sitting on some cold stone steps, after some vigorous exercise which produced considerable sweating. No other ætiological factor was discovered. A certain number of cases of transverse myelitis are attributed to vague exogenous poisons, and sometimes no cause can be adduced, even by the most ingenious.

More obvious causes of transverse myelitis are those due to compression. These cases are consequently spoken of as "compression myelitis". Disease of the vertebral column, such as tuberculous caries of the spine, or secondary deposits of malignant disease in the vertebræ, are not uncommon causes of paraplegia. Meningeal inflammation will occasionally produce sufficient thickening and swelling to press on the spinal cord. The condition called meningitis serosa circumscripta or adhesive arachnoiditis is obscure in nature but probably is the end result of some former meningeal inflammation and may cause compression of the cord. Meningeal tumours, of which fibromas, endotheliomas and psammomas are the commonest conditions of importance to bear in mind when considering compression paraplegia. Aortic aneurysm may, upon occasion, press on the spinal cord and rarely hydatid cysts will produce a similar result.

Compression myelitis is amenable to surgery quite frequently and its accurate investigation is therefore of great importance. Lumbar puncture

will frequently demonstrate a "block" in the circulation of the cerebro-spinal fluid: pressure on the jugulars will not produce the customary increase in rate of flow of the fluid. The latter may exhibit a yellow colour and will contain an increase of protein content without a corresponding rise in the cell count. This is the so-called Froin syndrome. Introduction of lipiodol, into the theca followed by X-ray examination may demonstrate the site of compression.

Disseminated sclerosis is an extremely common disease in Europe and America but, from all accounts, is very rare in this country. In England it has now supplanted syphilis of the central nervous system as the most common nervous disorder, whereas in India syphilis must be, far and away, the ætiological factor of greatest significance. Disseminated sclerosis produces paraplegia with great regularity, on account of its predeliction for the white matter of the cord.

Subacute combined degeneration of the cord is, like disseminated sclerosis, an example of a disease of which the main feature is demyelination of the white matter. It is almost invariably associated with a macrocytic anæmia, although some cases have been described without this form of blood picture. This disease, again, appears to be uncommon in India, but the diagnosis is made upon the discovery of a combined lesion of posterior columns and pyramidal tracts in a middle aged person, who exhibits a macrocytic anæmia with achlorhydria.

Syringomyelia is a condition which affects, not only the cord, as the name implies, but the brain stem as well. Whatever the essential pathological lesion is—an initial gliosis or not—the net result is the production of cavities, which occur in the neighbourhood of the central canal and thus involve the anterior commisural fibres. These carry the sensations of pain and temperature and their destruction gives rise to the well-known "dissociated anæsthesia". The patient retains the sense of touch but cannot appreciate painful stimuli or thermal ones. The "syrinx" or cavity is usually in the cervical region of the cord, and hence the anterior horn cells in the neighbourhood of C. 8 and T. 1 are involved, giving rise to wasting of the small muscles of the hand. The lateral columns which will be supplying the muscles of the lower limbs are also involved, with paraplegic symptoms. The ultimate picture is, therefore, a very characteristic one.

Distinguished from syringomyelia by the existence of gross sensory phenomena and a dissociated anæsthesia, is that form of motor neuron disease known as amyotrophic lateral sclerosis. Otherwise there is the same combination of upper and lower motor neuron symptoms and signs.

Hæmorrhage into the cord—hæmatomyelia—may occur as the result of an injury. Usually it follows some such trauma as results from diving into shallow water, but often there is no obvious ætiology. There is a sudden onset of numbness and weakness and then the features slowly develop into those closely resembling syringomyelia.

A condition described as "primary lateral sclerosis" is described in all text books and presumably this indicates an involvement of lateral columns, pure and simple, without sensory changes or disorder of other parts of the central nervous system. Many of these cases, with more careful

examination, would probably be found to fit into other diseases, such as disseminated sclerosis. Some are syphilitic in origin and probably are to be included under the name "Erb's syphilitic paraplegia", in which sen sory loss is slight, but in which bladder control is usually affected. The important point is that no diagnosis of primary lateral sclerosis should be made until all other diseases have been eliminated. In this connection, I would mention that, in this hospital, an unusually large number of paraplegias are seen in whom there appear to be no sensory signs or symptoms, and in whom the tests for syphilis in blood and cerebrospinal fluid are negative. Lathyrism can probably be ruled out, but it is not certain whether some vitamin deficiency or factor similar to that missing in subacute combined degenerarion of the cord is not responsible. This, of course, takes it for granted that the examination of the patient is thorough and complete, an assumption which I believe is sometimes unjustified, owing to the well known difficulties of sensory examinations in unintelligent subjects.

The above resume—includes most of the causes of the upper motor neuron type of paraplegia and now we have to mention some of the causes of the lower motor neuron variety. Peripheral neuritis comprises an important group and may be due to a variety of reasons. Acute anterior poliomyelitis may produce a flaccid paralysis of the musles of the lower extremities. Involvement of the lumbo-sacral plexus by tumour in the pelvis, an affection of the cauda equina or compression of the lumbar enlargment of the cord, may result in weakness of the lower extremities.

Finally there are a few instances in which paraplegia may be due neither to lesions of upper or lower motor neurons. For instance, in tabes dorsalis, which is a rarity in India, inability to walk will result from involvement of the posterior columns only. The curious malady known as Landry's paralysis, in which there is a spreading paralysis of the body, is probably another example of this category. Hysteria should always be suspected but this is not a diagnosis to be made lightly.

For the benefit of those who prefer a tabulated classification, the following is appended.

A. Causes of paraplegia in childhood·

1. Upper motor neuron lesions:

(*a*) Failure of development of cortical neurons, as in Little's disease.

(*b*) Congenital abnormalities—porencephlus, meningocele, spinabifida.

(*c*) Birth injury, as from forceps.

(*d*) Encephalitis, following upon some acute specific fever.

(*e*) Meningitis.

(*f*) Thrombosis of superior longitudinal sinus.

(*g*) Polioencephalitis, the cerebral variant of acute anterior poliomyelitis.

(*h*) Disease of the spine, as tuberculous caries

(*i*) Friedreich's ataxia.

2. Lower motor neuron lesions:

(*a*) Acute anterior poliomyelitis

(*b*) Peripheral neuritis

(*c*) Charcot-Marie-Tooth type of progressive muscular atrophy.

3. Lesions of primary muscular type

(*a*) Pseudohypertrophic muscular dystrophy

(*b*) Infantile and juvenile forms of muscular dystrophy.

B. Causes of paraplegia in adults.—

1. Upper motor neuron lesions:

(*a*) Encephalitis

(*b*) Bilateral cortical or subcortical hæmorrhages

(*c*) Bilateral cerebral thromboses

(*d*) Cerebellar tumour or abscess

(*e*) Meningitis.

(*f*) Transverse myelitis, numerous types

(*g*) Compression myelitis, various causes.

(*h*) Disseminated sclerosis

(*i*) Subacute combined degeneration of the cord.

(*j*) Syringomyelia

(*k*) Amyotrophic lateral sclerosis

(*l*) Haematomyelia

(*m*) "Primary lateral sclerosis"

(*n*) Erb's syphilitic paraplegia

(*o*) Lathyrism

2. Lower motor neuron causes:

(*a*) Peripheral neuritis, various types

(*b*) Acute anterior poliomyelitis

(*c*) Affection of the cauda equina

(*d*) Compression of the lumbar enlargement of the cord

(*e*) Involvement of lumbo-sacral plexus by pelvic mass

3. Causes other than motor neuron

(*a*) Tabes dorsalis

(*b*) Landry's paralysis

(*c*) Hysteria.

(A somewhat fuller list is given in French's Differential Diagnosis)

"Doctor", said the sick man, 'the other doctors seem to differ in their diagnosis of my case!

"I know", replied the medical man, cheerfully, 'but I am confident the *Postmortem* will show that I am right..... The Canadian Doctor.

He uses statistic as a drunen man uses lamp posts—for support rather than for illumination....." Reader's Digest.'

There is ampple evidence that if a woman is determined to keep a man, she will do it, or see to it that no other woman gets him.....Herb Graffis.

Ergot.

By B. M. KANNABIRAN, 1ST YEAR B.SC., PHARMACY CLASS,
Medical College, Madras.

The sclerotium stage of the fungus 'Claviceps purpurea' belonging to the Ascomycetes developed on the ovary of the Rye is called *Ergot.* The name 'ergot' meaning a Cock's spur was given to the sclerotium in reference to its shape. It is one of the most important drugs in the British Pharmacopœia and India is very unfortunate in depending entirely upon the foreign markets for its supply.

The Life History of the fungus Claviceps Purpurea.—The life of the fungus 'Claviceps purpurea' can be divided into three clear stages (1) Sphacelia stage, (2) Sclerotium stage and (3) Ascospore stage.

The first stage consists of the *ascospore* being deposited on the flowers of the Rye by the wind. The ascospores develop germ tubes that penetrate the epidermis and reach the base of the ovary resulting in the production of a white felt-like network of mycelium *Sphacelia.* This on further development produces slender rod-shaped bodies *Sterigmata,* which in turn give rise to tiny oval bodies *Conidia.* The conidia, which is capable of germinating into an independent mycelium appear in plenty on a mucilagenous sweet exudation 'Honeydew.' Bees and insects are attracted by this fluid, and they carry the infection to the other surrounding flowers during their search for honey.

The termination of the mycelium development produces on the base of the pistil of the Rye flower a shrivelled dark colour, which is the beginning of the *sclerotium stage.* This grows into a small, black, curved and horn-shaped body *sclerotium* with part of Sphacelia at its end, which drops down finally. If the matured sclerotium, which is about an inch in length is not collected at this stage, it falls on the ground and remains still till favourable conditions are offered to it. With sufficient moisture the sclerotium passes on to the next stage where small spherical bodies '*Stroma*' on slender stems of about an inch in length are seen to grow on the ruptured surface of the sclerotium. The stroma has its root—a tuft of hyphæ—at the inner surface of the sclerotium. The spherical body or the head, which is brown or orange in colour has numerous flask-shaped bodies *perithecia* under its surface. Each of the perithecium has in it a number of elongated club-shape *asci,* and each one of them contains six to eight thread-like *ascospores.* When the asci ripens, it bursts open liberating ascospores, which in turn get distributed to various flowers by the agency of wind.

Collection.—In Vienna experiment on artificial infection of the fungus on the Rye flower was carried out successfully by Mr. Hecke in the year 1921. He raised a crop of asci, and when the ascospores were about to appear, he received them on a gelatin medium, and suspending this gelatin in water sprayed it over Rye flowers and succeeded in thé dissemination of the fungus. But, the experiment remains to be tried on a commercial basis.

The chief centres for the collection of the drug in Russia are Tomsk,

Omsk and Viatka, in Spain the provinces of Orense and Leon, near Vigo and in Portugal and Poland; of which the Spanish and Portuguese varieties stand best. Ergot is also grown in Germany, Austria, occasionally in Canary Islands and rarely in England. Ergot is collected either by hand-picking or after the thrashing of the crops separation of ergot from grain is done in a machine. Separation is to be done very carefully and cautiously in countries like Germany and Russia, where Rye forms the staple food of the people, as consumption of Rye with minute quantities of ergot for a considerable time would produce in them the disease—*Ergotism.* The epidemics known as 'ignis sacer' and 'St. Anthony's Fire' were traced in 1747 to ergot. Ergot should be dried and stored in air-tight casks with camphor bits, to prevent the drug from deterioration.

