JOURNAL OF SOUTH INDIAN MEDICINE

(*Incorporating the Bulletin of the South Indian Medical Union*).

VOL. III JUNE 1936 No. 6

PUERPERAL INFECTIONS

T. S. A. PADMANABHAN, L.M.S.,

Badagara

Definition

The term 'Puerperal Infection' is applied to a group of local or systemic affections, occurring in puerperal women within the first three weeks of the post-partum period, and characterised by fever, abnormal lochial discharge, improper uterine involution and varying degrees of toxaemic, septicaemic, or, rarely, pyaemic, manifestations.

Aetiology

The very name 'Infection' reeks of micro-organisms. And, in every case, the underlying factor is some organism or other, though in some the retained products of conception and other debris undergoing autolytic decomposition play a conspicuous part.

The parturient canal in puerperium offers ideal soil for micro-organisms to grow and flourish. This is more so in the first week of puerperium and most so just after labour. The various tears and bruises and the ragged placental site afford them shelter and allow of their breedings; the blood clots and debris inside offer the pabulum for their growth and multiplication; and the gaping blood vessels and dilated lymphatics act as channels for their *demarche* and dissemination. The patient's defensive forces are below par. The very condition of conception, though a physiological process, is a deviation from the usual state; to it are added the various complications of pregnancy, all the drawbacks of difficult labour and the loss, in the lochia, of

the vaginal bacilli which normally protect the parts by their acid secretions. Great mercy, then, that, under such conditions, puerperal infections are not much commoner than they are to-day.

The organisms gain entrance into the field through auto-infections or conveyed ones. The nearness of the parturient canal to two bacterial factories, *viz*, the vulva and the anus; the ever-present possibility of accidental contaminations; the lowered vitality and the consequent liability of dormant lesions flaring up; all these are productive of auto-infections. But auto-infections are rare, the conveyed ones being much the commoner and by far the more serious. Infections are almost always conveyed to the uterus by the hands of the accoucher and the attendants; through ill-sterilized instruments and appliances; and from uncared or badly cared fields of operation. Protracted labours, especially in the debilitated, are particularly dangerous, as these exhaust the patient and may demand interventions. Indeed, as ARMSTRONG and BURTWHITE have pointed out, exhaustion and injuries that follow prolonged labour with or without instrumental delivery render the maternal tissues vulnerable to infections by otherwise harmless organisms.[1]

Lastly, infection may be conveyed through contagion, and that form of puerperal fever caused by *S. pyogenes* is distinctly and highly contagious.

The bacterial flora in puerperal infections are many and varied, and include such diverse cocci and bacilli as *staphylo*, *strepto*, *pneumo*, and *gonococci*, *colon*, *typhoid* and *diphtheroid* bacilli, and ***Bacillus aerogenes capsulatus*** in the exceptionally rare cases of gas gangrene—all these over and above a number of unidentifiable organisms always met with in the parturient canal. *S. faecalis* and *colon bacillus* dominate the picture in morbid puerperia, while *S. pyogenes* predominate in severe, and 'clinically septic' cases.

Pathology

The mischief usually begins in the uterus as an acute cervicitis or endometritis, and in less severe cases as a systemic absorption of the toxic substances produced therein. Rarely and especially in precipitate deliveries at the outlet, it starts in one or more of the tissue-breaks in the vagina or the perineum.

Whatever be the source of infection, the organisms, when they have gained an entry, soon multiply and try to set up colonies. But the

1. Edin. Med. Jour. 1928, Sept.

ever-vigilant leucocytes on the sentry-go scent the danger. The bolder and the more patriotic amongst them plunge into the arena and take the offensive, while the rest collect on the border to form, as it were, the second line of defence. The dormant and distant forces in the body are soon roused to activity, and each becomes engaged in its share of impending work. The subsequent events depend on the respective strengths of the body-foes on one side and the body-forces on the other.

Where the phagocytes and the protective elements are the more powerful, the organisms are either done to death and removed bag and baggage; or their activities become curbed and their very existence cribbed and cabined. In the latter event, the organisms remain entrenched in the tissue niches within the leucocytic barrier, biding their time, but all the same building thrombi, destroying locally and discharging into the blood stream their bombs of haemolytic and tissue toxins—all together tending to undermine and demoralise the body forces in a silent subtle way.

On the other hand, where the invaders outpower the defence (whether initially or at a later stage), the former break through the cordon of inflammation into surrounding structures, and further into the vascular system if and when conditions permit this. Once the circulation is reached, bacterial thrombi are formed from which small particles are detached and swept in the blood stream, producing the condition known as 'bacteriaemia'. The circulating particles become held up in the fine capillary meshes of the various organs and produce secondary foci there, similar in structure and properties to the primary ones.

The above depict the events in progressive infections with a mild or a moderately severe onset. In fulminant cases, however, the picture is an entirely different one. Here, the organisms are so powerful and disseminative, the onset so acute and sudden, and the onslaughts so grave and generalised that the body-forces, even if they find time and stimulus to organise, are unable to cope with the situation—as they are demanded on all sides and at all strategic sites simultaneously. In such life-and-death cases (the scenes of action constantly shifting, depending on the force, rapidity and virulence of the attack and the vulner-ability and functional importance of the part affected), the available leucocytes cannot be stationed at one spot but have to be sped up and down the blood stream to reach all momentous points. It should be obvious that the phagocytic barriers, so strong and evident in localisable lesions, should, in the cases under review, be conspicuous by an extreme thinness or an entire absence.

All things considered, it should be observed too that no one stage is an entity in or by itself. The course in puerperal infections is a continued one with an overlapping of different stages and allowing of no lines being drawn between the individual stages. In fact, the whole process is a progressive one proceeding either in a straight course or a closed one. In the former case, the continually increasing bombardments by the bacteria and their bombs suppress the defensive forces more and more till the whole defensive mechanism is completely broken down, when the conflict becomes decided against the body and the body succumbs with a picture of extreme bacteriaemia and toxaemia. In the latter event, the body stoops to conquer, succumbing temporarily, it soon rallies round and finally overcomes the foe—the reaching of the rallying-point and obtaining a complete recovery being however dependant on the zig-zags on the course, as such zig-zags tend to prolong the course and cripple the body.

The anatomical lesions in puerperal infections are never uniform, as the pathological changes underlying them ever vary. The condition being one of infection, the picture obtained is that of inflammation, and the findings in puerperal infections are those of inflammatory changes ranging from a mere cloudy swelling to actual necrosis. In morbid puerperia, (otherwise known as puerperal sapraemias or toxaemias), there are, over and above the local changes, a general congestion and cloudy swelling of all the organs and a rapid disintegration of red blood corpuscles as evidenced by the dark and imperfectly coagulated blood and the staining of the vessel-walls. In clinically septic cases, the changes at the primary site are little or nil, while general mischief is marked. All the organs are congested, oedematous and show punctate haemorrhages. The serous cavities contain haemorrhagic or sero-sanguinous effusions. The blood is of a tar-like consistence, coagulates with difficulty and shows a marked leucocytosis, but with a large number of leucocytes disintegrated or disintegrating. In fulminant and rapidly fatal cases, all anatomical changes are at a minimum; indeed, it might seem an axiom that the graver the infection the less the gross lesions. In affections that have become prolonged and chronic, the findings are those in chronic inflammations elsewhere, *viz.*, destruction of the parenchyma, increase in the fibrous tissue and a general matting locally, and amyloid and other degenerations in other organs.

Clinical Manifestations

In a disease with potentialities for spread and complications, the ymptoms and course are bound to overlap and be protean. Yet, for purposes

of description, one has to adopt some classification, and perhaps one may best classify the various symptom-complexes in purperal infections as:—

(I) Disturbances due to primary lesions;

(II) Disturbances due to local extension; and

(III) Disturbances due to systemic invasion.

Under the first we shall consider (*a*) Endometritis, (*b*) Vulvo-vaginitis, (*c*) Cystitis and (*d*) Gas-gangrene of the uterus; under the second, we shall deal with (*a*) Salpingo-oophoritis, (*b*) Cellulitis, (*c*) Peritonitis and (*d*) Phlegmasia alba dolens; and under the last, we shall discuss (*a*) Toxaemia, (*b*) Septicaemia, (*c*) Pyaemias, and (*d*) *B. coli* infection.

I. Disturbances due to Primary Lesions

(*a*) *Puerperal Endometritis.*—This is the most common primary lesion, and exhibits itself in two forms, *viz*, acute putrid endometritis and acute septic endometritis. We shall deal with the former under Sapraemia, and the latter under Septicaemia.

(*b*) *Puerperal Vulvo-vaginitis.*—This affection, much less common than the preceding, almost always starts in one or other of the perineal tears, and is often due to the mischievous stitches in cases savouring of sepsis. The symptoms and signs are much the same as in puerperal endometritis.

With the onset of infection, there appear headache, fever, malaise and other general symptoms; and signs of inflammation locally. The parts become swollen, hot, red and tender; in severe cases, the oedema and tenderness may become so great that the introduction of even the douche-nozzle into the vagina will be difficult and resented.

