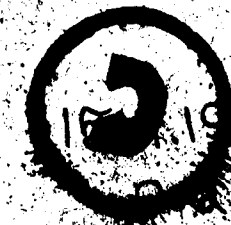


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PUERPERAL INFECTIONS (3)

T. S. PADMANABHAN, L.M. & S.

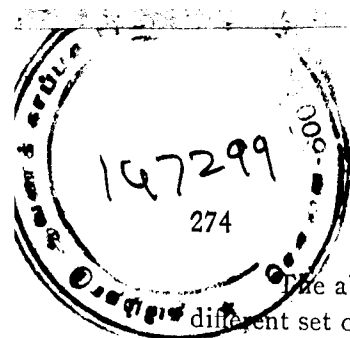
Badagara.

Diagnosis

The various symptom-complexes of infection met with in a puerperium may be the manifestations of an unalloyed puerperal infection or of some affection totally unconnected with the puerperium; between these two extremes there occur now and then cases where the apparent troubles are the results of a dire combination between a mild or a severe puerperal mischief and a superimposed extraneous disease.

FARQUHAR MURRAY¹ writes: "Always be on the look-out for sepsis. Influenza, pneumonia, debility, yellow atrophy of the liver and the breasts are often provided as an explanation for a sequence of events which are in reality due to septic processes. The headaches, shivers, malaise, and temperatures of a septic process are often attributed to influenza if it is prevalent. Chest conditions of septic origin are often manifested within three days of delivery, and to the inexperienced might be believed to be of accidental origin. Rapidity of pulse, with practically no other symptom than increasing weakness, can be due to hyperacute sepsis, but may be attributed to shock or haemorrhage. Jaundice, with only moderate pyrexia, or even in the absence of pyrexia, can be due to a hyperacute haemolytic streptococcal infection and not to hepatic derangement. Septic temperatures and engorgement of the breasts are common on the third day. Always be on your guard about sepsis; always suspect it; and always! look at the perineum and lochial discharges."

¹ British Medical Journal, 1933, Vol. I (Abstracted in The Indian Medical Gazette, December, 1933, p. 714.)



The above depicts one side of the picture. On the other side a different set of phenomena may be observed. Thus, a given case may be a fulminant condition of a flared up appendix very prone to be mistaken for a case of the less urgent *B. coli* excursions. Or, the case may be one of a let-in typhoid or a lit-up tubercle, both liable to be overlooked and so fraught with danger. Or, again, the case may be one of mixed infection as occurs when malaria complicates a morbid puerperium. Then, too, that ubiquitous worm may raise its head at a critical juncture in the course of a febrile puerperium and put the medical attendant on test and tension; for, the pain, vomiting and tenderness, especially when these are associated with a bloated abdomen and costive bowels, often do not admit of a timely diagnosis being made between worms and peritonitis in the absence of a microscope, and, on rare occasions, even in its presence.

Verily, under such circumstances, to clinch the diagnosis in a case of puerperal fever is often extremely difficult; yet, in no other instance, is a correct diagnosis so essential for happy treatment and brilliant results. An intelligent sifting after a patient hearing, a thorough examination on all available lines, a comprehensive survey of the various possibilities and a diligent filtering of the evidence—all these together only will enable one to arrive at a correct conclusion.

The diagnosis of puerperal infections may perhaps be best discussed under two main heads: (1) Differential diagnosis of puerperal infections with general symptoms predominating; (2) Differential diagnosis of puerperal infections with local symptoms predominating.

DIFFERENTIAL DIAGNOSIS OF PUERPERAL INFECTIONS WITH GENERAL SYMPTOMS PREDOMINATING

Apart from the exanthemata and the epidemics that may be prevalent at the moment, the fevers that generally disturb an otherwise normal puerperium and so require differentiation from the distinct variety are malaria, typhoid, influenza, pneumonia, and tubercle, and to a lesser extent, rheumatic fever, scarlet-fever and kala-azar.

The *exanthemata* and the *epidemics* can offer no great difficulty provided their presence is felt and remembered.

Malaria in the Tropics is always a sore point in the diagnosis of fevers, as it is almost endemic in many areas and may manifest itself in diverse forms. In the puerperium, septicaemia and, more so, *B. coli* infections are often exceedingly difficult to be clinically distinguished from malaria

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especially when the latter takes a quotidian course, or when the triple syndrome of rigor, fever and sweats tends to become irregular as in certain instances of malignant tertian cases. Fortunately, a case of malaria that is both quinine-resistant and refuses to show the parasites under the microscope is extremely rare, indeed, almost unknown. Further, a healthy uterine interior coupled with an arrest of all secretions in septicaemia, and the condition of the urine in *B. coli* affections will differentiate each from malaria.

The *enteric* group of fevers may offer real difficulties, especially during epidemics, in spite of their first-week four S's (Step-like temperature, Slow pulse, Spots on the abdomen and Spleen enlargement), and the later Widal reaction. The onset and the early course in sapraemias closely resemble those in the typho-paratyphoids: an insidious onset, a step-like temperature, intense headache, coated tongue, caecal gurglings, and lochial anomalies being common to both, while the rose-spots and the splenic enlargement are never constant in the enteric fevers. Clinically, the slow pulse is the only distinguishing feature between the two, although in a few cases early dysuria and delayed micturition may further help and point to the fever being more one of the typhoid group than of sapraemia. In the majority of cases, a careful blood-count and possibly an early blood-culture only will clinch the diagnosis; in typhoidal fevers, the former will show a relative rise in the lymphocytes without leucocytosis and the latter will evidence the bacilli, while in sapraemia the result will be different in either case. In many cases, however, especially in general practice, the fever becomes diagnosed on the 10th or the 12th day by the temperature coming down to normal on, or continuing beyond, that date. Frank septicaemia should be easy of differentiation from typhoid. Rigors which are constant in septicaemia are unknown in typhoid, except perhaps in the rare presence of a cholangitis or cholecystitis that may occasionally arise in a later stage of the disease, by which time, however, the fever will have been defined. Head-ache, a common initial symptom in typhoid, is rare in septicaemia. Further, an acute onset, great swings in temperature, a progressively rapid and feeble pulse, early brain symptoms, quick emaciation, a speedy grave general condition, euphoria, and, above all, a galloping nature of the disease—the whole course rarely exceeding 3 to 6 days—are all characteristic of septicaemia, and are the exact reverse of the features obtained in ordinary typhoid. In yet doubtful cases, the blood-count will clinch the diagnosis; whereas in typhoid there will be a lymphocytosis without increase in the total leucocytes, in septicaemia will be found an increase in the total leucocytes with particular rise in the polymorphonuclears. Little needs be

mentioned on the nervous types of typhoid; they are very rare, seldom suspected in the puerperium, and, even if suspected, the conditions obtained do not warrant an indulgence in the fineness of diagnosis; the case will have to be, and will be, treated as an hyperacute sepsis, due regard being paid to the life of the patient and the reputation of the attendant, and without indulging in heroic measures. Cases that start sapraemia-like and run an atypical course will offer very great difficulties, and more so if their onset had been some time before confinement. In such instances, one has to depend entirely on the blood count and the blood-culture, and, for consolation, the Widal test, as this last, with all its charm, often tends more to confuse than confirm the clearer clinical findings, and in many cases, becomes inclined to play to the tune of 'locking the gates'as by the time the results of the Widal are known, the patient has either recovered or succumbed. *B. coli* affections in puerperium, if not diagnosed early and treated properly, tend to become chronic, and in the absence of the grave signs of sepsis, are often dubbed as typhoid, as is generally done in all prolonged symptomless fevers. But care not to omit the urine examination in all its details will avoid the error; the picture of *B. coli* urine is typical, and has been already discussed under clinical manifestations. Besides, in a typical case of *B. coli* affection, the temperature will be characteristic; the pulse will be rapid; rigors may occur; the eyes, the expression, the tongue and the general condition may not indicate severe toxæmia; the patient will not feel ill for all her fever; dysuria and loin-pains may be extreme; and the blood will show a distinct leucocytosis. The diagnosis between the nephritic type of typhoid and the fulminant type of *B. coli* pyelitis may perhaps be possible at autopsy, but not in life; both are luckily rare.

Influenza, due to intrinsic as well as extrinsic vagaries, is often made the sinner in acute fevers with no definite signs. From a practical point of view it may require no great softening of one's brain, as its treatment is entirely symptomatic. Academically, an epidemic or pandemic prevalence; a sudden high temperature with chill or rigor and severe aching pains all over, early and peculiarly extreme prostration, speedy crisis with or without profuse sweating and a short duration of 1 to 3 days or at the most a week—in the general type; dyspnoea and respiratory distress out of proportion to the pulmonic signs which in some cases may be marked over but a small patch at the base or the apex showing roughened breathing and fine crepitations or sticking rales, and marked asthenia—in the respiratory type; a splitting headache and other symptoms suggesting a meningitis—in the nervous type; and sudden fever with choleraic symptoms and perhaps jaundice—in the abdominal

type; these features may help to recognize the possible presence of the disease. And, perhaps, blood counts and bacteriological tests may further help; the former will show a blood-picture without marked leucocytosis, and the latter will show *B. influenzae* in culture.

Pneumonia in the puerperium is not uncommon. The pulmonic lesion *per se* may not be difficult to diagnose, but its pathogenesis is not easy to discover, and, from a prognostic point of view, it is important to know if a given lesion is pneumococcal, influenzal or septic, as the last, occurring as an incident in a general septicaemia, is more serious than the other two. The fact that, in septicaemia, the pneumonic lesion tends to be lobular and not lobar does not ease the situation, as instances of small septic emboli causing infective lobar pneumonia or lesions resembling lobar pneumonia are not absent, and, besides, the pulmonic lesions produced by the pneumococcus and more especially the Pfeiffer's bacillus are not always lobar in type. Cultural tests will be of little avail, as in most cases a mixture of the organisms will be present. Blood-counts will not be highly elucidative, though in predominantly influenzal cases it may be suggestive. In most cases only a proper appreciation of the bed-side symptoms will help one to distinguish the septic type from the rest. In pneumonia born of septicaemia, there will be present an unusual severity of the symptoms, the usual signs of sepsis such as a swinging temperature, frequent rigors, recurring sweats, an increasingly rapid pulse, and an early collapse, or a continuance of the symptoms beyond the normal period of crisis.