Constituents.—The cells of the central tissue of the sclerotium consists of pseudo parenchyma formed of fine united hyphæ, and are laden with oil and alkaloids to which the importance of ergot is due. The most important of the alkaloids present are the crystalline alkaloids ergotoxine and ergotamine; and the less important ones present in such minute qnantities as not to effect any therapeutical action are Tyrosamine and histamine. Other constituents of ergot are acetyl choline, iso-amylamine, agmatine, ergothioneine (a sulphur compound), selero crystalline, scleroxanthin, sclererythrin (reddish violet substance), ergotinic acid, sphacelinic acid, glucoside clavicepsin and a fixed oil (about 30 per cent containing ergosterol.

Extraction.—Ergot in moderately fine powder is defattened by percolation with light petroleum (for about 24 hours) until 1 c.c. of the percolate leaves only a barely preceptible film when evaporated in a glass dish. The defattened ergot is dried by exposure to air till it is free from petrol odour. It is powdered again and macerated with 50 per cent alcohol containing 1 per cent tartaric acid for 4 to 6 hours in a closed vessel; and then it is packed in a percolator, and after macerating in more of the menstrum for forty to fifty hours is slowly percolated. The percolate is assayed, and if it contains more than 0·06 per cent of alkaloid ergotoxine, it is diluted with sufficient menstrum to bring it to the British Pharmacopœia standard. If the percolate happens to be weaker than 0·06 per cent, another percolate of higher concentration is made and mixed with this in such a way as to give a final strength of 0·06 per cent of alkaloid.

It is advised to use the Spanish and Portuguese ergot for the preparation of the liquid extract of ergot, as they contain 0·24 per cent of ergotoxine after defattening, and they impart proper strength to the liquid extract on maceration and completion.

The British Pharmacopœia preparations are:—

Prepared ergot (contains 0·1% ergotoxine).

Dose:—5-15 Grains.

Liquid extract of Ergot (contains 0·06—0·04% ergotoxine),

Dose:—10-20 Mins.

Action and uses.—The chief action of ergot is the stimulation of the plain muscle especially of the uterus and of the arterioles in peripheral parts of the body; it is employed to excite or increase uterine contraction and to control uterine haemorrhage.

REFERENCES.

1. *Pharmacognos* by Trease. 2. *Pharmaceutics* by Bently. 3. *British Pharmacopoeia* 1932.

A case of Pellagra from Maitland Ward under Dr. G. R. Dinker Rau, M.D.

Reported by S. RAMALINGAM, M.B.B.S., *House Physician.*

Name.—G.

Age.—45.

Sex.—Male.

Admitted.—24th October 1939.

Complaint.—Diarrhœa, difficulty of speech and insomnia.

Duration.—Six months.

History of present illness.—For the past 6 months patient has been having loss of appetite, nausea, vomiting and diarrhœa. He complains also of soreness of the tongue and of certain patches on the skin. He has insomnia. No complaint of any cramps in the muscles.

Previous illness.—Nil.

Family History.—No similar complaint in the family.

Habits.—Non-vegetarian, denies having taken alcohol at any time.

On Examination.—Patient is an emaciated individual, anæmic, slight œdema of ankles and legs present. Has a peculiar staring look and is rather stupied.

The Skin is rough and generally thickened slightly. But over the dorsum of the hands, of the forearms and of the feet, there are certain patches, brown in colour, irregular in size, varying from a quarter of an anna to big confluent areas, almost looking like sunburn. Over these areas, besides the change in colour, the skin is wrinkled, inelastic and thinned and there is a fine desquamation (*vide* clinical photograph).

The nails are atrophied and somewhat brittle.

Tongue is smooth, Papillæ atrophied showing loss of superficial epithelium due to old glossitis. There is also excessive salivation. No tremors of the tongue are present.

Abdominal Examination.—Nil abnormal.

Nervous System.—(1) Intelligence and memory.—Poor.

Sleep.—Insomnia.

Speech.—Slurred.

(2) Cranial nerves.—Nil abnormal.

(3) Motor functions.—Nil abnormal.

(4) Sensory functions.—Normal.

(5) *Reflexes.*

Superficial.—Abdominal reflex absent in all quadrants.

Plantar response: Flexor.

Deep.—All deep reflexes exaggerated especially the knee jerks.

No clonus.

Organic.—Normal.

(6) *Trophic.*—Nil.

(7) *Gait* is a little uncertain and somewhat of the spastic paraplegic type.

Skin Condition in Pellagra—*Vide* Note.

FRACTIONAL GASTRIC ANALYSIS GRAPH.

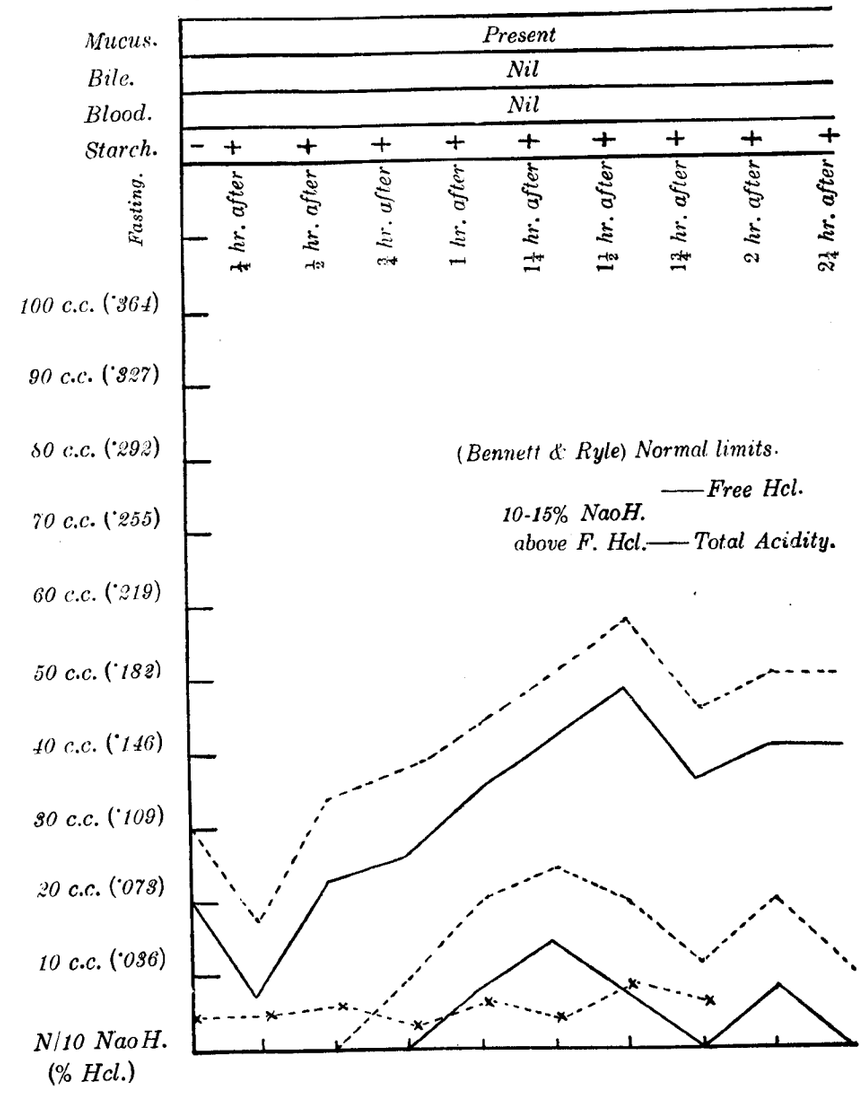
BIOCHEMISTRY DEPARTMENT,
MEDICAL COLLEGE, MADRAS.

S. No. 862 *Date.* 30-10-1939.

Name. Govindarajulu, Maitland extra.

Medical Officer. Dr. by Dinker Rao.

Hospital. G. H.



Cardiovascular and Respiratory Systems.—Nil abnormal.

Investigations.

Urine.—Acid Sp. gr. 1018, no Alb., Sugar present. Less than 1 per cent. No acetone.

Fæces.—Yellowish brown, loose, no mucus, no blood ; microscopically : no ova, no amæba, cellular exudate few.

Blood: *Smear.*—Anisocytosis, poikilocytosis.

Total R. B. C. = 3 millions.
Hæmoglobin = 70 per cent.
Colour Index = 1·17.

Total W.B.C. = 5,600 per c. mm.
Differential count.

Polymorphs = 35%
Lymphocyte = 50%
Mononuclear 15%.

Glucose Tolerance Test.

(1) 0·07 %
(2) 0·148% urine. Sugar trace.
(3) 0·191%
(4) 0·222%
(5) 0·200%

Lumbar Puncture.

C.S.F. Clear and not under pressure
Albumin = 30 mgms.%
Globulin = nil
Chlorides = 650 mgms.%

Fractional test meal shows complete absence of free HCl.

Blood Pressure—92/76.

Diagnosis.—The combination of the skin lesions (of the nature already described) with evidences of past glossitis, diarrhœa and the mental symptoms (such as stupidity and moroseness), suggest the diagnosis of Pellagra.

The Skin lesions in this case are very typical of Pellagra. They are the absolute symmetry sharply demarcated pigmented borders, and the distribution over those areas directly exposed to the sun's rays. The skin is wrinkled, inelastic and thinned.

It is on account of the roughness of the affected skin that the disease is called "Pellagra" [Pelle (Skin) and agro (rough)]

The Mental symptoms, though not marked, were present to a sufficient extent to help in diagnosing Pellagra. The vacant almost stupid staring look, the morose behaviour, and meaning less actions were diagnostic. Probably if the case were followed long enough, one would have seen a terminal confusional insanity.

The Nervous features.—The neurological features in Pellagra usually resemble those of a subacute combined degeneration of the cord, with spastic paraplegia, sensory disturbances, accompanied by increased knee jerks and ankle clonus absent abdominal reflexes and an extensor plantar response. The patient suffers from obstinate sleeplessness. There may also be tremors of the tongue and muscular cramps.

But in the case under discussion, except the increased knee jerks, absent abdominal reflexes and insomnia other features were absent.

Chvostek's Sign, mechanical irritability of the facial nerves (which is usually present in Pellagra) was absent.

Other minor diagnostic features are :—

(*a*) *Anæmia* of the secondary type, with leucopenia and a relative lymphocytosis. Sometimes a megalocytic anæmia with an increase in cell volume and a colour Index which equals or exceeds 1·0 is encountered in severe cases.

(*b*) Achlorhydria or hypochlorhydria, as shown by a fractional test meal analysis.

(*c*) Emaciation.

Differential diagnosis.—The glossitis, diarrhœa and emaciation, either singly or in combination can occur in Pernicious Anæmia, Sprue, Tropical macrocytic anæmia, Subacute Combined Degeneration of the cord or in Tubercular enteritis, but in none of them are these typical skin lesions present. The skin may show only evidences of malnutrition or avitaminosis such as a dry lustreless skin devoid of hair, and sometimes a fine desquamation.

Treatment.—The modern treatment for Pellagra is the administration of Nicotinic acid by mouth, in doses of 100 mgms. five times a day. The patient was accordingly put on Nicotinic acid. But as he had to be discharged three days after the commencement of the treatment, the results could not be observed.

I thank Dr. S. T. Achar, M.D., for his help in getting up this case report. My thanks are also due to Dr. G. R. Dinker Rao and the Superintendent, General Hospital, for permission to report this case and to the Barnard Institute of Radiology for the chemical photograph.

It is better to give than to lend, and it costs about the same.....Philip Gibbs.

Why, Sir, No man will be a sailor who has contrivance enough to get himself into a jail ; for, being in a ship is being in jail with the chance of being drowned. .....Dr. Johnson.

No man flatters the woman he truly loves.....Tuckerman.

Most women would be intolerable if they were not women.....S. W.

Japan wants peace we are told—Yes, a piece of China.

Patient in Doctor's Waiting Room : How do you do !

Second Patient : Oh, so so. I am aching from arthrities.

First patient : Glad to meet you. I am Payne from Chicago.

Teacher ; And where do we find mangoes ? "

Pupil : Wherever woman goes.