The typical lesion in vulvo-vaginitis is the highly-sensitive, slowly-healing, well-known 'puerperal ulcer' that makes its appearance about the perineum on the posterior wall or vagina, or over the labia, especially when irritating discharges are present; the ulcer itself is bathed in a dirty foul discharge.

Sometimes, the affection tends to become necrotic rather than ulcerative, when there occurs a generalised yellowish fibrinous exudate leading to a condition of 'exfoliative vaginitis', easily mistaken for diphtheria.

The course and prognosis in vulvo-vaginitis are never alarming; and the latter is decidedly better here than in endometritis. Sequelae are almost

Journal of South Indian Medicine

Vol-3 No-6&7

June
July 1986

nil; if any, they are more traceable to the complete or incomplete perineal tears than to the vulvo-vaginitis.

(c) *Puerperal Cystitis.*—This, in the majority of cases, arises out of an unwarranted or an injudicious catheterisation of the bladder. Occasionally, it forms part of a general infection by *B. coli*. And cases are not infrequent where the infection has spread by way of the urethra from an original vulvar lesion, in which case the urethral duct also becomes involved.

The predominating organism in all cases is the *B. coli.*

When once the mischief has set in, the affection runs the same course and leaves the same sequelae as any other acute attack of cystitis, showing in addition the typical features peculiar to the causative organism.

Cases wrought by *B. coli* are amenable to treatment, though it may entail time and patience.

(d) *Gas-gangrene of Uterus.*—This usually fatal, but rare, affection is caused by *B. welchii* or *B. aerogenes capsulatus*. The chief features are those of a severe 'live putrefaction'.

The onset is early and acute, and the most constant initial symptoms are the disproportionate pain and pulse. The local signs may be nil, and yet the patient will be writhing in pain; the temperature may be normal or febrile, and yet the pulse will be abnormally rapid and feeble.

At first, the endometrium is stained with a red-tinged serum and a dirty creamy tint is all that is visible on the uterine surface. Within 24 hours, the surface becomes covered by a dirty greenish-grey membrane exuding a thin discharge. At this stage, crepitations may be elicited and fine explosive sounds heard as the gas bubbles out to the exterior. Soon, oedema and gangrene appear, and a characteristic foamy offensive discharge exudes. Physometra develops, toxaemic collapse sets in and the patient sinks.

Prognosis is grave and a fatal outcome is the rule.

II. Disturbances due to Local Extension

(a) *Puerperal Salpingo-oophoritis.*—In the spread of puerperal infections, the tube or the ovary is seldom involved alone; a spread to the one invariably extends to the other. Indeed, in many cases, salpingo-oophoritis is but one part of an extensive pelvic inflammation involving the whole of

the reproductive organs, the pelvic cellular tissue and the peritoneum—the anatomical position of the salpinx however making the tube the first victim in the large majority of cases. The extension itself may occur through contiguity of tissue or through the lymphatics *via* the broad ligament.

The condition being but an incident in a large spreading affection, the symptoms and signs in puerperal salpingo-oophoritis are vague and indistinguishable, especially where the extension has been through the lymphatics. In tissue-extension, however, its onset may be suspected, if, after a week or ten days with features of mild infection, an acute exacerbation occurs with rigor, pyrexia and pain in the lower abdomen and iliac fossae. Physical examination at this stage usually reveals nothing definite to warrant a clear diagnosis. After a short or long period, the affection tends to be localised, when a bilateral or one-sided thickening with varying tenderness and mobility may be elicited.

The course tends to be very chronic. Prognosis, though not bad, is not rosy. Usually, there is no immediate danger to life; but all the sequelae of pelvic inflammations and a general run-down health consequent on slow poisoning may supervene.

(b) *Puerperal Pelvic Cellulitis.*—This affection, also known as 'Parametritis' (from the fact of the parametrium being involved the most), may occur as an uneventful incident in itself in the course of a general pelvic inflammation, or as the sole and important pelvic lesion starting and running a course of its own. In the latter event, the disease usually takes its origin in some *cervical* tissue discontinuity that has become infected; and as cervical injuries are much more common on the left side, the type of parametritis we are considering generally appears first on the left side.

When the mischief is but an incident in a wider malady, it possesses no distinctive features and its differentiation may be difficult. But, when it occurs alone, it presents certain well-marked characteristics.

In a typical case, after a varying period of 5 to 10 days of anxiety to the medical attendant consequent on an unsatisfactory progress of the patient for no ostensible cause, the disease comes out all on a sudden with or without a rigor. The temperature shoots up to 102°, 103°, or even 104°F; the pulse becomes proportionately raised; a sense of fulness and a dull aching or a severe pressing pain are complained of in one or other of the iliac fossae (usually the left for reasons already mentioned); and the patient exhibits all the features of a person acutely ill and toxaemic. Examination at this

stage usually reveals a uterus large for the day of the puerperium and painful on movement; pressure on fornices will excite pain and elicit tenseness or fullness on the affected side; and if the cervix is involved, there will be the usual signs of cervicitis.

In a day or two, effusion appears. The effusion may be confined to the original lesion in the paracervical tissues and grow there, forming a large inflammatory mass; or extend at its own level to the opposite side almost encircling the uterus, or posteriorly towards the rectum surrounding it like a horse-shoe, or again anteriorly to the space of Retzius showing itself on the anterior abdominal wall. With the increasing local disturbance, the general symptoms become exacerbated and additional symptoms due to pressure on the rectum, the bladder and the veins arise; examination now will reveal a variably displaced uterus and doughy elastic swellings on either or both sides of the uterus, about the rectum, or towards the anterior abdominal wall.

The usual fate of the disease is for the effusion to get absorbed and resolution to occur, leading to a complete recovery of the patient, although, in a small number of cases, traces of induration, an appreciable tilting of the cervix, a slight fixation of the uterus, and, rarely, stricture of the rectum are left behind as sequelae.

In a considerable number of cases, however, the effusion is not absorbed, but tends to suppurate and form abscesses. Such an event is marked by an increase in the temperature and a rise in the pulse. Ever after this and till the end of the course, the patient shows evidence of septic absorption; the temperature is remittent or intermittent, pulse is rapid, rigors and sweats occur, anorexia and asthenia increase, and general health becomes gradually impaired.

The abscesses are usually single, and, in the absence of spontaneous or surgical evacuation, burrow their way along the lines of least resistance, and point externally at different sites, depending on the site and nature of the extension of the original effusion. The most common situations are above Poupart's ligament, into the vagina or into the rectum; less commonly, it may point into the bladder. It may also rise on the anterior abdominal wall in front of the bladder, or posteriorly between the iliac crest and the last rib. Amongst other unusual sites are Scarpa's triangle, the buttocks, ischio-rectal fossæ—in which cases the travel has occurred along the exits of the main vessels through the femoral ring, the sacro-sciatic foramen and the obturator foramen. In rare cases, these abscesses may point into the peritoneal cavity or into any one of the hollow viscera in the vicinity; a case has

been recorded where a burrowing abscess ascending behind the peritoneum penetrated the diaphragm and reached the thorax.

The prognosis in an uncomplicated case of pelvic cellulitis is, for all its apparent frightenings, really good; indeed, when a case does not clear up, one can be almost certain that some other organ is involved. With proper convalescence (which may be prolonged) and thorough treatment complete recovery may be obtained; at the most, the conditions mentioned under the fate of the effusions may be left behind as sequelae.

(*c*) *Puerperal Peritonitis.*—This serious malady may occur in either of the two forms, *viz.*, the very grave and usually fatal general peritonitis, or the less dangerous (yet serious) localised pelvic peritonitis.

General Peritonitis.—This tragic event, when it occurs early and in an acute form, may result from an acute rupture of the uterus in prolonged labour and with chances for contamination; or from a rapid extension of a virulent infection directly through the uterine walls from a highly septic uterus. In other cases, the affection is due to a spread of infection from some primary focus *via* the lymphatics or through the mucous membrane of the tubes. In some rare instances, the rupture of some circumscribed or burrowing pelvic abscess directly inoculates the peritoneum.

The disease usually manifests itself some time between the second and the fourth day of the puerperium, depending on the virulence of the infection, the nature of the spread and the resistance of the patient. Where, however, the disease originates in the rupture of some pelvic abscess, it usually manifests late in the course of a mild or severe pathological puerperium. The symptoms, in most cases, begin as a limited or spreading peritonitis rather than a diffuse fulminant one. There is a sudden onset of abdominal pain and vomiting with a slight elevation in temperature and rapidity of pulse. When the disease becomes established, the symptoms become pathognomonic.