Tuberculosis in the puerperium is a double-edged scourge; it cuts off the patient usually, and often injures the attendant. Three typical instances cross the writer's mind in rapid succession where this disease brought disaster to the patient and discomfort to the attendant. Tuberculosis in the puerperium may manifest itself in two forms. A partially healed or closed lesion may become topically active after confinement; or, as more commonly happens (and almost always so in the young), an active lesion, becoming seemingly quiescent during pregnancy, suddenly flares up with the birth of the foetus and becomes acute galloping consumption or general miliary tuberculosis.*

* Most tuberculous patients, during pregnancy show an apparent improvement in health and a real increase in weight. It has to be noted, however, that the tissues are never healthy, not even subcutaneously thick as in thyroid deficiencies, but are flabby and hydraemic or 'gassy'. The pathology behind these is not clearly understood. The following are interesting to note:

(1) Perhaps, the tuberculous germs, always preferring the young and growing, spare the mother and concentrate on the foetus during pregnancy, and, when pregnancy becomes terminated, the sudden deprivation of their prey incites them to run riot. But intra-uterine deaths are not

Tuberculosis in the puerperium being severely prone especially in the young to produce successive multiple infections, the symptoms therein closely resemble those in acute puerperal sepsis. An acute and sudden onset, a swinging temperature, frequent rigors, profuse sweats, quick pulse, rapid anaemia, speedy emaciation, and a progressively waxy face are as much the features of acute sepsis as of acute tuberculosis; indeed, bed-side differentiation between the two is, in most cases, impossible to make. An early, dry, hacking, unproductive cough may be attributed to an associated bronchitis, or an imagined influenza, or at the nearest a "double pneumonia"—an euphemistic term for broncho-pneumonia (?). The first one or two sputum examinations may be negative. Blood-count and reading of blood-pressure can in no way better the situation. In general practice, a clear family history and an intelligent probing into the patient's pre-pregnancy health only will, in most cases, help the practitioner to appraise the true nature of the disease, while, in hospital practice, repeated sputum examination, cultural tests and, above all, a correct interpretation by an able radiologist of a

sweeping, full-term deliveries are common, and the children are born with a tuberculous diathesis. Probably, the instinct of self-preservation, present even in the minutest life, forces the foetus to rob the mother of the various anti-substances, but in a slow and filial way so as not to injure her, but vaccine-like, stimulating her to produce more and more of them for the benefit of both, and perhaps also parting with its own reserves when demanded—thus exhibiting the unique 'mother and child' relationship in its simplest form in its pristine existence in the womb (with, of course, the 'self' predominating!).

(2) Has the anterior pituitary anything to do? The Ascheim-Zondek findings have conclusively shown that in pregnancy there is a great increase in the quantity of prolactin in the blood, and that it can be recovered in the urine of the pregnant as long as the chorion is in biologic contact with the maternal circulation. The tuberculous during pregnancy invariably improve, and become bigger in size though in a different way from the gigantism or other known enlargements of anterior pituitary over-activity. It is possible that the anterior pituitary secretion has some entity in it that is inimical to tuberculous germs. And, it will be interesting to know if the urines of the normal pregnant and the tuberculous pregnant differ in their anterior pituitary content, either in quantity or quality.

(3) The hydraemic condition of the tissues in the tuberculous during pregnancy is explicable and perhaps inevitable. It is wellknown that, in pregnancy, the foetus deprives the maternal tissues of their calcium. Also, exudative phenomena like urticaria and angio-neurotic oedema disappear under calcium medication. Perhaps, the calcium deprivation in the normal para becomes continually replenished and the blood-calcium is always maintained at a healthy level, while these do not occur in the tuberculous, probably because, in the latter calcium deficiency always exists, but possibly also because some of the 'prolactin', being set against the tuberculous toxins, does not become available for the maintenance of the calcium metabolism. It has been found that anterior pituitary has a controlling influence on calcium and magnesium metabolism; but in what way these are effected, and what influence the anterior pituitary has on the parathyroids are matters still in the womb of time.

good skiagram will tell the diagnosis; in other cases, the disease will diagnose itself, provided the patient has vitality enough to suffer and live sufficiently long.

Acute rheumatism is an affection of doubted existence in India, albeit one, now and then, comes across cases of hyperpyrexia with typical rheumatic joint-swellings of text-book descriptions, as also cardiac cases with typical rheumatic histories. The following is an interesting case of rheumatic hyperpyrexia in puerperium. The patient, a Mussalman primi, aged 14 years, got headache and fever during the fifth month of pregnancy and in a few hours there appeared abdominal pain and vaginal bleeding, for which she was put under a lady medical attendant, who, however, finding abortion inevitable completed the process the next day through ecbolics. The abortion was complete, the whole of the embryo and the placenta having come off; and with it, the bleeding and the pain were considerably improved. The fever, however, continued, neither increased nor decreased, and on the third day of the abortion, the patient began to complain of diarrhoea, having passed 5 or 6 loose rather painful motions the previous day. The most arresting feature at the time of the examination was the wavy præcordial pulsation heaving over the jacket. The throbbing heart was enlarged transversely, the apex was ill-defined, the cardiac impulse was diffuse, and the sounds were foetal in character; epigastric pulsations were present, there was no thrill, and no murmurs could be heard. The other conditions were as follows: the patient thin and emaciated, temperature 102° F, pulse 120, eyes not anaemic nor jaundiced, expression assuring, tongue clean, sudamina present; respiration and lungs normal, palpitation present, no pain anywhere in the chest; abdomen not tympanitic but showing general tenderness and borborygmi; urine high-coloured but otherwise normal, no oedema anywhere; uterus not felt on the abdomen and vaginal discharge sanguineous but neither excessive nor foul; headache and general pains were complained of, there was no mention of rigors, slight sweats were said to have occurred now and then, and the 'womb' was said to have been reported normal by the lady attendant (being a purdah-lady, vaginal examination was not possible, and, besides, the history of the complete abortion and the nature of the vaginal discharge did not demand it). On probing into the previous history, it was elicited that, from her childhood, the patient was having periodical attacks of high fever with joint-swellings which used to last for 3 to 7 days every time. The heart condition, as it did not fit in with the clinical findings but fitted in well with the history of 'rakta-vatam', was put for a myocardial dilatation of 'rheumatic' origin, and the

fever was considered to be a continuation of the pre-abortion condition, whatever it was. Accordingly, the patient was kept under observation, and put on hot lysol vaginal washes and four doses of dram measure of castor oil in emulsion with 5 grains salol for each dose to control the diarrhoea and fever, as it was found that no laxative had been given to account for the diarrhoea. On the 4th morning, the temperature was 99.5° F. the pulse was 112, the diarrhoea had stopped, and the patient was feeling better; she was put on four doses of ordinary diaphoretic with Sod. benzoas 10 grains, Tr. Nux vomica 10 mins. and Tr. Digitalis 7 mins. for a dose. That evening, it was reported that the temperature had gone up to 103° F. and that the patient complained of some sore-throat and had slight pain and swelling over the elbow joints. (It is worthy of note that no rigors, not even a chill, was reported as having accompanied the rise in the temperature). Formamint tablets for the throat, and menthol and methyl salicylas ointment for the joints were ordered and 7 grains Sod. salicylas and 3 grains Sod. iodide per dose were added to the remaining two doses of the morning mixture while the digitalis and the nux vomica were raised to 10 and 15 mins. respectively per dose. Next morning, when the patient was examined, her temperature was 104° F, pulse 145, respirations hard, elbow-joints red, tender, swollen and non-oedematous, and the wrist-joints following suit; the patient had severe general pains, the whole skin was hyperaesthetic, and she was toxæmic to a degree; the vaginal discharge had diminished and was sero-sanguineous, but could not be termed 'pathological'. A doubtful diagnosis of acute rheumatism in the course of a sapraemic puerperium or an acute sepsis with septic arthritis was made, and novalgin and omnadin were injected alternately and each twice, beginning with novalgin, all injection being six-hourly and every omnadin or novalgin being preceded by 1/30 gr. strychnine and ½ c.c. pituitrin in one injection—all these in addition to general symptomatic measures. The temperature fell to 100° F. twice and each time within 2 to 3 hours of injecting the omnadin, but it rose as quickly as it fell and reached the pre-injection level on both the occasions. The patient had a restless night, but there was no delirium and she was mostly conscious though stuporous at times. Next morning, her temperature was 105° F, pulse was 150, elbow, wrist and knee joints were affected, and the general condition was very bad. A distant lady-specialist was called in, and, when she came in the evening, the patient was semi-comatose with an axillary temperature of 104° F. and a pulse of 140. Some arguments passed; she, not having noticed the joint-swelling, and having forgot to enquire into the history and the patient's shifting conditions, but only witnessing the then state, was a bit up in spirits, but became perplexed when she was appraised of the

whole facts. The uterus was explored, an intrauterine douche was given with the return flow bringing a few small dark-grey shreds which, in all reason, could not have produced such great mischiefs, ½ c.c. pituitrin was injected, and Ol. gaultheria with Ol. terebinthine was prescribed for the joint condition. The patient died in coma that night, and with very high fever (according to report). Perhaps, the case is unique in medical experience, perhaps it is not. And perhaps bold suspicion of sepsis and early energetic measures might have saved the patient; yet, perhaps, the best that could be done was done, and nothing on earth could have saved one with such a heart as our patient had. The interest of the case, however, lies in the facts that hyperpyrexia with joint swellings does occur in puerperium, and that, when there is a clear history of longstanding 'rheumatism', the case but rarely offers for correct diagnosis, and perhaps never in mofussil practice, where, microscope being a rarity and cultural results being ever 'an hour late,' and yet the case demanding urgent attention, and all diagnoses have to be made on the spot and from the bedside findings.

Scarlet fever in puerperium has been sufficiently discussed under 'clinical manifestations' when 'puerperal scarlet fever' was being considered. There is no fresh ground to be covered except perhaps to mention that, in the findings of some, throat-troubles, pathognomonic of typical scarlet fever, are rare in scarlet fever of puerperal origin, while vomiting and a rapid pulse-rate are much more frequent in puerperal scarlet fever than in streptococcal puerperal erythema.*

Kala-azar, common in many areas and endemic in some, may put in its appearance in the course of a puerperium. The disease can usually be much more easily distinguished from puerperal infections than any other fever. A symptomless fever with double diurnal rise and no great immediate strain on the patient is kala-azar, and, in doubtful cases, the Gel test and a spleen puncture will make the diagnosis clear.

