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PUERPERAL INFECTIONS* (2)

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Badagara.

III. DISTURBANCES DUE TO SYSTEMIC INVASION

(a) *Puerperal Sepsaemias or Toxaemias*.—These are the results of systemic absorption of toxins produced at some focus of mischief in the parturient canal. The toxins may originate in the decomposition of the various products retained or accrued in an entire or injured parturient canal; or, they may arise from some damage caused by the saprophytes that have gained entry into the tissues. In either case, the centre of the mischief is fixed, and the whole process may be compared to a cannonade as opposed to a cavalry charge. It should be obvious, however, that where saprophytes are present, the mischiefs are worse, for, as JELLET has pointed out, "It is probable that in many cases of putrid endometritis, irritation by saprophytic poisons is the first stage towards an infection by septic organisms, which in their turn produce superficial necrosis of the uterine wall and thereby supply further pabulum for the saprophytes."

The history in all cases is typical, and will speak of prolonged first and second stages with perhaps perineal tears, or a ragged placenta when removed, or an incomplete expulsion of the membranes, or there may be some devitalising factor like excessive haemorrhage.

The general symptoms are characteristic and more or less uniform, while the focal signs vary with the varying cause.

The onset is insidious and appears any day between the second and fourth, depending on whether the toxic absorption is from pent-up lochia or retained products of conception. The initial and the most urgent symptom

* Continued from June 1936, p. 167,

dependant. But, in more severe forms, more than one lobe may be involved, and, in such cases, the onset is usually earlier and the symptoms are severer.

The symptoms in 'puerperal mastitis' are as in acute inflammations elsewhere. There are the usual chill or chilliness, malaise and headache. The temperature shoots up and the pulse goes high correspondingly. The affected breast is visibly swollen, heavy, tense and very painful, especially in the usual hanging position. The skin over the involved segment is hot, red, oedematous and very sensitive to touch.

The course of the disease is generally short, and usually tends towards resolution. The initial high fever gradually comes down until the temperature becomes normal within 48 to 72 hours; and, with the abatement of the fever, the other symptoms also disappear except perhaps slight local tenderness which lasts for a few days longer. Should the fever continue or should there be recession in the decline of the fever, either there is pus-formation or the inflammation is spreading to an adjacent lobe; the former will manifest itself in the development of a fluctuating abscess while the latter will be evidenced by the shifting of the signs to the new zone. In cases of pus-formation, the abscesses may become localised, constituting 'mammary' abscesses, or burrowing their way to the cellular tissues underneath they may become 'sub-mammary'.

The prognosis is usually not bad. But, unless early and efficient measures are adopted, troubles may arise and permanent damage to the mamma may be caused.

Milk-Fever.—This phenomenon, as its very name indicates, is a usual accompaniment of the onset of active functioning of the breasts in a lying-in-woman. Its constancy and mildness have caused it to be considered as innocuous and almost functional, though there are many who consider the fever as distinctly pathological with an underlying mild infection for cause. In this connection, the following will be of interest, "Although the breasts exhibit evidence of activity from the early days of pregnancy, during which a secretion is usually passed out into the ducts, an abundant secretion is rarely present before the third or fourth day after delivery. (*The usual period for the onset of the 'milk-fever'!*—T. S. P.) The factors that determine this remarkable phenomena are still not understood. The hyperplasia of the secreting elements of the breasts, which occurs during the nine months of the pregnancy is believed to depend on the activating influence of some biochemical material which is elaborated in the placenta or in the tissues of the child, and which reaches the breasts by the blood-stream. It is sometimes stated as a partial explanation of lactation that during preg-

nancy this biochemical influence has an anabolic effect on the breast, and that its removal by delivery is followed by a katabolic change with the pouring out of the secretion. It is evident, however, that this explanation is insufficient to account completely for the phenomena".* It is a well-known fact that all katabolic changes are associated with the liberation of some amount of heat; this should happen much more then where the katabolism is high and occurs suddenly in tissues engaged in prolonged anabolism. No wonder then, "Often the milk appears in the breast suddenly, causing a great deal of discomfort and a rise of a point or two in the temperature".*

The affection, at any rate, is not serious. Excepting the fever which is mild and a heaviness of head, there are no other morbid symptoms, and the fever itself rarely lasts for more than 24 to 48 hours. The incident has been discussed here only to distinguish it from distinct sapraemias on the one hand and 'Mastitis in Puerperium' on the other; from the former it is differentiated by an absence of all local and the severer general signs, and from the latter by an absence of all inflammatory signs in the breasts.

(b) *Puerperal Septicaemias.*—A case of septicaemia is many times more serious than a case of sapraemia. In septicaemia, the organisms are not encaged, but are engaged; in other words, we have here a regular cavalry-charge as opposed to the cannonading we witnessed in sapraemia. Moreover, the organisms in septicaemia are of high virulence, and, in most cases, the resistivity of the patient is very low.

Acute puerperal septicaemia is usually the result of a rapid and widespread extension of septic infection from some original focus along the lymphatic spaces and into the lymphatic vessels and glands whence it enters the blood-stream. Less commonly, the blood-stream becomes directly inoculated as in cases of manual removal of placenta where the hands are brought into direct contact with the placental sinuses which are as large as or even larger than the vena cavae. Lastly, instances are not rare where a case of puerperal infection begins as sapraemia and ends in septicaemia.

In primary cases, the onset of symptoms is abrupt and acute. On any evening between the second and fourth day of the puerperium, fever ushers in with rigor. But, where the condition is one super-imposed on an existing saprophytic infection, the onset may be on any day between the third and seventh, when there occurs a sudden exacerbation of the symptoms already present. It is interesting to observe too that, after the seventh day, acute septic infections of the type we are discussing are rare.

* "Combined Text-book of Obstetrics and Gynaecology", by Kerr, Fergusson, Hendry and Young, First Edition. p. 507.

In a typical case, the temperature is irregular from the beginning and shows frequent and wide variations, ranging between hypo- and hyper- pyrexias. Towards the end, in severe cases, the hyperpyrexia is almost continuous and may reach even 110° F. Rigors, indicative of fresh infections, are frequent and troublesome. Sweats in the early stages are invariable and profuse, but, later on, they may cease altogether and the skin may become dry and shrivelled. The pulse is rapid, feeble, irregular and of low tension and is distinctly thready or wiry at the end. It rises with each rise in temperature but shows no corresponding fall, and so soon becomes out of proportion to the temperature. The face is hot and hectic at first, but as the disease progresses and the patient gets very ill and exhausted, it becomes worn and waxy. The eyes are ominously brilliant and the conjunctivae are red or pale yellow. The tongue at the onset is sore and red, but quickly gives place in most cases to an aphthous condition. The skin shows erythematous or distinctly purpuric spots, and, in exceedingly severe infections, typical scarlatinous eruptions may appear.

Heart sounds are feeble, rapid and irregular; and cardiac disturbances due to toxic myocarditis will be present. Breathing is laboured and respirations are rapid. Congestive bronchitis is common, but cough may or may not be present. Typical pneumonia is rare, and, if present, is usually lobular. Breasts are lax and flaccid; and mammary secretion is completely absent if the disease has set in before the secretion has commenced, and is much lessened in other cases.

The abdomen is painful, tender, tense and distended. The pain may be slight or severe, and localised or general. The tenderness is present all over, but is particularly severe over the flanks—probably due to tubal mischief. The distension is not great and is more marked above the navel, being caused chiefly by a distended colon with inhibited peristalsis. Liver and spleen may be palpable, and are tender to pressure. Constipation is the rule in the early stages, but uncontrollable diarrhoea may set in towards the end.

Urine is scanty and smoky and may show signs of actual nephritis, especially in scarlatinal infections where there may be even actual suppression.

Headache which is so constant and troublesome in sapraemia is usually severely absent here; indeed, the presence or absence of headache in puerperal infections is a feature of extreme diagnostic importance. Delirium is a pretty early symptom, and in many cases is curious in that it centres round serpents and death; and, what is important from a prognostic point of view, such cases rarely recover. The delirium, as a rule, does not last long;

within 24 to 48 hours coma sets in, and, when that occurs, the end can pretty safely be predicted in most cases. An ominous brain symptom, that is more frequent in chronic cases but not uncommon in the acute ones, is the pose of extreme well-being of the patient both in the early and last stages. In the early stages, the patient looks surprisingly well for all her temperature and other grave clinical manifestations. But this does not last long, and sooner or later she begins to show evidences of rapidly growing weakness. Yet, in her last stages, when the higher centres are dulled and euphoria sets in, she again becomes wonderfully hale and cheery—but, alas, with the final healer Death at the threshold!

The local signs follow an inverse course to that of the virulence of the attack. All discharges per vaginum are completely absent in purely septic cases, and when saprophytes are also present they are scanty—almost to nullity. In any case, there is no foulness about them; on the contrary, they may show a peculiarly sweetish odour.

Vaginal examination will show a variably mobile, normal-sized uterus, rather doughy and often tender to pressure. In slight cases where the primary uterine lesion has become well-marked and in early stages where the infection has just spread beyond the uterus, the uterus shows all the signs of "septic" endometritis—the surface is covered by a dirty, yellow, fibrinous exudate as in ulcerative tonsillitis, and small pus-collections may be present; the muscle tissue is turgescient, oedematous and shows distinct metritis; there may be acute tenderness over the lower abdomen as a result of parametric complications; and the very involution of the uterus may be retarded, especially where saprophytes too are present, when the organ will be large, its walls thickened, its tissues friable, and the exudate purulent or bloody. In severe cases, on the other hand, the uterus is severely normal with a strange smooth interior, firm walls and no sloughs. Other pelvic organs like the tubes and the ovaries may or may not show any definite signs. The pelvic veins may be thrombosed, especially in cases of manual removal of placenta. The thrombus may be limited to uterine and ovarian veins or may spread to the iliac ones, and can be felt per vaginum or per rectum. In the presence of thrombosis, the pulse exhibits a peculiar 'picking' or 'plucking' character—a phenomenon first described by MAHLER and so known sometimes as 'Mahler's' symptom; it more or less resembles the rapid motion of the fingers of one who plucks feathers (JELLET). In such instances, the temperature too shows an irregular rise, and micturition may become painful.

The course of the disease depends on the severity of the infection. In a very virulent type, the temperature rises with each rigor and the pulse

gets more and more collapsed and indeterminable. In two or three days, brain symptoms supervene, and the patient becomes restless and delirious. A day or two more, and the picture entirely changes; the bouts of fits are no longer present, and there creeps in a long and continuous state of unconsciousness which lasts for 12 to 24 hours and ends only with the patient's end. The whole course is galloping, so to speak; and the duration rarely exceeds four or five days, though it may, in some instances, last a week.