Pain, the ushering and the most constant symptom, is, like all pains of serous inflammations, stabbing or lancinating in character. It is agonising, continuous and generally diffuse; the slightest contractions of the abdominal wall starting or increasing it, the patient resents even the gentlest manipulation by the medical attendant. Indeed, the pain imposes an absolute immobility on the sufferer, and the patient, to keep her abdomen relaxed and rested, adopts a characteristic decubitus; she prefers to lie on her back, often with knees drawn up, and taking short shallow breaths superficially and

intercostally to avoid using the diaphragm as that excites pain. *Vomiting*, an early and prominent feature, is usually painful, and often of a peculiar 'pumping' character; it usually persists, adding to the distress of the patient, and may become incessant in the later stages when the patient may be seen involuntarily pouring out a few ounces of an intensely foul fluid every few minutes. The vomit, consisting of stomach-contents at first and then a bile-stained greenish or brownish fluid, may become distinctly stercoral in the end.

Constipation, nil or slight at first, may become absolute with the onset of intestinal paralysis when there will be no flatus passed and no borborygmi present. Cases are not infrequent where an initial diarrhoea ends in a later paralytic constipation.

The appearances and physical signs of the patient vary with the time of the examination. In the early stages, one sees a patient, suffering and supine; her skin fevered; her cheeks hot and flushed; her expression featuring intense agony; her looks anxious and apprehensive; her ominously bright eyes instilling fear and alarm in the mind of a careful and cautious medical attendant; her thin, moist, tremulous tongue bespeaking an acute illness; and her vigorously working alae nasi and intercostals suggesting an impending crisis. Fever is present but its height varies; and fever by itself is of no import unless it be subnormal or hyperpyrexial. Pulse is always rapid and often out of proportion to the temperature. Respiration is rapid and heaving, with intercostals very active; chest is usually free but may show basal congestion. Abdomen is 'resty', rigid and tender; distension is absent or only slight; borborygmi will be present. Blood shows a marked leucocytosis and the number of active leucocytes marks the prognosis. Local signs are usually nil.

As the disease progresses, the picture also changes; and towards the end, typhoid state sets in and 'facies hippocratica' develops. The bed which was so full and exciting but 36 to 48 hours ago is now sullen, and contains in its small middle or a gloomy corner the thin haggard, shrunken patient, listless, muttering and fast-sinking. Her pale face is cold and languid; the nose is pinched and drawn; the malar bones are prominent; and beads of sweat stand out. The eyes sunk in their sockets are dull, and stare out if open; reflexes are lost and corneal opacity may be seen spreading over. The mouth is drooping, and mucus or saliva may be seen dribbling through parted lips; the tongue is dry or slimy and thick with flakes of sordes; the oral cavity may be extremely foul, especially if stercoral vomiting had been present. The thin shrivelled limbs are hard as if frozen, and are flaccid.

The cold, clammy, lustreless skin is loose and inelastic; in certain cases, a peculiar induration of the subcutaneous tissues may be present. The skin temperature may be subnormal, normal or febrile, but the thermometer in the rectum will usually show hyperpyrexia; the oral temperature is difficult and risky to take. The pulse is small, soft, thready and wiry; may be imperceptible and uncountable. Respirations are weak, sighing and irregular, or may be unnaturally calm, the abdominal breathing being synchronous with the thoracic; examination of chest will show rales, especially at the bases. The abdomen is usually tense, distended and tympanitic; borborygmi are usually absent. Liver dulness may be lost, and in certain cases, especially in those caused by rupture of the uterus, shifting dulness indicative of fluid in the peritoneal cavity may be elicited. Urine may be suppressed or the bladder is distended. Uraemic symptoms and terminal hiccough may supervene. Nervous prostration is extreme, and subsultus tendinum is the usual terminal event. If the patient is conscious (which is extremely rare) the patient does not complain—the pain has completely disappeared—and answers questions feebly and unconcernedly. Blood shows slight or no live leucocytosis, most of the leucocytes being disintegrated and the rest disintegrating.

The course is rapid, and prognosis is particularly grave. Fatal termination is the rule, and death occurs within 48 hours. Unfavourable features are— early onset; grave initial symptoms; a rising and progressively feeble pulse; a very high or a very low temperature; persistent and stercoral vomiting; diarrhoea followed by absolute constipation; suppression of urine; onset of subsultus tendinum; and a low leucocytosis. Of favourable import are conditions contrary to the above and a tendency for the disease to become localised, evidence of which will be found in the short quick extra-abdominal respirations giving place to quiet, natural, easy breathing, and in the diffuse abdominal signs getting more and more confined to fixed areas.

Pelvic Peritonitis.—This affection, once also known as 'Perimetritis', is again an infection of the peritoneum. But here, the organisms being not so virulent, or the local resistance being superior, or because of both, the disease tends to become localised; and in consequence of such localisation, the symptoms are less tragic and the prognosis less grave.

The etiological factors are much the same as in general peritonitis, except that a rupture of the uterus rarely starts a *local* lesion. Besides these, a preceding cellulitis occasionally flares up a local peritonitis. In fact, in most cases, para - and peri - metritis co-exist within widely varying limits, and the disease in any particular case takes its name after the more obvious of the two.

The clinical features are similar to those in the diffuse variety; but they are seldom so tragic. The stabbing or lancinating pain, though severe, necessitating the patient to lie on her back with knees bent, is never so intense nor so diffuse as to compel her to suspend all abdominal breathing; it is localised in the lower abdomen, and becomes increased by pressure in the iliac fossa. Vomiting too is never so severe as in general peritonitis; in many cases, it may amount to a mere sickness; it is seldom stercoral. Constipation, though usually present both early and late, is due more to other causes than intestinal paralysis.

The patient is acutely ill, but not so alarmingly as in general peritonitis. The skin is hot and flushed. The face is congested, and may be cyanotic in severe cases. The eyes are injected and bright, but not ominously so; they may be distinctly jaundiced in the late stages. The expression is anxious, but rarely apprehensive. The tongue is dry and thickly coated. Typhoid state and facies hippocratica are rare. The fever at onset may be higher, but hyperpyrexia and subnormal temperature are unusual. The pulse is full, bounding, rapid, and usually corresponds to the temperature; in very few cases only it becomes soft and thready. The respirations are short, shallow and rapid, but are not usually confined to extra-abdominal breathing. The abdomen is tender, but the tenderness is more limited than diffuse. Rigidity may or may not be present; if present, it is not diffuse and is usually best elicited at a level well above the pelvic brim. Distension, tympanitis and borborygmi are present to a variable extent. Urine is not suppressed. The bladder may become distended in the presence of effusion-pressure when the rectum, and the veins too, may become affected.

Local examination will show the vaginal vault resisting, full and tender; pressure on, and movement of, the cervix will be painful. The rectum may be tender, and rectal examination distressing. Other findings will depend on the course of the inflammation.

In mild instances, the exudate being slight, more fibrinous in character and with a distinct tendency to form adhesions, there occur a central fixation of the uterus and a general matting of the pelvic tissues involving the gut the most, which becomes almost glued to the other pelvic viscera. In severer cases, on the other hand, the effusion is large, the exudate is more cellular, and the inflammatory products have a tendency to become purulent; in consequence, pockets of pus form in various sites in the pelvis between the coils of the gut and the reproductive organs. The pus, forming large collections, may gravitate to the pouch of Douglas, where the

adhesions already formed encyst them to form localized abscesses; very rarely, the pus collections tend to travel to the utero-vesical fossa, which becomes then the seat of abscesses. It is noteworthy that, whereas the effusions in pelvic cellulitis appear to have a tendency to become associated with the anterior part of the pelvis, those in pelvic peritonitis seem to show a preference to the posterior. The abscesses may be felt per vaginum or from the rectum as the case may be. Vaginal examination will show a bulging in and a narrowing of one of the fornices, most commonly the posterior; and the finger in the fornix will feel the tumescence behind. If the abscess is in the pouch of Douglas, as it almost always is, it lies in front of and pressing the rectum; rectal examination in such cases will show a swelling in front of the rectum and not surrounding it as is obtained in cellulitic mischief. The abscesses themselves remain chronic. When they rupture, they do so most into the rectum and rarely into the vagina; intra-peritoneal ruptures usually result in general peritonitis.

The course of the disease is decidedly chronic. The prognosis is far from good; it is indeed positively bad in most cases. A fatal event, though rare, is not nil; and the patients that escape immediate death are often invalided for life. Dyspareunia, dysmenorrhoea, dysuria, dyschezia and a host of other 'dys-activities' of pelvic organs are the usual legacies of the disease. As a rule the acute inflammation disappears, but not before inflicting permanent damage on the reproductive organs and enough on the bladder and rectum. Pyo-salpinx, hydro-salpinx, salpingo-oophoritis, peri-salpingitis and perimetritis; less frequently, inflammations of or injuries to the bladder and rectum—these are some of the common sequelae of non-fatal pelvic peritonitis. The collections of serum or pus between the coils of the intestines constitute a grave danger, as these tend to produce adhesive peritonitis, ileal kinks and similar serious troubles. Sacculated exudations and encysted peritonitis are not uncommon; they are difficult of diagnosis and may appear anywhere in the abdomen or pelvis, though most usually in the vicinity of the navel. A low inflammation of the serous coats of the abdominal and pelvic viscera; small or large localised peritoneal thickenings of cartilaginous hardness; a chronic induration of the whole peritoneum; displacements and disorganisations of the organs in the abdomen and the pelvis—these are some of the rarer complications of the disease; and if persent, they trouble the patient much and tax the medical attendant to the maximum as their diagnosis is extremely difficult. The general health of the patient suffers considerably as a result of a slow poisoning from the unresolved suppuration, or as a result of the pain and other symptoms incidental to the secondary complications, or as a result of both.