Other febrile disorders like acute indigestion and tropical liver may now and then infest the normal course of a puerperium. But that is rare, and, besides, in such cases, the fever will be less urgent, and in most cases, the

* Since writing my last notes on 'puerperal scarlet fever', there has come to my notice an illuminating paper on 'Scarlet Fever in Bombay' read by Dr. P. T. Patel before the Indian Science Congress of 1931 (Jan.) and published in the 1931 June issue of *The Indian Medical Gazette* (p. 306). In that paper Dr. Patel establishes through facts and figures that "Scarlet fever is an indigenous disease in India, particularly in big cities, hill-stations, and other places". And he further says that it has little tendency to spread and that the infecting streptococcus is not generally virulent and haemolytic.

presenting symptom will not be fever at all; being so that may be passed over in our discussion.

The zymotic and other extraneous fevers having been excluded and the case proven as one of definite puerperal infection, to distinguish the nature of the systemic invasion is comparatively less arduous, as each entity has distinctive features of its own, usually identifiable at the bedside and with the aid of the ordinary consulting-room tests. We discussed at some length the symptoms of each under "clinical manifestations," and therefore all that needs be done here is, more or less, to recapitulate them.

A case that gives a history of prolonged labour, or of the expulsion of a ragged or incomplete after-birth, and presents the following features, *viz.*,—an insidious onset with intense headache, but no rigors nor any constant chill; a temperature, which, slight at first, but rising step-like reaches a maximum of about 103° F on the 3rd or 4th day and maintains that level for the next 5 or 6 days; a pulse often dicrotic, always full, regular, of good volume and tension, and keeping pace with the temperature; the eyes assuring; the expression encouraging; the tongue moist, and clear or coated; the skin, hot, but healthy otherwise; the breasts functioning normally, or engorged; the chest always, and the abdomen usually, keeping normal; micturition, remaining free and easy, and the urine showing only high colour; the uterus, involuting slowly or not at all; the vaginal discharge, keeping normal in quantity, or showing excess, but never becoming scanty, and presenting such pathological features as foulness, darkness in colour and clots; and a comparatively short course, with little or no complications, and the temperature reaching normal by lysis between the 10th and the 13th day of the puerperium—such a case is *puerperal sapraemia*.

In *septicaemia*, the history in hospital cases will usually be a difficult labour with all the evils attendant on it, while, in general practice, it will usually point to some dire mischief by some meddling inexperienced. In a typical case, the clinical features will be somewhat as follows:—Onset, with rigor and fever, but no headache; temperature, irregular from the beginning, ranging between hypo- and hyper- pyrexias, and accompanied by shiverings and sweats; pulse, raised, soft, becoming progressively rapid and feeble and getting out of proportion to the temperature; eyes, brilliant; face, flushed; expression, anxious; tongue, sore and red; skin, moist, pungent and perhaps showing rashes; breasts, lax, empty, and not functioning; respiration, laboured; chest, showing rales or other adventitious sounds;

heart, featuring toxic disturbances; abdomen, tender, painful, usually tympanitic, often rigid with liver and spleen palpable and bowels generally constipated; loins and flanks, tender and usually painful; urine, scanty and smoky, at times distinctly nephritic; brain, affected early, and featuring delirium, coma and perhaps euphoria; uterus, generally normal; vaginal discharge, nil or scanty, and in the latter event, usually peculiarly sweet-smelling; and course, short in the vast majority of cases, not lasting for more than a week at the most, and ending in coma and death, generally accompanied by hyperpyrexia, wiry pulse, extreme anaemia or jaundice, aphthous tongue, terminal diarrhoea, and other severe toxæmic features.

Pyæmia is but a sister form of septicaemia; in fact, it is septicaemia with distinct foci of suppuration. If the multiple abscesses are visible, the case will be clear; if they are visceral, the diagnosis may be difficult, especially when the abscesses are small and miliary. At any rate, the clinical features of septicaemia are evident in pyæmia too; and the former have been discussed already.

Bacillus coli affections have one or two redeeming features; if they are remembered, they will not keep us long in suspense, and if they are remembered sufficiently early and treated properly, they will gracefully leave the scene. The features that characterise typical *B. coli* affections are a sharp onset with generally a rigor, a spiked and steepled temperature chart, a slow pulse, symptoms pointing to lesions of the urinary tract, and an uncertain course especially if discovered only late; in other respects, they resemble those in a case of sapraemia.

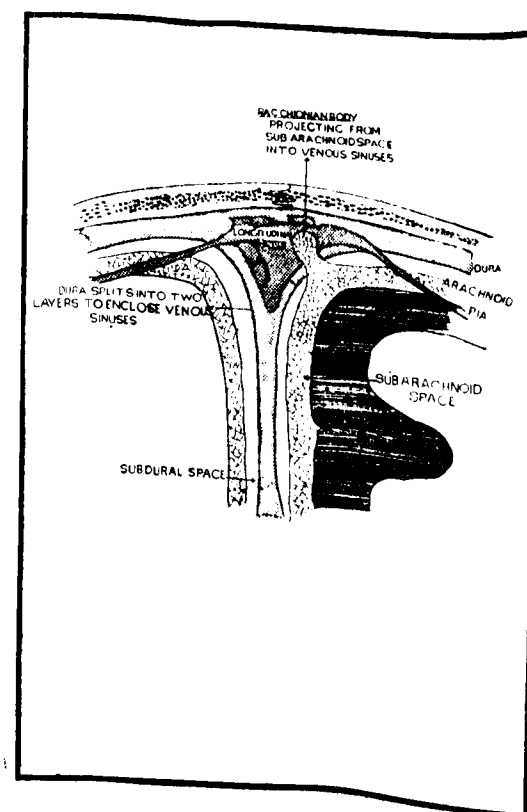
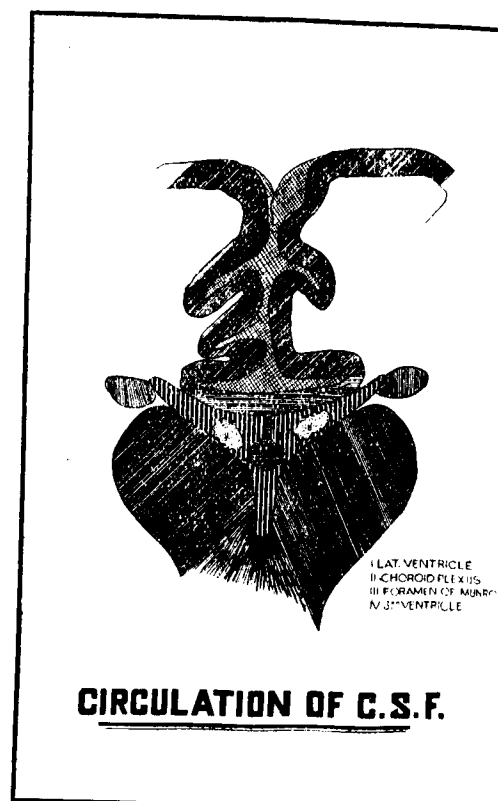
SOME ELEMENTS OF NEUROLOGY (11)

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CEREBROSPINAL FLUID
CIRCULATION OF CEREBROSPINAL FLUID

1. Cerebrospinal fluid is formed by choroid plexuses in lateral ventricles.
2. Flows through each foramen of Monro into the third ventricle.
3. Thence *via* aqueductus Sylvii into the fourth ventricle.



4. From the fourth ventricle out into subarachnoid space *via*

(1) Foramen of Magendie—in middle line,
and (2) Foramina of Luschka—placed laterally.

5. Cerebrospinal fluid also passes out *via* anterior and posterior perforated spots, but these are too small by themselves to deal with the cerebrospinal fluid if the other foramina are obstructed.

6. Cerebrospinal fluid is absorbed from subarachnoid space into venous sinuses *via* pacchionian bodies which are projections of subarachnoidal villi with contained cerebrospinal fluid into venous sinuses.

Cerebrospinal fluid may be obtained or removed by

- (1) Lumbar puncture ;
 - (2) Cisterna puncture ;
- or (3) Ventricular puncture ;

LUMBAR PUNCTURE

1. Spinal cord ends at lower border of the first lumbar vertebra.
2. You can therefore safely go into the space between the second and third, third and fourth, or fourth and fifth usually.
3. Vary the site for repeated lumbar punctures lest irritation and blockage results.

Technique :

1. Join iliac crests by line.
2. Line passes over upper part of the fourth lumbar vertebra.
3. Space below = between fourth and fifth.
4. Depth at which cerebrospinal fluid is generally obtained = 2 inches in adults.
5. Go on gradually withdrawing stylette after 1 inch, 1½ inches, etc., to see if fluid comes out.

Uses of Lumbar Puncture :

1. Diagnostic.
2. Lipiodol injections demonstrate subarachnoid block caused by tumour.
3. Encephalography.

4. Therapeutic.
5. Production of spinal anaesthesia.
6. Relief of increased intracranial pressure.

1. DIAGNOSTIC.

Examination of the cerebrospinal fluid is necessary for the diagnosis of—

1. Meningitis.
2. Encephalitis—(1) Poliomyelitis.
(2) Encephalitis lethargica.
(3) Post-vaccinal encephalitis, etc.
3. Parenchymatous syphilis—(1) Tabes
and (2) General paralysis.
4. Intracranial tumours—normal cerebrospinal fluid under slightly increased tension.

Compression of the Cord by Tumour or Abscess.

Cerebrospinal fluid shows fairly characteristic reactions—

1. Total protein is considerably increased; normal = 0.02 – 0.04 per cent.
2. But cells are not increased; normal = 2 – 4 per cmm.
3. This is called “The *Albumin-Cell* dissociation phenomenon.”
4. This excess protein with no increase in the number of cells may also occur in—(1) Meningitis;
(2) Polyneuritis;
(3) Cerebrospinal arteriosclerosis.

Froin's syndrome :

In addition to excess of protein—

1. Cerebrospinal fluid may be yellow—xanthochromia
- and 2. May even coagulate spontaneously on standing.

II. INTRATHECAL INJECTION OF LIPIODOL.

1. To demonstrate subarachnoid block by tumour, etc.
2. Lipiodol—(1) A heavy oily substance opaque to X-rays;
(2) Consists of iodine and oil of poppy;
(3) Harmless;
(4) Very slowly excreted.

Technique :

- (a) 1. Inject lipiodol through a lumbar puncture.
2. Invert patient so that lipiodol flows down towards cervical cord.
3. See flow of lipiodol with X-rays.
4. Lipiodol held up at a point = subarachnoid block at that level.
- (b) Or
1. Inject lipiodol through a cistern puncture;
2. Allow to gravitate down towards lumbar region.
3. Light lipiodol can be used. It floats up after a lumbar puncture.

