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THE CADUCEUS

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STREPTOCIDE IN THE TREATMENT OF MENINGOCOCCAL MENINGITIS.

by

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INTRODUCTION.

The cases of meningitis described in this paper were admitted to hospital between August and December 1938. No attempt at selection has been made, and the one point they all have in common is that they were all proved bacteriologically to be cases of meningococcal meningitis.

Two lines of treatment were adopted, one group being treated with streptocide only, the other with streptocide and anti-meningococcal serum. The series is too small to allow conclusions of value to be drawn from it, but clinically there is little doubt that recovery is more uneventful in those cases treated with serum and streptocide. Lumbar puncture was carried out in all cases for the first two or three days after admission, and as much cerebrospinal fluid as would flow readily was drained each day. Lumbar puncture appears to be a valuable adjunct to this combined form of therapy for these reasons. It enables one in most cases to tell whether block has or has not been established in the cerebrospinal fluid system and it enables one to relieve pressure in the ventricular system. The cerebral cortex is so intensely hyperaemic and softened during the first week of the disease that structural deformations of the ventricular systems occur readily. If treatment is not prompt and effective, block, either complete or partial, is only too often established in the first weeks of the disease and once established it leads inevitably to internal hydrocephalus. This happened in two cases treated earlier in the year, and was almost certainly due to ineffective treatment during the first two weeks of the disease. The importance of this measure can hardly be overstressed, and in all cases manometric observations were made at each puncture, and Queckenstedt's test was carried out. It has also become

routine practice to have the patient put into the sitting position with the lumbar puncture needle still in situ. This manoeuvre enables one to tell at a glance whether drainage is free, and it also has the advantage of increasing the amount of cerebrospinal fluid obtained at each puncture. The indications for omitting lumbar puncture daily are the disappearance of fever and the clearing of the cerebrospinal fluid.

Subarachnoid lavage was carried out daily in some of the cases in the early stages of the disease. It is a measure of undoubted value, and the indication for its use is the appearance of signs of impending "block." If Queckenstedt's phenomenon from being positive becomes negative, or if the amount of cerebrospinal fluid obtainable by lumbar puncture becomes negligible then subarachnoid lavage is indicated. Both lumbar and cistern punctures are done and the two manometric readings are recorded: normal saline warmed to body temperature is then *slowly* and gently injected into the lumbar theca. It is useful to have the saline tinged blue with indigo-carmine as the colour is then readily detected in the outflowing fluid from the cisternal needle. Many observations have shown that on an average 18-22 ccs. of coloured saline have to be injected into an adult lumbar theca before the outflowing cisternal fluid becomes blue. It is well to wait a little after injecting 20 cc. and to keep the nozzle of the syringe in the lumbar needle. Not infrequently one is able to note the sudden overcoming of the "block" by a rapid increase in the rate of flow from the cisternal needle. When one considers how often in the course of meningococcal meningitis localised matting prevents a free flow of fluid from a lumbar puncture needle and how readily the introduction of 2-3 c.c. of normal saline will produce a free flow by breaking down inflammatory loculi, it is easy to visualise the effect of such matting when it extends over the whole length of the spinal cord. The manoeuvre just described is really nothing more than a large scale adaptation of the method of introducing small amounts of saline into a lumbar puncture needle to induce a free flow of cerebrospinal fluid.

The beneficial effects of subarachnoid lavage are two-fold. First, it enables the circulation of the cerebrospinal fluid to continue and thereby lessens the risk of block becoming permanent; second, it enables serum or drugs introduced by the lumbar theca to gain access to the base of the brain and the whole cerebro-spinal axis. Incidentally the use of streptocide intrathecally has been abandoned. It appears to cause very severe root pains and to be no more effective by this route than by oral administration.

In carrying out subarachnoid lavage it is essential to have the patient as still as possible, and if delirium or restlessness were marked evipan was always given before marking the punctures in adults, open ether in children. No ill effects have been seen as a result of

giving evipan to patients suffering from meningococcal meningitis, although it does tend temporarily to increase tremor if tremor be already present.

Anti-meningococcal serum has been used both intrathecally and intravenously. Patients admitted to hospital seriously ill were usually given 15-20 c.c. intravenously in addition to an intrathecal dose of 20-30 c.c. It is uncertain whether serum intravenously is of value, but at any rate it seems rational to give it by this route to those patients who show petechiae on admission. We know that the serum contains bacteriolysins, bacteriotropins and anti-endotoxins. It is, therefore, obviously reasonable to combine it in the early stages of the disease with a bacteriostatic agent such as streptocide. Theoretically it is desirable to confer some degree of passive immunity and if this measure can be conjoined with the exhibition of a drug which inhibits the growth of the casual organism, recovery should occur in a high proportion of cases.