In a good proportion of cases, the patient survives the first shock, and the disease takes a chronic course, but, in the majority of cases, only to allow the patients to die a death of slow and prolonged poisoning—complete recovery resulting in but a limited few. In such cases, the fever continues, is never high, and becomes definitely septic, with great remissions or intermissions. Rigors and sweats are inconstant. Pulse is distinctly asthenic; it is small, soft and easily compressible. The general appearance of the patient is typical of one in prolonged ailment—she is emaciated almost to skin and bone, with eyes sunk, face pinched, malar bones prominent, and expression languid and lustreless; progressive haemolytic anaemia or jaundice is quite apparent, and, in prolonged cases, extreme; the skin is dry, sallow, shrunk, and scaly or branny, and shows rashes as in malignant endocarditis. Chest signs are invariable. Dyspnoea and palpitation are common. Dilatation with embryocardia and murmurs (organic or haemic) are usual, especially towards the end. Productive bronchitis and troublesome cough are frequent; septic broncho-pneumonia is not uncommon; hypostatic congestion is rarely absent. The abdomen will be scaphoid and usually show generalised pains which may be severe over the flanks and below the umbilicus. Signs indicative of loss of 'digestive fire' will be always evident: thus, anorexia will be extreme, and nausea or vomiting may be severe, especially in presence of acidosis or alkalosis; motions are usually loose and foul and may contain undigested particles, and the diarrhoea in many cases becomes lienteric and terminal. Urine will generally show signs of damage to the kidney or the bladder. Uterus is usually normal; the adnexa will be found to be affected to a small or great extent, but they are usually impossible to be defined in the general matting of the pelvic tissues. Thrombosis of the veins in the pelvis will be marked and the thrombosed veins will be felt per vaginum or per rectum as raised thickened cords—as thick as one's fingers. The treacherous symptom of *euphoria* is very common in chronic puerperal sepsis, and very often puts the medical attendant off his or her guard in pronouncing a prognosis. In the ups and downs of the disease there are periods indicative of imminent death quickly followed by short or long periods of abatement, or semblant recovery. The patient in these

cases does not as a rule suffer much and complains little; and in her last stage, when death has placed its first heavy foot on the threshold and waits for but the last light wind to blow out for the completion of its work, the patient rallies round wonderfully and becomes exceedingly cheerful and optimistic—as in the case of a dimming light that would shine with more than its wonted brilliance when its end is nigh.

Prognosis in acute puerperal septicaemia is always grave, only a small percentage escaping death. When resolution occurs, recovery is usually complete.

In any case, the ominous features are—the time of onset (the earlier this is, the more serious will be the prognosis); frequent rigors; great swings in the temperature; a progressively feeble and rising pulse; brilliant eyes; severe skin eruptions; suppression of urine; early onset of brain symptoms, especially visions of snakes and death and euphoria, if it is, as is rarely, present.

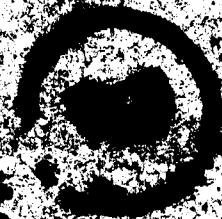
In chronic cases, one should always be guarded in pronouncing an opinion. Bright and dark phases are common, and quickly alternate. And in coming to a conclusion, one should always be guided more by the severity of the general signs and the type of the organism found than by the subjective symptoms and local signs, as the latter are particularly vagarious. The prognosis in chronic puerperal sepsis is usually bad, and especially so in the presence of diarrhoea and cardiac manifestations. A few recover; but, of these, a good number are crippled for life, as the various degenerations in the course of the disease leave lasting impressions on some vital organ or other.

Puerperal Scarlet Fever.—Puerperal women are not immune to this specific affection; on the other hand, they are exceedingly vulnerable to it. But it is a moot point if any high fever with throat and ear symptoms and scarlatiniform rashes in the course of a puerperium can indiscriminately be dubbed 'Scarlet Fever' or 'Puerperal Scarlet Fever'. Unfortunately, the specific organism in the specific scarlet fever has not, as yet, been identified*; in puerperal infections, a variety of organisms comes into play; and amongst

* "A. H. G. Burton and A. R. Balmain found that 191, or 75 per cent out of 265 cases of scarlet fever on admission to hospital showed haemolytic streptococci in the fauces. Of these 46.1 per cent could be classified under one of Griffith's four groups (of which types 1 and 3 caused severe attacks, and 2 and 4 mild attacks), the remaining 53.9 per cent being inagglutinable with the type sera. Certain of these strains became agglutinable on repeated sub-culture. In view of the large percentage of inagglutinable haemolytic streptococci, Burton and Balmain regard it as impossible to accurately correlate the type of streptococcus with the clinical type of scarlet fever." J. D. Rolleston in 1931 *Medical Annual* (p. 421).

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the organisms found in puerperal infections, there are some hæmolytic streptococci, which (though they do not belong to any of the groups of the organisms found so far in scarlet fever) do cause lesions typical of scarlet fever. Under the circumstances, it is questionable how far the general nomenclature 'Puerperal Scarlet Fever' will suit all conditions. It would seem better to group all such fevers under one general head of 'Scarlatinal Fevers in Puerperium,' reserving the name 'Puerperal Scarlet Fever' strictly to cases of specific infection that may occasionally appear in the course of a puerperium, and calling the rest "Puerperal 'Para-Scarlet' Fever" or by some such name. This is of special importance and interest to us in India, as, in this country, the specific scarlet fever is not common, while high fever with scarlatiniform rashes and with or without throat and early symptoms (the values of which are, as we shall see, uncertain) are not uncommon; indeed, under the changed conditions, we shall have only 'Scarlatinal Fevers in Puerperium' or 'Puerperal Para-Scarlet Fever,' and no Puerperal Scarlet Fever.

The position of the entity known today as 'Puerperal Scarlet Fever' can be gauged from the following words of BECKWITH WHITEHOUSE and J. D. ROLLESTON, in 1931 *Medical Annual*, and the position has not revolutionised since then. According to BECKWITH WHITEHOUSE on page 390, "The relationship, clinical and pathological, which scarlet fever bears, if any, to certain cases of puerperal streptococcal infection has long been of importance to the obstetrician. The clinical details associated with two small recent epidemics of scarlet fever at the Lariboisiere Hospital, Paris, are therefore of interest. Six cases occurred at this hospital, *all in primiparae*, during an interval of two months. In one instance, infection followed an abortion. The remaining women were at term.....The second epidemic consisted of four cases.....L. DEVRAIGNE, L. BAIZE, and M. MEYER (*Bull. Soc. d'Obst. et. Gynecol*, 1929, xviii, 337), who report the epidemics, note that *the contagiousness appears to have been slight*, since the wards were crowded at the time, and persons at a distance contracted the disease—not those in close contact. *In each case, a very definite infection of the genital tract was present, and none of the women had the type of pharyngitis usually present at the onset of scarlet fever.* The genital tract therefore appeared to be the portal of infection. The physician, however, who contracted the infection developed a characteristic sore-throat. *Discussing the differential diagnosis between scarlet fever of puerperal origin and a streptococcal puerperal erythema, the authors lay stress upon vomiting and a very rapid pulse-rate in the former. Streptococcal erythemas usually appear only in cases of severe generalised puerperal infection with all the signs of a grave septi-*

caemia. In all the Lariboisiere cases the actual puerperal infection was very discreet. None of the infants contracted scarlet fever.....In this report an obvious criticism is that no reference is made to the bacteriological investigation of the genital tract. *The occurrence from time to time in parturient women of cases typical of scarlet fever and of others showing scarlatiniform erythema with streptococcal septicaemia appears to point to local infection of the genital tract with different strains of the same hæmolytic streptococcus.* The favourable results attending treatment with **Anti-Scarlet-Fever Serum** in the majority of cases of streptococcal infection of the parturient canal without clinical erythema afford further confirmation of this." J. D. ROLLESTON contributes on p. 422, "According to P. BAIZE and M. MEYER (*Presse Med.* 1929, 1007) who report six cases in women aged from 17 to 39, all but one of whom were primiparae, the etiological features of *puerperal scarlet fever* are as follows: *Two-thirds of the cases occur in primiparae. In most cases, the source of infection cannot be detected.* In the great majority, the disease sets in on the third to the fifth day after delivery. *The symptoms differ but slightly from those of ordinary scarlet fever.* The severity of the attack varies in different epidemics. As a rule the prognosis is likely to be severe when the interval between delivery and the onset of symptoms is short. As regards the pathogenesis, *two groups of puerperal scarlet fever may be distinguished: the first consists of cases of ordinary scarlet fever which has developed as a mere coincidence in the puerperium, while in the second and much larger group the attack has resulted from an endometritis which is usually slight. These two groups can be distinguished from puerperal scarlatiniform erythema by the Schultz-Charlton reaction.*"*

(c) *Puerperal Pyaemias.*—These are met with where the blood-stream is riddled with organisms, or where fragments of broken thrombi circulate in the blood.

The symptoms, unlike those in septicaemia, are distinctly remittent and appear later, generally after the seventh day of the puerperium, and often preceded by manifestations of some local septic lesion. The chief features are wide swings in the temperature, recurrent rigors, metastatic foci of infection and multiple abscesses. The temperature ranges from hypopyrexial to hyperpyrexial states, and in some cases the remittency becomes persistent and continuous. The abscesses may be formed anywhere—wherever the broken thrombi lodge. The commoner sites are the lungs, and the pleura and the joints; but the brain, the kidney and the spleen are not exempt.

* In the quotations the italics are mine.—T. S. P.

The course and prognosis depend on the virulence of the attack, the resistance of the patient, and the site and the severity of the metastatic lesion. In severe cases, the picture is one of lymphatic sepsis with abscesses, and such cases are always grave.

(d) *B. Coli Infections in Puerperium*—Infections with *Bacillus coli* in puerperium are fairly common. According to GREEN-ARMYTAGH of the Eden Hospital, Calcutta, *B. coli* infections after obstetric and gynaecological operations are a common feature of the tropics, and he has proved bacteriologically that 30·2 per cent. of the fevers subsequent to confinement in the tropics are due to *B. coli* infections.*

As regards the aetiological features the same writer remarks "The greatest pains have been taken to investigate the aetiology of these infections: the catheter, though possibly an occasional factor, could only be blamed in less than 5 per cent of cases, for the greater percentage of the patients had no catheter passed. The infections are just as frequent in Indians as in Europeans; consequently, diet can be no causal factor. It occurs in private patients with their own nurses as frequently as in ward cases. The most predisposing cause would seem to be retention of urine for more than eight hours."

Bacillus coli is a normal and usually harmless inhabitant of the bowels; but, under certain conditions, it strays and becomes distinctly pathogenic, giving rise to the 'entero-renal' syndrome. During pregnancy, the pressure of the gravid uterus on the ureters causes a stasis of the urine there, and leads to a damming back of it in the kidneys - both affording ideal conditions for the thriving of the bacilli which therefore make their egress to their new and more congenial abode when conditions demand. In the process, it is generally the right kidney that becomes selected—probably because the colon covers a larger area of and comes in closer contact with the right kidney, and also perhaps because the rather fluid contents of the ascending colon help the bacilli to bore their way easily through the mucosa there. But it should not be forgotten that the anatomical position and the growth of the gravid uterus are such as are liable to cause greater damage to the right ureter. The pregnant uterus, as it grows, tends to turn round its vertical axis slightly towards the right, thereby causing some kinking of the right ureter; and, besides, the right ureter, owing to its position of being farther away from the mesial line, is afforded less protection against pressure by the pelvic bay. In a large number of cases in pregnancy, however, the

infection is haematogenous. The proverbial constipation in the woman becomes much increased by pregnancy, and the resulting stasis in the intestines probably produces slight abrasions or otherwise injures the mucosa. The organisms, now that the intestines are damaged, force themselves into the blood stream to seek fresh and more congenial spots; and when the system excretes them by the kidneys, the damming back of the urine and the damage to the pelvis and the ureter afford them a favourable medium to stay. The same phenomenon happens whenever the intestinal mucosa is injured. And, indeed, as the author in the *Combined Text book of Obstetrics and Gynaecology** has remarked, "Very frequently, there is a history of some enteritic disturbances before the attack of pyelitis takes place."