III. ENCEPHOLOGRAPHY.

1. Introduction of air into subarachnoid space through lumbar puncture needle.
2. After effects are often severe.

IV. SPINAL THERAPY.

1. Meningitis—(1) Anti-meningococcal serum.
(2) Anti-pneumococcal serum.
2. Neuro-syphilis—Salvarsanized serum.
3. Poliomyelitis—Convalescent serum.
4. Tetanus—Anti-tetanic serum.

V. SPINAL ANAESTHESIA

VI. RELIEF OF INCREASED INTRACRANIAL PRESSURE.

1. Uraemia.
2. Apoplexy, etc.

Significance of blood in cerebrospinal fluid :

Haemorrhage into subarachnoid space may occur—

- (1) During the performance of lumbar puncture ;
- (2) Prior to the performance of lumbar puncture.

1. Haemorrhage during the performance of lumbar puncture :

- (1) Needle was pushed too far—
- and (2) Damaged plexuses on dorsum of vertebral column.

2. Haemorrhage prior to the performance of lumbar puncture—

- (1) Apoplexy ;
- (2) Haemorrhage from a previous lumbar puncture ;
- (3) Subarachnoid haemorrhage from a leaking aneurysm.

(a) During lumbar puncture blood in cerebrospinal fluid is due to going in too far and striking venous plexus—go in gradually.

1. Cerebrospinal fluid—(1) thick with blood at start ;
(2) clears if you wait and withdraw needle a little.

2. Centrifuged or allowed to stand—

- (1) Red blood corpuscles not haemolysed ; therefore sink.
- (2) Supernatant fluid clear.

(b) Prior to lumbar puncture—

1. Cerebrospinal fluid equally coloured ;
2. Centrifuged and allowed to stand—

- (1) Haemolysis of red blood corpuscles ;
- (2) Supernatant fluid—red or yellow.

Significance of yellow cerebrospinal fluid :

The yellow colour is due to bilirubin formed from hæmoglobin—

1. Previous haemorrhage
- or 2. Xanthochromia.

Xanthochromia and loculation syndrome :

1. Xanthochromia—The peculiar colour of cerebro spinal fluid found in certain cases of obstruction to the spinal canal by—

- (1) Tumour ;
- (2) Meningitis, etc.

2. Blood vessels running longitudinally upwards are compressed ;

3. And behind the compression, *i.e.*, below the block,

- (1) Red blood corpuscles
- and (2) Protein

escape from the congested vessels into the cerebrospinal.

4. The red blood cells—(1) Are haemolysed,
(2) Converted into bilirubin.

5. Bilirubin gives the yellow colour.

6. Protein content may rise to a very high figure.

7. The cerebrospinal fluid clots on standing.

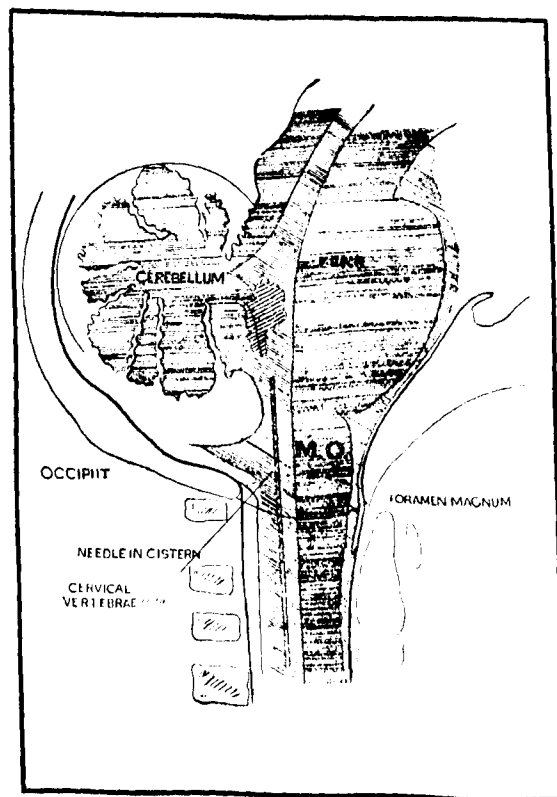
CISTERN PUNCTURE

1. *Cisterna magna* is a large space filled with cerebrospinal fluid situated behind the medulla and underneath the cerebellum.

2. Cistern puncture consists in introducing a needle into this space.

Technique :

1. Usually the second cervical vertebra in the first palpable vertebra.
2. Introduce an ordinary fine lumbar puncture needle below the first cervical vertebra or atlas and above the second cervical vertebra in middle line of neck.
3. Pass it up inside ring of atlas.
4. It enters foramen magnum,
5. and pierces cisterna magna.
6. Take external auditory meatus as your line of direction.
7. You will obtain fluid at a depth of 1½ inches.
8. To avoid striking the back of the medulla—vital centres, etc.
 - (1) Do not go too far forwards ;
 - (2) Do not go too far upwards.



(1) Do not go too far forwards :

1. Direct the needle more vertically rather than in the direction of the external auditory meatus.
2. Thus, instead of striking foramen magnum at first shot, you will strike occiput.
3. Now, gently direct the needle forward, when it will slip over edge of foramen magnum and enter cistern.
4. Thus, you will not damage spinal cord as you pass through foramen.
5. Nor will you push needle too far and enter the floor of the fourth ventricle
6. There is a danger of injury to the cerebellum, but even if needle did enter cerebellum it would probably have no effect as cerebellum is a "silent area" of the brain.

(2) Do not go too far upwards :

1. To avoid so doing introduce needle to a distance considerably short of that at which you expect to strike cistern, *e. g.*, 1 inch.
2. Take out stylette.
3. See if fluid is obtained.
4. If no fluid, push it on $\frac{1}{4}$ inch.
5. Take out stylette again and see if fluid is obtained.
6. Thus, you cannot go too far as cistern is never dry.

Advantages of Cistern Puncture :

1. Alternative to lumbar puncture when difficulty is experienced in getting cerebro-spinal fluid therefrom, *e. g.*, occasionally in old people the lumbar vertebrae may be almost fused together into one bony mass like sacrum.

2. Lipiodol may be injected through cistern puncture in diagnosis of subarachnoid block by spinal tumour—

- (1) Lipiodol gradually sinks down spinal canal, and
- (2) Is held up at level of obstruction, *i. e.*, tumour on X-ray.
- (3) Compare cerebrospinal fluid above block with that below block.

3. Cistern puncture relieves headache in such cases of tumour.

4. Therapeutic sera may be introduced *via* cistern puncture and so get more directly to seat of trouble, *e. g.*, base of brain in meningococcal meningitis.

VENTRICULAR PUNCTURE

Fontanelle Technique in Infants :

1. Use an ordinary fine lumbar puncture needle.
2. To avoid the sagittal or longitudinal sinus you must *not* go in the middle line.
3. Go in about $\frac{1}{2}$ inch to either side of middle line, depending on which ventricle you are aiming at.
4. Drive needle downwards and slightly outwards.
5. You will reach the lateral ventricle at a depth of about $1\frac{1}{2}$ inches.

N. B.—Depth is about the same for all punctures—lumbar, cistern, ventricular.

Trephine in Adults :

1. Trephine skull in occipital region.
2. Direct needle forwards.
3. No apparent damage to brain or blood-vessels results. Probably they are pushed aside as brain during life is semi-fluid.

Advantages :

1. Therapeutic—In meningococcal meningitis in infants get serum direct into ventricles as well as by lumbar route.
2. Air ventriculography—
 - (1) Replace cerebrospinal fluid in each lateral ventricle by air.

- (2) By changes in symmetry you can lateralize and localize tumours.
- (3) Internal hydrocephalus shows symmetrical dilatation of both ventricles.
3. Ventricular estimation—
 - (1) Withdraw as much cerebrospinal fluid as possible from each ventricle.
 - (2) Carefully measure and compare : normally equal on two sides.
 - (3) Pronounced difference indicates partial or complete obliteration of that ventricle by tumour.
4. Intraventricular injection of dyes—in hydrocephalus and tumour :
 - (1) Inject phenolsulphonephthaleine.
 - (2) Estimate amount excreted in urine in first two hours after injection.
 - (3) Decreased excretion = obstruction to ventricular outflow.

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AN ORGANIZED MEDICAL PROFESSION

We have often referred in these pages to the unorganized state of the medical profession in this country. We have previously touched upon some of the evil consequences of this want of organization. Further, whenever a few medical practitioners meet, they rarely disperse without commenting on this unfortunate condition. The evil is probably much greater than we are aware of. For it has attracted the attention of the British Medical Association. And it is sending one of its prominent members to this country to examine the condition of the profession and to suggest measures to remedy the evil. We are sure the profession in this country would welcome such help. But before we could profit by advice, it is essential that we should have as clear an idea as possible about the defects which are in the way of such an organization. Dr. Anderson who will be in our midst in a few days is the Medical Secretary of the British Medical Association. He is therefore well aware of the difficulties of bringing together a vast number of medical men who have differing aims and ambitions. He may therefore more easily detect the real obstacles which hinder a better organization of the profession here. But eminent and experienced though he be, Dr. Anderson is still a stranger. And unless we here are able to lay our difficulties before him definitely, he might arrive at conclusions which may not be entirely satisfactory.

The British Medical Association is primarily an Association of British medical men. And its object is naturally to safeguard and further the interests of the British medical men in their own country. In recent years it has extended its activities to parts of the British Empire where its influence could

prevail. Its influence on the medical politics of this country has been telling. It is therefore natural that the Association should endeavour to increase its influence still further. But whether that influence could be useful to the progress of the profession as a whole is for the Indians to consider.

It is a common belief that the presence of the licentiate group of medical practitioners is a great hindrance to the solidarity of the profession in this country. Nothing could be further from facts. The licentiates form the vast majority of medical men in this country. The graduates of this country and foreign qualified medical men are a mere handful against the licentiates. If there be any complaint, it must be from the licentiates that the small number of the former, forming a class apart prevents the unity in the ranks of the profession. But we have not heard any such complaint. That the existence of practitioners of modern medicine of varying standards of education is not in the way of the unity of the profession is exemplified by the fact that even among the various groups, if groups they can be called, there is not that cordiality and singleness of aim which one would expect. But undoubtedly there is disunity and dissatisfaction in the profession. The cause should be sought elsewhere.