The Madras Medical College Magazine.

"INCIDENCE OF CARCINOMA IN MADRAS AND BENGAL."

THE Bengal Cancer Institute, Calcutta, publishes in No. 1, Vol. II, May 1938, the following appeal to doctors "Please help us in our fight against Cancer by sending us cases as early as possible."

Similar associations, in various countries of the world, have realised that the early diagnosis is the most important item in the programme of their campaign. Appeals to the lay public, education of their constituents by the Life Assurance Companies, and realisation by members of the medical profession of the importance of early diagnosis may be expected to bring in cases early for treatment.

Of an analysis of 860 cases of carcinoma in this hospital (carcinoma cervix cases not being admitted) 72 per cent were oral carcinoma. Oral carcinoma involves the cheek, tongue and the lips, parts which are looked on so frequently either by oneself in the mirror or by other members of the household or by friends parts which even in the earlier stages of the disease, interfere with the ordinary daily routine such as, cleansing of the mouth, drinking and eating ; it is suprising that patients do not take advice in the early stages of the disease.

In an analysis of 152 cases in Calcutta, Carcinoma of the Larynx and the hypopharynx formed the largest group (32 out of 152) ; next in order were Carcinoma of the oral cavity 27, of the pharynx 15, of the tonsillar region 14, of the tongue 14, *i.e.*, 70 out of 152 in the oral cavity and pharynx.

The age incidence in the Calcutta series was

22%	between	31 and 40
32%	do	41 and 50
24%	do	51 and 60

Carcinoma of the penis affected only Hindu patients and all but one of them had phimosis ; this incidence seems to bear a close relationship to the custom of circumcision, which is not practised by the Hindus.

75 per cent laryngeal and hypopharyngeal carcinoma cases were smokers ; cheek Carcinoma cases had the habit of chewing pan with tobacco and keeping the resultant mass as a bolus between the cheek and the gum. Each patient had one particular favourite spot where the bolus was lodged. The carcinoma appeared at that very spot.

The pan habit is most common in Bengal, Orrisa and Southern India.

Pyorrhœa was present in 90 per cent of the cases. The prophylactic measures are obvious. It becomes the duty of the members of the medical profession to educate our own people on the dangers of this universal bad habit.

CLINICAL CASE-TAKING

ALL clinical teachers and all regulations of examining bodies rightly insist that clinical case taking is a very important method of learning clinical, medicine and surgery. Anybody visiting a foreign clinic will be impressed by the fact that a careful History taking and a methodical clinical Examination, recording the physical signs elicited is the essential routine in all clinics. *Doing rather than listening* should be the motto of the student. The following points from the Lives of two eminent men are of immense use to the student in the execution of his clinical work.

Hughling Jackson (1835—1911) was always careful to record only what he saw and what could readily be demonstrated to an unbiassed observer.

In the field of neurology his observations had been carefully made, step by step. He recorded only what he saw and consciously avoided generalities. This method of procedure left no grounds for objection so that his work was practically unhindered and undiverted by any bickerings over this or that terminology or interpretation.

J. C. Lettsom (1744-1815) was most diligent in his observation of the patients, of whose cases he made notes, a custom not then usual (Sir Charles Ballance—1906.)

Personal.

Our hearty congratulations to Lt.-Col. M. M. Cruickshank, M.D., Mch., F.R.C.S., F.R.S.E., I.M.S., on whom was conferred the fellowship of the American College of Surgeons in October 1939.

Dr. A. Vasudevan, M.B B.S., Curator of the Pathology Museum goes on leave from 1st January preparatory to retirement after a continuous hard work of twenty years at the museum.

Dr. D. Govinda Reddi, M.D., is posted as the Curator, Dr. S. K. Sundaram, M D., is Assistant Physician to the Professor of Medicine, Dr. Vaydianathan, M.D., is the Assistant Physician to the Professor of Therapeutics.

OBITUARY.

It is with deep regret that we record the demise of Major Ebden, M.D., M.S., F.R.C.S., IM.S., on 10th November 1939 who was previously a Surgeon and Professor of Operative Surgery in this Hospital and College. He was a frank and kind-hearted good teacher with an all-round knowledge of Architecture and Engineering. The College was closed as a mark of respect.

College Comments.

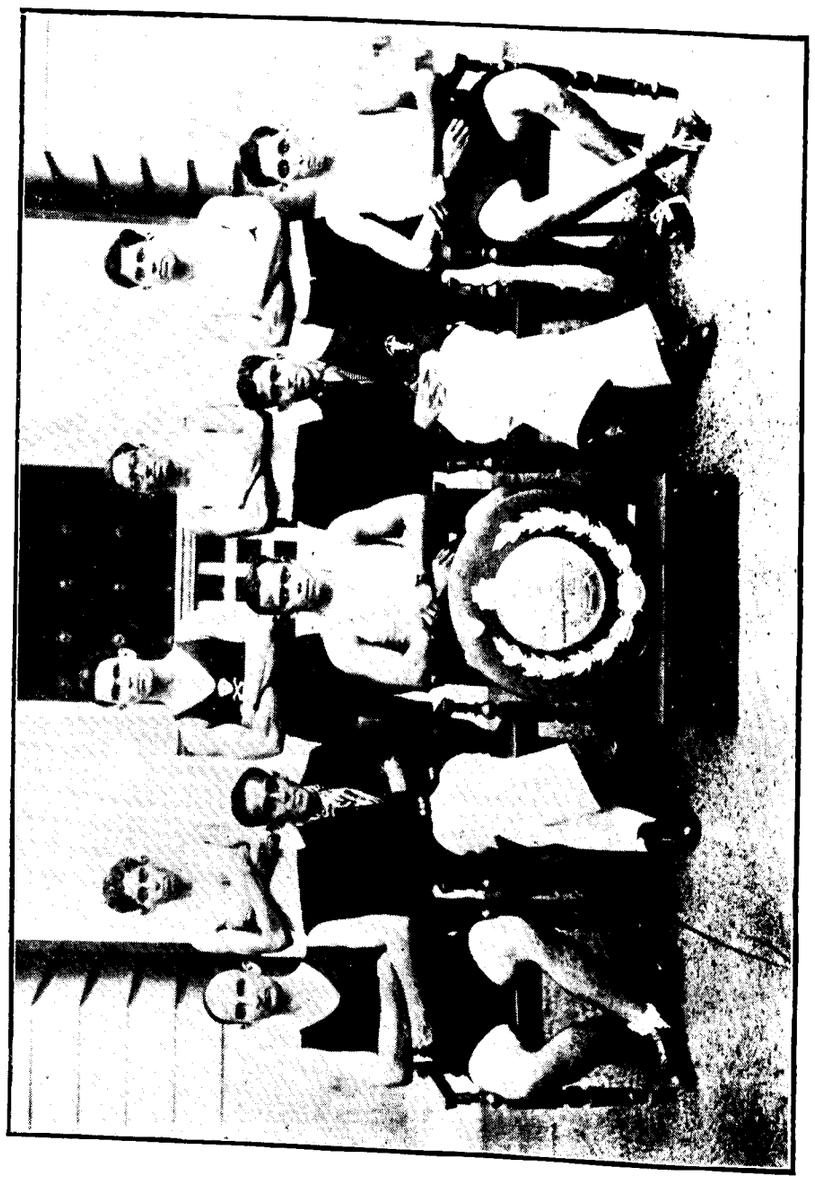
The New Year.—Though very late we wish to offer all our readers a very, very happy and successful New Year.

The Inter-Collegiate oratorical contest.—In the above contest in oratory our College was represented by Miss Jayalakshmi of the second year class and Mr. C. K. Ramachandran who is in the third year. It is very creditable indeed to the College that one of the representatives should get the special prize. Miss Jayalakshmi deserves our congratulations for ably representing our College. She may not have won the prize but she won the applause of all which is most deserving to her courage.

Rangachari Memorial.—Dr. S. Rangachari was an old student of the College and was also a Surgeon and Teacher of the College. His name is perpetuated by a number of scholarships to deserving students of the Colleges. The Public of Madras subscribed for a statue, and the life-size bronze statue was unveiled by H. E. the Governor and the statue stands in front of the gate of the General Hospital. He was a strong silent man who believed in action than in utterances. That should serve as a great example to all students past and present.

Our Hostel.—It is nearly a year since our hostel at Kilpauk has been started and it is very gratifying to note that it has been going on very well. The hostellers always in the fore in college functions be they receptions, meetings, or other things deserve to be congratulated. Their endeavours have been most happily rewarded when our popular Principal Dr. Mudaliar threw over a Deepavali dinner in the Hostel. The hostel day will be coming off soon and as funds are rather low (It is the same with boys all over the world, we dare say) one could not appreciate anything better than generous donations from the members of the staff. In this connection we may say that the Warden and the hostellers have made valiant efforts to start a Library of their own but they have not had any encouragement from the outside and all that the Library can boast off are a few odd books,—contributions from the Warden and the hostellers. We would like to appeal through our College Magazine for generous donations from all our readers.

Athletics.—This year our College has not been very successful in the matter of sports. However, the athletes of the College are to be congratulated as theirs have been no mean efforts and we can be very well proud of the results. It is true we did badly at football but we have won the inter-collegiate championship in cricket and shared honours with the Y.M.C.A in Hockey. But it is to be remembered that the College footballers had to play hockey and were not available for football. Mr. Shambushetty, the captain, is to be congratulated for gathering up an eleven amidst his many great difficulties and putting up very bold show indeed. In the inter-'varsity sports held at Anamalai our College won the championship and Mr. Nicholas, the individual champion.



THE MADRAS MEDICAL COLLEGE ATHLETIC TEAM.

Winners of the Inter-Varsity Championship held at Annamalai.

The All-India Surgeons' Conference ; and the Indian Science Congress.—Both these conferences were held in the College buildings sometimes back and students of the College from all classes, both boys and girls, did yeomen service as volunteers. The volunteering work for the Surgeons' Conference was entirely by the Medical Students. In the Science Congress most of the Volunteers were from the College and the entire success of both conferences were no little due to the work of the volunteers. We can very well be proud of the little service we have rendered to the organizers of these conferences. These conferences, we believe, would thrust new spirit and enthusiasm towards the scientific developments and researches of the world in general and India in particular after having met very many eminent scientists and surgeons from various parts of India.

Examinations.—The examinations are all over and worse still the results are out. We offer our congratulations to the successful and extend our sincere sympathies to the failed students. Their efforts may not have been deserving of success but theirs have been no less sincere than the others and they are none the losers for the experience of defeat shall only better fit them for greater success. Congratulations to all the prize winners.

The University Training Corps.—This year we had the U. T. C. camp as usual but with various interesting changes. We had the Camp at Avadi instead of at Pallavaram.

Even the parades were not so boring as last year because the new Adjutant was particular in giving us a practical demonstration and then giving us the chance of practising them.

Competitions were keenly contested and it is very creditable that we got the GUARD MOUNTING CUP which shows our unique excellence in military training.

Besides these we had the "ever-ours" TUG OF WAR cup which looks as though specially reserved for MEDICALS. The winning of the INTERIOR ECONOMY CUP is another feather to our cap this camp. This is specially indicative of the sustained effort we put forth on every day and every hour of the fortnight of camp. Our Company also won the Hockey Cup.

Our officers took a keen interest in coaching us up for the various competitions and this was solely responsible for our success this year in the camp.

Revised University Regulations.—There have been certain changes in regulations regarding the medical examinations. The more important of them are being published below. Will the students make a note.

Scheme of Examination.—The examination shall include:

(*a*) Inorganic Chemistry—One paper (3 hours) and a practical examination not exceeding three hours.

(*b*) Physics—One paper (3 hours) and a practical examination not exceeding three hours.

(*c*) Natural Science—One paper (3 hours) and a practical examination not exceeding three hours.