To sum up, the conditions in pregnancy conduce to make the urinary tract (especially the pelvis of the kidney and the upper part of the ureter) cosy for the *B. coli*, and this happens to a much greater extent in presence of gross lesions like tumours and displacements, as these lead to disturbances of micturition or leave residual urine. When there occurs in the intestines any disturbance that breaks the equanimity of the bacilli there, they force themselves out and seek fresh spots; they may do so directly or, as more often happens, via the blood-stream, and in the latter event come to stay in the already prepared spots in the urinary tract as they are being excreted by the system. The stage becomes thus set in pregnancy for the play of these during the puerperium.

BERRINGTON and WRIGHT have shown that operations on the urethra are followed by bacteraemia, and, according to JOHN W. H. EYRE, "A slight and transient bacteriuria due to *B. coli communis* and one usually passing off without any treatment can frequently be observed following operative measures upon the rectum or anus or the organs of generation."† And delivery, whether natural or aided, practically constitutes an operation on the uterus!

In puerperium, the *B. coli* infections, if the kidneys are intact, are almost always haematogenous. But, where the kidneys always harbour the organisms, the infections may arise in their pelvis. Injudicious catheterisation is responsible in not a few cases. It is suggested that, in some cases at least, the infection is a direct one from the anus via the perineum to the urethra and thence to the bladder and the kidney. Superficial findings, like the almost next-door neighbourhoods of the anus and the urethra, the frequent injuries to the perineum or its weakened state consequent on its stretching during labour, the use of unclean vaginal pads constantly met with in

* *Indian Medical Gazette*, 1928, p. 155.

* 1923, p. 268.

† An Index of Differential Diagnosis of Main Symptoms 1923, p. 70.

outside cases, and a host of such other findings may lend support to the suggestion of direct infection—indeed, may suggest that in puerperium, the urethral route is the commonest. But facts observed are otherwise. Clinical urethritis is unknown, and where both pyelitis and cystitis exist, the former has almost always forerun the latter, which could not have been the case if the infection had been from the bladder to the kidney. Such direct infections, therefore, seem to be exceedingly rare.

With such potentialities for infection, it is small wonder that *B. coli* affections are so common in parturient women. It has been found that the bacillus grows well in urine with a pH of 5 to 9 and that a pH of 4.6 to 5 on the acid side and a pH of 9.2 to 9.6 on the alkaline side are inimical to it. Perhaps further bio-chemical researches on the pH values of the different tissue-fluids under varying conditions and with particular reference to the behaviour of the colon bacillus to them may throw some light on the high incidence of *B. coli* infections in the tropics.

The affection may manifest itself as bacillaemia or as bacilluria and may be accompanied—though not always so—by gross lesions in the kidney or the ureter or the bladder.

Bacillaemia occurs when the organisms are in the bloodstream, and sets in sharply on the second or third day of the puerperium and is often attended by rigor. Within 12 to 24 hours the temperature falls, but only to rise again within another 12 to 24 hours. The picture continues and the temperature chart becomes a spiked and steepled one. With the fever, there appear the usual febrile manifestations like headache, malaise, prostration etc. The pulse is slow for the temperature, of good volume and tension, and regular. Tongue is generally clean, eyes are bright and normal, expression is assuring and the general condition is good. Chest will be normal and the breasts will be functioning as usual. Abdomen may show flatulence, but tenderness and rigidity are common; there may be slight pain on pressure over the loins (especially the right) if the kidney is affected to any extent. Vaginal discharge may be increased and is usually stinking; it may be blood-stained or frankly haemorrhagic. The condition of the urine will depend on the state of the kidney. The course in bacillaemia is usually short. The organisms are either eliminated or they settle themselves down in some viscera, usually the kidney or the gall-bladder. Rarely, the affection tends to continue with intermittent exhibition of symptoms.

Bacilluria occurs when the urine freshly voided contains *B. coli*. Where bacillaemia has just preceded it, it is usually a necessary complication of the latter; in fact, in such cases, bacilluria begins where bacillaemia ends.

In old infections with the bacilli well settled, bacilluria starts in some depot in the urinary tract. The onset and symptoms in bacilluria are much the same as those in bacillaemia, but with these differences—the initial rigor may be absent, the fever is usually less, and the spikes in the chart are less conspicuous; the pain in the abdomen, if present, is central and below the umbilicus, and not in the loins; frequency of, and pain or burning during, micturition will be present, and urine may be tinged with blood and will show rod-shaped bacilli under the microscope. The course in bacilluria is treacherous, and is to a large extent controlled by the efficiency of the treatment adopted and the time at which it was instituted. Discovered early and treated adequately, many cases recover completely.

It is important to note that the finding of *B. coli* in urine *per se* does not mean infection of, or injury to, the kidney. If such a comparison may be instituted, the presence of *B. coli* in urine connotes what that of sugar in urine does, and nothing more nor less; even as glycosuria *per se* does not mean damage to the pancreas, bacilluria does not mean damage to the kidney. And indeed as glycosuria is due to the blood-sugar rising beyond the renal threshold, likewise bacilluria is due to bacillaemia getting beyond the renal control. In other words, both are 'excretion of the excess' phenomena. Further, just as a glycosuria may in course of time bring about renal injury, so also a bacilluria may cause damage to the kidneys.

The above affords us a clue for the inconstant behaviour of the urine and the urinary tract in most *B. coli* infections. So long as the phenomenon is merely excretory, the urinary tract remains uninfected, and the urine is only febrile. In many cases, however, the organisms irritate the mucosa as they are being discharged and then the urine may show the following features—it may present any tint from smoky to bright red by reason of its admixture with blood; the reaction is usually acid and the acidity is often of such a degree as will inhibit the growth of the organisms *in vitro*; albumin may be present to a varying extent; and the centrifuged deposit may show under the microscope blood-cells and renal epithelium in addition to the *B. coli*, but leucocytes beyond their normal limit and degenerate leucocytes will be absent. The process of excretion and consequent irritation, when prolonged, may result in inflammation, in any part of the urinary tract—usually (and for anatomical, histological, and possibly also bio-chemical reasons) the pelvis of the kidney, which then becomes the nidus for gross infections. When this occurs (for the matter of that, when any part of the urinary tract becomes infected), the symptoms already present become exacerbated, and fresh symptoms peculiar to the region affected supervene. The urine shows

marked changes, depending on the severity of the infection. In moderately severe cases, it is usually of a straw colour with a shimmering or shot-silk opalescence, and emits a peculiarly fishy odour, especially if kept long. The reaction is usually acid, but, in the presence of pathogenic saprophytes, it is usually alkaline, due to the ammonia from the decomposition of the urea by the saprophytes; occasionally the day urine alone is neutral or faintly alkaline, the cause of which is not known—probably metabolic or biochemical. The specific gravity is low; blood is absent, albumin is present, and pus is constant; the pus, when measured by the centrifuge, may be little or amount to a good percentage of the total bulk of urine. The centrifuged deposit will show, in addition to the germs, a large number of leucocytes, live and in different stages of destruction, tube-casts, and disintegrated epithelium.

The prognosis in *B. coli* infections is always uncertain. In rare instances, either at the onset or during the course, a fulminant type occurs where an initial hyperpyrexia and complete or partial suppression of urine leads to early coma and death. Barring in such instances, much depends on the early diagnosis and adequate treatment. Many cases recover, but also an equal number relapse with symptoms cropping up intermittently. Among those that recover, many retain bacilluria and remain healthy, while there are others in whom evident pyuria lingers with no untoward symptoms. There are cases too where chronic intestinal disorders originate from a first attack of *B. coli* infection. The following is a typical example of the same. A thin but active young lady aged about 30 years and mother of eight children had irregular fever for three days, the temperature ranging between 101° and 103°F., and diarrhoea, consisting of six to eight large, loose and almost painless motions a day. On close enquiry, it was elicited that the diarrhoea was one of 8 years' duration, dating from her fourth confinement which had a febrile puerperium, and that she had no other trouble whatsoever since then except occasional burning during micturition which was present now too. Examination revealed general tenderness over the abdomen which was however particularly marked about the caecum. Her urine was examined, and it showed all the features of bacilluria minus the microscopic findings (which were not carried out). Helmitol and alkaline diuretics alternately controlled the fever in 48 hours but the diarrhoea continues though it has much improved.

SOME ELEMENTS OF NEUROLOGY (10)

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POLYNEURITIS *

DIPHTHERITIC PARALYSIS

1. The exotoxin of diphtheria is highly selective for nervous tissue and some form of paralysis occurs in a very high proportion of the cases.
2. The intensity of the paralysis bears no relation to the severity of the local infection, for cases in which the original disease has passed unnoticed may be followed by serious damage to the nervous system.
3. The nervous manifestations of diphtheria fall into three distinct groups—
 - (1) Local = Paralysis of the soft palate;
 - (2) Specific = Paralysis of accommodation;
 - (3) Generalized = Multiple neuritis.

I. Local = Paralysis of the Soft Palate

1. Local paralysis occurs in parts related anatomically by nervous connections to the site of the diphtheritic lesion.
2. In faucial diphtheria, the local palsy appears in the palate.
3. In extra-faucial diphtheria, *e. g.*, infected sores on the limbs, the local palsy appears in the muscles supplied by the segments of the cord to which afferent nerves from the infected focus pass.
4. The reason for this is: the toxins elaborated by the Klebs-Loeffler bacillus ascend from the primary focus to the cord or the medulla.
5. Having reached the central structures, they diffuse to neighbouring motor cells and by injuring them cause paralysis of the muscles they supply.
6. Paralysis of the palate, therefore, does not occur except in faucial diphtheria.

* Continued from page 212.

Paralysis of the Soft Palate

As faucial diphtheria is the commonest form, the most frequent nervous symptom is paralysis of the soft palate.

1. It is shown by—

(1) The nasal quality of the voice,

and (2) The regurgitation of fluids through the nose.

2. As a rule, the weakness is bilateral and equal.

3. But in some cases it is greater on the side on which the local lesion is more severe.

4. Onset—(1) About the end of the second week ;

(2) But it may come on as early as the 4th day and as late as the 6th week.

5. The soft palate is relaxed and its movement on phonation is diminished.

6. The palate may be insensitive and its reflex is often diminished or lost.

7. Recovery usually occurs in a few weeks.

8. In rare instances there is paralysis of—

(1) The muscles of the pharynx,

and (2) The vocal cords.

9. Together with palatal palsy, it is common to find weakness and tenderness of the sternomastoid and masseter muscles—these are also local effects.

II Specific = Paralysis of Accommodation

1. The specific manifestation of diphtheria is paralysis of accommodation ;

2. Like trismus in tetanus, it is not due to a local lesion, but occurs in many cases, whatever the site of origin of the toxins ;

3. It is present in cases of both faucial and extra-faucial diphtheria ;

4. And is the local effect of exotoxin accepted from the general blood stream ;

5. Its onset is about the same time as the palatal palsy, perhaps a few days sooner ;

6. The reaction of the pupils to accommodation as well as to light can almost always be obtained ;

7. The trouble is subjective and is shown by defects of near vision, e.g., by inability to read small print or thread a needle ;

8. Hypermetropes suffer great inconvenience ;

9. In myopes it may pass unnoticed ;

10. Paralysis of any of the extrinsic ocular muscles with strabismus and diplopia may occur and this may be either nuclear or peripheral in type.