There was no evidence of this discontent in the early years of this century. Medical men were then few and they were almost entirely in government employ. If there was then any grievance, it was merely a scramble for a healthier or more lucrative station. But at present in addition to the large number of medical men and women in government services, there is a large number of practitioners outside the various services. Medical men are therefore divided into two large classes, *viz.*, those who are in government services and those who are not thus employed, *i.e.*, the independent practitioners. There has been an increasing dissatisfaction between these two classes.

Medical men in government employ draw salaries. In addition they have the privilege of engaging in practice. There was no grievance on this question so long as the salaried men were the only available medical men in many parts of the

country. But with increase in number of independent practitioners who depend on their practice for their living, the government medical men who are in charge of hospitals and dispensaries feel that these practitioners encroach on their privileges of practice and thus to a certain extent curtail their emoluments. While the independent practitioners feel that these salaried medical men interfere with their means of earning a living by their engaging in practice instead of confining their attention to government work. The men in service fight to retain their rights. The independent practitioners struggle to maintain themselves usefully. The government encourages its employees to live well, but is unwilling to provide the bulk of the practitioners with facilities for a mere living. Is there any wonder that there is no agreement between these two groups of practitioners? It is enough to engender bitterness.

The conditions in the various medical services are such as to set one against another. Equally efficient and capable men are set in disagreeable combinations. Ambition in one is looked down upon as presumption in another. High place and larger emoluments are not infrequently conceded to the less worthy and more incapable. And yet, those who look upon such conditions with perfect equanimity are often loudest when they bemoan the absence of unity in the profession. The profession is not merely divided horizontally into the licentiate, graduate and foreign qualified groups. It is again divided into service men and independent practitioners. And the services are again divided into superior, inferior and subordinate officials. The profession is in layers, in columns and in squares; and there are different forces which jealously guard against any possible merge, even by rare accident. The profession might gaze on this horrid monster in check suit with thin, thick and stout lines in lurid colours. Some one may suggest a more pleasing clothing.

believe that he has to deal with a case of acute pelvic infection or appendicitis. What potentialities the disease has to confound the attendant may be gathered from what HERBERT FRENCH says in "*An Index of Differential Diagnosis of Main Symptoms*": "*Coli bacilluria* is now a familiar difficulty in the differential diagnosis of appendicitis; although theoretically the pain would be referred mainly to the kidney, generally the right, it is quite common for patients suffering from this condition to refer their pain not to the back or loin at all, but to the front of the lower part of the abdomen, and particularly over the right iliac fossa, in such a way that acute appendicitis is simulated very closely. Even though the urine be examined and a haze of albumin found, together with an excess of leucocytes, the acuteness of the condition may be such that the surgeon may not feel justified in waiting for cultures of a catheter specimen of the urine to be made, to see whether the *Bacillus coli communis* is present in pure culture; and not a few patients of this kind are unavoidably operated upon for acute appendicitis when the appendix is perfectly normal." *We shall see presently the difficulties of distinguishing appendicitis from salpingitis. It is, however, in less urgent cases of the pyelitis, or when the case is seen after the acute stage, that confusion with inflammations of the uterine appendages will arise. The pain, the fever and the urine abnormalities will be present in both, and, unless care be bestowed on the details, the case is apt to become misjudged. In pyelitis, the pain is usually unilateral, generally over the right loin, and is often colicky, even simulating renal colic; the fever is remittent or intermittent; and the urine, in most cases, will show frank pyuria and *B. coli* under the microscope, while the dysuria and the frequency of micturition are intrinsic in character, pointing to a lesion of the kidney. On the other hand, in pelvic lesions, the pain will be ill-defined, felt over a wide area below the umbilicus and in the flanks, seldom unilateral and never colicky; the fever will be more irregular; and the urine may not show frank pyuria, and the *B. coli* cannot be seen under the microscope under normal circumstances, while the dysuria and other urinary troubles will be of an obstructive type, or cystitic if the bladder has got involved. Other features such as a more rapid and irregular pulse and a greater toxæmia in the septic pelvic lesions; sapraemic features of vaginal discharge, and perhaps also of the breasts, in pyelitis; and a blood-count and urine culture will further help to differentiate the two conditions.

Acute appendicitis is one of the worst calamities that can befall a puerperal woman and her medical attendant, for in the conglomeration of the various puerperal complications, it seldom stimulates one's memory, and even

* HERBERT FRENCH.—*Index of Differential Diagnosis*, Fourth Edition, 1928, p. 559.

if it comes to be remembered, its discovery at the proper moment is none too easy; indeed except in a very limited number of exceptionally lucky cases, its presence becomes felt at a time when it is too early to be dogmatic or too late to do positive good. The condition, on the one hand, will closely simulate *B. coli* pyelitis or inflammation of the uterine appendages, and, on the other, it may resemble the conflagration of peritonitis in its early stages, from all of which it has to be studiously excluded. Whereas pyelitis and pelvic inflammations will allow of expectancy being followed, and purely medical measures adopted, the latter especially in pyelitis, a case of appendicitis will nearly always demand surgical intervention, and to a greater extent in puerperium. The need to distinguish appendicitis from peritonitis, though both are catastrophic, will be appreciated when we compare the inflamed appendix in puerperium to an angry cobra by the side of an invalid, and the peritonitis to the invalid already under the influence of the venom; it should be obvious that, while ridding of the cobra is the need in the former, the rallying of the patient is the indication in the latter. The three credentials in the diagnosis of an appendix lesion in puerperium are: (1) to remember the existence of the vestige and its potentialities for mischief; (2) to suspect its probable peeping in when a fever in puerperium is accompanied by acute lower abdominal pain, more so when the pain is circumscribed over the right iliac fossa, and, most so, when it predelicts and points to McBurney's point; and, (3) to set aside all notions of prestige and seek the advice of an expert or colleague in all doubtful cases—the last, especially in general practice, and both for the good of the patient and the good name of the practitioner. When these together as basis, usual symptoms as adjuvants, and Rovsing's sign and other special findings as correctives, a good prescription may be got for correct diagnosis. The cardinal features in an acute appendix are: rapid onset of severe pain, usually at McBurney's point but varying with the position of the organ; tenderness and rigidity over the right iliac fossa, compelling the patient to lie still with the right knee drawn up and the abdominal breathing partially or completely suspended; a diffuse palpable fulness or a distinct swelling in the right lower abdomen; and general symptoms such as moderate fever, raised pulse, furred tongue, vomiting* and constipation. Sometimes, especially when the inflamed organ is low in the pelvis, additional symptoms of urinary troubles may appear. In the diagnosis of appendix lesion, Rovsing's sign is of importance and is positive, if, on pressure being applied on the pelvic colon in the left iliac fossa, pain is complained of in the right iliac fossa which is due to the gas being

*The writer has seen two cases with temperature 102° F, pulse 100 and no vomiting; both were operated in the Headquarters hospital.

forced backwards into the caecum. Another suggestive sign is the prominence of the congested superficial circumflex iliac vein on the right side. The difficulty of distinguishing appendicitis from pyelitis has been already pointed out under *B. coli* pyelitis. Perhaps, a positive Rovsing's sign, and, in appendicular lesion of low pelvic type, the discovery of a tender right-sided fulness or swelling per vaginum or rectum, may help to some extent. Acute salpingitis or other allied pelvic lesion will offer the same difficulty. HERBERT FRENCH, in discussing the differential diagnosis of 'pain in the right iliac fossa' says, "Acute salpingitis or inflammation of the right ovary is generally secondary to some other pelvic inflammation... The skilled gynaecologist may, by vaginal palpation, be able to determine the cause, though doubts may exist as to whether the condition is one affecting the right uterine appendages or the vermiform appendix, until laparotomy is performed."* It may be noted, however, that a much less precipitous onset and, perhaps, in the course of a general sepsis, general symptoms as severe as, or severer than, the local ones; ill-defined features per vaginum or rectum, showing bilateral troubles of the uterine adnexa and a general matting of the pelvic tissues, the uterus itself remaining pretty normal; greater fever, and a more rapid pulse; less intense suffering, and practically no vomiting; negative Rovsing's sign, and possibly a more marked leucocytosis and less fulminant course—are all features of some acute puerperal pelvic lesion as opposed to those of acute appendicitis in puerperium. Peritonitis at its onset and possibly also in the early stages when the storm is but gathering, will be difficult to differentiate, especially to the un-initiated, from appendicitis; but, it should not be so when the disease has become established and the storm is raging—generally speaking, diffuse rigidity and tenderness, incomparably severe pain, a purely thoracic breathing, marked tympanitis, hyperpyrexia generally, a very fast pulse, and the characteristic peritoneal facies will all evidence the fury of the tempest. All things considered, the diagnosis of acute appendicitis in puerperium will yet remain elusive. RENDLE SHORT† observes "That we would do well to improve our diagnostic methods is shown by the confession that of 1,298 cases sent into hospital at Zurich as acute appendicitis (in five years), 252 (roughly 20 per cent.—T. S. A.) were incorrectly diagnosed as such and that 6 per cent. of the patients operated on had no appendicitis (Clairmont)." The same author‡ further observes, "R. Soupault and G. Seille call attention to pelvic appendicitis. They are very important, because so apt to mislead the medical attendant. There is neither pain, tenderness, nor rigidity in the right iliac fossa. If the appendix is low in

* HERBERT FRENCH—*Index of Differential Diagnosis*, 4th Edition, 1928, p. 559.
 † *Medical Annual* 1931, page 46.
 ‡ *Ibid* 1932, page 55.

the pelvis, there will probably be frequency and painful micturition, and there is tenderness on rectal examination. If it lies high in the pelvis, these signs fail, but lower abdominal pain of acute onset with vomiting and fever, and some tenderness in the hypogastric region, are enough to warrant the diagnosis in boys, girls, and men. In women, acute salpingitis has to be considered." One may not entirely agree with the warrant for diagnosis of high pelvic appendicitis, as we cannot entirely ignore the nomadic *B. coli*, especially in boys and girls, but, the rest are uncontested and well worth carrying in our memory. It is noteworthy too that the observations noted above are on cases of appendicitis in otherwise normal persons. It may be imagined how often we shall get confused and how often we shall have to confess when we are dealing with the affection in puerperium. Under such circumstances, no sentimental apology needs to be offered for having allowed the vermiform vestige to take up so much space in our discussion.