(*d*) *Phlegmasia alba dolens.*—This affection, known also as 'white leg', arises from a blocking of the circulation in the affected limb, due to some deep thrombo-phlebitis or lymphangitis. The disease, pre-eminently infective in origin, may arise as a direct extension of some low mischief at the uterine sinuses, or as the result of the damage to the fine endothelial vessel-walls by the irritating toxins in the blood; it may also arise from an exudate either within or without a vessel causing compression on an adjacent one.

Phlegmasia, like cellulitis, is often but an incident in a protean pathological puerperium. But, in many cases, it occurs as the only lesion of import and runs a course of its own.

In a typical case, after a varying period of 15 to 20 days of an unsatisfactory puerperium manifested by symptoms of slight *febrileness* and no obvious mischief, the disease ushers itself in suddenly about the end of the second week or the beginning of the third with or without an initial rigor. The temperature shoots up to 103° or 104°F and the patient complains of cramp-like pains on the calf of one leg, usually the left (the cervical lesions are usually left-sided; the pelvic colon lies towards the left and the peculiar arrangement of the veins of the broad ligament and leg on the left side favours venous stasis). Within 24 hours of onset, the affected limb becomes considerably swollen, appears white and glistening, with the skin over it greatly stretched, and feels heavy, painful and tender. The swelling, pain and tenderness vary. Where the cause has been a direct extension, the whole leg becomes enormously swollen, and the pain and tenderness may start well within the pelvis as a dull aching, becoming acute as they pass the groin. In other cases, they are slight; the swelling may be confined to the ankles, and the pain and tenderness referred in patches along the course of the vessels. The nature of the swelling too is not uniform. If the mischief be in the veins, the leg will be oedematous and pit on pressure, while in cases of lymphatic mischief, the limb will not pit on pressure and be peculiarly brawny. Sometimes, the mischief lies superficial; in such cases, there may be visible on the limb-surface red knotted streaks with erythema around along the course of the vessels, and in extreme cases the whole vessel from the groin downward may be felt as a red raised thickened cord.

The general symptoms subside in a week or so. The local mischief however continues for some time more. Sometimes, when the affected limb is resolving and the patient is recovering, there occurs a recrudescence in the healthy limb when the whole process becomes repeated, though usually in a milder degree and covering a smaller period.

The course of the disease is prolonged and the prognosis is not bad. There is no immediate danger unless, as occasionally happens in ill-managed cases, a detached clot causes embolism in some vital organ. The remote prognosis will depend on the severity of the initial lesion and the care adopted in the management of the case; in bad cases and badly managed cases, the affected vessels become permanently damaged, there is a constant tendency for the swelling to recur on undue exertion and whenever the patient is run down, and there ever hovers the risk of some detached clot causing embolic manifestations.

SOME ELEMENTS OF NEUROLOGY (6

M. GERARD KELLY, CAPT., I.M.S.

Physician, General Hospital, Madras.

FRIEDREICH'S ATAXIA

This is characterized by—

1. A slow, clumsy ataxia ;
2. Absent knee-jerks ;
3. Positive Babinski's sign ;

and 4. Deformities.

Etiology :

1. Hereditary and familial.
2. Insidious onset : in early childhood before 6th year ; occasionally not until puberty.

Pathology :

1. Degeneration of the dorsal columns—
   (1) Loss of knee jerks—reflex arc gone ;
   (2) Dynamic ataxia—conscious muscle sense gone.
2. Degeneration often of the pyramidal tracts—Babinski's sign present.
3. Degeneration of the cerebellar tracts—
   (1) Static ataxia ;
   (2) Cerebellar signs.

Symptoms :

1. " The child was never strong on his legs, he could never run and play ".
2. The legs are affected first whereas in progressive muscular atrophy and syringomyelia the arms are affected first.
3. Ataxy is always the first sign to appear.

I. Ataxic Paraplegia :

(Dorsal columns and Pyramidal tracts)

(1) Ataxic gait ;
(2) Romberg's sign ;
(3) Absent knee-jerks ;
(4) Babinski's sign invariably present.

II. Charcot's triad :

(Cerebellar tracts)

III. Deformities :

Pes cavus and spinal curvature ; due to spasticity or loss of tone.

IV. Sensation :

Usually normal ; sometimes impaired.

Vibration sense may be lost.

V. Sphincters :

Normal.

Differential Diagnosis :

1. *Juvenile Tabes*— (1) Lightning pains ;
   (2) Eye signs ;
   (3) Charcots's triad absent.
2. *Disseminated sclerosis*— (1) Never occurs in childhood ;
   (2) The deep reflexes are never lost ;
   (3) Spinal deformity does not occur.

Treatment :

1. Tonics ;
2. Fraenkel's exercises.

CEREBRAL DIPLEGIA

This is due to agenesis of the cortical cells.

There are *two varieties* :

1. Spastic diplegia : an upper-motor-neurone lesion of all four limbs.

2. Spastic paraplegia or Little's disease: an upper-motor-neurone lesion of legs only.

3. There are other rare forms of diplegia also.

Symptoms:

1. Upper-motor-neurone lesion of limbs with crossed-leg (scissor-leg) progression due to the very marked spasticity.

2. Mental deterioration, especially in spastic diplegia.

Prognosis:

1. Spastic diplegia tends towards imbecility.

2. Spastic paraplegics—cases of Little's disease—are sometimes nearly normal both physically and mentally by the age of puberty. Some cases tend to remain stationary and non-progressive.

MYASTHENIA GRAVIS

(Asthenic Bulbar Paralysis)

Myasthenia gravis is a chronic malady of adult life, characterized by—

1. A variable paralysis of muscles which is produced or rapidly increased by exertion and which tends to disappear slowly during rest.

2. A permanent paralysis which—

(1) Succeeds the variable paralysis;

(2) Shows no improvement with rest;

(3) May be very local in distribution;

and (4) May be associated with atrophy of the muscles.

3. The affected muscles on strong Faradisation soon cease to respond to Faradism but remain excitable to Galvanism: Erb's myasthenic reaction.

Etiology:

1. It is clinically associated with Grave's disease, the ophthalmoplegias of which closely resemble those of myasthenia gravis.

2. A condition identical with myasthenia may occur in myeloma.

Pathology:

1. A large persistent thymus or thymic rests have been found.

2. Lymphorrhages in the muscles.

3. There is probably a defect of the motor end organs, such as is produced by curare.

Symptoms:

1. A variable paralysis with fatigue phenomena: the school-master—the house-maid—the theatre-goer.

2. Eye signs:

(1) Diplopia;

(2) Ptosis is the rule;

(3) Sometimes retraction of the lids with Von Graef's and Stellwag's signs, etc;

(4) Nystagmoid movements;

(5) An ocular paralysis which is well each morning on walking and which develops in the course of each day, is always due to myasthenia;

(6) Permanent nuclear ocular paralysis follows the variable paralysis.

3. *Myasthenic Facies*:

(1) Lack of facial expression;

(2) Inability to close or pucker the eyes and mouth;

and (3) A peculiar weak nasal smile.

4. The motionless and slightly dysarthria speech is unmistakable.

5. Asthenic bulbar paralysis supervenes.

6. Wasting occurs only in muscles that are markedly and permenently paralysed.

7. The reflexes are normal;

8. Also sensation;

9. And sphincters.

Differential Diagnosis

1. The different types of bulbar paralysis.

2. Encephalitis lethargica.

JOURNAL OF SOUTH INDIAN MEDICINE

JUNE 1936

THE HONORARY MEDICAL SCHEME

In our review of the last Triennial Report on the administration of the civil hospitals and dispensaries we made a passing reference to the appointment of honorary medical officers to the State hospitals and dispensaries. Some recent events affecting this scheme have produced some misgivings in the minds of the profession and the public. We therefore propose to trace the beginnings and course of this scheme.

The scheme of appointing suitably qualified practitioners on the staff of the State hospitals was seriously inaugurated at the end of 1923. The scheme has therefore been in existence for a little over a dozen years. Within this short period, varying counsels have prevailed among the advisers of the Government. When it was decided to initiate the scheme, the Presidency hospitals were selected for the experiment since it was natural to presume that suitable medical practitioners would be more easily available in the City. The then Surgeon-General and the Senior Medical Officers who were consulted by him were very particular that the honorary officers appointed to the teaching hospitals should take an effective part in the imparting of clinical instruction to the medical students working in those hospitals. And in the rules that were framed in the beginning to govern these appointments, the duty of imparting clinical instruction is specifically laid down. Further, the hours of attendance of honorary medical officers in the teaching hospitals is even now more or less dependent on the hours of clinical instruction.