Streptocide has been given in heavy doses, because earlier experience has shown that it is not merely ineffective in small doses, but it actually seems to make subsequent recovery more difficult if recourse is finally had to serum. The aim of treatment in this disease, as in any other infection, is to enable the patient to produce his own antibodies, and the reason for combining the two forms of treatment is to prolong life sufficiently to enable these antibodies to be produced. Streptocide merely inhibits the growth of the infecting organisms while this is happening, and if the drug is discontinued too soon relapse occurs. This is well shown in Case 1A where streptocide was discontinued too early, a relapse occurred, and was overcome by streptocide without further serum. In this case presumably the antibody response of the patient had progressed to a certain point, and was enabled to progress to full recovery by the readministration of streptocide.

As most of the patients admitted have been ill for at least three or four days, it seemed irrational to wait before giving streptocide. One was justified in the majority of cases, in assuming that some degree of immunity had either been established already, or was never going to be established, and there was therefore no reason to delay giving the drug. An attempt was made earlier in the year to obtain better results by waiting 12 to 18 hours after the first dose of anti-meningococcal serum before giving streptocide, but the method did not appear to give better results than combined therapy from the start.

Adequate dosage and properly spaced dosage are essential if streptocide is to be of value. The drug is excreted very rapidly and must be given at least 4 hourly if results are to be obtained. It appears to be well tolerated, especially by children, and in only one case was intolerance of the drug severe enough to make it necessary to reduce the dose. All patients taking streptocide were given to drink 5%

glucose saline containing 240 grs. of sodium bicarbonate to each pint. This measure prevents the onset of acidosis and also helps to make up for some of the salt loss which results from the loss of cerebrospinal fluid by lumber puncture during the first few days of treatment. Several of the younger children in the series became dehydrated in the early stages of the disease, mainly through inability or unwillingness to swallow the requisite amounts of fluid.

The toxic effects noted were vomiting, nausea, cyanosis and prostration. In only one case did vomiting occur although several of the children complained of nausea during the first week of taking the drug. The cyanosis was marked in only two cases, and was never very striking. Undoubtedly the most constant feature in the mild grades of intoxication is the prostration. Patients who have become afebrile and have lost their pain and rigidity complain bitterly of "tiredness." They are much too weary to attempt to sit up or read or employ themselves in any way and this stage of prostration may last for a week or ten days. It seems to pass off even if the dosage is maintained at the high level of 6 grms. every 24 hours. It is not infrequently accompanied by pronounced anorexia.

Blood examinations were not carried out as a routine owing to lack of facilities, but in three cases blood films showed no abnormalities nor were any changes detected on spectroscopic examination of the blood.

One of the striking things about the use of streptocide in conjunction with serum in this disease is the comparative rapidity of the response observed clinically. Fever usually abates, if not entirely, to a low level within 30 hours of the beginning of treatment, and severe headache is rarely complained of after 48 hours of treatment. There is a perceptible lag in the clearing of the cerebrospinal fluid. Patients may have become free from symptoms several days before their fluid is clear, and this lag is probably to be correlated with the development of immune bodies by the patient himself. It has been so noticeable in several of the cases that we have come to speak of it as the "latent period of streptocide." When the cerebro-spinal fluid does begin to clear, and that is usually about 3 to 4 days after the institution of treatment, it clears with surprising rapidity, and often at the end of the first week of treatment the only naked eye change is a pale yellow colouration.

The appended abstracts of the case histories and the charts of each case make it easier to see how gratifying the response to combined therapy was in some of these cases. The most important facts about the cases are shown in tabular form.

CASES.

GROUP A. TREATED WITH STREPTOCIDE AND SERUM.

CASE IA.

Lam Kam. Female. Aged 35. Admitted 23. VIII. 38.