An old *salpingeal lesion* may become lit up in puerperium to one of its acute phases, or a pyosalpinx may become burst, giving rise to all the consequences incidental to it. The diagnostic features of the resulting lesions will be those of acute salpingitis in the former, and probably peritonitis in the latter; sufficient has been said on both.

Acute indigestion with raised temperature and pulse, vomiting, abdominal pain, and initial constipation may simulate some serious abdominal malady, especially if the helminths too are active. In puerperium, acute indigestion in its early stages will have to be distinguished from peritonitis in its early stages, neither condition in the later stages offering much difficulty in diagnosis, as the diarrhoea and possibly the symptoms of dehydration in one, and the toxæmia and the facies Hippocratica in the other, will be pathognomonic. In the differentiation of the two conditions, the history, and such phenomena as rigidity and distension on one hand, and peristalsis and borborygmi on the other are of little value. The history in all acute indigestions will generally be some offending food. But, in puerperium, under normal circumstances, it is doubtful if such a history will be obtained, as a puerperal woman is under strict dietetic regime, unless it so happens that personal idiosyncrasy has caused disagreement with some food, the possibility of which, however, will seldom be suspected in the absence of allergic symptoms like urticaria. Though rigidity and distension are the special features of peritonitis, in many cases of acute indigestion, the abdomen is distended and the abdominal muscles are reflexly contracted and rigid; besides, rigidity, as SNOWMAN* points out, is not usually well-marked in puerperal peritonitis. Peristalsis and borborygmi, the common features of

* *Manual of Emergencies* by J. Snowman, 1926, page 184.

acute indigestion, are not uncommon in the early stages of peritonitis. In the differential diagnosis of acute indigestion and peritonitis, greater attention is to be paid to the pain, the character of the respiration, the facies and the blood count, and the greatest reliance is to be placed on the pulse. The pain in indigestion is paroxysmal, colicky, courses round the umbilicus, and may shoot up to the shoulders, the front of the chest, or the interscapular space, depending on the direction and the velocity of the gases in the bowels; the writhing patient welcomes pressure on the abdomen, and prefers to lie prone, and with pillows beneath. Breathing is normal, often controlled and grunting, and usually thoraco-abdominal. Facies is one of conscious suffering with features contorted and expression wretched. Blood-count is unaltered. In peritonitis, on the other hand, the pain is constant and continuous, stabbing or lancinating and agonising, diffuse and generalised, and confined to the abdomen; it imposes immobility on the patient and compels her to adopt the characteristic still and supine decubitus with knees in an attitude as will put the least strain on the abdominal muscles. Respirations are soft, sighing, rapid and extra-abdominal, with the intercostals and *alae nasi* active, and abdomen free. Facies is one of agony, with features apprehensive and expression anxious. Blood-count will show a marked leucocytosis with a great increase in the polymorphonuclears. In either case, the pulse, as has been already said, will give the best indication; if, on counting it every ten minutes, it is found to rise at successive counts, peritonitis should be the diagnosis of choice and safety. It should be observed, however, that the above are the features in typical cases. But, atypical cases are many, some indistinguishable except through laprotomy. In all doubtful cases it will be safer to exhibit patience and placebos than to indulge in purgatives and paregorics till the incubation period is passed.

Having excluded all other acute infective conditions of the abdomen, and labelled correctly the given case as one of distinct puerperal infection with predominant local symptoms, we have to discover the particular nature of the lesion, and this involves a consideration of peritonitis, perimetritis and parametritis, as salpingo-oophoritis, the only other condition of note, seldom occurs alone and has its features merged in those of the associated perimetritis.

Peritonitis and *perimetritis*, being the general and the circumscribed lesions respectively of the same serous peritoneum, differ but in degree in their clinical manifestations; in fact, many cases begin as circumscribed perimetritis, and the resistance to the invasion being unequal, becomes spread into a general peritonitis. Being so, they should be difficult of diagnosis in their early stages. But one will not be kept long in suspense, as the original lesion, if at all it is to flare up, will do so within 24 hours of onset,

when all the symptoms like pain, tenderness, rigidity, distension and vomiting, as also the temperature, pulse, respiration, and the general condition will become worsened and show themselves in all their direness and frightfulness. The chief distinguishing features, however, will lie in the findings per vaginum. These will be nil in general peritonitis. In pelvic peritonitis on the other hand, all the organs of the pelvis will be tender and painful, the vaginal vault full, the cervix in all probability centrally fixed, and the pelvic tissues matted, oedematous, or showing small swellings corresponding to the pockets of pus; in later stages, there may usually be felt a bulging in a narrowing of the posterior fornix and a tumescence behind in the pouch of Douglas, due to the collections of pus having gravitated to the bottom and formed an abscess there. The other helpful features are: whereas in general peritonitis paralytic phenomena of bladder and rectum such as anuria and 'achezia' respectively will be prominent, and a short, tragic, fatal course will be the rule; in perimetritis, obstructive phenomena such as dysuria and dyschezia as also pressure on pelvic veins with consequent swellings of legs will be evident, and a slow, toxic, invalidating course will be manifested.

The differentiation of parametritis and perimetritis is important from a prognostic point of view, as the former is usually benign while the latter is seldom so. But, unfortunately, the two conditions often co-exist, and as such their identification will not be easy—at any rate in the early acute and subacute stage. The following, however, are noteworthy. In parametritis, the onset is late (usually between the 5th and the 10th day of the puerperium), the general symptoms are not alarming, the toxæmia and the suffering are not great, and the pain (the lesion being cellulitic) is of a constricting or compressing nature. Further, the local lesion being generally unilateral and in the vast majority of the cases left sided, the vaginal findings will generally be on the left side, at least in the early stages; the uterus will be (if it is to be) tilted and fixed laterally, and not centrally, and the pelvic tissues and the organs of the pelvis will be free, or at their worst be pressed upon by the effusion to produce obstructive symptoms. The swelling formed by the effusion is usually single, has a great tendency to rove, and prefers to do so anteriorly (by virtue of the anatomical distribution of the cellular tissues in the pelvis); if, however, it chooses to wander behind, it will surround the rectum like a horse-shoe, and not settle on it in front and pressing it. The inflammation itself will be found to be of a low grade form, the fibrinous elements preponderating and consequently the swelling tending to remain

hard and indurated, unless, of course, pus forms, which, however, is not very common. The swelling, whether indurative or suppurative, has a great tendency to burrow along the lines of least resistance, or rupture spontaneously especially if an abscess has formed. The natural course of the disease is towards resolution, recovery without untoward sequelae occurring in most cases. In perimetritis, on the other hand, the onset is early and sudden (usually abrupt on the 2nd or 3rd day of the puerperium), the general symptoms are severe, the toxæmia and the suffering are great, and the pain (the lesion being serous) is stabbing. The pelvic lesion will be bilateral, diffuse and indefinite. The uterus may be tilted to any side, but will usually be fixed centrally. The pelvic tissues will be bathed in serous exudations, and consequently will be spongy or matted and riddled with multiple miliary abscesses, showing thereby that the inflammation is of a high grade. The pelvic organs may be glued or adherent and generally pressed upon. The swelling formed by the gathering of the small pus collections will be soft, elastic and fluctuating, and show no tendency to rove, but, gravitating to the bottom, will settle as the well-known pelvic abscess in the pouch of Douglas in front of the rectum and pressing on it. The abscess is chronic, has no tendency to burrow nor to rupture spontaneously, and will induce a slow poisoning in the patient. The course is prolonged, complete sterility as the sequelae is the result in most cases. We have discussed in some detail the main features in the differential diagnosis of the symptom-complexes in puerperal infections. But, the discussion will not be complete without a final word of warning. Let it ever be borne in mind that face-to-face facts, in actual practice do not always agree with the studied and well-arranged descriptions in theses and text-books; piquant situations will arise when we feel pricked and become inclined to burst forth. For instance, scanty and a peculiarly sweet-smelling vaginal discharge in puerperal septicaemia, and a profuse foul bloody discharge with clots and debris in sapraemia are the time-old text-book descriptions; but, there may not be one amongst us but that he or she has not been confronted with a case where facts went a different way. In puerperal infections, the cause of the infection, in most cases, does not lie in the soil, or the seed, or again with the sower individually, but rest on an unwitting, yet formidable, combination of all the three; as such, no two crops can ever agree. It is for the mower to discriminate and be on the *qui vive* for all eventualities, if he wants to make the best out of a bad job—not to say, to win kudos! Faith, Hope and Charity are the triads of salvation; Facts, Judgment and Clarity shall be those of diagnosis.

SOME ELEMENTS OF NEUROLOGY (12)

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

Physician, General Hospital, Madras.

CEREBRO-SPINAL FLUID : COMPOSITION

Composition of Cerebro-spinal fluid similar to that of blood plasma : { 1. Minus almost all the protein.
2. Plus 2 to 4 cells, chiefly lymphocytes, per c.mm.

Protein :

1. *Albumin and Globulin :*
 1. Mere traces.
 2. Approximately equal proportions.
2. *No Fibrinogen :*
 1. Normal cerebro-spinal fluid will not clot on standing like blood or lymph.
 2. Clotting means that fibrinogen has escaped from the blood-vessels into the cerebro-spinal fluid as a result of excessive dilatation.
3. *Clotting therefore points to an inflammation of the meninges :*
 1. Meningitis;
 2. Poliomyelitis;
 3. Reaction after injection of sera, e.g., anti-tetanic serum into theca.

4. *In searching cerebro-spinal fluid for tubercle bacilli in tubercular meningitis you will often readily find the tubercle bacilli in the tiny clot in which they seem to get entangled.*

(If you suspect tubercular meningitis add sodium citrate to prevent clot.)

Tests for Protein :

Heat and Coagulation Test for Albumin and Globulin :

Normal cerebro-spinal fluid gives only the merest opalescence on boiling.

Nonne-Appelt Reaction for Globulin :

1. Half saturate cerebro-spinal fluid with ammonium sulphate—"salting out."
2. Ammonium sulphate is more soluble than the protein.
3. Protein is merely a colloid.
4. Hence, the protein is knocked out of solution and precipitated by the ammonium sulphate which takes its place.
5. The bigger globulin molecule is more easily knocked out than the small albumin molecule.
6. Therefore, in the Nonne-Appelt reaction only globulin of cerebro-spinal fluid is precipitated.

Technique :

1. To some cerebro-spinal fluid gently add an equal quantity of saturated ammonium sulphate solution.
2. A white ring of precipitate appears at the junction of the two fluids if the globulin in the cerebro-spinal fluid is increased.