A few years passed and the public were already impressed with the usefulness of the honorary scheme, and in their enthusiasm sought for its extension. A question was asked in the Legislative Council about the success of the scheme and the possibility of its extension. This innocent question brought forth the amazing reply that honorary medical officers were unsuitable for teaching hospitals. The public were perhaps not aware that between then and the

introduction of the scheme, there was a change in the head of the administration of the medical department. And with the change in the personnel there was a change in the view. A perusal of the proceedings of the local Legislative Council of those days would certainly be instructive. The reply was long, and the view expressed therein was sought to be fortified with extracts from the opinions of the superintendents of the various hospitals on the working of the scheme. Their opinions were specially asked for before the reply to the question was published on the floor of the Legislative Council. The view of the Surgeon-General was so unexpected that the Government agreed to have the whole question considered by a committee of official and non-official medical men and members of the Legislative Council. We are not aware of the findings of that committee, nor of the Government's decision. But the scheme was left alone and the honorary medical officers continued to function. When Major-General Megaw became the Surgeon-General of the Province, things seemed to brighten up for the honorary scheme. A number of new honorary appointments were made. He was not satisfied with that. He did not consider that honorary officers were unsuitable for teaching hospitals. Far from it, whenever there was an opportunity and suitable men were available, honorary medical officers were appointed to act as lecturers and professors in the schools and colleges attached to the hospitals. And at the farewell dinner given to him by the profession in Madras he spoke appreciatively of the honorary scheme and was confident that it would grow rapidly under the guidance of his successor.

Major-General Megaw was fully justified in that confidence. Major-General Sprawson who succeeded him as the Surgeon-General with the Government of Madras evinced a very great interest in the honorary scheme. His influential association with the new University of Lucknow had made him familiar with the successful possibilities of "direct" appointments to the faculty of Medicine. He did not subscribe to the common view that only the existing services could supply suitable candidates to fill up vacancies in the medical department, academic or administrative. With his distinct academic flair, he sought more and more to develop an academic atmos-

phere in the teaching institutions. In continuance of the policy of his predecessor he recommended to the Government that honorary medical officers attached to the teaching hospitals should automatically be put on the roll of the staff of the colleges and schools concerned. He further recommended that some at least of the senior officers who were engaged in important teaching should be paid a teaching allowance. A few of these recommendations were certainly not very much to the liking of some members in the services, though the Government of the day approved of most of his suggestions regarding the honorary scheme.

With the advent of the present Surgeon-General, Sir Frank Connor, there was some misgiving in the minds of the honorary officers and the independent profession. He came from Calcutta, and its atmosphere was not known to be very friendly to the honorary scheme. In fact, early after he assumed office it was given out that the new Surgeon-General was not as enthusiastic about the honorary scheme as his immediate predecessor. But it soon became evident that the nervousness of the profession was unnecessary, and that the vague rumours were groundless. Vacancies in the honorary services have been promptly filled up, and more practitioners have been given opportunities to serve in the various hospitals. The appointment of the honorary surgeon to the Government General Hospital is noteworthy in this connection. This post was created when the honorary scheme was inaugurated, but the actual appointment was made only about a year ago. Further, in the day to day administration, District Medical Officers and superintendents of hospitals have had occasion to complain against junior honorary officers, while the latter had sometimes to represent to the Surgeon-General some of their difficulties and disadvantages. On all these occasions we have very good reasons to believe that the head of the administration has been sympathetic and kind to the honorary officers concerned.

We are therefore surprised at some of the recent orders of the Government. The Government have sometime ago issued orders to reclassify the grade of junior honorary medical officers, especially the Licentiate members. Secondly, the Government seem to be

undecided in their mind about one of the recommendations of the last committee appointed to consider the rules governing the honorary scheme and the possibilities of its extension. The committee unanimously recommended that honorary officers attached to the teaching hospitals should also be eligible for appointment to professorships and lectureships. The Government seem be to unwilling to accept even this modest suggestion. Thirdly, the Government have intrepreted literally the rule that they might terminate the services of honorary officers after giving three months' notice without assigning reasons. None of these decisions might strike the superficial observer as serious change of policy. But they interefere so fundamentally with the spirit of the honorary scheme. We therefore propose to deal with these questions in detail in a subsequent issue.

GOVERNMENT OF MADRAS

EDUCATION AND PUBLIC HEALTH DEPARTMENT

(Public Health)

Order—No. 639, P.H., dated 5th June 1936.

The Committee appointed by Government in G.O. No. 554, P.H., dated 10th March 1933, to consider the question of the further extension of the scheme of employment of Honorary Medical Officers in Government Medical Institutions in the Presidency, recommended, among other things, that an honorary medical officer of a hospital attached to a teaching institution should be considered also for appointment to the academic post in his speciality when such a post becomes vacant and that he should be paid for such teaching work, if appointed. The Government have now carefully examined the Committee's proposal and have decided that it should be deferred for the present.

(By order of the Government, Ministry of Medical Administration and Public Works)

C. H. MASTERMAN,
Secretary to Government.

JOURNAL OF SOUTH INDIAN MEDICINE

(Incorporating the Bulletin of the South Indian Medical Union).

VOL. III JULY 1936 No. 7

SOME ELEMENTS OF NEUROLOGY (7)

M. GERARD KELLEY, CAPT., I.M.S.

Physician, General Hospital, Madras.

THE EXTRA-PYRAMIDAL SYSTEM

The Extra-Pyramidal System consists of—

1. The Basal Ganglia—(1) Optic Thalmus,
   and (2) Corpus Striatum;
2. Other masses of grey matter of the Mid-brain:
   (1) Red Nucleus,
   (2) Substantia Nigra,
   and (3) Sub-thalamic body;
3. Deiter's Nucleus in the Pons;

and 4. Three of the important Extra-Pyramidal Tracts—
(1) Rubro-Spinal,
(2) Tecto-Spinal,
and (3) Vestibulo-Spinal—

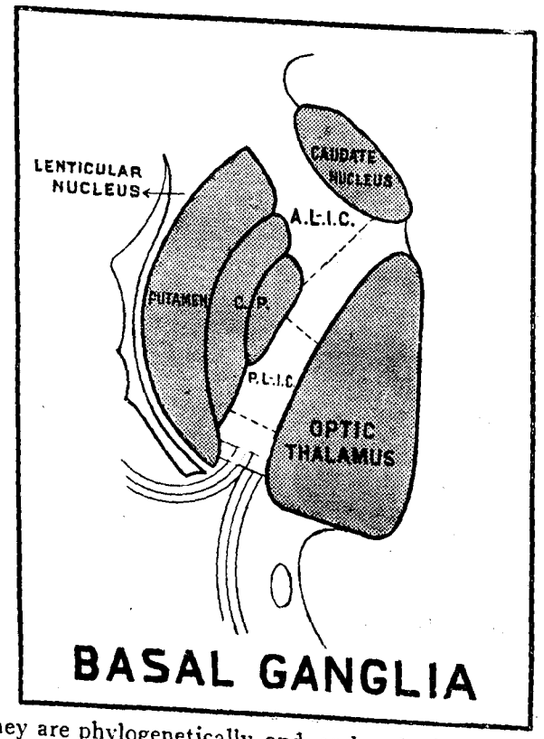
to the Anterior Horn Cells.

5. The Cerebellum and its connections are closely linked to the Extra-Pyramidal System.

The Basal Ganglia

1. Optic Thalamus; *2. Corpus Striatum.*

1. The basal ganglia are masses of grey matter more or less completely embedded in the substance of the brain.



BASAL GANGLIA

2. They are phylogenetically and embryologically much older than the cerebral cortex.

3. In the lower vertebrates they represent the higher motor and sensory centres of the brain—the *optic thalamus* constituting the reception centre for afferent impulses, and the *corpus striatum* the centre for regulating afferent impulses.

4. In the higher vertebrates these centres become subordinated to the cerebral cortex.

5. Their cells act as cell-stations for both afferent and efferent fibres of the cerebral cortex.

The Optic Thalamus :

1. The essential organ of the thalamus is a centre for recognition of certain crude sensations.

2. The sensory cortex is responsible for the finer discriminative aspects of sensation.

The Corpus Striatum :

1. The Corpus Striatum consists of—

(1) The Caudate Nucleus,

and (2) The Lenticular Nucleus.

2. The Lenticular Nucleus is divided into—

(1) Putumen—outer portion,

(2) Globus pallidus—inner portion.

3. The corpus striatum receives all its afferent impulses from the optic thalamus.

4. The efferent fibres arise solely from the globus pallidus and run as the ansa lenticularis to the sub-thalamic region to end in the substantia nigra and red nucleus.