Candidates other than those exempted under Regulations 8 must at their first appearance present themselves in all the three subjects.

A candidate shall be declared to have passed the examination if he obtains not less than 35 per cent in the written and 35 per cent in the practical tests in each of the subjects : Inorgnaic Chemistry, Physics and Natural Science. All other candidates shall be deemed to have failed in the examination.

Candidates for the examination who fail but obtain 40 per cent in each of the written and the practical parts in any subject shall be exempted from re-examination in that subject.

Candidates who pass the whole examination at the first appearance shall be ranked in the order of proficiency as determined by the total marks obtained by each and shall be arranged in two classes, the first consisting of those who have obtained not less than 50 per cent of the aggregate marks, and the second consisting of all others.

Candidates who pass in the first class and who obtain not less than 60 per cent of the marks in any subject shall be declared to have passed with distinction in that subject.

Candidates who pass the whole examination at a subsequent appearance shall be ranked only in the second class.

A candidate shall be declared to have passed Part II of the Final M.B. & B.S. Examination if he obtains—

(*a*) (i) not less than one-half of the marks in the written and oral examinations in Medicine taken together ; and

(ii) not less than one-half of the marks in the clinical examination in that subject ;

(*b*) (i) not less than one-half of the marks in the written, practical and oral examination in surgery taken together, and

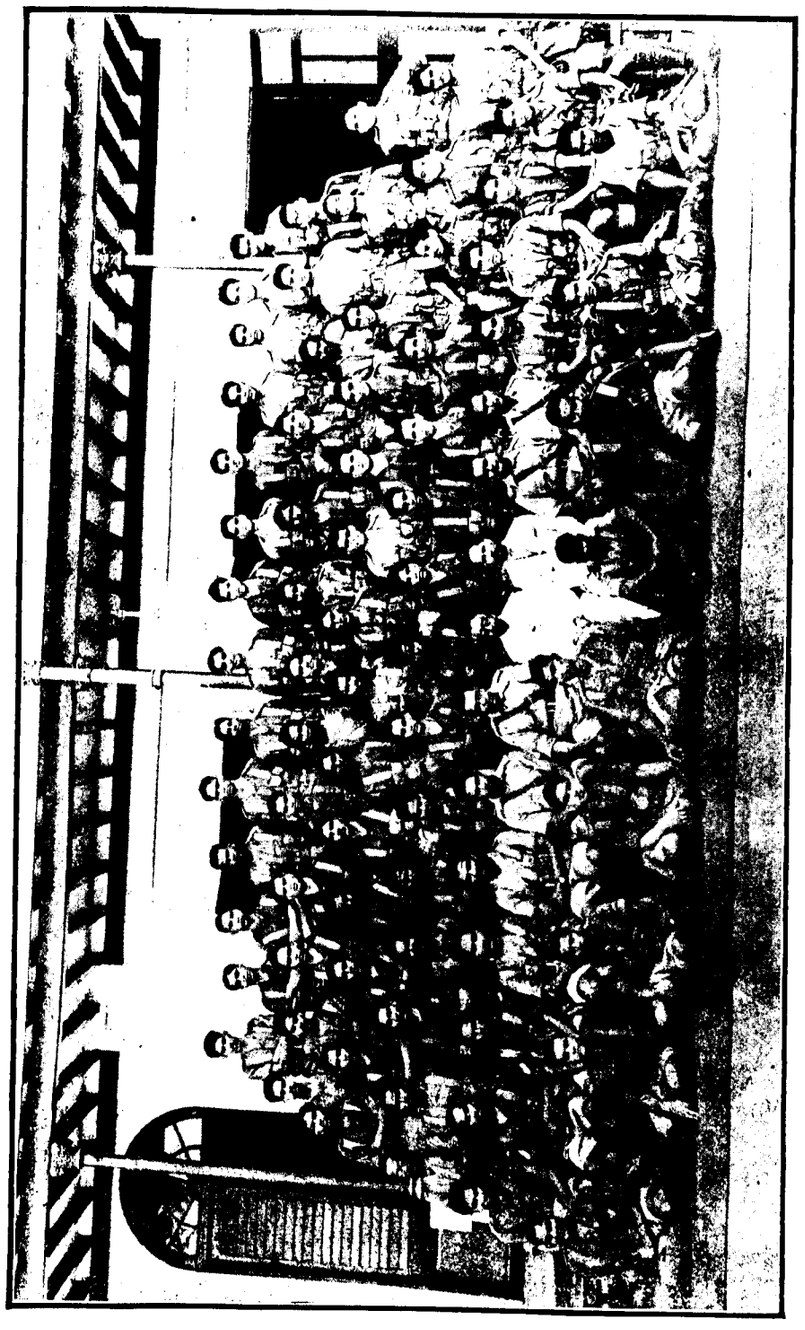
(ii) not less than one-half of the marks in the clinical examination in that subject ; and

(c) (i) not less than one-half of the marks in the written and oral examinations in Obsterics and Gynæcology taken together, and

(li) not less than one-half of the marks in the clinical examination in that subject.

All other candidates shall be deemed to have failed in the examination.

The Student Editors



Group Photo of the Medical Contingent. A-Coy. 5th Madras University Training Corps taken before the departure to Camp at Avadi.

Extracts.

SPECIAL REPORT SERIES, No. 232
MEDICAL RESEARCH COUNCIL.

Appendicitis

A Statistical Study

BY MATTHEW YOUNG AND W. T. RUSSEL.

In spite of the high incidence of such conditions as gastric and duodenal ulcer, appendicitis and colitis, we remain remarkably ignorant of their causes. We still know too little about the predisposing factors to devise effective methods of prevention. The figures relating to the frequency of appendicitis not only confirm that it is very prevalent, but show that it has apparently been on the increase in recent years. There is evidence, moreover, to suggest that the increase is real, and not due merely to greater knowledge and skill in diagnosis; for instance, amongst the insured population of Scotland the annual incidence of appendicitis increased appreciably even between 1930 and 1935, although this was not accompanied by any upward trend in the general sickness rate. Nevertheless, the mortality rate from appendicitis has remained relatively constant during the last twenty years, while the case fatality rate has fallen considerably, no doubt largely owing to the improvements in treatment, and especially in surgical treatment, which have occurred during this period.

Though the mortality from appendicitis seems at a casual glance to be of small account as it is less than 1 per cent of the annual general death rate in England and Wales, the disease is, nevertheless, an important cause of death. In comparison with that from tuberculosis of the respiratory system its quota is small, but it is quite appreciable when contrasted with that from peptic ulcer. The male death-rate from appendicitis was 60 per cent of the corresponding value for peptic ulcer and the female rate was actually 71 per cent in excess.

The Size of the Standardised Death-rate from Appendicitis in comparison with that from some other diseases in 1935.

Diseases.	Standardised Death-rates per 1,000,000.	
	Males.	Females.
Appendicitis	80	53
Ulcer of Stomach and Duodenum (Peptic Ulcer)	124	31
Cancer	1,058	959
Tuberculosis of Respiratory system	627	486
Diabetes	89	117
Pernicious Anæmia	32	43
All causes	1 0,167	8,036

JL

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Mortality during 1915—1918.

The mortality from appendicitis resembles that from diabetes in-as-much as it decreases with descent in the social scale. In view of this "class" correlation it was of interest to study the history of the mortality during the war years to see if it bore any resemblance to that from diabetes. During the years 1915—1918 the diabetic mortality declined at the older ages and the decline has been ascribed to the lessened food consumption in that period because, when the food restrictions were lifted, the mortality subsequently increased. Did the age specific death rates from appendicitis follow a similar course? In view of the difficulty of obtaining an accurate enumeration of the males exposed to risk, the examination was confined to the female mortality. The death rates per million in the two contrasted periods 1911—1914 and 1915—1918 were:—

	Ages.								
	0—	5—	15—	25—	35—	45—	55—	65—	75—
1911-14 (A) ...	24	86	64	45	45	58	69	84	87
1915-18 (B) ..	24	82	64	39	44	63	58	62	71
B as % of A. ...	100	95	100	87	98	109	84	74	82

Although the rates declined at some of the younger ages the reduction at ages above 55 years is the more pronounced. This reduction at advanced life, similar to that for diabetes, was only a temporary phase because the mortality at these ages in the post-war period increased appreciably. There is thus some indication that an excess of food, or possibly certain types of food, may play a part in the incidence of appendicitis.

MORTALITY AND SOCIAL STATUS.

The next aspect of the subject to which attention may be directed is the social distribution of the mortality from this disease. The definition of the five social classes distinguished by the Registrar-General and the mortality amongst males in these categories at ages 20—65 years for the triennial period 1921—1923 are shown in Table 8. In juxtaposition with the rates for appendicitis are the corresponding mortality rates for peptic ulcer (gastric and duodenal ulcer jointly) which is a disease very similar to appendicitis as regards conditions of diagnosis and treatment.

In appendicitis it will be seen that the mortality rate in the highest social class (Class I) is greatly in excess of that shown in the lowest social class (Class V), the ratio of the rates being nearly 5:2. The mortality rates in the intervening social classes show a progressive decline with the social order. The mortality from appendicitis thus falls with exceptional severity on people of high social grade. This divergence, has as been pointed out in the report of the Registrar-General, cannot be attributed to varying efficiency of diagnosis, as such a varying efficiency would be found also

in the case of peptic ulcer and has seen in the table, the mortality in males from that disease shows a steady increase with decline in social class. It would seem therefore that there must be some real causes, whatever they may be, to account for the higher mortality from appendicitis experienced in the upper ranks of society. It is possible indeed that the class difference in the mortality returns is under-estimated owing to the fact that the more prosperous classes would be likely, other things being equal, to have more prompt and efficient treatment.

Showing the Standardised Mortality for Males at Ages 20—65 for the period 1921—1923.

	Appendicitis.	Peptic Ulcer.
Social Class I (Upper and Middle) ...	15·1	14·3
Social Class II (Intermediate) Civilians only ...	12·7	15 3
Social Class III (Skilled Workers) Civilians only ...	7·9	15 3
Social Class IV (Intermediate) ...	7·5	16·6
Social Class V (Unskilled Workers) ...	6·2	19·8
All Males	8 9	16·5
All occupied and Retired Civilian Males ...	8·9	15·8

In the poorer classes but that its diagnosis, especially at advanced ages had made greater progress among the more prosperous.

Recent history of appendicitis mortality shows that there is at present a strong tendency to increased recognition of this malady in later life, and if it is supposed that this tendency has progressed further with the professional attendants for the wealthier than of the poorer classes, it must follow that the excess for these classes recorded at all periods of life will increase to a greater or lesser extent as age advances.

The degrees of association have been determined by the use of the correlation coefficient applied to the comparative mortality figures for the diseases studied in males from 20—65 years engaged in different occupations.

All these coefficients are of much the same order, except that for appendicitis and peptic ulcer which is less than 0·2 but not significantly less than the others. The interpretation of the meaning of correlation coefficients derived from data such as those used is by no means easy. Since, however, the association between appendicitis and peptic ulcer, in the causation of which some common factor might possibly be involved, is not higher than that between appendicitis and nephritis, in which from clinical experience it appears much less likely, if not improbable, that a common causal factor exists, little emphasis can be laid on the importance of the coefficients found.

Clinicians in this country seem to hold very divergent views regarding the importance of appendicitis in the aetiology of peptic ulcer. Moynihan stated there was evidence of some association in about 66 per cent of cases of peptic ulcer.

There is no evidence in the English data that the mortality has any relationship to season of the year, a fact

which seems to be at variance with the inference that would be drawn from the correlation found between appendicitis and temperature; but it may be recalled that this correlation became insignificant when the diarrhœa mortality was held constant.

In the figures for/of New York there is a suggestion, though it cannot be considered anything more, that the mortality may be rather higher in summer and autumn than in the colder parts of the year. In contrast with the mortality from appendicitis, the mortality from gastric ulcer in England and Wales appears to show a relative excess in the colder seasons of the year.