Significance of Increased Protein :

1. Increased protein in cerebro spinal fluid is due to increased permeability of blood vessels.
2. Hence, its causes are the same as those of clotting: any meningeal inflammation, *e.g.*, meningitis, poliomyelitis, etc.
3. Usually albumin and globulin are increased hand-in-hand.
4. Excessive increase in globulin alone = syphilitic meningitis.
5. Slight increase in protein and strong Nonne-Appelt = syphilitic meningitis.

Chlorides :

1. Slightly more than in plasma, *i.e.*, 0.75 per cent (for purposes of maintaining the osmotic pressure of a fluid poor in colloids.)
2. *But the chlorides are not the only salts in the blood although they form the majority.*

Significance of Reduction in Chlorides :

1. *Blood chlorides :* Fall in any general infection, and are an index of the patients' resistance.

2. So chlorides are reduced in meningitis :

1. Below 0.7 per cent strongly suggests meningitis.
2. Reduction to 0.6 per cent or under : is absolutely diagnostic of tubercular meningitis.

Estimation of Chlorides :

Use a standard solution of Silver Nitrate, and Potassium Chromate *as an indicator.*

Sugar :

As in plasma = 0.08 per cent.

Significance of reduction of sugar content :

1. Dead pus cells liberate glycolytic ferments.
2. Hence reduction of sugar points strongly to meningitis.

Estimation of Sugar :

1. In ordinary way, *e.g.*, by McLean's method.
2. More usually a simple Fehling's test is employed.

Technique :

1. Make a Fehling's solution in the ordinary way using equal parts of Nos. 1 and 2.
2. Take 1 cc. cerebro-spinal fluid.
3. Add *one drop* of Fehling.
4. Boil the mixture.
5. Normal cerebro-spinal fluid gives a yellow or red precipitate a few seconds after bringing to the boil = Immediate reduction with Fehling.
6. If the sugar is reduced in amount
 1. No precipitate occurs,
 - or 2. Is very delayed in appearing.

Boveri Reaction for Tubercular Meningitis :

Specific for tubercular meningitis.

Technique :

1. A solution of permanganate of potassium is made up in water to give a light port wine colour, strength = 0.025 per cent.
2. It does not keep, so it is better to make it up fresh each time, judging the strength roughly by the colour.

3. To some cerebro-spinal fluid add an equal quantity of the permanganate solution.
4. Mixture decolorises within 3 minutes = positive boveri = tubercular meningitis.

Method of counting cells in cerebro-spinal fluid :

1. Use Thoma-Zeiss or Burkner instrument for counting blood cells.
2. Cerebro-spinal fluid must *not* be diluted.

Technique :

1. The whole field = 9 sq. mm.
2. Search the whole field—normally you find only a few cells.
3. Divide by 9 to bring it to 1 sq. mm.
4. Distance between cover slip and ruled lines = $1/10$ mm.
5. Therefore, multiply by 10 to bring the figure to 1 c. mm.
6. In doing an accurate count, repeat several times, washing slide and repeating with fresh drop of cerebro-spinal fluid each time.

Example :

Suppose the figure for successive counts are 3, 2, 5, 2, for whole field.
 Average = 3 per whole field.
 Divide by 9 = $3/9$ per sq. mm. = $1/3$ cell. per sq. mm.
 Multiply by 10 = $1/3 \times 10 = 3$ cells (approx.) per c. mm.
 Count may rise to several hundred cells per c. mm. in inflammation of the meninges, e.g., Meningitis, Poliomyelitis, etc.

Method of distinguishing type of cells : lymphocytes or polymorphonuclears :

1. Centrifuge some cerebro-spinal fluid.
2. Take a drop of deposit on slide.
3. Dry, fix and stain with methylene blue.

Increased lymphocytes = acute non-pyogenic infection :

1. Tubercular meningitis.
2. Poliomyelitis.
3. Encephalitis lethargica.
4. Syphilitic meningitis.

Increased polymorphonuclears = acute pyogenic meningitis :

1. Meningococcal,
2. Streptococcal,
3. Pneumococcal, etc.

Sterile Meningitis :

1. Irritation of meninges results from intrathecal injections of antitetanic serum.

2. Resulting cerebro-spinal fluid is exactly like that in meningitis :

- i. Quite turbid.
- ii. Cell count up to several hundreds per c.mm.

3. This sterile meningitis may have a curative effect.

4. Possibly, anti-meningococcal serum acts in this way, i.e., its curative effect in cerebro spinal fever may not be a specific one and might just as readily be obtained with horse-serum or diphtheria antitoxin.

5. This is supported by the fact that there is no complement in cerebro-spinal fluid (Boxwell).

6. Serum cannot act in the absence of complement.

Wassermann Reaction.

1. Wassermann reaction substance in blood is combined with the colloid globulin.

2. It cannot therefore under normal conditions leak through the capillaries of the meninges and choroid plexus, into the cerebro-spinal fluid.

3. But when there is meningitis with congestion of and damage to the capillary walls you get cerebro-spinal fluid with

1. Increased protein,
2. Globulin,
3. Positive Wassermann,

and 4. Lymphocytosis.

Colloidal Gold Reaction—Lange's Curve.

1. In parenchymatous syphilis, globulin is greatly increased.

2. As a direct result of increase of globulin the cerebro spinal fluid is able to precipitate colloidal suspensions.

3. Hence, *Lange's Colloidal Gold Curve*.

(Clotting is due to the escape of fibrinogen from excessively dilated blood vessels into the cerebro-spinal fluid).

3. Sugar is reduced because dead pus cells liberate glycolytic ferments.

4. Chlorides are reduced, as the blood chlorides which are an index of the patient's resistance, fall in any general infection.

5. Cells are increased (beyond the normal few lymphocytes not exceeding 6 per c. mm.)

(1) Lymphocytes are increased in acute non-pyogenic infections :

- i. Tubercular meningitis;
- ii. Poliomyelitis (later stages);
- iii. Encephalitis lethargica.
- iv. Syphilitic meningitis.

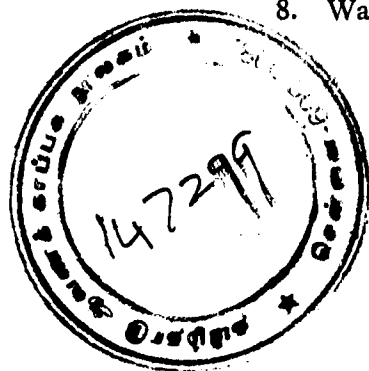
(2) Polymorphonuclear leucocytes are increased in acute pyogenic infections—

- i. Meningococcal;
- ii. Streptococcal;
- iii. Pneumococcal.

6. Causal organism.

7. Boveri reaction for tubercular meningitis.

8. Wassermann reaction.



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Technique :

1. Make various dilutions of cerebro-spinal fluid $\frac{1}{10}, \frac{1}{20}, \frac{1}{40}, \frac{1}{80}, \frac{1}{160}$.
2. Add the diluted cerebro-spinal fluid to the Colloidal Gold Suspension.
3. Note any change in colour.
 - No change of colour = 5 (i.e., complete precipitation).
 - Blue = 2.
 - Pink or Red = 1.

Normal cerebro-spinal fluid :

No flocculation in any dilution.

Luetic Curve—1 1 2 4 4 2 1 1.

- | | | |
|--|---|-------------------|
| <ol style="list-style-type: none"> 1. Tabes. 2. Syphilitic Meningitis. 3. Dessiminated Sclerosis. | } | Meningitic Curve. |
|--|---|-------------------|

Paretic Curve—5 5 5 4 3 2 1 1 0 0.

General Paralysis.

Colloidal Benzoin.

1. Cheaper ;
2. Easier to perform ;
3. More certain results.

Also—Collargol, Mastic, and Bi-coloured Guaiac tests.

Note.—

1. Lymphatic drainage of parenchyma of central nervous system is into subarachnoid space.
2. Hence in parenchymatous syphilis you get in the cerebro-spinal fluid
 1. Lymphocytosis ;
 2. Increase of protein ;
 3. Globulin in abnormal amounts ;
 4. Typical Colloidal Gold Curve ;
 5. Positive Wassermann. Wassermann Reactive substance is combined with the colloid globulin.

MENINGEAL SYNDROME.

1. Symptoms of fever ;
2. Symptoms of increased intracranial pressure ;
3. Symptoms of anterior root irritation ;
4. Vasomotor disturbances.
5. Cerebro-spinal fluid changes.

I. Symptoms of fever :

Modified by slow pulse, etc.

II. Symptoms of increased intracranial pressure :

Except optic neuritis.

III. Symptoms of anterior root irritation :

1. Stiffness or spasm of the neck muscles produces head retraction, and pain when the head is acutely flexed on the chest.

2. Stiffness or spasm of the hamstrings is responsible for Kernig's sign or inability to extend the knee with the thigh flexed at a right angle on the abdomen.

3. Brudzinski's two signs—

(1) Neck sign—When testing for neck stiffness both legs tend to be drawn up or flexed.

(2) Leg sign—When doing a Kernig's on one leg, the other leg is drawn up or flexed.

4. Attitude—The legs are drawn up.**IV. Vasomotor disturbance.**

Tache cerebrale.

V. Cerebro-spinal fluid.

1. Under tension (Normally, 60 drops per minute).
2. Albumin is increased due to increased permeability of the blood vessels : globulin is increased in syphilis.

Captain Inglefield Dall, Superintendent, Borstal School, Palamcottah. This is the first time that a Health Exhibition was held in the Borstal School, Palamcottah.

Lieut. Col. T. S. Shastry, I.M.S., the President of the Association welcomed the guests and requested Mrs. Dall to open the Health Exhibition. He explained to the audience the prevalence of Cholera in the District and how to prevent the same. He also explained the causation and prevention of other infectious diseases such as Tuberculosis, Small-pox, etc. Dr. P. S. Srinivasan, explained in easy Tamil the gist of what Col. Shastry said.

Dr. E. Arumainayagam, L.M.P., and Dr. P. Ananta Nayanar, sub-assistant surgeons of the Borstal School, explained in Tamil, the posters on the prevention, causation of Tuberculosis, Flies and Cholera for the benefit of about 400 inmates and families of the warders.

This was very much appreciated by the gathering.

THE MEDICAL PRACTITIONERS' ASSOCIATION, NUZVID.