III. Generalized = Multiple Neuritis

1. Multiple neuritis follows extra-faucial as well as faucial diphtheria ;

2. It is also a result of the action of exotoxin circulating in the blood ;

3. Multiple neuritis usually comes on 2 to 3 weeks after recovery from the throat infection ;

4. Its presence is often detected when patients begin to exercise themselves during convalescence.

5. Common *early symptoms* are—

(1) Weakness and aching pains in the legs ;

(2) Unsteadiness in walking ;

(3) Clumsiness in performing fine movements with the hands ;

(4) Feelings of pins and needles in the extremities.

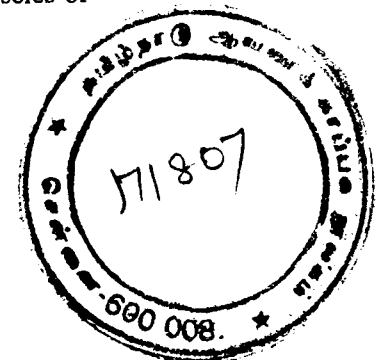
6. *Weakness* affects in varying degree the muscles of—

(1) The neck ;

(2) The trunk ;

and (3) Limbs.

7. It is generally slight in degree ;



8. And is greater in the lower than in the upper extremities and greater in the extensors than in the flexors.

9. Marked local atrophy is uncommon.

10. In severe cases, life may be endangered by paralysis of the Inter-costals and of the Diaphragm, but fortunately one set of muscles has usually begun to recover before the other is seriously affected.

11. The small muscles of the hands and feet and the muscles of the calves and forearms—

(1) Are almost always tender on pressure;

(2) They are soft and flabby;

and (3) Often show partial Reaction of Regeneration.

12. *Sensory Ataxy*:

i. Sensory ataxy is almost always present and is often severe when the paralysis is trivial.

ii. It causes the patient great inconvenience as it interferes seriously with walking and with the finer movements of the hands.

iii. Objective examination reveals sensory impairment of the "glove and stocking" type.

iv. On the hands and feet—

(1) The loss to slight tactile stimuli is often complete;

(2) Pain and temperature being less affected.

v. As the limb is ascended, sensation gradually becomes normal.

vi. Even when the sensibility of the skin is but little diminished the sensations of position and of passive movement in the extremities are often seriously impaired and the sense of vibration is often lost.

13. *The Reflexes*:

i. Compared with the symptoms already described, loss of the tendon reflexes is a late sign.

ii. In the early stages they are exaggerated but are lost later in every case.

iii. Their return is often long delayed and it is common to see patients, months after recovery of normal power, in whom the knee-jerks are still absent.

iv. It is common also to find them absent many months after an attack of diphtheria in patients who give no history of nervous symptoms during the attack.

v. The skin reflexes are usually retained.

vi. The plantar response is the normal "flexor".

14. *Eye Signs*:

i. The external ocular muscles are sometimes paralysed.

ii. The most common defect is weakness of the internal recti, which causes a slight divergent squint, like that of myopia.

15. *Cardiac Failure*:

i. Is a grave but uncommon complication.

ii. It is not yet decided whether it is attributable—

(1) To paralysis of the vagus (neurogenic);

or (2) To degeneration of the heart muscle (myogenic).

16. *Vasomotor and Trophic Disturbances* which occur so often in other forms of peripheral neuritis, are never seen in diphtheria.

Prognosis:

In those that survive the attack, complete recovery from the nervous troubles always occurs.

DIABETIC NEURITIS

1. In many patients with glycosuria symptoms are present which point to:

(1) Changes in the peripheral nerves,

or (2) In the fibres of the posterior roots.

2. In many respects they resemble *talcs* rather than peripheral neuritis.

Pathology:

1. The peripheral nerves show degenerative changes in some cases ;
2. Whilst the intramedullary portion of the posterior roots show degeneration similiar to that found in tabes.

Symptoms :

1. In some cases the only symptom is neuralgic pain in the distribution of one or more peripheral nerves.
2. This is commonest in the lower limbs, where it simulates sciatica, and sugar is found in the urine in the absence of any other sign of diabetes.

In severe cases of diabetes :

1. The knee-jerks or ankle-jerks are diminished or lost in more than half the cases.
2. This may accompany subjective sensory troubles in the lower limbs, or it may appear as an isolated symptom.
3. The muscles are often very tender.
4. The vibration sense in the feet is frequently absent.
5. To objective examination, the sensibility of the skin is usually intact.
6. Perforating ulcers of the feet have been observed.

THE DIAGNOSIS OF MULTIPLE NEURITIS

1. The diagnosis of multiple neuritis from other diseases rarely presents any difficulty.
2. It is made from the combination,
 - (1) Symmetrical flaccid paralysis ;
 - (2) With sensory loss of the " glove-and-stocking " type ;
 and (3) Tenderness of the muscles and nerves, confined to or most intense in the distal parts of the limbs.

Differential diagnosis— from Tabes :

1. Tabes may be suggested—
 - (1) When sensory disturbances and diminished tendon reflexes are prominent symptoms,

and (2) muscular weakness is slight.

2. The resemblance is still greater when ataxia is present.
3. Difficulty usually arises when the distinction has to be made between tabes and alcoholic neuritis in a patient who has courted both diseases.
4. In most instances the diagnosis can be made from the nature and distribution of the sensory changes:
 - (1) The lightning pains of tabes are unmistakable.
 - (2) Anaesthesia of the extremities is common to both diseases.
 - (3) But diminished sensibility around the nose and across the chest is peculiar to tabes and is present in almost every case.
5. The calf muscles and nerve trunks—
 - (1) Are tender in neuritis ;
 - (2) Show greatly diminished sensibility in tabes.
6. Tabes is also suggested by
 - (1) Hyperaesthesia to touch and temperature ;
 - and (2) Great exaggeration of the abdominal reflexes.
7. Argyll-Robertson pupil or optic atrophy, though in favour of tabes, is not decisive, as both have been found in chronic alcoholism.

CHANGES IN EMOTION IN NERVOUS DISEASES

1. Emotion does **not** express itself *via* the pyramidal tracts.
2. Both these tracts may be completely severed :
 - (1) The person still displays emotion ;
 - (2) In fact it is displayed in an exaggerated form.
3. It is uncertain what part of the brain has to deal with emotional expression.

Two views about Centre for Emotional Expression.

- I. Centre for emotional expression lies in *corpus striatum*.
- II. Centre for emotional expression lies in cerebral *cortex*.

- (1) Outgoing path is not along with the pyramidal tract ;
- (2) Outgoing path is by a special tract.
- (3) This special tract passes down from under surface of frontal lobes ;
- (4) Proceeds internal to optic thalamus ;
- (5) Through mid-brain, pons and medulla ;
- (6) Finally, gets lost in spinal cord ;

It is uncertain which view is correct.

But it is known however that cerebral cortex through pyramidal tracts exerts some *controlling* influence on emotion in the same way that it controls "tone."

Disturbances of Emotion

- I. May be—(1) Physiological departures from the normal ;
- (2) Pathological alterations is emotion.
2. Each of these alterations, whether physiological or pathological, may be
 - (1) Towards an exaggerated display of emotion ;
 - (2) Towards a lack of all display of emotion.

Physiological Disturbances

I. Exaggerated Emotional Display :

1. When a person's cerebral cortex exerts very little control on his emotional centres.
 - (1) He may then feel very little sorrow, and
 - (2) Yet display much outward emotion, *e.g.*, "bursting into tears."
2. We may compare this "release" of emotion with the "release" of tone in decerebrate rigidity.
3. Such people do not necessarily *feel* anything like the sorrow they are outwardly displaying, and so, as a rule, they receive very little pity.

4. In the same way, persons of a rather "brainless" type, *i.e.*, with very little cortical control, may show unnecessary laughter over a feeble joke.

II. Lack of Emotional Display :

1. In the opposite direction many persons show an unusual degree of cortical control—a considerable degree of "sorrow" control is expected in the male sex.
2. Such people may feel acute sorrow without showing it at all.
3. Such intense control of emotion may have a very fatiguing effect on the cerebral cortex.
4. And we are all familiar with the intense release of emotion which takes place in such a person when the control of the cerebral cortex snaps.

Pathological Disturbances

I. Exaggerated Emotional Display :

1. In a lesion of *both* pyramidal tracts control of emotion is removed.
2. And the person displays exaggerated emotions, *i.e.*, laughs and cries very easily.
3. It is comforting to remember that such patients do not *feel* anything like the anguish they exteriorize.

Diseases with Exaggeration of Emotional Display—*i.e.*, diseases with bilateral involvement of pyramidal tract :

1. Disseminated Sclerosis.
2. General Paralysis of the Insane.
3. Friedreich's Ataxia.
4. Double Hemiplegia.
5. Spastic Diplegia and Little's Disease.
6. Spastic Bulbar Paralysis.
7. Amyotrophic Lateral Sclerosis.
8. Primary Lateral Sclerosis.

In unilateral lesions, e.g., hemiplegia, emotional expression is not affected.

II. Lack of Emotional Display :

1. In paralysis agitans, parkinsonism, etc., acute sorrow may be felt and yet patient may not be able to exteriorize it because muscles of face, etc., are rigid.

2. They may enjoy a joke as well as anybody else but only their eyes show it by a twinkle.

3. If you wait long enough their stiff facial muscles may show the semblance of a smile.

4. They are just the opposite of patients with bilateral pyramidal tract lesion.

5. We should be especially careful not to hurt such people's feelings as they do not outwardly demonstrate the anguish they suffer.

A CASE OF ACUTE PULMONARY OEDEMA

B. SRIRAMULU, M.B.B.S., B.S.SC.

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Mrs. M. Y., Muslim, aged 30 years, pregnant, full term (3rd para, previous pregnancies and this one so far uneventful, the last one being about 8 years previously) was getting feeble labour pains from 10 p.m., on 12-6-1936. At 11-45 p. m. she went to the latrine to urinate, after which she lay down on the bed, the pains being still feeble. Suddenly she felt a choking sensation, and immediately expectorated a large quantity of white frothy fluid which was mistaken by the relatives for a vomit. Soon she was extremely dyspnoeic and the face and extremities became cold and clammy and were bathed in sweat.

At the time I was called in, which was about 15 minutes later, I found her condition as follows :

1. Pulse : rapid, feeble and barely perceptible.
2. Heart : not enlarged ; no murmurs, but sounds feeble.
3. Extreme dyspnoea and the patient was complaining of a choking sensation and a sensation of impending death.
4. No pain anywhere in the chest.
5. Frequent expectoration of copious quantities of white frothy fluid (there was already half a pint of sputum in the vessel) ; the sputum was not blood-stained.
6. Rales all over the chest but no areas of dullness.

I diagnosed the condition as one of Acute Pulmonary Oedema and adopted the treatment given below :

1. An ampoule of Cardiazol Ephedrin (Knoll) at once ;
2. Followed immediately by 1/100 gr. atropine sulphate.

I resisted the temptation to give Adrenaline to combat the collapse because of the known effects of adrenaline on pulmonary circulation, viz., that of dilatation of the pulmonary capillaries, which would aggravate the condition.

13-6-'36-015 a.m. Pulse a bit better and the respiratory distress

0.45 a.m. Pulse better, sweats disappeared, body felt warmer, respiratory distress less, and the patient was getting real labour pains.

Gave an injection of Camphor-in-oil.

2.00 a.m. Patient much better, pulse vastly improved, respiratory distress much less. She was getting now strong labour pains and she was removed to the local hospital where she gave birth to a female child at 9 a.m. by which time the respiratory distress and expectoration ceased.

Puerperium was uneventful and no further attacks occurred either during her stay in the hospital (for 7 days) or afterwards.