Incidence of Septic Types of the disease in childhood and later life.

It is commonly believed, and there is much clinical evidence in support of the belief, that children are particularly susceptible to septic complications of appendicitis. The gravity of acute appendicitis in childhood, and the consequent high fatality at this period of life, are usually attributed by surgeons to the delay entailed by difficulty in diagnosis and the frequent administration of aperients by the mothers in the belief that the symptoms are to be ascribed to some trivial complaint. Consequently, the proportion of children admitted to hospital with acute appendicitis in whom the appendix is perforated or ruptured is much higher than that in the adult.

There was no evidence of a seasonal influence on the incidence of appendicitis among the boys and girls attending the public schools whose records were examined by the School Epidemics Committee of the Medical Research Council.

The Sex Incidence and Fatality.

The case records of the hospitals in London indicate, on the reasonable assumption that males and females are probably equally likely to be admitted to hospital, though it is possible many hospitals have more beds for male patients, that appendicitis is of more frequent occurrence in males than in females. In the cases from the Edinburgh hospitals, males also predominated over females even when correction was made for age distribution of the general population. On the other hand, Hobler states that in his series of cases from American hospitals the incidence in the sexes was about equal, whereas Reid holds the view, also based on American statistics, that appendicitis is twice as common in males as in females. In all these series of cases the case fatality was definitely higher in the male sex.

"My chicken's laid an egg," boasted one little girl at the tea-party.

"Mine laid two eggs," said another, not to be outdone.

But their small hostess had the last word.

"My daddy laid a foundation stone," she announced.

* ON THE EFFICACY OF THE GONADOTROPIC HORMONES IN THE TREATMENT OF WHOOPING COUGH

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and
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It is characteristic of whooping cough that its incidence is confined chiefly to children below 10 years of age, though occasionally it has also been reported in old people. Persons above the age of 10 and right through healthy adult life are generally immune to the disease, in spite of living in intimate contact with infected children, even though they might not have been attacked previously by any disease having the vaguest semblance of whooping cough. Second attacks in children have also been recorded though only rarely. Further, the vaccines prepared from the organisms isolated from the droplets of the upper respiratory passages of infected children have not been as effective therapeutically as they ought to be if the disease were caused only by infection.

These considerations suggested that, besides any immunity that might have been acquired, there is some other factor which is peculiar only to the period of life between the age of 10 and the end of the adult age that determines the power to resist an attack of whooping cough during this period. One of the dominant features of life at this stage is the activity of the sex glands, and the assumption that the immunity displayed is associated with such activity would be justified, if it could be demonstrated by any means that a temporary stimulation of the activity of the gonads in persons of the susceptible age would act as a curative, as well as a prophylactic, agent. Results of the clinical trials carried out so far have provided sufficient evidence for the correctness of this view.

In the clinical investigation reported in this paper, the stimulation of the gonads was effected either by the oral administration of a standardized preparation of the gonadotropic harmones of pregnancy urine which is described in detail below, or by the injection of Antuitrin 'S'. The latter could not be tried as a routine measure on account of the prohibitive cost and was resorted to only when the oral medication had been ineffective.

Five hundred to nine hundred rat units, the exact requirement depending upon the virulence of the disease and the individual variations, were usually found sufficient to alleviate the characteristic symptoms. Where parenteral administration was indicated, 0.25 c.cm. of Antuitrin 'S' was injected daily. After six to twelve injections, the vomiting and the whooping stopped completely. The residual cough was treated symptomatically. The criterion of cure applied was the total disappearance of the whoops and vomiting and a diminution of the cough.

There was a marked leucocytosis of 12,000 to 30,000 per c.mm. with 46 to 76 per cent of lymphocytes. Twenty cases out of the 23, or 87 per cent, were cured by oral treatment with the

Extract from *The Indian Medical Gazette*, Vol. LXXIV, No. 5, May 1939 page 271

hormones or by injection of Antuitrin 'S'; in three cases, or 13 per cent, either treatment failed. The proportion of probable cures to failures in this series is 5·1:1. In the therapeutic doses prescribed, the preparation of the hormones did not produce any undesirable symptoms.

No case of whooping cough in old age has come to our notice so far.

The administration of the hormones as a prophylactic measure to contacts conferred immunity on them.

In another series of 113 cases which were treated by qualified allopathic medical practitioners in the city and the *mofussil*, 70 cases were reported to have derived benefit by the oral medication and 13 more by the injection of Antuitrin 'S', as judged by the alleviation of the distressing symptoms such as whooping, vomiting and cough. Blood examination could not be carried out in those cases for lack of laboratory facilities. The percentage of cures in this series works out to 73·5 per cent.

For the whole series of 136 cases which have been treated with the gonadotropic hormones of pregnancy urine, the percentage of cures effected is 80·3.

The relief from the most distressing symptoms of whooping cough effected in such a short time as about a week, by the administration of the gonadotropic hormones appears to warrant the conclusion that in these hormones has been found a useful and unsuspected remedy for combating the harassing disease.

WAR GASES.

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In April 1915 the Germans first used the chlorine gas. It was let out from cylinders emitting a hissing sound which gave rise to faint greenish cloud hanging lower on the ground. Later on in 1917 mustard gas came into the field. It was known as yellow cross shell gas. Both these gases caused heavy casualties during the great war as no preventive methods were known at that time except the old form of respirators which were invented in 1917–18. It is therefore necessary to give a brief resume of the war gases, the pathological results of their action, and the treatment and prevention of the casualties in case of gas air raids.

Classification of War Gases.

I. *Non-persistent*:—When released these gases disperse quickly, and cease to be effective after a short period, the length of which depends on the weather conditions and the nature of the soil.

II. *Persistent gases*:—They are generally in liquid form and contaminate the area in which they are released and continue to diffuse in vapour form for quite a number of hours and sometimes even days.

The gases can also be grouped according to the effects they produce:

(*a*) Eye irritant or tear gases.
(*b*) Nose irritant or sternulators.
(*c*) Lung irritant or choking gases.
(*d*) Blister gases.

(*a*) *Eye irritant or tear gases* such as chloraceto—phenone (C.A.P.) ethyl-iodo-acetate (K.S.K.) bromo benzyl-cyanide (B. B. C.)

The tear gases, as their name signifies, cause a copious flow of tears and also a smarting pain in the eyes. They are generally used by the police for dispersing unruly mobs. The action is immediate, selective and temporary, recovery taking place on removal from the infected area.

(*b*) *Nose irritant group or sternulators*:—They are arsine compounds (D.A., D.M., D.C.). Chemically they are known as Diphenylamino-cyan-arsine Diphenyl-amino-chlor-arsine and Diphenyl chlor-arsine. This group has aslo a selective action on the nose. In high concentration or after lengthened exposure burning pain spreads to the naso pharynx and chest. Beyond a mental condition of depression which may last longer, recovery takes place on removal to a freed atmosphere. For both groups an efficient respirator is cent per cent protective.

(*c*) *Lung irritant or choking gases*:—The lung irritant gases are very lethal in their effects; they cause an initial irritation of the eyes, nose and throat, amounting to burning and lachrymation but their predominant effect is on the respiratory passages and lungs in particular. They inflict grave injury and prove deadly to the unprotected. The chief gases of this group are phosgene (C.G.) chemically di-chlor-carbonyl; chlorine which was the agent in the first cloud gas attack in April 1915; diphosgene, used mostly by the French and German; chloropicrin (P.S.) also called "Vomiting gas", tri-chlor-nitro-methane and acting very much like chlorine.

Phosgene and di-phosgene have a delayed action on the lungs, a most important feature from the treatment standpoint. The pathological changes following severe exposure to the long irritant gases, particularly phosgene, are:—

1. Emphysema, of varying degree, but permanent.

Fibrosis of local or more general type according to the complications than ensued.

3. Persisting bronchial conditions, when the upper passages are effected.

A.R.P. Centres.—In large cities and industrial areas there should be A.R.P. centres in suitable buildings preferably attached to the police stations. The A.R.P. establishment in a town should consist of:—

1. Air-raid Wardens—Their duty is to enquire and report air-raid damages.

2. First Aid and Rescue Parties—for bringing in and giving first aid to persons injured in an air raid.

3. Decontamination squads.—Their duty is to treat the places contaminated with gases in order to make them harmless.

4. Fire Brigade.

A stock of gas masks, protective clothing, etc., should be kept in each station. Treatment centres should be set up for treating persons contaminated with the gases.

Gas Mask. Most of the war gases attack the nose and the eyes; so it is necessary to protect them. The best means for the protection of the nose and the eyes is the use of a good gas mask (Respirator). It will prevent gas vapour from passing through it. Two types of gas masks are available now, one for the general citizens, *civilian type*, and the other the *service pattern* for those who will have to work for long hours in area contaminated with poisonous gas.

Service Respirator.—The service respirator consist of *a face-piece and a container containing charcoal and cellulose to absorb gases.* The facepiece covers the eyes, nose and mouth. *It is made of rubber and is shaped to fit the face. In front of the face-pieces there is an outlet valve.* Attached to this valve is a flexible tube through which the air comes from the container. The eye-pieces are made of glass discs which do not splinter. The face-piece has elastic bands for fitting it on the head. The tin container is filled with layers of charcoal and cellulose. The cellulose catches up the smoky particles in the gas; while the charcoal absorbs the gas itself. The container thus serves as a filter of poisonous gases contained in air. The air enters and passes through the container. It is thus freed of any poisonous gas. After purification the air passes through the flexible tube into the rubber face-piece.

2. *The General Civilian Respirator* is smaller in size; and its tin-container is placed just in front of the face piece. An oblong celluloid sheet passes across the eyes.

If no gas mask is available then ordinary lint piece soaked in water and tried to the mouth will prevent to certain extent the injury by the gases.

4. Lack of tone in the myocardium though not necessarily a myocarditis, but leading to tachycardia, and lack of cardiac response.

(*d*) *Blister gases.* There are two examples of this group, *viz.*, mustard gas or (H.S.) and lewisite.

Mustard gas (H. S.) (Yperite in French and yellow cross in German).

Chemically this is dichlor-ethyl-sulphide $(CH_2\ Cl.\ CH_2)_2S$. It is a heavy, oily fluid, clear when pure, darker in the impure state, with a boiling point of 217° C., and having a faint smell or garlic or mustard. It first came into use in July 1917, and then predominated in gas actions, either utilized by itself or in mixtures, till the end of the war. It proved to be the most potent of all gases and largely replaced those in use at earlier periods.

Mustard gas has no selective action like phosgene but either in the form of vapour or liquid, effects every tissue structure which it comes into contact. It is readily soluble in fats and oils and owing to this high lipoid solubility its power of penetration are rapid and marked. *The skin for instance absorbs it, not by Corrosion but by lipoid solubility, skin to blotting paper soaking up in ink.* Owing to the delayed action some hours may elapse before parts are affected and unless protected by a mask the eyes and the respiratory passages bear the brunt of injury. The external skin lesions, erythema, swelling and acute inflammation, blister formation and secondary infections causing even necrosis, are very common. The exposure also effect the mucous membranes conjuctivæ and upper respiratory passages. The trachea and larger bronchi may be the sites of necrotic tissue destruction and slough formation while in some cases of the upper bronchial tree have been coughed up. The lung structure itself, beyond perhaps some areas of emphysema, or collapse, may escape. In contra-distinction therefore to phosgene, mustard gas affects the respiratory tract from above downwards. Secondary infections of the lungs are likely to result in broncho or septic pneumonia, with pleurisy.