The Neo-striatum—Putumen and Caudate Nucleus :

1. Histologically the cells of the putamen resemble those of the caudate nucleus.

2. They, therefore, constitute the neo-striate or non-pallidal portion of the corpus striatum.

3. The neo-striate portion is sensory in type.

4. It may exert a restraining influence on the much older globus pallidus which is motor in type, and whose cells bear no resemblance to those of neo-striate portion.

The Paleo-striatum or Globus Pallidus :

1. The globus pallidus is the oldest part of the corpus striatum.

2. It consists of large multipolar and pyramidal shaped cells somewhat resembling anterior horn cells.

3. It is motor in character and inhibits postural tone and tonic activities.

4. When the restraining influence of the globus pallidus is removed, we get another release phenomenon, namely, the setting free of the tone path, with the result that there is great increase in tone all over the body.

5. Striatal rigidity, however, differs greatly from decerebrate rigidity and, therefore, cannot be due to the release of the tone path in quite the same way.

STRIATAL RIGIDITY:

1. Striatal rigidity affects mainly the big muscle masses and the proximal parts of the limbs.

2. It affects agonists and antagonists—flexors and extensors—alike.

3. Though the rigidity is almost universal in distribution, its incidence is more marked on certain muscle groups—

(1) Sternomastoid,

(2) Biceps,

(3) Knee-flexors,

(4) Facial muscles—

with the result that a characteristic flexed attitude is produced.

4. The posture is not modified by altering the position of the head.

5. No signs of an upper-motor-neurone lesion will be present.

TYPES OF STRIATAL RIGIDITY:

1. Cog-wheel rigidity elicited by rapid passive movement=rigidity and tremor.

2. Pipe-stem rigidity=the same resistance in flexion and extension.

The Red Nucleus

1. Each red nucleus receives fibres—

(1) From the opposite cerebellar cortex by way of the superior peduncles,

and (2) From the globus pallidus by the ansa lenticularis.

2. The rubro-spinal tract takes origin in the red nucleus.

DECEREBRATE RIGIDITY:

1. The higher parts of the brain down as far as the red nucleus tend to depress the tone reflex thereby keeping the limbs in the normal position and maintaining a normal state of tone in the muscles.

2. In lesions of

(1) The red nucleus,

(2) The rubro-spinal tracts,

or (3) The pyramidal tracts,

the tone reflex is released from this controlling influence, normal postural tone is lost, and the decontrolled action, especially of Deiter's nucleus, brings about exaggerated tone in the antigravity muscles which are normally used in maintaining the body against gravity=decerebrate rigidity.

Substantia Nigra

Paralysis agitans is believed to be due to a primary neuronic degeneration of the cells of the substantia nigra.

The Cerebellum and Deiter's Nucleus

These are the centres for tone.

INVOLUNTARY MOVEMENTS

1. Three main types of involuntary movements occur in voluntary musculature.

(1) Tremor;

(2) Chorea;

(3) Athetosis.

2. They are never present when the muscles are completely paralysed, *i.e.*, there must be relative integrity of the pyramidal tracts.

3. They result from lesions of the extra-pyramidal paths when the pyramidal pathways are relatively intact.

Tremor

1. Tremor consists of fairly regular, rhythmical, alternating contraction of a muscle group or groups and their antagonists.

2. Types of Tremor—(1) Static or Striatal;

(2) Intention or Cerebellar;

(3) Senile;

(4) Cerebral;

(5) Toxic;

(6) Functional.

3. Note—(1) Rate,

(2) Amplitude,

and (3) Distribution of tremor;

and (4) Age of patient.

Static or Striatal Tremor:

This is due to the released activity of a supra-spinal centre situated in the mid-brain.

Chorea and Athetosis

Chorea: irregular and spasmodic involuntary movements of groups of muscles of a simple nature occurring during rest and also superimposed upon voluntary movements, which they render inco-ordinate.

Athetosis: involuntary movements of a more complicated nature, but quite purposeless—writhing, grimacing type—slower and more stereotyped than the movements of chorea.

1. It is obvious that the two conditions must have a very similar iology.

2. It is surprising that they do not alternate more frequently in the me person, when one kind is present, *e.g.*, chorea occurs in rheumatism t rarely athetosis.

3. Nevertheless, the two are sometimes met with in the same person.

EORIES OF CAUSATION:

1. Lesion in the neo-striate.

2. Lesion of cerebello-cortical fibres.

(1) *Lesion in the Neo-striate*:

1. The globus pallidus, being motor in type, has the function of performing certain involuntary movements.

2. The neo-striate normally has a restraining influence on the globus pallidus and prevents these involuntary movements from occurring.

3. In lesions of the neo-striate, the globus pallidus is set free and so originates the involuntary movements.

(2) *Lesion of the cerebello-cortical fibres*:

1. These involuntary movements originate in the cerebral cortex.

2. They use the pyramidal tracts for their outgoing paths.

3. Neither chorea nor athetosis can occur when both pyramidal tracts are destroyed.

4. But athetosis is common in old hemiplegias, *i.e.*, when the lesion of the pyramidal tract is only partial.

5. An internal capsular lesion causing hemiplegia produces athetosis not by damaging the pyramidal tract but by destroying other fibres in the region of the thalamus passing from the opposite cerebellum towards the cerebral cortex, namely, the cerebello-cortial fibres.

6. The cerebello-cortical fibres exert a restraining influence on the cerebral cortex in much the same way as the neo-striate does on the globus pallidus.

7. When they are destroyed the cerebral cortex is released and gives rise to involuntary movements.

Objection—These involuntary movements sometimes occur during sleep or descent of inhibition to the mid-brain.

PARKINSONISM

(EXTRA-PYRAMIDAL SYNDROME)

James Parkinson was an erudite English Physician practising at Hoxton. In 1817 he published his classical essay on the "Shaking" Palsy. He must have been a very exceptional clinician, for there are few features observed now after over 100 years which escaped his attention.

In 1917 Von Economo practising in Vienna described a small outbreak of Encephalitis Lethargica, the symptoms of which are roughly the same, And thus was introduced the term 'Parkinsonism' which indicates a *Syndrome* and not a *Disease*.

1. The lesion is in the extra-pyramidal pathway in the region of the basal ganglia—putamen, globus pallidus, substantia nigra and red nucleus.

2. The symptoms are roughly the same.

3. But there are different varieties according to the nature of the lesion—

(1) *Idiopathic*—Paralysis Agitans;

(2) *Inflammatory*—Encephalitis Lethargica and Post-Encephalitic Sequel;

(3) *Arterio-sclerotic*;

(4) Syphilitic;

(5) Neoplastic;

(6) Senile rigidity in extreme old age;

(7) Hepato-lenticular degeneration;

(8) Manganese poisoning;

(9) Parkinsonism associated with epilepsy.

4. The Syndrome of Parkinsonism indicates the anatomical diagnosis.

5. The etiological diagnosis must be determined by the additional evidence presented.

Four Cardinal Symptoms:

1. Rigidity;
2. Movement disorders;
3. Tremors;
4. Weakness.

I. RIGIDITY; II. MOVEMENT DISORDERS

(See Striatal Rigidity)

1. Rigidity of the facial muscles.
2. Rigidity of the muscles of the larynx, tongue and lips.

3. Rigidity of the neck muscles.
4. Stiffness of the trunk muscles.
5. The gait.
6. Rigidity in the hand.

1. *Rigidity of the facial muscles:*

1. The face assumes a fixed, anxious, mask-like expression.

2. There is an absence of wrinkling of the forehead.

3. The eyes look forward with a fixed reptilian stare owing to infrequent blinking.

4. In the parkinsonian facies, it is not so much that there is loss of expression but rather that there is a lack of ease (poverty of movement) in passing from one expression to another, so that the normal play of emotions is not exteriorized.

2. *Rigidity of the muscles of the larynx, tongue and lips*:

1. The voice loses its inflexions.
2. The speech becomes slurred and monotonous.
3. But there is no defect of articulation.

3. *Rigidity of the neck muscles:*

1. The patient carries his head and neck in one piece with his trunk as if he were a statue.

2. He never inclines or raises it in the customary manner.

3. If he turns round to look at anything he tends to move the whole trunk round with the head.

4. In looking sharply to one side the eyes move before the head, whereas, under normal circumstances, the coarse adjustment of this movement is done just by the neck muscles and the fine adjustment subsequently by the eye muscles.

4. *Stiffness of the trunk muscles and of the upper extremities*:

1. Stiffness of the trunk muscles gives a stooping attitude with the head inclined forward.

2. Stiffness of the muscles of the upper extremities causes the shoulders to be rounded and the arms carried with the elbow semiflexed and pressed into the sides.

3. Poverty of movement—*the parkinsonian stays put*—he does no move about in his chair, cross and uncross his legs, twist his monocle, cares his nose, or stroke his hair.

5. The gait:

1. The gait is deprived of spring and suppleness on account of rigidity of muscles.

2. He walks like a statue with a slow shuffling gait—all movements are slow (bradykinesia), irregular or jerky and limited.