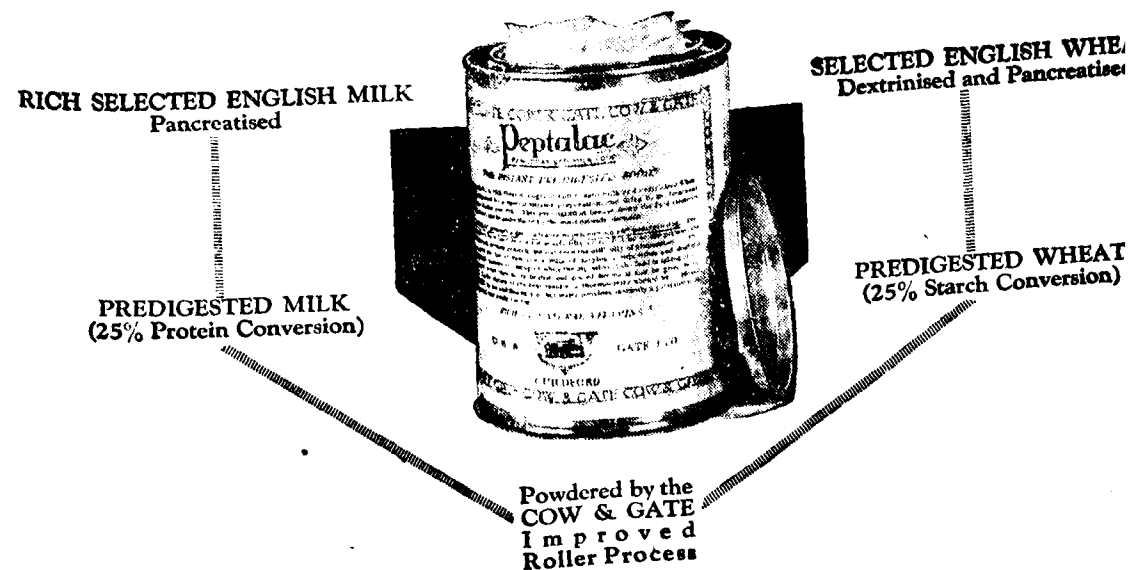
A monthly meeting of the above Association was held at Nuzvid in the Local Government Hospital on Sunday the 29th November 1936 with Dr. P. Anjaiah Naidu, L.M.P., in the chair. After light refreshments offered by Dr. T. Satyanarayana, L.I.M., who was at home to the members, the meeting commenced at 3 p.m. when Dr. N. Rama Rao, L.M.P., Secretary, read the minutes of the previous meeting which were duly adopted. The members then began to discuss on several professional topics of which were the following.

1. Mr. N. Rama Rao reminding the members of the case of neuralgia of lower extremity simulating sciatica that was demonstrated by him during the last Anniversary Meeting, said how the case was sent for consultation to Dr. Guruswami Mudaliar of Madras who diagnosed the same as Fibrositis due to faecal concretions resulting out of chronic constipation and also an old though transient attack of Gonorrhoea from which the patient claims a radical cure ever since. Under treatment by Gonococcal vaccine constipation relieving remedies and anti-neuralgic sedatives, the patient improved a good deal.

2. Mr. P. Anjaiah Naidu mentioned about a new born child of only 2 days ago that was born with moderately well developed upper and lower incisor teeth and as such caused much inconvenience to the mother while suckling and so he said he had to extract the teeth.

3. Mr. V. Ananthachari narrated the clinical course of a peculiar case that simulated Malaria for a time and then the fever began to come every Wednesday about a month. Anti-malarial remedies were not of much use but as the patient gave a history of syphilis some time back and also of a rat that scratched his sole just superficially as not to cause any bleeding the doctor was tempted to give Neosalvarsan which effected a relief rather surprisingly.

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5. PEPTALAC is therefore invaluable for infants, young children, the aged, convalescents, and the weak in health, who are prone to contract milk-borne diseases from ordinary milk or preparations made with it.
6. PEPTALAC is pleasing to the most fastidious palates—salt or chocolate may be added for further flavouring if desired.

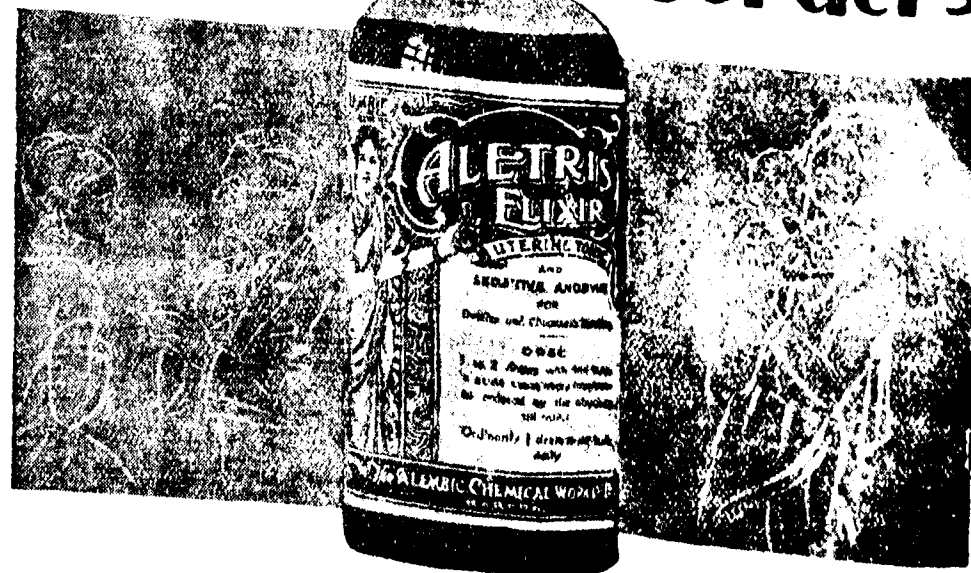
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ASSOCIATION NOTES.

THE TINNEVELLY MEDICAL ASSOCIATION, PALAMCOTTAH.

(Affiliated to the Indian Medical Association.)

The Tinnevely District Medical Association held its monthly meeting on Saturday the 21st of November 1936, at the St. John's College Hall, Palamcottah. Members numbering 54 including 7 woman doctors gathered from all parts of the District.

After Tea Party and sweet music, the meeting began under the Presidentship of Lieut. Col. T. S. Shastry, I.M.S. at 6 p.m. The Secretary read the minutes of the monthly meeting held at Srivaikuntam on Sunday the 31st of October 1936 which were passed.

Dr. P. G. G. Unnithan, M.B.B.S., B.S.C., D.P.H. (Hopkins), D.T.M. & H., (Eng.), District Health Officer, Ramnad, gave an interesting lecture on newer knowledge of "NUTRITION."

The following clinical cases were demonstrated:—

- | By whom | Details of the case. |
|--|---|
| 1. Lieut.-Col. T. S. Shastry, I. M. S. | A specimen of a case of Ovarian Cyst. |
| 2. Dr. P. S. Srinivasan, L.M.P. | A case of Duodenal Ulcer and "Burst Abdomen." |

Dr. P. S. Srinivasan, L.M.P., sub-assistant surgeon of the Government Headquarters Hospital, Palamcottah, read case notes of (1) A case of Septic Meningitis as a result of fall, (2) A case of Chronic Duodenal Ulcer and "Burst Abdomen," and (3) A case of rupture posterior tibial artery right leg. Dr. Miss A. Lazarus, L.M.P., of the Vannarpet Hospital, read case notes of a case of Ovarian Cyst, which was operated on by Lieut.-Col. T. S. Shastry, I. M. S.

The above cases and the lecture were fully discussed by the members. Then the President brought the meeting to a close by his remarks on the subject of the learned lecturer and summed up the salient and important features of all the cases. He thanked the Principal, St. John's College, Palamcottah, for placing the College Hall, at the disposal of the Tinnevely District Medical Association for holding the meeting.

Thereafter, an excellent Dinner brought the happy gathering to a close late at night.

HEALTH EXHIBITION.

Under the auspices of the Tinnevely District Medical Association, in connection with its monthly meeting at the Headquarters (Palamcottah), a Health Exhibition was arranged at the Borstal School, Palamcottah, on Saturday the 21st of November 1936 at 10 a.m. under the Presidentship of



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4. Mr. T. Satyanarayana demonstrated a female patient that was operated for cervical adenitis (excision) in the General Hospital, Madras, which later on resulted in a temporary paresis of the shoulder muscles. Now the patient, he said, is getting weaker and emaciating though she does not manifest definite tubercular lesions anywhere except that she complains of impaired movements at the left tip joint with limple which he suspects to be of tubercular origin though in its very early stage.

5. Mr. S. Hanumantharau mentioned a case of Amenorrhœa primarily and then developed very bad obesity and the later condition got controlled by Thyroid medication, though Amenorrhœa still persists. Very many topics of interesting nature were brought on discussion and the members decided not to have the next meeting in December being Xmas season. The meeting came to a close by 7 P.M.

THE COIMBATORE MEDICAL ASSOCIATION.

(Registered).

The eleventh annual meeting of the above association was held in the premises of the Moses Gnanabharanam Eye Hospital, Coimbatore, on the 5th December 1936. After a group photo at 5 P.M., the general body met under the presidency of Dr. C. N. Ananta Ranga Rao. The Annual Report was read and adopted, and new office-bearers for the coming year were elected. Dr. Rao Sahib Y. P. Vasudevan was elected President who now took the chair and the following office-bearers were elected.

Patron.—Major A. I. Cox, I.M.S.

President.—Rao Sahib Y. P. Vasudevan, M.B.B.S., B.S.SC.

Vice-President.—Dr. R. Sundararajan, M.B.B.S.

Secretary.—Dr. L. Seshadrinathan, M.B.B.S.

Asst. Secretary.—Dr. J. K. Gnanaolivu, M.B.B.S.

Committee Members.—Dr. N. Krishnaswami, L.M.S.; Dr. S. Gurbatham; Dr. K. Pratap; Dr. Mrs. Jayakaran and Dr. C. N. Anantaranga Rao.

Resolutions appreciating the services of the Patron, the outgoing President and Secretary were placed on record.

A resolution of condolence on the sudden demise of Dr. M. K. Sambasivan of Kumbakonam was passed the members standing.

A donation list for funds for the Association to have a building of its own was opened, and about Rs. 700 subscribed on the spot.

There was a musical entertainment after this which was followed by a Dinner. There was a large attendance, and the gathering dispersed after a pleasant time.

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