There is no evidence to show that any general or systemic infection occurs from absorption of toxic products of mustard gas, apart from supervening sepsis. The consequence is that systems other than the respiratory remain practically unaffected. The abdominal organs show little change, beyond perhaps some petechial hæmorrhages. Occasionally tainted food and drink have been held to be responsible for continuance of gastro-intestinal symptoms. The kidneys show no characteristic change, though a transient albuminuria due to septic absorption may be present. Beyond a leucocytosis present due to the same cause, no particular changes occur in the blood.

Lewsite.—(*B*-chlor-vinyl-Di-chlorarsine). A colourless liquid which gives off an invisible gas, is rapidly destroyed by water and any alkali and penetrates materials in the same way as mustard, has a low freezing point. Characteristics are similar to that of mustard contains arsenic, invisible in vapour form, smell of geraniums, has irritant action on eyes, stinging sensation on skin. It is persistent in action like mustard gas. It must be remembered that lewsite though not used in the last war, is likely to be a weapon of the future.

There are differences of importance in its action, but the general effects correspond to those of mustard gas, with which it is classed as a blister gas. The fluid in the blisters however, contains arsenic. The late results are practically the same, except that arsenical absorption may lead to neuritis.

Sequelae, and after effects of gas poisoning.—The after results mainly occur in the

1. *Respiratory system.*—Sequelae, when occurring are similar in the main to those so commonly seen in cases of long continued bronchitis. From a clinical standpoint, only an authentic history would distinguish. That is to say the emphysema initiated at the "Gassing" is progressive, bronchiectasis of varying type and extent, increases, and fibrosis, usually at the bronchial tree, distribution, proportionally develops, increasing dyspnoea, this usually having an asthmatic character. The clinical features noted with the more severe types of bronchiectasis are likely to be acute prolonged and very serious. Persistent pyrexia of a septic type, much blood stained sputum, and occasional profuse hæmoptysis, loss of weight with continued general deterioration, may readily suggest the presence of supervening tuberculosis. The sputum, however, remains negative, and autopsy has shown in such cases the absence of tuberculous lesions. Bronchiectatic cavities or pulmonary abscess, even gangrene of the lung, may develop, but are not to be distinguished from similar conditions due to other causes. It was early anticipated that pulmonary tuberculosis would have a high incidence in men who had been gassed, but the general experience of the chief combatant nations since the war has been quite the reverse. No material increase of pulmonary tubercle in ex servicemen can be attributed to gassing.

The development of dyspnœa of the asthmatic type deserves special mention. Various factors tend to induce it. Increasing emphysema, lack of cardiac response, pulmonary fibrosis, vascular congestion and anoxæmia are important. Reflex effects from the gastro-intestinal tract, including nasal and oral conditions are also common.

2. *The eyes.*—After an initial short delay, exposure to the gases mentioned causes intractible acute conjunctivitis, haziness of the cornea and (according to the severity of the exposure) followed by keratitis, after ulceration with hypopyon and iridocyclitis.

Photophobia.—This varies from a mild to an extreme degree, in the latter instance requiring the use of tinted glass.

Lachrymation.—Varying according to the severity of the condition present. Visual acuity is always involved.

Congestion of the conjunctiva is a feature in the case of the bulbar portion, for this is marked with enlargement of the vessels; in some cases there is a nævus-like formation. No vitreous changes have been noticed, and so far as can be observed the fundus is normal. The prognosis is bad, and not infrequently enucleation for the relief of pain and the removal of a useless organ has had to be performed.

3. *The Skin.*—Any persistent or relapsing skin conditions are unusual; in fact so few cases are known that beyond an occasional persistence of a mild dermatitis or chronic furunculosis, the latter from secondary infections, no trouble of serious import need be anticipated. This is perhaps surprising, considering the severe skin effects that characterised mustard gas lesions.

4. *D.A.H. and Irritable Heart.*—Though these conditions with precordial pain lack of exercise response and dyspnœa were persistent and frequent after effects during the convalescent periods, following gassing from lung irritant and mustard gas, time and treatment appeared to remedy these conditions.

5. *Functional Nerve Conditions.*—These conditions are the sequelæ of the original gas effect but persist afterwards due to physical causes. Thus aphonia would readily occur from laryngeal inflammatory effects from the irritant gases, and persist either as that, or even mutism physically induced or partly influenced by the early requirements of treatment. Stammering is another form in which the same factors might be a dominant influence. Blepharospasm readily might find origin in the ocular spasm from eye irritant gases, or even extend subsequently to habit spasms. Migraine attacks have on occasion been ascribed to vomiting when gassed.

Detection of Gases.

The gases may sometimes be detected from the nature of the cloud formed at the time of bursting of a bomb, peculiar smell, effect produced on the person, and by means of special test.

1. *By Visible Signs.*—A gas bomb has a thin shell with a large amount of gas in it. It is made especially to explode and discharge its contents on the surface of the earth. In some cases, a *cloud* appears on the part where the bomb drops and drifts down wind. The cloud of gas is dangerous, but as it travels down with the wind and becomes more and more diluted, it soon loses its power of doing harm. *Non-persistent gases* like

chlorine, phosgene, sneezing (nose-irritant) gas, choking gas and one type of tear gas from clouds of this nature. When however a gas bomb containing *mustard gas* bursts, there a is *splash of liquid* at the place where the bomb drops. The liquid drops, sprinkled on the ground or any object, continue to give off poisonous vapour for a long time until the liquid is all evaporated.

2. *By the effect on the body.*—Tear gases only give rise to a copious flow of tear from the eyes. Sneezing gases (nose irritant) produce sneezing and coughing Phosgene and chlorine cause a choking sensation. Lewsite—produces a *burning sensation in the eyes and nose.* It is however easily detected from tear or nose-irritant gases by its strong *geranium-like smell.* Mustard gas does not produce any immediate sign, and is therefore the most dangerous and treacherous of all the gases used in war.

3. *By Testing Apparatus.*—There is a chemical which has been found to change from yellow colour into red on coming in contact with liquid blister gases. Advantage is taken of this quality for the detection of these liquid gases. The chemical detector may be had in the form of a *yellow paint.* If the liquid content of a bomb containing *blister gas is splashed* on anything painted with this yellow detector, the *yellow colour is at once changed into red.* By this means it is possible to find out the presence of liquid mustard gas or lewsite whether sprinkled after the bursting of a bomb or sprayed from above. *Electric and telephone poles and letter boxes in the streets should be painted yellow with this chemical. The window ledges of buildings in towns and cities should also be painted with it.* After an air raid the change in colour of the window ledges will help a citizen to take immediate measures for counteracting the bad effects of the poisonous liquids.

SURGERY OF THE HAND

Injuries and infections of the hand have an importance which is rarely recognized in surgical literature but which is well known to those who are interested in workmen's compensation. On the French railways they account for over 46 per cent. of sums paid in compensation, while in Zurich the highest accident mortality occurred in a period of ten years in wounds of the fingers. At the international Congress on Industrial Accidents held at Brussels in 1935 statistics were given regarding more than 40,000 accidents, and of these wounds of the hands represented 8 per cent. and those of the fingers 33 per cent. Wounds of the hands and fingers thus represented 41 per cent. of all industrial accidents, and they cost the insurance companies 30 per cent. of their total expenditure and represented a total cost of not less than seventy million crowns. It is therefore obvious that the field covered by these two volumes is of enormous importance.—*B. M. J., July 15, 1939.*

STAPHYLOCOCCAL INFECTION OF THE KIDNEY.

Staphylococcal infections of the kidney, apart from the pelvicalycal infections associated with stones, are relatively rare, usually cortical, and hæmatogenous, their source being commonly a boil in the skin or a carbuncle. Early diagnosis is difficult, for in a great many cases no pus or bacteria are found in the urine, and the patient is usually not seen until a secondary perinephric abscess has developed. Reoognized staphylococcal lesions of the kidney include multiple, bilateral, minute, pyaemic cortical abscesses; a subcapsular triangular septic infarct, probably present when perinephric abscesses resolve after simple drainage: and, thirdly, the deep cortical "carbuncle," drainage of the associated perinephric abscess often resulting in a persistent sinus, which either does not heal without nephrectomy or else heals only to be followed by recurring symptoms. Renal carbuncle is commmoner in men than in women, the favourite age being 20 to 35, and there is usually an interval (during which, presumably, the renal lesion forms) of two to eight or more weeks between the initial skin lesion and the onset of symptoms, which at first are merely those of a general infection—rigor, malaise, pyrexia, tachycardia. After a further interval, the duration of which apparently depends on the distance of the lesion from the surface of the kidney (the deeper the focus the slower the development of symptoms), there are local phenomena, pain, tenderness, rigidity, and, finally, swelling in the loin. Renal carbuncle, most commonly due to *Staphylococcus aureus* is usually unilateral, single, and situated in one of the poles, where it bulges on the surface. Its surroundings of dense fibrous and granulation tissue explain alike the rarity of rupture into the pelvi-calycal system (and thus of pyuria), and the lateness of development of perinephric abscess. Important diagnostic points are the history of a primary staphylococcal lesion, the early absence of urinary symptoms, a high leucocytosis, and (with a deep or large carbuncle) deficient or distorted pyelograms, obtained by intravenous or ascending routes; commonly one or more calyces are widened or obliterated. Even at the late stage, when there are local physical signs in the loin, by which time a perinephric abscess has usually formed, pyelography is useful as a guide to treatment. This, according to Girling Ball[1] should be as conservative as possible—for example, drainage of a perinephric abscess. If complete restoration of health or of pyelographic deficiencies does not follow, the affected kidney should be removed if its fellow be sound. But the discovery of a large carbuncle before the formation of a perinephric abscess suggests early nephrectomy which will avert a prolonged illness. Spence and Johnston[2] lay stress on urography as the greatest single objective aid in the diagnosis of renal carbuncle, and point out that both enucleation of the carbuncle and incision and drainage are being performed, more commonly than hitherto, their impression being that, unless more than one-third of the kidney is involved, some such conservative procedure seems preferable as the first step in treatment. It is noteworthy that the operative mortality has dropped of late. Yet statistics are variable as regards the differential mortality between nephrectomy on the one hand and conservative enucleation or incision and drainage on the other. What is certain is that, apart from operation, the general progress of renal carbuncle is to a fatal issue from subphrenic abscess, septicaemia, exhaustion, or peritonitis, whereas with timely operation the prognosis is fairly good.—*B. M. J. July 29, 1939.*

1. Brit. J. Mdl. December, 1938.
2. Anv. Surg 1939, 109, 99.

RECENT WORK IN UROLOGY

The 1938 issue of this year-book well maintains the high standard of its predecessors as a readable synopsis of world urology, with good illustrations, copious references, and useful indexes of subjects and authors. The importance of haematuria is stressed by the fact that in 798 consecutive patients suffering from it 32 per cent were found to have genito-urinary neoplasms, 11 per cent renal tuberculosis, and 16 per cent lithiasis. There are sane discussions of the dangers, as well as of the value, of the more recently introduced drugs. For instance, the administration of mandelic acid, perhaps one of the least harmful of such drugs, is not infrequently followed by the presence of albumin, casts, and even blood in the urine. As regards the statement that the presence of pus and organisms in the urine is among the outstanding features of renal carbuncle, the opposite is usually the case, except late in the disease.

It is illuminating to find a radiologist advising, and a urologist condemning, routine ascending pyelography in renal tuberculosis; surprising to find a recommendation of aspiration biopsy in renal growths and comforting that the frequent presence of large dilated veins round a renal neoplasm signifies neither malignancy of the growth nor invasion of the renal vein. One may disagree that in the case of a suspected renal neoplasm urography should, if possible, be ruled out in favour of ascending pyelography. Even if seen clinically early most renal neoplasms are pathologically late, and excretion urography gives obvious confirmation of their presence without the risk, involved in ascending pyelography, of dislodging neoplastic cells into the circulation by pyelovenous back-flow. Ascending pyelography can be reserved for cases, such as early ones, where excretion urography findings are inconclusive.