I publish these notes for the following reasons :

1. To point out the absence of any obvious cause for the occurrence of the condition, there being no hyperpiesia, valvular disease of the heart, nephritis, acute infections as rheumatic fever, asphyxiating gases, injection of adrenaline, cerebral embolism or thrombosis, thoracentesis, or abdominal paracentesis, in all of which the above condition can possibly occur. (Her urine was examined and she was under the observation of a lady doctor for the previous 2 months).

2. To discuss the use of morphia here along with atropine, which is recommended by all authorities and in all text-books.

What is the exact action of morphine in the above condition? If, as is suggested by some, morphia allays the apprehension and quietens the patient, what about its action on the respiratory centre?

Is it advisable to give it, when it is known that in certain instances of acute pulmonary oedema, death may occur by suffocation before the patient could expectorate anything and when, in fact, expectoration is supposed to be of good prognostic significance?

In the experimental research on the development of oedema of lung following injection of adrenaline, it was found that in the dog oedema was prevented when the respiratory centre was inhibited by morphine (*Medical Annual*, 1933, p. 275). Even supposing that it can so prevent, can it be of any use once oedema has occurred.

In the above case, I did not give morphine in spite of the usual teaching. I shall be highly grateful if any of the readers of the JOURNAL can enlighten me on the above points.

CULTURAL MEDICINE*

OLD TRAILS

II

THE RENAISSANCE.

The golden dawn of what we know as modern medicine followed the Italian Renaissance of the fifteenth century. The revival of real learning led to a ruthless scrapping of that part of the Arabian and Galenic medical heritage that was useless, and an intensive inquiry after new truths (particularly anatomical truths). Mere authority as such was boldly discounted and the best features of the Hippocratic methods reapplied.

The most determined opposition to this rebirth came, not from the Church, but from reactionary university men, but the bitter battle staged by them seemed only to stimulate the avalanche of revelations that began to pile up.

One of the most romantic of the early figures of the sixteenth century was Paracelsus, who was born in Switzerland in 1493 and died in 1541. He was the great clinical reformer of the period, as Pare conferred a new prestige on surgery and Vasalius vitalized anatomy. At the same time, he represented the Metaphysical Division in the evolution of medicine, proclaiming an *Archaeus*, or spirit, which governed all life. The perturbations of this spirit caused disease.

Paracelsus gave a great impetus to the study of chemistry and pharmacy and scotched dogmatism almost unto death. The reputation for charlatanry that has clung to him down the centuries was in part affixed to him by enemies whom he violently belaboured, for Paracelsus appears to have been one of the most adept dispensers of abuse in history. But even his writings were burlesqued. Then his preoccupation with chemistry at a time when alchemy prevailed invested him with a mystical glamour. It is true, none-the-less, that he was greatly interested in occult matters and wrote extensively upon hermetics, so that between what was true and what false concerning this picturesque personage the mystical legend became more or less inevitable.

Paracelsus supplemented his education at Basel and Ferrara with much travel about Europe and the Orient. He developed into a great Luther-like figure of forceful personality, complete intellectual independence—except for his enigmatical subservience, in the spirit of the times, to occult nonsense—notable knowledge of chemistry, and great medical skill. "Man," he said shrewdly, "is a chemical composition, for which reason it is necessary to use chemical means to combat disease."

He achieved three professorships, all of which were terminated because of his hard-hitting proclivities and uncompromising independence of thought. It is to be remembered that this man was a blatant, heretical and offensive innovator in an age that clung madly to authority. Galen and the Arabians were worshipped by the reactionary rank and file and here came Paracelsus with his ribald but sound criticisms and startling innovations. His position was based upon original observations and experience and the bludgeon that he wielded was guided by clear thought and good motives. He burned the old books, and some of them literally, and counselled the personal acquisition of knowledge of things themselves and their properties. He seems to have had a Messianic confidence in his mission and proclaimed to all his contemporaries, "You shall follow me, and the monarchy shall be mine."

* *Medical Times*, Vol. 64, August 1936, p. 305.

He was one of those extraordinarily gifted persons who appear from time to time against the world's horizon, with a strange mental equipment that spells great achievement at the same time that it spells paranoia.

Paracelsus greatly advanced practical medicine. He denied that his chemical studies were to discover the means of making gold and silver, but "to develop the supreme sciences and to direct them against disease." This noble motive is well borne out by his discovery in some cases and introduction into medicine in others, of zinc, mercurial compounds, calomel, flowers of sulphur, iron, antimony, lead, arsenic, copper, and, last but not least, laudanum. The last-named is probably the key to some of the many "cures" that he wrought. It is at any rate easy to understand how the relief of pain that he was thus enabled to bestow upon great sufferers must have seemed magical to his patients and their friends.

We still speak of galenicals, meaning drugs derived from the vegetable kingdom. Paracelsus, in his revolt against the dominance of Galen, was led to investigate the chemical world, with results that are felt to this day. But he also made tinctures and alcoholic extracts commonplace.

Paracelsus wrote a book about mercury as a cure for syphilis, a very good treatise on surgery, and other works on the diseases of miners and on mineral baths. He understood the transcendent importance of cleanliness in surgery and the nature of goitre and cretinism.

One good old Hippocratic principle to which Paracelsus was wholly devoted was the *vis medicatrix nature*, or the healing power of nature.

Paracelsus did at least two jobs so well that even we of today, in medicine, feel their consequences deeply; he laid the ghost of dogma and authority, and he won again the battle for the rational interpretation and management of the phenomena of disease. "The business of the doctor was to know the different kinds of sicknesses, their causes, their symptoms and their right remedies. This he would teach, for he had won this knowledge through experience, the greatest teacher, and with much toil. He would teach it as he had learned it and his lectures would be founded on works which he had composed concerning inward and external treatment, physic and surgery" (Stoddart).

We must discount heavily the bizarre slant toward occultism in Paracelsus. Despite it, his contribution to modern medicine was vast and momentous.

THE SIXTEENTH CENTURY.

Now comes before us a very great figure in the Pantheon of Medicine—Andreas Vesalius (1514-1564). In 1543, at the age of twenty-nine, Vesalius published his great work "On the Fabric of the Human Body," of which Osler wrote "Modern Medicine begins here," and also "Vesalius grasped...the cardinal fact that to know the human machine and its working, it is necessary first to know its parts—its fabric."

The Alexandrians, as we have shown in an earlier chapter, initiated dissection. Mondino (Mundinus of Bologna), in 1315, dissected two human bodies before his pupils—perhaps the first public dissection in seventeen hundred years. We also know that men like Guy de Chauliac dissected more or less. Leonardo da Vinci dissected over a hundred bodies with the idea of writing a treatise on artistic anatomy. Jacob Beranger Carpi (discoverer of the appendix), Sylvius (teacher of Vesalius and one who gave many muscles and blood vessels names that they retain to-day and will retain forever), Achillini, Benedetti and Vidius, although devout Galenists, nevertheless had dissected to some purpose. But it was Vesalius, determined to demonstrate the anatomical errors of Galen—something requiring incredible courage in the face of that worthy's prestige—

who gave us the first authoritative work on human anatomy, beautifully illustrated, the "Fabrica" already alluded to. He was the real maker of modern anatomy.

Vesalius was trained at Louvain, Paris, Venice and Padua. His abilities as a youngster may be judged in the light of the fact that he was elected to the chair of surgery and anatomy at Padua before he had reached his twenty-fourth year.

It is well to remember that the art of printing from moveable letters had been invented only a short time before by John Gutenberg. The oldest book known to have been printed from moveable type was a Latin copy of the Bible issued from the press of Gutenberg and Faust at Mainz between the years 1454 and 1456; within thirty years thereafter, over eight hundred medical works appeared; so that by the time Vesalius was ready to publish the art of printing had reached its full artistic flower at Basle, in Switzerland, and from the press of Oporinus, in 1543, appeared the sumptuous *De Humani Corporis Fabrica* seven hundred pages in length and with three hundred woodcuts made by van Calcar (or Kalkar), a pupil of Titian.

Despite his profound knowledge of anatomy, Vesalius had no notion as to a general circulation of the blood; that great discovery was reserved for Cesalpinus. For Vesalius, the arteries still carried air (!), since they were always found empty after death (because of the fact that all the blood then enters and is retained in the great veins).

So great was the reaction against the impudent and sacrilegious scholar, Vesalius, on the part of various university men of the time, that he was obliged to flee from Padua to Brussels and to renounce further anatomical blasphemies. The last phase was in Spain, from which country he was also hounded, to die of exhaustion and fever, after shipwreck, in the fiftieth year of his age, on the island of Zante. His death on Greek territory, home of scientific ideas, seems symbolic of the man. Thus closes one of the most pathetic of medical tragedies.

Ironically enough, it was largely through attempts of Galenic adherents to disprove the findings of Vesalius that further anatomical progress was made in the period immediately following his death.

Thus Bartolommeo Eustachio (1524-1574), in his efforts to discredit Vesalius, was led to discover the Eustachian tube, the four pulmonary veins, and a number of other important structures.

Realdo Columbus (1490 (?)—1559), in the course of his attempts to bolster Galenic anatomy, discovered that the pulmonary veins contain blood.

The discovery of the *lesser*, or *pulmonary* circulation, was made by Medical Servetus (1509-1543). This, of course, was a step toward Cesalpinus's and Harvey's work on the *general* circulation.

Gabriel Fallopio (1523-1562) was a loyal pupil of Vesalius who before he died at the early age of thirty-nine, acquired a fame which is still fresh to-day to every physician, since his name is immortalized in some of the most important structures of the body. He discovered the ovaries and many other organs.

A famous pupil of Fallopio (or Fallopius) was Fabricius (1537-1619), whose studies of the valves of the veins at Padua greatly influenced his student Harvey.

The greatest surgeon of the sixteenth century was Ambroise Pare (1510-1590), a Frenchman. This genius was quite as independent intellectually as Paracelsus and Vesalius, and made extraordinary contributions to the science and art of surgery. Finding himself on the battlefield

at night—he was a surgeon in the army of Francis I—without boiling oil, with which gunshot wounds were then treated on the theory that they were poisoned, he dressed them simply and found that they healed well, which scrapped barbaric methods. He devised ligatures wherewith to tie bleeding vessels, thus enormously simplifying amputations. Pare was an extremely versatile operator and much of his writing has a classic value.

Varolius (1542-1575) did very important work on the anatomy of the brain.

Midwifery was advanced during the sixteenth century by the revival of Cæsarean section (known to the ancient Greek and Roman world) and the introduction of podalic version. But the general ignorance of the physiology and mechanism of labour was such that about half the mothers died as a consequence of normal labour, the conduct of which was in the hands of midwives. What the mortality must have been in abnormal labour may easily be conjectured.

In the works of the sixteenth century one begins to read for the first time of whooping-cough, scurvy and syphilis.

The sixteenth century did two outstanding things in medicine; it shattered authority and it achieved an accurate knowledge of how the human body is made. In it was discovered the circulation of the blood. From that discovery flowed all future advances in physiology.

THE SEVENTEENTH CENTURY.

The scientific methods which came into vogue during the seventeenth century were the result of the teachings by Francis Bacon of the philosophy which substituted experiment for speculation (induction for deduction) and by Rene Descartes of the philosophy which invited skepticism to take the place of authority, but it would seem that the philosophers took their cue from such men of action as Vesalius, Pare, and Paracelsus, who really created the *spirit of the times*.

The progress made during the seventeenth century was in large part made possible by the relatively greater social quiet that prevailed, outside of such disorders as the Thirty Years War in Germany.