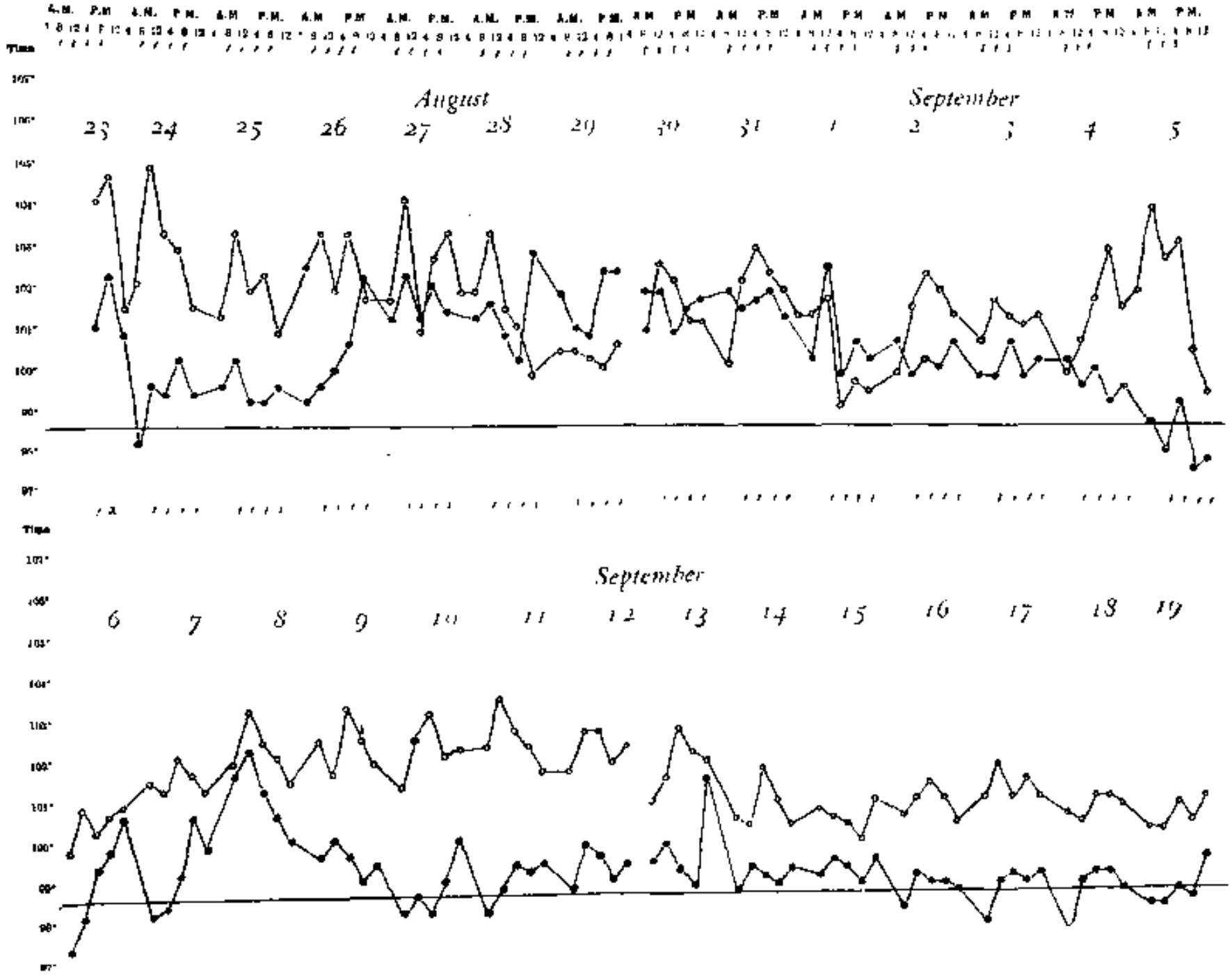
History. The woman said she had been taken ill suddenly 4 days ago. She had a rigor and fever at onset but no vomiting or nausea. She also said that headache and stiff neck had been present since the onset, and that "her fingers and legs had gone numb." There had been no sphincter trouble and no involvement of eyes or ears.

Condition on admission. She lay in the dorsal decubitus and on admission her temperature was 100.8°, pulse 128 and respirations 26. Her face was flushed and a little bloated and she rambled in speech. She showed no head retraction or opisthotonos. There was "soldering" of the cervical vertebrae and Kernig's signs was positive on both sides. Tâche was readily elicited. The cranial nerves showed no abnormality and the pupils were equal and active. The knee and ankle jerks were absent; plantar responses were flexor.

Course and Treatment. Lumbar puncture was done under local anaesthesia, and turbid cerebrospinal fluid was obtained at a pressure of 300+. Queckenstedt's sign was positive. Meningococci were found in smears, and after 35 c.c. had been drained 30 c.c. of A.M.S. were given intrathecally. 20 c.c. were also given intravenously and 10 c.c. intramuscularly. She received in all 3 grms. of streptocide by mouth on the day of admission.

Next day her temperature had dropped to normal but she was still delirious. Lumbar puncture yielded only 5 c.c. of fluid and Queckenstedt's sign was negative. Cistern puncture yielded 48 c.c. of excessively turbid fluid in which meningococci were found. Subarachnoid lavage was attempted unsuccessfully and 30 c.c. of A.M.S. were given intra-cisternally. 10 c.c of 2½% of streptocide were given intramuscularly and 4 grms. of streptocide were ordered daily by mouth, and for the first five days in hospital she received 1 grm. of streptocide daily at 8.00 a.m., noon, 4.00 p.m. and 8.00 p.m.