It is impossible to do more than mention a few of the points of this admirable compendium, which should prove attractive and easy reading, not only for the urologist, but also for the general surgeon and practitioner.—*B. M. J. July 29, 1939.*

[Review of the 1938 year book of urology in the B. M. J., July 29, 1939].

MEASUREMENT OF ARTERIAL BLOOD PRESSURE.

A Standard Technique.

THE JOINT REPORT.

We print below a joint report of the committees appointed by the Cardiac Society of Great Britain and Ireland and the American Heart Association on standardization of methods of measuring the arterial blood pressure.

Introduction.

It has long been realized by many teachers and practitioners of medicine that the wide variations in blood pressure records of a single subject were due not only to changes in the pressure from time to time under different conditions, but also to the different methods used by the observers. The interpretation of the sounds heard on auscultation is an important cause of divergent readings. A recent survey of Wright and others (American Heart Journal, 1938, 16, 469) revealed a serious lack of agreement as to the correct technique for taking and interpreting the blood pressure. Similar uncertainty was found among insurance companies as to what they should require of their examiners in this regard. Experiments with multi-aural stethoscopes showed that recent years have brought little if any improvement, as the discrepancy among the recently qualified was as great as among the seniors.

The committees for the standardization of methods of measuring arterial blood pressure appointed by the American Heart Association and by the Cardiac Society of Great Britain and Ireland have tried to bring about a crystallization of the best available thought on this subject. If feasible, the committees were asked to make joint recommendations which might be accepted as a standard for practising physicians, medical teachers, and insurance companies.

After a careful study of the material available from many sources and after much discussion the committees jointly recommended the following procedure as the standard method for taking and recording blood pressure readings in man. Some of these recommendations are dealt with in more detail in the explanatory notes. The British and American Committees have decided to publish over their joint signatures reports which differ slightly in wording and phraseology. In the few instances where there was any difference of opinion the views of both committees have been included in each report.

The British and American Committees have both accepted many suggestions where they thought the other had made useful additions or had expressed the recommendations more clearly; both, however, have retained to some extent their own phraseology where they thought this more appropriate to their country and have set forth with different emphasis the three remaining points in which agreement was not reached. Two of these three are simple: the British Committee expressing a preference for the mercurial manometer as less likely to get out of order, and expressing no choice as to whether the patient is lying down or sitting provided he is comfortable; the American Committee preferring that he should be seated, but expressing no preference between the mercurial and aneroid instruments, provided they are in good condition.

The third more important difference is in the method of recording the diastolic pressure. Both committees agree that the point where the loud, clear sounds change abruptly to the dull and muffled sounds should be recorded. The British Committee think this is the only point that should be recorded and that it can nearly always be detected accurately, though at times only with difficulty. The American Committee, influenced perhaps by this difficulty, recommend that the point where the sounds disappear should also be recorded.

Recommendations.

The Treatment.—The equipment to be used for measuring arterial blood pressure, whether of the mercurial or of the aneroid type, must be in good condition and requires to be calibrated at frequent intervals against a standard mercurial manometer. The British Committee think that the mercurial type is the most dependable. The proper care of these instruments and the importance of a standard armlet are dealt with in the explanatory notes that follow.

2. Position of the patient.—The patient should be allowed time to recover from any recent exercise or excitement. He must be comfortable and may be either lying or sitting. The American Committee recommend that he should be sitting and that, if not, a special note should be made. The arm, relaxed and comfortably supported at the patient's side, must be laid bare to the shoulder to avoid any construction by a rolled-up sleeve and to facilitate proper application of the armlet.

3. Application of the cuff.—The cuff, must be of standard size, with a rubber bag at least 12 cm. wide. The cuff, completely deflated, should be applied with the middle of the rubber bag over the inner side of the arm, and its lower edge one inch above the bend of the elbow. It should fit closely and evenly round the arm to ensure against bulging at the sides when it is inflated.

4. Determination of the systolic pressure by palpation.—A preliminary approximate reading of the systolic pressure should be taken by palpation as a check on auscultation; the pressure in the armlet should be raised quickly in steps of 10 mm. until the radial pulse ceases, and then allowed to fall rapidly.

5. The application of the Stethoscope.—The brachial artery should be located by palpation, and the stethoscope applied lightly and accurately over it, just below but not in contact with the cuff. The hand may be pronated or supinated according to the position yielding the stronger brachial pulse.

6. Determination of the systolic pressure by auscultation.—After inflating the cuff quickly to a pressure about 30 mm. above the level of the systolic pressure as found by palpation, auscultation should be conducted during slow deflation. The systolic pressure is the highest level at which successive sounds are heard.

7. Determination of the diastolic pressure.—With the pressure continuing to fall slowly and uniformly, the sound increases to its maximum intensity and then decreases, at first gradually and later suddenly, and soon disappears. The point where the loud clear sounds change abruptly to the dull and muffled sounds should be taken as the diastolic pressure. The American Committee recommend that if there is a difference between this point and the level at which the sounds disappear completely the latter

reading should be regarded also as a measure of the diastolic pressure. This should then be recorded in the following form—Rt. (or Lt.) 140/80-70 or 140/40-0, or if these two levels are identical as 140/70-70. The British Committee disagree with this recommendation and think that except in aortic regurgitation it is nearly always possible to decide the point at which the change comes and that this is the only reading that should be recorded.

EXPLANATORY NOTES.

The Committees feel that certain other considerations in addition to these specific recommendations should be taken into account, and for convenience they are numbered in the same way as the relevant recommendations.

1. *The instrument*:—Frequent discussions have taken place as to relative merits of various types of blood pressure apparatus. The mercury manometer and the aneroid type of apparatus are both capable of correct readings if in good condition, and both may be inaccurate if they are not. This is sometimes neglected in reference to the mercury manometer, which should be tested at intervals in the following ways:—

(*a*) The level of the mercury at rest should be at the zero mark. If any mercury has leaked out it must be replaced.

(*b*) If the small air vent at the top of the glass tubing becomes clogged there may be a lag which will give false readings.

(*c*) The blood pressure box must be on a level surface, since tilting of the manometer will lead to mistakes; it should also be level with the observer's eye.

The aneroid type of instrument, if used, must be calibrated frequently against a mercurial manometer. The needle should stand at zero when the rubber tubing is deflated, and move immediately when inflation begins. A stop pin at the zero mark makes it difficult to check its accuracy. In both types of instrument the valve must be competent. The entire system, including the rubber tubing and bag, must be free from leakage. The rubber cuff must be at least 12 cm. wide; it must be 23 cm. long and its covering must be of inextensible material and, if not of leather or made rigid with metal ribbing, should extend as a band 15 cm. wide for a distance gradually to an apex during a further length of 30 cm. If bulging occurs the reading may not be accurate. For children the rubber cuff may be narrower and the covering shorter. New types of cuffs, using a zipper mechanism or rubber hooks on a ribbed extension the same width as the cuff, may prove to be more satisfactory than the long tapering cuff end.

2. *The Patient*.—The sitting position was selected by the American Committee because for practical purposes it simplified the taking of large numbers of blood pressure readings. The British Committee did not think there was any significant difference between the readings obtained in the sitting and lying positions. Certain physical and psychological factors should be considered, enquiry should be made as to the patient's activity just before the examination, as strenuous exercise may produce changes in the figures recorded. Blood pressure observations taken immediately after meals differ from those taken before meals. There are that for research purposes observations should be made with the same

relationship to meals, sleep, exercise, and allied factors. A rest period of from 10 to 15 minutes before taking blood pressure readings would eliminate or minimize certain of these factors. Tachycardia in itself sometimes causes a raised systolic pressure. Appreciation of the stress or anxiety through which the patient is passing is important to the examiner. Any appearance of concern on his part may unduly alarm the patient, thereby increasing the pressure. Apart from this, the first reading is often much too high because of the patient's nervousness.

3. *Application of the cuff*.—Venous congestion must be avoided so far as possible; there should be no constricting bands on the patient's arm and the pressure cuff should not be kept inflated longer than is necessary to take the reading; it must be deflated completely before any further determinations are made. In obese subjects special care in the application of the cuff is necessary to prevent bulging. Deflation should be at the rate of about 3 mm. of mercury per second.

4. *The systolic blood pressure*.—If the method of palpation is used before the auscultatory method as recommended, the unusual case with a silent gap will not be missed. In these cases after the first sounds have been heard there is an auscultatory gap below which the sounds reappear. This silent gap is not very uncommon in cases of aortic disease and hypertension.

Recommendations for taking blood pressure in Cardiac Arrhythmia and Aortic Valvular Disease.

The determination of blood pressure in these conditions is more difficult and the following recommendations are made:—

(*a*) Where there are extrasystoles the higher pressure of the beat that follows should be ignored.

(*b*) In auricular fibrillation only approximate blood pressure readings can be obtained; the systolic value should be taken at the point where the majority of beats appear, and the diastolic (if at all) at the point where they become dull and muffled. The American Committee suggest that the average of a series of such readings should be noted as the systolic and diastolic pressures.

(*c*) Alternation in the strength of the beats (pulsus alternans) must be looked for carefully. It must be distinguished from the alternating values produced by regular alternate extrasystoles (pulses begeminus).

(*d*) In aortic stenosis with a slow rising or anacrotic type of pulse, auscultation may give a false systolic reading and palpation may be more accurate; often a satisfactory diastolic reading cannot be obtained.

(*e*) In aortic regurgitation with a collapsing pulse the diastolic and point is marked by a less obvious change in the quality of the sounds, which may be difficult to appreciate.

General.—In speaking of the changes which the sounds undergo, the term *points* is suggested for use instead of the commonly used term

phase, since the latter implies a period of time, whereas the *points* are in fact the precise moments at which one phase changes into the next. The use of a single figure for a pressure that varies so rapidly cannot be accurate to within from 5 to 10 mm. of mercury. For special accuracy several determination should be made and the highest and lowest should be recorded. If the pulse feels different on the two sides the blood pressure should be taken in both arms. In the presence of unexplained high pressure in the arm it should be taken in the leg also, so that coarctation of the aorta may be detected. A special cuff is necessary to record the blood pressure easily and accurately in the leg. The rubber bag should be 15 cm. wide and its covering should be 17 cm. wide and 30 cm. longer than in the case of the armlet. The patient should be in the prone position with the leg extended and the sounds should be auscultated over the popliteal artery. In detailed researches on blood pressure the use of a basal pressure might be considered after preparation similar to that used for basal metabolism. It should be determined 10 to 12 hours after the last meal of the previous night, and is necessary to record the blood after resting half an hour in a warmed room. Difficulties in the use of this procedure in practice are obvious, and observations suggest that after 15 minutes the pressure has generally fallen to a basal level. A defect of hearing in the examiner may call for the use of a stethoscope with some form of amplifier.

It is hoped that a critical attitude towards this subject on the part of physicians generally will result from the efforts of these committees, so that every precaution will be taken to obtain conscientious and unbiased records through the use of the standard technique suggested here. These recommendations are not intended to discourage other methods of observation when indicated in special situations.—*B.M.F., July 22, 1939.*

When we give ourselves the airs of angels, we generally end by wearing the tails of asses.—' Arthur Baryard.

The fickleness of the women, I love is only equalled by the internal constancy of the women who love me.—G. B. S.

A doctor is an individual who puts drugs about which he knows nothing into a body of which he knows much less.

A fly may sting a horse and make him wince, but one is still an insect and the other a horse.—Dr. Johnson.

" Doctor ", my wife has insomnia very badly, she very often remains awake until two in the morning what shall I do for her?

Doctor, " Go home earlier."

There is ample evidence that if a woman is determined to keep a man, she will do it, or see to it that no other woman gets him.—Herb Graffis.

A lacedomion man when asked how he had preserved his life so long answered 'By my ignorance of medicine '.—Moontaigue.

Diabetes and the Treatment of Diabetic Coma.