The background of all medicine is physiology, and it was William Harvey (1578-1657) who finally established physiology as a science. The anatomical foundation had been securely laid, as we have seen, by Vesalius and his disciples, and the functional foundation by Cesalpinus.

Praxagoras (384-322 B. C.) had taught that the arteries carried only air, which was drawn in by the lungs, to pass through the bronchial tubes to the air cells of the lungs, thence to the heart, from which point it entered the arteries. Blood spurting from a cut artery was explained on the theory that by the cut an abnormal condition was produced which determined blood to the artery.

We should honour Andreas Cesalpinus (1519-1603) as the discoverer of the general circulation. He wrote with the utmost clarity concerning both the systemic and pulmonary circulations in the course of his arguments against Galenism. The blood, he said, was "brought through the veins to the heart" and "distributed through the arteries to the body at large." Moreover, he conceived of the heart as a kind of pump, whose systole ("contraction") made the blood circulate.

Harvey's work was as painstaking and thorough as any that can be cited in the whole story of medicine. He was educated medically in Cambridge and Padua. While at the University of Padua the demonstrations of the valves of the veins and of the effects of placing ligatures on the veins by his teacher Fabricius undoubtedly inspired a particular interest in the mechanisms of

the blood stream. That this great "harvester" was acquainted with the pioneer work of Cesalpinus seems an inescapable assumption.

After about fifteen years of animals experimentation, Harvey issued, in 1628, his *Exercitatio anatomica de motu cordis et sanguinis in animalibus* ("Concerning the Motions of the Heart and the Blood").

Since he lacked a microscope, there was a missing link in Harvey's great demonstration. It was not definitely known how the blood made its way from the finer arteries into the smaller veins. This point was soon cleared up by the Father of Histology (microscopic anatomy), Marcello Malpighi (1628-1694), a great Italian scientist, who discovered the capillaries, those tiny tubules which form the connecting links in the circulation between our two sets of vessels. Very much of our knowledge, by the way, of the blood corpuscles, lungs, taste organs, skin structures, bronchial tubes, liver, spleen, and kidney dates back to Malpighi, working with the microscope. He also founded the science of descriptive embryology and shares with Robert Hooke and Nehemiah Grew the credit of foreshadowing the cell-doctrine, around which so much of modern science has revolved.

It is in order at this point to explain that although the microscope had been devised by the Janssens (1590) and Galileo (1610), and improved by Cornelius Drebbel (1621), it had not come into medical use at the time that Harvey did his work.

It took a generation to force acceptance of the doctrine, and Harvey lost much practice and was thought to be demented. But happily he lived to see the restoration of his fortune and to command great prestige.

Chemistry made great strides during the seventeenth century at the hands of Robert Boyle (1627-1691), Rudolph Glauber (1604-1688), van Helmont (1577-1644), and Sylvius de le Boe (1614-1672).

Boyle proved that life is dependent upon oxygen and for the first time systematized chemical knowledge.

Glauber still lives in the salt that bears his name (sodium sulphate).

Van Helmont's mission was to continue the chemical researches and metaphysics of Paracelsus. He was a strange mixture of the mystic and the scientist. Despite his fanciful ideas he was an experimentalist. He introduced the term "gas," discovered carbon dioxide, formulated the principles of gas analysis, placed chemical therapeutics on a good basis and opposed blood-letting.

This tradition of chemical investigation was continued by Sylvius de le Boe (or Franciscus Sylvius). He lives forever in the Sylvian fissure in the brain, for he was an anatomist as well as a chemist. He, too, was of the mystical school of thinkers but he did much to start physiological chemistry safely on its way. His was the first chemical laboratory. He had a small infirmary of twelve beds at Leyden, and here he gave ward instruction to medical students quite in the modern fashion.

Upon the whole, the seventeenth century was a practical era, going to the length of attempting to express every phenomenon of life by a chemical formula (iatrochemical school) or a physical law (iatriophysical school). The mystical survivals were pretty much neutralized by common sense. The exponents of the prevailing schools consciously guarded themselves against their own speculative tendencies, seldom permitting lapses in practice. Santorio Santorio, of Padua (1561-1636), usually called Sanctorius, affords an example of this cautiousness. He initiated

the study of metabolism by conducting a series of weighing experiments upon himself, with particular reference to perspiration. He also made investigations with thermometers and hygrometers, and attempted mechanical measurements of the pulse. And then there was Baglivi, to whom the circulatory system was a hydraulic machine, the breathing apparatus a balloon, and the digestive organs a sieve. Yet these men usually kept their heads when they approached the bedside and were nothing if not rational clinicians, considering their paucity of data. It was paucity of data, to be sure, that accounted for their speculations.

Thomas Sydenham (1624-1689), the "English Hippocrates" and the "Prince of Physicians," played the greatest part in shaping the practice of the seventeenth century. Educated at Paris and Montpellier, he was essentially a practitioner. He had no use for theories and but little for books. As a clinician, guided solely by accurate bedside observations, he first clearly described and made familiar such conditions as rheumatism, scarlatina, erysipelas, Saint Vitus' dance, gout, pleurisy, broncho-pneumonia, and hysteria. His classification of symptoms as cardinal (due to nature's attempts to effect a cure) and artificial (due to treatment) was a highly scientific step forward. The tablet over his grave, erected by the College of Physicians, fittingly records him as *medicus in omne aevum mobilis*—a medical nobleman for all time.

Thomas Willis (1621—1675) first described typhoid and child-bed fevers, paralytic dementia and myasthenia gravis, and noted sugar excretion in diabetes. His name will never be forgotten because of the "circle of Willis" and the "nerve of Willis" (eleventh cranial, or spinal accessory).

Many finely detailed anatomical advances were made during the seventeenth century by men after whom various parts of the body are named: Steno, Lower, Vieussens, Pecquet, Wirsung, Wharton, Glisson, Peyer, Brunner, Nuck, Meibom, de Graaf, Rivinus, Havers, and Spigelius.

Great advances in physiology were accomplished by Hooke, Bartholin, Schneider and some of the anatomists mentioned in the preceding paragraph.

It was during this century that blood transfusion was introduced. Denys of Paris did a human transfusion on June 15, 1667, while Richard Lower performed the second one on November 23, 1667 (although he had transfused animals in 1667). The use of cinchona bark was another important innovation (popularized by Sydenham), although quinine was not isolated from it until 1820.

It is an odd fact that intravenous medication was introduced during this century by Sir Christopher Wren, himself not a physician nor even a student of medicine, but one who had been closely associated with Thomas Willis, whose book on the brain and nervous system he illustrated.

Surgery languished during the seventeenth century, despite the great work of Pare in the sixteenth. But Fabry of Hilden (1560-1624), in Germany, did some clever things: he extracted an iron splinter from the eye with a magnet and wrote a fine collection of case-records. Richard Wiseman (1622-1676), called the Pare of England, first described tuberculosis of the joints, operated for stricture (external urethrotomy) and wrote "Several Chirurgical Treatises." But surgery's great stride forward awaited the eighteenth century.

During the sixteenth century child-birth was presided over by midwives, even in the best walks of society. In the seventeenth century there were some celebrated midwives, e.g., Louise Bourgeois, who delivered Marie de Medici six times, Justine Siegemundin, who published a treatise on obstetrics in 1690, and Jane Sharp, author of the *Compleat Midwife's Companion*

(1671). During the eighteenth century this speciality began to be practised more and more by male obstetricians, and just as soon as they interested themselves in the art and science of midwifery, just so soon was notable progress registered. The pioneers left great names: Mauriceau, Portal, Van Deventer, Ould, Smellie, Leveret, Fried, Roederer, Baudelocque, Camper, Boer, Saxtorph, Palfyn, De La Motte, Sigault, Sirbold.

It seems odd that in a field in which women held almost undisputed sway down to the time of the galaxy of obstetricians listed in the foregoing paragraph there should have been so few distinguished midwives. Lack of educational facilities comparable to those enjoyed by men cannot wholly account for the failure of obstetrics to make notable progress at the hands of women, for even at the school of Salernum, dating from the ninth century, there were women as well as men professors. The names of women students and teachers are also met with in the early history of the ancient school of Bologna, among them Alessandra Giliani, the first anatomist to practice the injection of the blood vessels with coloured liquids.

Here is an instance in which women may be said to have had what practically amounted to a monopoly in practice for hundreds of years, without a single achievement that has stood the test of time to their credit. Not until Van Deventer was the mechanism of labour so much as considered (1701).

It is possible that the feminists can explain this curious phenomenon easily, and we are willing and even anxious to be enlightened by them, if they can qualify. But at the present writing the matter would seem not greatly unlike the position of vast numbers of privileged women, in, say, the field of music, where they have had golden opportunities and have displayed great talent but little creative power.

Even in a man-made world it would seem that women's creative record should be better, if she possesses inherent powers and has enjoyed exceptional opportunities and training at times. She is nowhere eminent in obstetrics to-day.

No clever midwife of the old dispensation was ever faced by greater resistance than was Semmelweis, who, about 1840, discovered that uncleanness was the chief factor in the etiology of child-bed fever, and who was ridiculed, hounded and persecuted to death (literally).

It will not do to plead that behind every creative man there has always stood some woman, at once a source of energy and an indirect participant in the production of results.

And it will not do to plead biological creativeness—consecration to motherhood—tremendous sapper of energy though it be.

Woman the midwife and nurse—man the obstetrician. That has always been the situation in medicine. Is it so in all other departments of life?

What say the feminists, themselves privileged in great part—and, in the main, sterile in more senses than one?

By 1670 male obstetricians were supplanting the old-time midwives. Francois Mauriceau had published the first scientific treatise on the subject in 1668. One Peter Chamberlen invented the obstetric forceps about 1670, but its nature and use were kept more or less secret by him and his son Hugh until 1721, when John Palfyn invented the real original of the modern

It was in the seventeenth century that the spermatozoa were discovered by a medical student, Johann Hamen, in 1674, although the complete description of them was written by Antony van Leeuwenhoek, of Delft, who also described the crystalline lens of the eye. Van Leeuwenhoek was not a physician, but a man who had inherited wealth made in the brewing industry and who devoted himself with the utmost intensity to the grinding of lenses and the advancement of microscopy. He is said to have had two hundred and forty-seven microscopes and four hundred and nineteen lenses.

The red blood corpuscles were discovered by Swammerdam, a Hollander, in 1658. Malpighi studied them thoroughly in 1665 and van Leeuwenhoek gave a good description of them in 1674.

The science of physiologic optics was developed by seventeenth-century astronomers (Kepler and Scheiner) and the philosopher Descartes. They worked out the functions of the retina and of the crystalline lens, as well as the mechanisms of accommodation and refraction.

It was not until 1650 that physicians trained in European universities began to settle permanently in America, although they had accompanied expeditions and tarried for awhile in colonies. These early practitioners trained their successors through a system of apprenticeship. Lectures on anatomy were given by Dr. Giles Firmin at Harvard about 1646, and these were followed by a partial course in medicine.

The first hospital in what is now the United States was erected on Manhattan Island in 1663. But Cortez had built a hospital in the City of Mexico in 1524 while Canada had established hospitals in 1639 and 1644.

The seventeenth century produced no American medical literature.

"The best work of the seventeenth century," says Garrison, "was either conceived from some deep source of original inspiration or else sprang from a fresh, native wonderment over the newly revealed marvels of nature, as when old Pepys declared himself 'with child' to see any new or strange thing."