3. There is a marked absence of the movements of co-operation—the arms are not swung on walking.

4. He takes small gliding steps, displacing his centre of gravity as little as possible.

5. *Propulsion*, *retropulsion* and *latero-pulsion*—if he trips or is pushed, his centre of gravity is displaced and he often has a difficulty in regaining it and so he continues the movement of necessity, until he falls or is stopped by some object by which he can arrest himself and restore his balance.

6. *Festination* is the quickening of the pace sometimes seen in this attempt to overtake the displaced centre of gravity.

6. Rigidity in the hand:

1. The interossei are most affected and so the hand tends to assume the "interosseal position":

(*a*) Fingers pressed together;

(*b*) Thumb adducted;

(*c*) Metacarpo-phalangeal joints flexed;

(*d*) Intercarpal-phalangeal joints extended.

2. From the rigidity of the hand, the handwriting becomes small, cramped and tremulous and is not in a straight line.

III. TREMOR

1. *Rate*: is about 6 per second.

2. *Amplitude*: is $\frac{1}{8}$ to $\frac{1}{4}$ inch.

3. *Rhythm*:

(1) It is a regular rhythmical contraction of the muscles alternating with the opposing group.

(2) The rhythmicity is seen in the pill-rolling, drum-tapping, bread-crumbling or cigarette-rolling movements, and pronator-supinator tremor.

4. *Distribution*—

(1) It usually commences in the hand and forearm and is not conspicuous in other situations.

(2) But it may be seen in the face, tongue, jaw, neck and feet.

(3) In rare cases it may be universal.

(4) It is always distributed where rigidity is least marked.

(5) In the eyelids there is lid retraction and blepharoclonus or rapid blinking on asking the patient to close the eyes tightly—he is unable to maintain a steady contraction.

(6) In the eyeballs, punctate jerky movements.

(7) The tongue is tremulous on protrusion.

(8) In the arms there is tremor, clumsiness and difficulty in performing fine movements.

(9) Tremor of the legs.

5. *Tremor is present*—

(1) At rest;

(2) Continues when the limb is supported.

6. *Tremor ceases*—

1. On voluntary movement;

2. On alteration of position;

3. On painful stimulus;

4. During sleep.

7. *Tremor increases*—

With emotion, excitement, self-consciousness.

8. *Tremor is little marked or absent*—

Where rigidity is very conspicuous.

Conversely, when tremor is universal, rigidity is less—antagonism between tremor and rigidity.

Differential Diagnosis :

1. Idiopathic parkinsonism—paralysis agitans ;
2. Post-encephalitic parkinsonism ;
3. Arterio-sclerotic parkinsonism ;
4. Syphilitic parkinsonism ;
5. Neoplastic parkinsonism.

I. Paralysis Agitans :

1. There are three diagnostic points—

i. Facies, attitude and gait.

ii. The rhythmic rolling tremor which continues during rest.

iii. The absence of any signs of organic disease of the central nervous system.

2. In "hemiplegic" paralysis agitans—

i. The aspect is parkinsonian ;

and ii. There are no signs of an upper-motor-neurone lesion.

3. The onset is gradual between 40 and 60 without any history of acute illness.

4. Tremor is much more prominent than rigidity.

5. Sialorrhoea is not present.

6. There are no definite mental symptoms apart from simple depression.

7. Paralysis agitans tends to a progressive downward course.

II. Post-encephalitic Parkinsonism.

1. There is a history of preceding febrile illness.

2. The patient is under 40.

3. There is greasiness of the skin.
4. Sialhorroea.
5. Oculo-gyric crises.
6. Involuntary movements.
7. Sleep abnormalities.
8. Respiratory disturbances.
9. Gait abnormalities.
10. Mental changes.
11. Trophic disturbances.

Oculo-gyric Crises :

1. These are recurring attacks of tonic conjugate deviation of the eyes, most commonly upwards.

2. They last from a few seconds to 4 hours or more.

3. They may occur frequently or at intervals of several days.

4. Any attempt to move the eyes downwards is accompanied by violent clonic movements of the eyelids.

5. Impulsive thoughts may occur during the crisis.

6. They may proceed to torsion spasm of the trunk.

7. Oculo-gyric crises occur only in encephalitis lethargica and may constitute, with some slight facial parkinsonism, the sole sequel of this malady.

Involuntary Movements :

1. Myoclonic—1. The corner of the mouth.
   2. The deltoid.
   3. One rectus.
2. Choreiform.

or 3. Torsion type.

Sleep Abnormalities :

1. Lethargy ;
2. Insomnia ;
3. Inversion of the sleep rhythm.

Respiratory Disturbances :

1. Disorders of respiratory rate—tachypnoea, causing tetany.

2. Respiratory dys-rhythmias—apnoeic pauses and breath-holding spells.

3. Respiratory tics—yawning, sniffing, coughing and spitting.

Gait abnormalities:

1. Deformities of the feet ;

and 2. Curious bizarre attitudes not unlike some of the attitudes in torsion spasm.

Mental changes :

1. In adults—loss of emotion, periods of extreme depression and excitement.

2. In children—" behaviour disorders " : " *apache* children " ;

1. Deceitfulness, cruelty, stealing and sexual offences.

2. Their acts seem to be committed impulsively and are not pre-meditated.

3. Self-mutilation may occur.

Trophic disturbances :

1. Loss of weight ;

or 2. Adiposity with polyuria and polydipsia like a pituitary tumour ;

3. Hyper-secretion of sweat and sebum.

III. Arterio-sclerotic Parkinsonism :

This is diagnosed by :

1. The concomitant vascular changes ;

2. The pronounced rigidity ;

3. The absence of tremor ;

4 The psychical changes—memory defects and general deterioration ;

5. The frequency of catatonia ;

and 6. The ***marche a petits pas*** (shuffling gait).

IV. Syphilitic Parkinsonism :

1. There are the features of parkinsonism in addition to

2. Those of a syphilitic lesion of the central nervous system—Argyll-Robertson pupils, absent tendon jerks, and a positive Wassermann reaction.

V. Neoplastic Parkinsonism :

1. Features of parkinsonism ;

and 2. Signs of a tumour in the basal ganglia.

Hepato-Lenticular Degeneration

(Kinnier Wilson)

1. Occurs between the ages of 7 and 27.

2. The primary and essential lesion is in the liver (Portal cirrhosis).

Symptoms :

1. Parkinsonism.

2. Involuntary movements due to lesion of neo-striate and liberated globus pallidus.

3. Symptoms of portal cirrhosis.

4. Green rim round edge of cornea.

Treatment

1. Hyoscyamine as Gelhyoscyamine for Rigidity.
2. Hyoscine or Genoscopalamine for Tremor.
3. Tinct. Belladonna m. 10—30 t. d. s.
4. Tinct. Stramonium m. 30—80 t. d. s.

R/

Sod. bromide	10 grains.
Hyoscine hydrobromide	1/200 grain.
Tinct. belladonna	10 minims.
Tinct. hyoscyamus	10 minims.
Aq. chloroform to make	½ fluid ounce.

Sig.—Half an ounce thrice daily after food.

Push above to limits of tolerance—till dilation of pupils and dryness of throat occur.

NOTICE
THE MADRAS MEDICAL COUNCIL

"It is a grave breach of professional propriety for a medical practitioner to take any part in the manufacture, or sale of a proprietary food or medicine or to have his name in any way associated with it. Medical men should also be cautious how they lend themselves to the puffing of these articles."

(Extract from the Code of Medical Ethics—Section 22.)

ASSOCIATION NOTES

THE TINNEVELLY DISTRICT MEDICAL ASSOCIATION, PALAMCOTTAH.

(Affiliated to the Indian Medical Association).

The Tinnevelly District Medical Association held its monthly meeting at Palamcottah on Saturday the 23rd May 1936. Members numbering 54 including 5 lady doctors were present on the occasion.

Demonstrations in the Operation Theatre were given by Lieut.-Col. T. S. Shastry, I.M.S, from 9 A.M. to 12 Noon. The following interesting operations were demonstrated on the occasion :—

1. Right reduciable Inguinal Hernia—Fascial Suturing.

2. Ligature II, one side for Prostatic enlargement (or, Steinachs' Rejuvenation Operation).

3. Skin-grafting of a Motor Injury case.

4. Application of Plaster of Paris.

About 15 doctors attended the clinical demonstrations in the Thearte and wards. The following are some of the interesting clinical cases demonstrated to the members and explained to them by Lieut.-Col. T. S. Shastry, I.M.S :—

1. Radical Operation for Cancer of Breast—primary skin-grafting.

2. Partial Gastrectomy, and secondary Intestinal Obstruction and Laparotomy.

At 4-30 P.M., the Tea-party began and the meeting started under the Presidentship of Lieut.-Col. T. S. Shastry, I.M.S., at 5-30 P M.