P. UMAMAHESWARA RAO—*Vth Year.*

Diabetes which means 'a running through', is a disorder of carbohydrate metabolism as a result of which the sugar of the food is not converted into energy and heat as it normally ought to; and the main symptom of this disease is glycosuria. Glycosuria itself may be a hyperglycæmic glycosuria or a renal glycosuria; the latter is said to be a benign condition though this type may end as a hyperglycæmic glycosuria. Injection of the drug phloridzin can induce renal glycosuria by lowering the threshold of the kidney for sugar to zero. This fact has been proved experimentally on animals, and on man; in some rare cases however there is a renal glycosuria occurring ***normally*** which is not explained.

Hyperglycaemic glycosuria, which is a more serious condition may result from various causes. It may occur as a result of an emotional reaction as excitement of an order experienced by students before an examination, the suprarenal gland secretes adrenalin which mobilises sugar into blood. If this sugar be not utilised it is excreted in urine as the blood sugar may have risen above the renal threshold. Alimentary glycosuria, which is a very debatable entity, theoretically occurs as a result of eating too much of carbohydrate food when there is a temporary increase in the blood sugar. Normally a man may consume (if he can) 400 grams of sugar without showing even a trace of it in his urine. Pathologically, glycosuria may occur in diseases of the pituitary especially that manifestation of pituitary dysfunction termed 'Acromegaly'. Amongst the various harmones secreted by the anterior pituitary is one of the diabetogenic harmone the hypersecretion of which is said to be responsible for this glycosuria. In the Thyrotoxic manifestation of Graves' disease there is a decreased glucose tolerance with a resultant glycosuria; Chronic Pancreatitis (a rare disease in itself) Bronzed Diabetes (an adrenal dysfunction). Syphilis and other chronic diseases of the liver, especially in children who have huge palpable livers, are the other pathological conditions where there is hyperglycæmic glycosuria with a lowered glucose tolerance.

Pathology.—"The most striking feature about the pancreas of a patient who has died of diabetes is that it looks perfectly normal. And yet the pancreas may be replaced by a malignant tumour without giving rise to a trace of diabetes, for very little islet tissue seems to be sufficient for the needs of the body." The experimental removal of the pancreas in dogs produces hyperglycæmic glycosuria; but the removal of the pituitary as well does not produce this condition. The diabetogenic harmone stimulates certain hypothetical 'sugar centres' in the brain which send down secretory stimuli to the suprarenal gland. Adrenalin thus secreted influences the two functions of the liver concerned with sugar metabolism, *ie.*, glycogenic and glycolytic functions. The glycolytic function is stimulated so that glycogen is converted into glucose and passed into circulation. Insulin has

two functions to perform. It stimulates the glycogenic function of the liver; also it acts as a catalyst in the muscles and secreting glands where sugar may be broken up for purposes of energy and resulting in the final end waste products of water and carbondioxide. Naturally enough the insulin action of the pancreas is antagonised by the pituitary and adrenalin, also the thyroid acts in a similar manner antagonestic to insulin. Insulin is secreted by the islets of langerhans which contain three types of cells the Alpha, Beta and Gama cells, all of which are granulated. The Beta cells are responsible in main for the insulin production. The purpose of the other two cells is still unknown. The only lesion that may be seen in the pancreas is that the Beta cells show hydropic degeneration. The cells are vacuolated and the vacuoles may unite into one and the nucleus is flattened and pushed to one side.

This hydropic degeneration of the Beta cells is a sign of exhaustion for in experimental animals rest by means of starvation can bring them back to the normal. These experimental results cannot be too closely applied to man for these are acute experimental lesions while the disease in human beings is a chronic process and the most confounding thing being that "many diabetics do not die of diabetes, but of arterioselerosis, gangrene, pneumonia or tuberculosis."

The action of insulin and Adrenalin on the glycogenic, and glycolytic function of the liver are antagonestic and in the normal person are in balance producing a condition wherein there is no glycosuria. In diabetes this balance is lost and there is either a relative or absolute change in the quantitative production of insulin and the diabetogenic harmone. The cause for this is unknown. It may be hereditary or it may be otherwise. It may occur in the old or in the young. Obesity is said to predispose. Perhaps it is the result of a "slowly acting poison of unknown nature."

Symptoms.—The Diabetic may be one of the two types. In the one type the person is 'normal' in all respects and does not know that anything is wrong with him and may come in for some other complaint and his unfortunate condition is accidentally discovered during the routine examination when the doctor detects a Fehling's solution reducing substance in his urine. The other type is very typical and the condition is soon evident. He is thin and emaciated with a dry skin and a furred tongue. He is highly cachectic and complains of an increased appetite, of polyurea, and of great muscular weakness. The cause for the polyurea is not understood; it cannot be the result of osmotic equilibrium of the sugar in urine, for the people of the first type rarely suffer from polyurea. In Diabetes insipidus, a disease of the pituitary, there is extreme polyurea; considering that the pituitary is also intimately connected with sugar metabolism may not this polyurea be a result of some minor pituitary dysfunction? The extreme thirst is the result of the polyurea and considering that the patient is losing large quantities of sugar the hunger can be easily understood. Unfortunately however a good number of cases of the first type sooner or later pass into the second type. The normal healthy man soon feels easily tired, he wastes and in a short while is quite emaciated with bones showing out. Sugar appears in his urine and if the condition had been for some time

acetone as well appears in his urine and his breath smells of acetone. His blood chemistry shows a high blood cholesterol.

Diagnosis.—Diagnosis is easily made if the patient's glucose tolerance test be done but one should however realise that this test is not fool proof but has falacies; for instance, a healthy individual can produce a diabetic curve by starving for 48 hours; also a diabetic can produce a normal curve by starving for 48 hours.

Complications.—The complications of the disease are many and the chief danger to a diabetic is the many complications rather than the disease itself which produces fatal results in but a few cases. Arterio sclerosis, diabetic coma, septic complications, eye complications like cataract, diabetic retinitis, etc., nervous disturbances like peripheral neuritis, lung complications like pneumonia, tuberculosis, kidney disease, are but a few of the complications that occur. The main cause being a lowered resistance due to lowered vitality, excess of sugar in blood which offers a good culture medium for pathogenic organisms and most important of all the arterial changes due to which the main and most common complication gangrene occurs so frequently in this disease.

The Glucose Tolerance Test.—The patient takes 50 grams of sugar in 200 c.c. of water first thing in the morning on an empty stomach. Take some blood from a superficial vein and examine it for its sugar content. Also empty the bladder. Thence take blood every half-hour and examine a specimen of urine at same time. The test is expressed as a curve. The normal blood sugar is 70 m. gm. though total reducing substances in blood come to 100—120 m. gm. per cent. This is the resting figure. In a normal person on taking 50 gms. of sugar the blood sugar rises up to a maximum of about a 180 m. gms. per cent in an hour's time comes down to normal goes down the base line and is back again to normal in about 2 hours time. There is no sugar in the urine. The fall of blood sugar below normal is due to secretion of insulin. In the diabetic the resting blood sugar may be normal (mild) or may be high. The blood sugar rises well above a 180 m. gms. per cent and comes down very slowly taking sometimes six hours to come to the base line (mild) in the more severe cases it never comes to the base line. If however the patient be a renal glycosuric case he will show a normal resting blood sugar. The blood sugar on taking the glucose goes up to about 130 m. gms. per cent and comes down to normal but at this low figure of 130 m. gms. there is glycosuria due to the low renal threshold, which is perhaps only 120 m. gms. per cent.

Diabetic Coma and Treatment.—Diabetic coma has to be diagnosed from coma due to other causes but that subject being comprehensive cannot find a place here. Hence one shall confine oneself to the differentiation of coma of diabetes and coma as may result from an injudicious use of insulin. In a diabetic due to the great danger of the coma passing on to death there is a great necessity for energetic and prompt treatment. In all cases of coma the examination a catheterised specimen of urine shall form the basis of diagnosis. Catheterised urine in either case may show sugar but bears not much import and the examination for acetone by Rothera's Nitropumshide test is paramount. Rother's test may also be positive in both types of coma for it must not be forgotten that the urine catheterised

may have been collecting for some time in the bladder and the sugar and acetone may be of precomatic origin.

The appearance of the patient may help in the diagnosis. In diabetic coma the patient is usually thin and exaciated with a dry and scaly skin, his tongue is dry, raw and beefy, his pulse is fast and feeble, the tension in his eye ball is low and his blood may show a low alkaline reserve. In the hypoglycæmic condition the body may be flushed, surface capillaries dilated, the intraoccular tension tends to be high and the pulse is full and bounding. A second specimen of urine is drawn some half an hour later. Presence of sugar and acetone indicates diabetic coma.

The coma is due to ketosis and not due to the excess of sugar and the aim of the treatment is to remove this ketosis. Further treatment should be towards combating the hyperglycæmia and shock. The Blood Sugar in these cases may by near 300 m. gms. per cent and so 40 to 50 units of insulin will not be unsafe. It may have to be given by the vein if the condition be very acute or sometimes the subcutaneous route may suffice. The dosage of insulin that has to be given is to be judged by the amount of sugar in the urine and the Blood Sugar. Examine a specimen of urine, obtained by catheter if necessary, for sugar and ketone bodies. At the same time remove some blood for analysis. Give 40 to 50 units of insulin subcutaneously. Insulin injections are given every four hours and a specimen urine is examined for sugar each time before the injection is given. If the sugar in the urine is only a trace, no more insulin should be given without a further blood test, as the patient may insensibly be wafted from hyperglycacamic to hypeglycacamic coma. The use of glucose intravenously or by mouth or stomach tube also depends on the amount of sugar in the urine.

The treatment of shock is as important as the treatment of coma. The patient must be kept warm with hot water bottles or under a cradle with electric lamps. Stimulants like coramine, strychnine and also digitalis are to be given to combat respiratory and circulatory failure. Dehydration which is also a prominent feature must be tackled; water in as large amounts as possible should be given by stomach tube and if necessary normal saline intravenously or subcutaneously. Estimation of the haemoglobin will determine if enough fluids are being given. In coma the alkaline reserve is very low and sodium Bicarbonate will be useful; pass a stomach tube, wash out the stomach and put a dram or two of Soda Bicarb in 10 ounces of water and if necessary half an ounce of Mag. Sulph. may be added to relieve the constipation. The treatment of coma does not end with the recovery of the patient from coma. For at least two days the patient must be put on a rigid orthodox diabetic diet.

In presenting this article one offers no pretences for originality or even completeness of the subject. This is based mainly on the lecture "Diabetes and its complications" delivered by Dr. Dinker Rao, M.D., under the auspices of the Medical College Association.

Association of Surgeons of India.

The Association of Surgeons of India offers an annual prize of the value of Rs. 100 to the best essay based on original work on a subject to be decided by the Governing Body of the Association and announced at the beginning of every year.

The following are the conditions of the award:—

1. The competition is open to all qualified medical practitioners registered in India, who have been in practice for not more than 10 years after qualification.

2. The essay should be based on original work and should be written in English.

3. It should be type-written on one side of the paper only and should not contain the name or other indication of the identity of the competitor.

4. The name, address and qualifications, however, should be written on a separate sheet of paper and enclosed with the essay.

5. The subject for 1940 is "**Blood changes in surgical inflammations**". Candidates may, however, restrict themselves to any one aspect of the subject, and the essay should reach the Secretary before the 1st December 1940.

6. The copyright for the winning essay will remain with the Association of Surgeons of India. Other essays will be returned to the senders if accompanied by stamped addressed envelopes.

7. The Governing Body may at its discretion withhold the prize during any year if the essays submitted do not come up to the standard.

8. All communications regarding the above are to be addressed to the Secretary, Association of Surgeons of India, Binfield, Kilpauk, Madras.

C. P. V. MENON,
Hony. Secretary.