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OCTOBER 1936

THE CONTROL OF TUBERCULOSIS

In a country which is the victim of innumerable diseases, one does not know where to begin preventive measures. The area is vast, the population large and poverty appalling. The Sanitarian might well despair in this depressing environment. In spite of the odds against him, he tucks up his sleeves and sets to work. His courage is admirable and he deserves the sympathy and the unstinting support of the public. But the public is generally ignorant of the scientific background of public health measures. And denied the hope that springs out of this knowledge it is generally inclined to be fatalistic. In these circumstances, the duty of the Profession is very urgent. The medical man has not merely to cure the sick but he has more and more to play the rôle of the public health propagandist. The practitioner himself is bewildered. He is in the midst of malaria and the chronic invalidism which it creates. He witnesses the rural population enfeebled and made apathetic by the ravages of hookworm infestation. He meets innumerable cases of so called dyspeptics—victims of chronic manifestations of dysenteries. And almost daily he helplessly looks on while the young and the old, the poor and the rich, the beloved and the pets are stricken and carried away by tuberculosis.

Something is being done to check the ravages of hookworm infection. The State makes futile efforts towards controlling malaria. The chronic dysenterics suffer in silence. But tuberculosis does not allow any room for complacency. And if some useful measures are not taken here and now, the outlook is certainly gloomy. While the incidence and mortality of tuberculosis are decreasing in other countries, they are rapidly rising here. And should there be further serious havoc before attempts are made to control the disease in this country?

The gravity of the situation has been well realized, and since the reign of Edward VII there have been efforts to study and control the disease. There has been some feeble imitation of the methods adopted in the West against this disease. A few

dispensaries, "institutes" and sanatoria have been opened here and there. Individuals have been appointed to these places so that they might study the various aspects of the problem. But after the lapse of more than twenty-five years, we know that the advance has been negligible. In the meantime, the disease is spreading and takes an ever increasing toll of human life.

In the prevention and cure of tuberculosis, the State, the Public and the Profession suffer from serious handicaps. Our knowledge about many aspects of the disease is very meagre. All that is known definitely is that the tubercle bacillus is the cause of the disease. Everything else in almost wholly or partly controversial. In spite of laborious and expensive research, the cure of tuberculosis is still very unsatisfactory. The methods adopted range from sun-cure to synthetic drugging. While these methods give fair results in a proportion of cases in different countries, the time and trouble and the expense involved are almost prohibitive for adoption on a general scale in a poor country like India. A method which was at first cautiously and modestly used is coming to be more and more relied upon in certain countries. *Collapse therapy* is giving increasingly good results in a larger number of cases. We are not concerned here with the relative merits of various therapeutic measures. We wish to concentrate upon some methods which would ultimately help to lessen the incidence of the disease.

In the First Anti-Tuberculosis Conference held in New Delhi in 1934, the prominent workers from different parts of India met to discuss the various aspects of this question. But the ordinary man who goes through the proceedings cannot help feeling that there is no indication as to how they propose to approach the problem of prevention of tuberculosis. "Organizations for the treatment and prevention of tuberculosis are now more or less standardized throughout the civilized world, comprising notification, clinics (for diagnosis, special treatment of the ambulatory case, and post-institutional care), diagnostic laboratory service, sanatoria, and hospitals, and home visitation by trained nurses." But to carry out an efficient scheme of this kind requires large financial resources. Even in rich countries like

America it has been found impossible for small municipalities and counties to undertake a scheme on these lines. For one thing, this scheme requires sanatoria and hospitals with a large number of beds. For instance, in New York State with a population of about six millions, there are large sanatoria and hospitals run by various bodies with beds for the accommodation of about 9,000 patients at a time. In addition to these institutions there are other organizations like children's preventoria, open-air schools and summer camps for the tuberculous. If a similar efficient scheme is to be organized in this country, we should have sanatoria and hospitals with 900 beds for the city of Madras alone for the exclusive use of tuberculous patients. We wonder if there are so many beds together in all the general hospitals in the city. Therefore, to look for even a fourth of the required accommodation is to wish for something unobtainable for a long while.

It is now definitely known that intimate contact of susceptible people with patients who cough about indiscriminately is the most important single factor in the spread of human tuberculosis. The possibility of massive infection is enhanced by the crowded and ill-ventilated environment which goes along with poverty and squalor, though the disease is equally frequent and fatal among the rich who do not lack hygienic amenities. The prevention of crowding and provision of tolerably healthy environment can only follow economic improvement. In other words, the prevention of contacts is a matter of large funds. But we may achieve prevention of the disease in an equal measure, if we can succeed in diminishing the source of the infecting sputum. This may be secured by isolating and treating early cases in sanatoria and housing and treating advanced cases which are chiefly instrumental in bringing up large quantities of heavily infected sputum and thus spread the disease to those around them. We have already pointed out the practical difficulties in the way of financing such expensive schemes. We are, therefore, compelled to seek other and less costly measures to secure this object. Fortunately, *collapse therapy* by the induction of artificial pneumothorax offers great possibilities in this direction. It is cheaper, but therefore not less useful. This method of con-

trolling tuberculosis has many advantages apart from its inexpensiveness. The unfortunate patients can remain in their own homes while undergoing the treatment—a factor which has a very great effect on their happiness. Within a short period they improve so well that they could resume their occupation and thus continue to earn the bread for the family, thereby easing the tax on the State. And when the treatment is successful, they do not bring up any sputum. The radiating centres of infection are thus rendered innocuous. The ambulatory pneumothorax clinic at the Henry Phipps Institute working in the slum quarter of Philadelphia in the midst of poverty and poor hygiene treats about fifty patients every morning with results that are said to compare favourably with those of well ordered sanatoria. The field pneumothorax clinic of the Chicago Municipal Tuberculous Sanitarium and similar clinics in other cities report similar results.

An extensive and rationally organized scheme for the utilization of this method requires a large number of medical men well trained in the technique and well conversant with the few complications that might arise in the course of this treatment. A contemplation of this need is likely to frighten the authorities into the belief that what is saved in sanatoria and hospitals may have to be spent on the large medical staff and the necessary apparatus. Money is required for the apparatus and inexpensive habitations. But as for staff, we have a large number of practitioners whose services are not properly utilized by the Public or the State. These medical men could be given the necessary training by the Government. Some of them might be given a helpful subsidy, while many would be willing to carry on without any monetary help from the Government. But this would not be possible if the Government adopts the same ungracious attitude towards the practitioners as they sometimes do towards the honorary staff of the various hospitals. We would desire that in return for the services of the medical men, the Government should help to organize the profession and distribute its services over the country so that the practitioners might not be hampered by any selfish combinations, or by the abuse of the elastic conditions in the hospitals under their direct care.

MEDICAL RESEARCH WORKERS' CONFERENCE

The Fourteenth Conference of the Medical Research Workers' Conference will be held in the Library of the School of Tropical Medicine, Calcutta, from the 30th November to the 5th December, 1936, with Major-General Sir Cuthbert Sprawson, Director-General, Indian Medical Service, in the chair. Colonel A. J. H. Russell, I.M.S., Public Health Commissioner with the Government of India, will act as Secretary to the Conference.

The Scientific Advisory Board of the Indian Research Fund Association will also meet at Calcutta at the same time.

There will be a meeting of the Scientific Advisory Board for a preliminary discussion before the proceedings of the Medical Research Workers' Conference actually commence. The Conference itself will be opened by the Chairman with an address, and this will be followed by a general discussion in which the research workers present will each give details of research activities during the preceding year. Each item of research work will be discussed and the officer in charge of the work will make his proposals for the next year. The proposals will then be considered and recommendations made to the Scientific Advisory Board whether and how far the suggestions for the following year be carried out.

In order to ensure that each item of research work receives close and adequate consideration, the Conference will set up a number of advisory committees each dealing with problems appertaining to a particular subject. Thus there will be advisory committee on leprosy, cholera, maternity and child welfare, malaria, plague, nutrition and rabies. It has not yet been decided if there should be an advisory committee on tuberculosis, but if facilities of discussion so require, a committee will also be set up on that subject.

The recommendations of the Conference will next be considered by the Scientific Advisory Board of the Indian Research Fund Association, which will meet for the purpose on the 5th December, before the workers assembled for the Conference disperse; and the Board will in its turn make its recommendations to the Governing Body of the Indian Research Fund Association which meets in Delhi early in March next.

The Conference will also discuss a series of resolutions dealing with the public health problems of India. These resolutions, too, will be considered by the Scientific Advisory Board of the Indian Research Fund Association, and passed on to its Governing Body with its recommendations. The Governing Body will again consider the matter, and such of the resolutions as they approve will next be forwarded to Education, Health and Lands Department of the Government of India for its consideration.

It may be noted that as Major-General Sir Cuthbert Sprawson retires in February next, this will be the last Medical Research Workers' Conference over which he presides.

ASSOCIATION NOTES

THE RAMNAD DISTRICT MEDICAL ASSOCIATION

(Formerly Chettinad Medical Association)

The usual monthly meeting of the above Association was held at the Lady Pentland Hospital, Kanadukathan, on Sunday 19th July at 5 P.M. under the Presidency of Dr. B. V. Varadachari, B.A., M.B.C.M., D.M.O., Ramnad. About 40 doctors from all over the District including four doctors from Kumbakonam were present.

After tea arranged by the host Dr. Miss Ivatts, Dr. T. S. Shetty, the Secretary read the minutes of the previous meeting which was adopted. Then there were a few case reports—one was of a big myxomatous tumour of the hard palate in a girl of 14 years operated by Dr. T. S. Shetty in his clinic and which was discharged cured after having had severe bleeding on the 7th and 9th days of operation due to the slough opening the inferior alveolar vessel.

Another case was also from his clinic—a complicated case of Subperiosteal abscess which was operated by him.

After this Dr. M. K. Sambasivam, M.B.B.S., Chairman, Municipal Council, Kumbakonam, delivered a lecture on "Non-padded plaster casing in the treatment of fractures" with practical demonstrations. There were discussions and doubtful points were cleared by the Lecturer. Captain G. S. K. Aiyer who came with him gave out a few important points with regard to the subject. This over, the members witnessed a magic performance and then adjourned for dinner.

The members spent a very enjoyable evening with the excellent arrangements made by the host Dr. Miss Ivatts, the Medical Officer of the Lady Pentland Hospital.

THE TINNEVELLY DISTRICT MEDICAL ASSOCIATION

PALAMCOTTAH.

(Affiliated to the Indian Medical Association.)

The Tinnevelly District Medical Association held its monthly meeting on Sunday the 27th September 1936 at Sankarankoil, a holy place of this District. Members numbering 29 gathered from all parts of the District.

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ASSOCIATION NOTES

XXXV

After Tea Party and sweet music, the meeting began under the Presidentship of Lieut.-Col. T. S. Shastry, I.M.S., The Secretary read the minutes of the monthly meeting (President's Function) held at the Headquarters on 29-8-1936, which were passed.

The following Clinical Cases were demonstrated :—

By whom.	Cases.
Dr. P. S. Srinivasan, L.M.P., Sub Assistant Surgeon, Government Headquarters Hospital, Palamcottah.	1. Penetrating Wound Chest. 2. Malignant Tumour Left Thigh upper third and Hobnailed Lungs. (Specimen shown.)

Case notes of the following cases were read by Dr. P. S. Srinivasan :—

1. Perforated Duodenum.
2. Malignant tumour left thigh upper third and Hobnailed Lungs.
3. Intussusception.
4. Penetrating wound chest.