The following Resolution regarding the demise of Dr. M. A. Ansari, M S., M.D. (Edin.), was moved by the President in a feeling speech, and passed, all the members standing :—

"Resolved to place on record the Association's keen sorrow at the sudden and untimely demise of Dr. M. A. Ansari, M.S., M.D. (Edin.), who

was one of the ablest Physicians and Surgeons of the Country and a Vice-President of the Indian Medical Association."

The Secretary read the minutes of the monthly meeting held at Tiruchendur on 25 4 36, which were passed. The Resolution of Dr. V. Sankarasubban, M.B B.S., regarding the subscription was then taken up; and after keen and exhaustive discussion, the resolution was put to vote —six voted for and thirty-four against it and so it was declared lost. The subscription therefore stands at one Rupee per mensem, and the President requested the members to pay up now without hesitation all their arrears.

Dr. Rama Rao's Bill on College of Physicians and the Rules of the Tamil Districts' Medical Federation were postponed to the next meeting.

Dr. T. K Nilakanta Ayyar, and Dr. Miss Lazarus read very interesting notes of the cases which were operated on by Lieut.-Col. T. S. Shastry, I.M S., *e.g.* Mastoid abscess, Head Injury—Fracture of Middle Fossa—Decompression Operation.

Dr. K. R. Venkoba Rao, L.M. & S., B.S. Sc., demonstrated to the members the full apparatus for testing Blood-sugar made by Carl Zeis. Dr. N. Isaac read an interesting paper on 'Tobacco and its Medical aspects.'

Dr. P. R. Venkatarama Ayyar, M.B & C M., District Medical Officer, Nellore, paid a visit to the meeting. He was welcomed heartily by the President and warmly introduced to the Association as President of the Nellore District Medical Association and as a Tinnevellian The Guest replied very cordially commending very highly the career and work of the Tinnevelly District Medical Association.

Thereafter Lieut.-Col. T. S. Shastry, I.MS., summed up the salient and important features of all the cases and addresses and clearly explained the useful measures to take as first aid in Head Injuries and pointed out the salient points for diagnosing Mastoid disease. He also stressed the need for every Doctor writing notes of his examination of his cases, of the details o his treatment, as this clinical record will serve as his own teacher in futur and as the storehouse which he could place at the disposal of the Distric Medical Association for the advancement of Professional knowledge.

Thereafter an excellent Dinner brought the happy gathering to a clos at 11 P.M.

TWO CASES OF DYSENTERY

1. A Madrasi patient aged about 45 came to me for treatment of dysentery. He had frequent stools mixed with mucus and blood. The temperature was 101° and the patient complained of severe pain in the abdomen and tenesmus. He was first given anti-dysenteric serum 20 c.c. but as no improvement followed in his condition I started him only on Bacte-Dysenteric-Phage A. F. D. One ampoule of 2 c.c. in half a glass of Vichy water was administered every 3 hours. The next day blood and mucus stopped. Pain in the abdomen and tenesmus disappeared. On the third day the patient was perfectly free from the trouble and he passed well formed stools.

2. A child 2½ years old was brought to me suffering from high fever with diarrhœa. The stools were mixed with mucus and blood. The complaint was of eight days' duration during which time I persume he was treated elsewhere. The condition had become very grave I at once started him on Bacte-Dysenteric-Phage as I believed he must have taken all sorts of other treatments during those eight deys. In all 10 ampoules of 2 c.c. were given, the intervals between the ampoules being 3 hours. The symptoms subsided in a remarkably short period. Fever disappeared, the stools become of normal colour and consistency in two days. The child was convalescent in 3 days' time.

From the Diary of a Medical Practitioner.

THE EAST GODAVARI DISTRICT MEDICAL ASSOCIATION, COCANADA.

Minutes of the meeting held at Amalapur on the 30th May 1936.

A meeting of the East Godavari District Medical Association took place at Amalapur on 30-5-1936. 31 members attended the meeting. After an enjoyable tea the proceedings of the meeting commenced with Captain J. S. McMillan, I.M.S., in the chair. Dr. A. Umapathi Mudaliar of Amalapur exhibited a very interesting case of Pulmonary stenosis in a boy aged 12. A case of Congenital syphilis in a child of 2 presenting bony prominences in the skull and another case of Malignant growth neck, probably Lymphadenoma, with pushing forward of the posterior wall of the pharynx and protrusion of one eye-ball. Dr. N. S. Reddi of Amalapur read a paper on Typhoid fever, Dr A. Perisastry of Amalapur one on Tetanus. There was dinner after which the guests dispersed.

THE TRICHINOPOLY DISTRICT MEDICAL ASSOCIATION.

(Registered).

A monthly meeting of the above Association was held at 5 p. m. on Saturday the 27th June '1936 in the E. R. High School, Teppakulam, Trichinopoly, when about 25 members were present, Dr. R. Srinivasan, of Kulitalai, was 'At Home' to the members and Capt. Sangham Lal, I.M.S., F.R.C.S., District Medical Officer, Trichinopoly, presided.

After the demonstration of a few X-Ray films and discussion of intersting cases Dr. R. Kalamegham delivered a lecture on 'Diphtheria'. Half a dozen members took part in the discussion that followed.

With a vote of thanks to the Chair, Host, and Lecturer of the evening the meeting terminated.

THE TINNEVELLY DISTRICT MEDICAL ASSOCIATION, PALAMCOTTAH.

(Affiliated to the Indian Medical Aseociation).

The Tinnevelly District Medical Association held its monthly meeting on Saturday the 27th June 1936 at Papanasam (Ananda Vilas) one of

At 6 P.M. a Health Exhibition was arranged at the Thirthapathy High School, Ambasamudram, which was opened by M. R Ry. M. D. T. Kumarasawmy Mudaliar Avl., President, District Board, Tinnevelly. Colonel Shastry presided and welcomed the large gathering and explained the need for a campaign on Nutrition and Diet, pointed out the serious defects in our dietary and pleaded for immediate reform as the first step in the prevention of disease. The President, District Board, expressed in a short speech that such exhibitions are very useful and beneficial to the masses The exhibits consisted of posters ralating to Cholera, Tuberculosis, Malaria, Small-pox, etc., and a lecture on Tuberculosis was delivered in Tamil by Dr. N. Lakshmanan, L. M. P. This was very much appreciated by the gathering which consisted mostly of citizens and school boys.

the most sacred places of the District, noted for wild scenery. Members numbering 43 including 2 lady doctors gathered from all parts of the District. All the members had an early bath at the Falls and had light refreshments.

The meeting began at 10 A M. under the Presidentship of Lieut-Col. T. S. Shastry, I.M.S. The Secretary read the minutes of the monthly meeting held at the Headquarters (Palamcottah) on 23rd May 1936, which were passed. Then Dr. Rama Rao's Bill on College of Physicians and Surgeons was taken up. The following resolution was passed unanimously :—

"The Tinnevelly District Medical Association considers that no useful purpose will be served by the institution of a College of Physicians and Surgeons"

The Draft Rules of the Tamil Districts' Medical Federation was read by the Secretary and after discussion the following resolution was passed unanimously :—

"The Tinnevelly District Medical Association approves of the idea and supports the organising of the Tamil Districts' Medical Federation."

Interesting case notes by Dr. P. Janardhana Rao, L. M. & S., on (1) Typhoid Fever complicated by cerebral and respiratory symptoms and aphasia, (2) Stabwound of chestwall with protrusion of omentum, (3) Early case of Leprosy with cracks and Keratosis of tips of fingers and toes were read. Colonel Shastry read case notes on a case of Myxoedema. There was a lively discussion on these cases. Dr. K. Padmanabha Ayyar, L.M.P., of Tenkasi, read an interesting paper on Constipation. After discussion Lieut.-Col. T. S. Shastry, I.M S., summed up the salient and important features of all the cases. Dr. Swaminatha Sastry, L. M & S., of Tichinopoly and Prof. Nilakanta Sastry of the Madras University were present on the occasion and M. R. Ry. M. D. T. Kumaraswamy Mudaliar Avl., the President, District Board, Tinnevelly, joined us about the end of the meeting.

After a hearty vote of thanks to Dr. P. Janardhana Rao, L. M. & S, and his co-workers the meeting dispersed, and all had an excellent dinner at 1 P.M. Thereafter enjoyable music at 3 P.M. by M. R. Ry. Venkateswara Ayyar Avl., B.A., B L., Vakil of Ambasamudram, brought the happy gathering to a close.

JOURNAL OF SOUTH INDIAN MEDICINE

NOTICE

CONTRIBUTIONS of medical interest—original articles case notes, clinical memoranda—are invited. They should be addressed to the EDITOR, JOURNAL OF SOUTH INDIAN MEDICINE, EGMORE, MADRAS, and should be accompanied by a stamped addressed envelope if their return is desired in case of non-publication.

Articles are accepted for publication on condition that they are contributed solely.

MANUSCRIPTS must be written clearly or preferably type-written, with double spacing, on one side of the paper only.

Books and pamphlets for review should be sent to the Editor, who will be pleased to receive also prints of which abstracts will be interest to the profession. Journals for exchange or review also should be addressed to the Publicity Officer.