The above cases were fully discussed by members. Then Lieut.-Col. T. S. Shastry, I.M.S., summed up the salient and important features of all the cases.

Dr. K. Rama Iyer, M.B.B.S. (Andhra), gave an interesting lecture on "The Role of X-Rays in Medicine and Surgery."

The President brought the meeting to a close by his remarks on the subject of the learned address and proposed a vote of thanks to Dr. T. R. Srinivasa Iyengar, Selection Grade Sub-Assistant Surgeon, for his splendid arrangements.

Thereafter an excellent dinner brought the happy gathering to a close.

HEALTH EXHIBITION.

Under the auspices of the Tinnevely District Medical Association, in connection with its monthly meeting at Sankarankoil, a Health Exhibition was arranged at the Board Gomathy Edward High School, Sankarankoil, on Sunday the 27th September 1936, at 10 A.M. under the auspices of Mr. Ghulam Mahammad, B.A., Revenue Divisional Officer, Koilpatti. This is the first time that a Health Exhibition was held at Sankarankoil.

Lieut. Col. T. S. Shastri, I. M. S., the President of the Association, welcomed the guests and requested Mr. Ghulam Muhammad to open the Exhibition.

Mr. Ghulam Muhammad in declaring the Exhibition open said that such Exhibitions are very useful, instructive and beneficial to the public and school children and promised to devote his best attention hereafter to encourage Health Propaganda.

Mr. Maduranayagam, the Health Inspector, explained in Tamil, the exhibited posters on the prevention and causation of Tuberculosis, Flies, and Cholera, for the benefit of about 200 school children and public.

This was very much appreciated by the gathering.

THE TRICHINOPOLY DISTRICT MEDICAL ASSOCIATION

(Registered.)

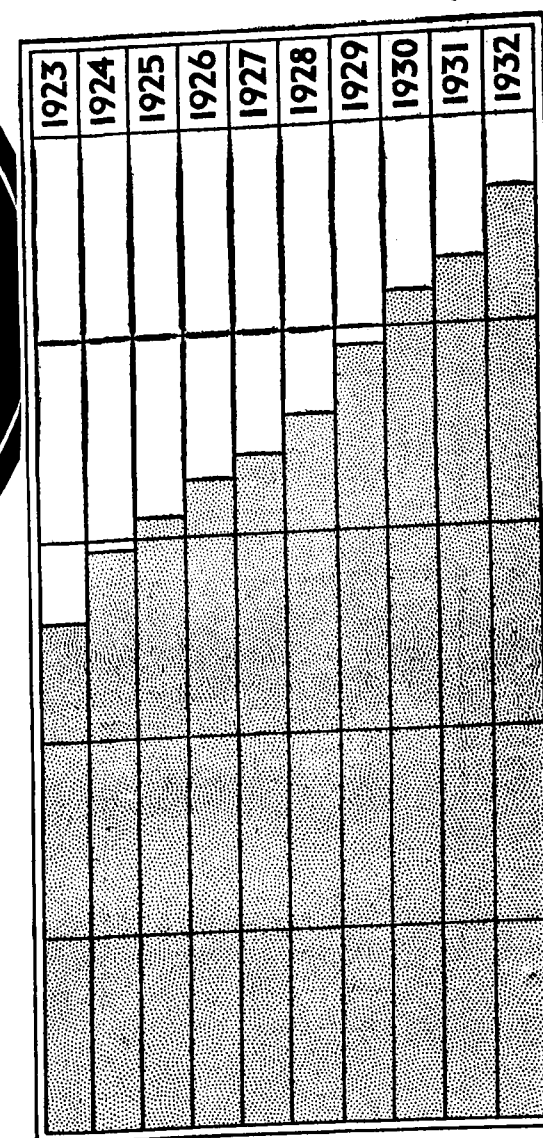
A monthly meeting of the above Association was held at 5 P.M., on Sunday the 27th September 1936 in the E. R. High School, Teppakulam, Trichinopoly, when about 25 members were present. Dr. T. V. Swaminatha Shastri, L.M. & S., presided and Dr. Mrs. D' Costa, L.M. & S., was 'At Home'. Dr. T. N. Subramanyan, L.M. & S. delivered an interesting lecture on 'Laboratory as an Aid to Diagnosis', in the course of which he said that though clinical methods are essential for proper diagnosis yet the Laboratory was useful to confirm or correct the bed-side findings and appealed to Medical men to make more use of Laboratory methods.

Dr. Raghavan gave his experiences mostly of cases in which the Laboratory diagnosis was contrary to clinical findings and treatment based upon Laboratory findings are invariably correct as shown by the results. In his concluding remarks the Chairman stressed upon the need of well equipped Laboratory in such a place like Trichinopoly and hoped that Medical men will make use of Laboratory methods for correct diagnosis of cases.

Miss Sakunthala of Mysore, a girl of 6, then gave a demonstration of mental feats such as mathematical calculations, etc., which was much appreciated.

With a vote of thanks to the Hostess, the Lecturer, the Chairman of the evening and the Manager of the premises the meeting terminated.

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THE TRICHINOPOLY DISTRICT MEDICAL ASSOCIATION

(Registered.)

A monthly meeting of the above Association was held at 5 P.M. on Friday the 23rd October 1936 in the Mission Hospital Compound, Woriyur, Trichinopoly, when about 35 members were present.

Dr. Miss Edith Little was 'At Home' and Dr. C. E. R. Norman presided.

Dr. W. R. Aykroyd of the Nutrition Research Laboratories, Coonoor, delivered an interesting lecture on 'Nutrition'. He said that the average Indian of the poorer classes suffered from Malnutrition. Malnutrition and hunger are not analogous, as hunger could be satisfied without nourishing food. The effects of malnutrition are likely to be most severe in growing children, in the manual worker and the pregnant and lactating mother. Indian diets are often inadequate both in quantity and quality. A satisfactory diet must contain adequate quantities of protein, fat, vitamins and mineral salts.

Protein is an important food element for the growth and repair of tissues. Rice is not rich in protein, pulses contain more. Leafy vegetables contain more protein, minerals and vitamins, than ordinary vegetables. Keerai is as valuable as the costly cabbage lettuce, spinach, etc. Milk, meat and fish contain more proteins but are not usually within the reach. Animal fats such as ghee, cod liver oil and butter, contain more vitamin A and D than vegetable fats such as gingell oil, cocoanut oil, etc.

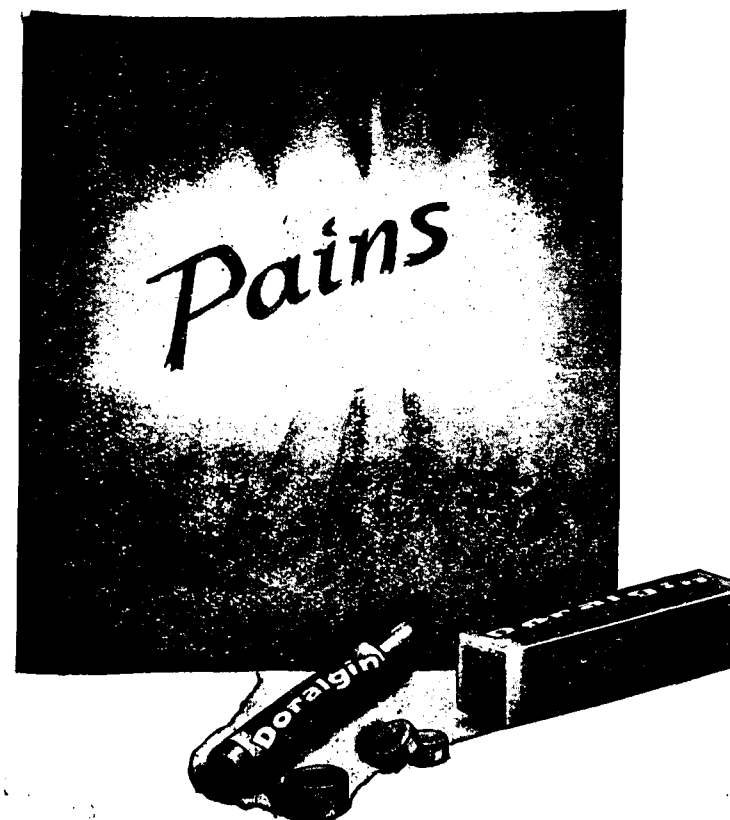
Deficiency of vitamins causes various diseases. Lack of vitamin A causes Keratomalacia and certain skin conditions which are curable by the administration of liver-oils and green vegetables. Lack of vitamin B-1 causes Beri-Beri and Neuritis in lactating women, and lack of vitamin B-2 causes sore mouth which is curable by yeast milk. Lack of vitamin C leads to Scurvy which can be prevented and cured, by the administration of citrus fruits such as lime, etc. Deficiency of vitamin D causes Rickets and perhaps dental caries. Vitamin D is found in milk, fat, liver-oil and may also be obtained through the action of sunlight on the skin.

Minerals especially calcium and iron are also essential and deficiency of these leads to various pathological conditions. Much anaemia in pregnant women was associated with a deficient intake of iron.

After the lecture there was an interesting discussion in which many members took part.

The Secretary then moved a resolution sending fraternal greetings on the occasion of the Annual of the South Canara District Medical Association which was passed unanimously. In a moving speech he proposed another resolution which was passed all standing at the sudden and untimely demise of Dr. M. K. Sambasivan, M.B.B.S., Chairman, Municipal Council, Kumbakonam, and who was actively connected with the Association for a number of years.

With a vote of thanks to the Lecturer and the Hostess, the meeting terminated.



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The Dr. B. S. Shroff Memorial Gold Medal

OF THE BOMBAY MEDICAL UNION

The following subject has been selected by the Bombay Medical Union for competitive thesis for the above Prize for 1937:—

“The dietetic deficiencies and excesses at present prevalent in different parts of India, and their effects on health, with suggestions for improvements of such dietaries.”

The award will be in the form of a Gold Medal called the DR. B. S. SHROFF MEMORIAL GOLD MEDAL of the BOMBAY MEDICAL UNION.

The competitor must be (i) a duly qualified member of the Medical Profession holding a degree or degrees and diplomas from Indian and other Universities created by statute, or (ii) a duly qualified member of the Medical Profession holding the diploma of Membership of the College of Physicians and Surgeons of Bombay.

The thesis must be sent in six typed copies so as to reach the Honorary Secretaries, Bombay Medical Union, Blavatsky Lodge Building, French Bridge, Chowpatty, Bombay, on or before the 15th March 1937 the latest.

The thesis should be designated by a motto instead of the writer's name and should be accompanied by a sealed cover containing the name of the writer and his Post-Office Address.

The name of the Prize, the year of competition, the subject of the thesis, and the writer's motto should be superscribed on the cover.

No study or essay that has been published in the medical press or elsewhere will be considered eligible for the Prize, and no contribution offered in one year will be accepted in any subsequent year unless it includes evidence of further work.

The accepted thesis shall be the property of the Bombay Medical

All other thesis shall be returned if not accepted provided the returned postage expenses are paid in advance by the writer.

In the award of the Prize to the successful candidate, the decision of the Committee, shall be final.

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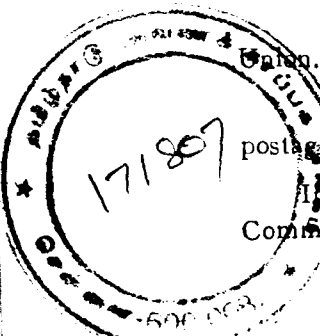
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