On the 25th her signs were unchanged and she still wandered in speech. Lumbar puncture produced only 10 c.c. of fluid. Cistern puncture yielded 50 c.c. of fluid which was less turbid than that of the day before. Subarachnoid lavage with indigo-carmine saline showed that the block was being slowly overcome. 20 c.c. of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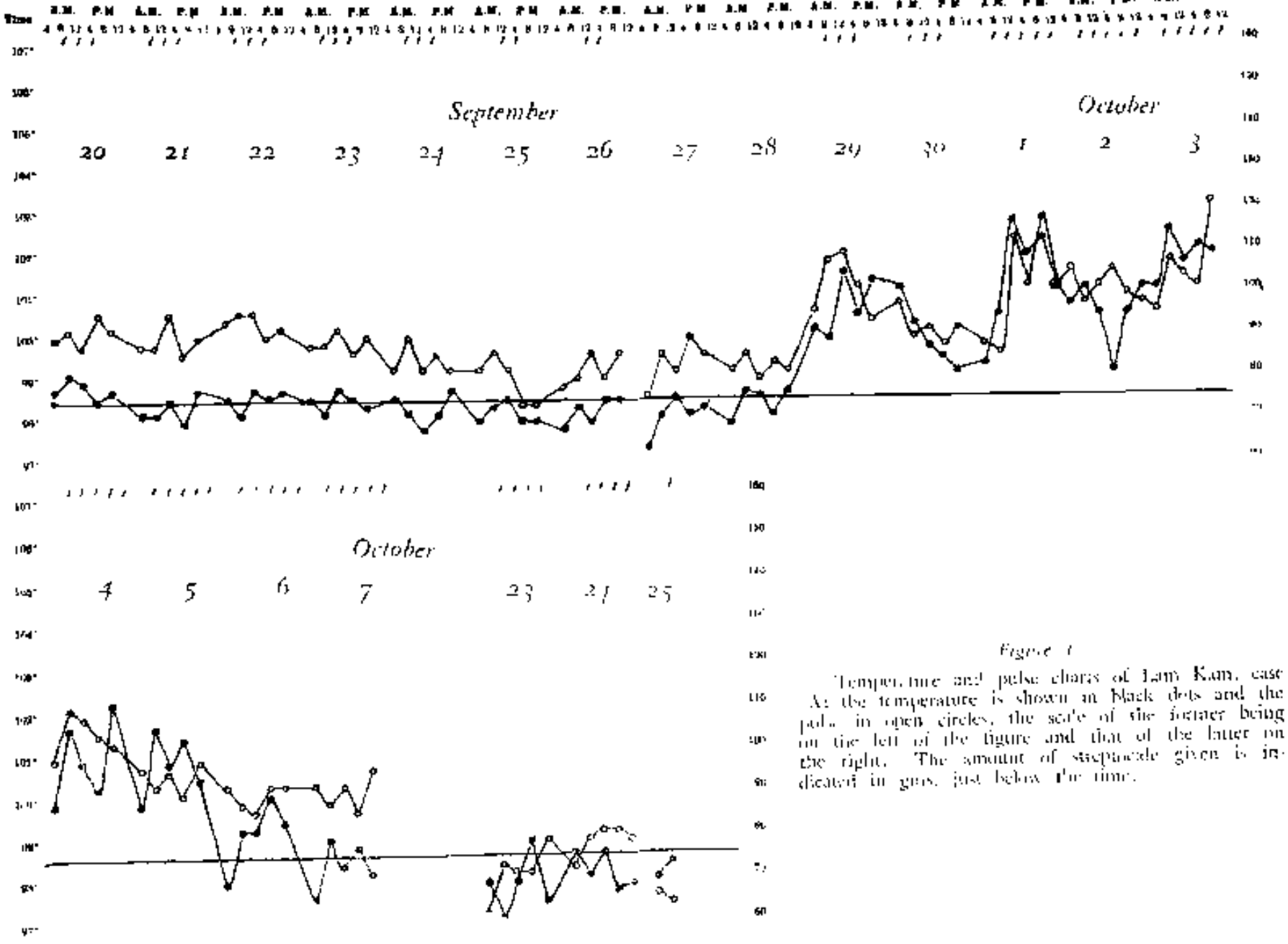


Figure 1

Temperature and pulse charts of Lam Kam, case A: the temperature is shown in black dots and the pulse in open circles, the scale of the former being on the left of the figure and that of the latter on the right. The amount of stegomyia given is indicated in guts, just below the time.

A.M.S. were given intra-cisternally. 15 c.c. by the lumbar needle. Restraint was necessary at this time as she was very restless, and she was also doubly incontinent. 10 c.c. of 2½% streptocide were given intramuscularly. By the 26th Queckenstedt's sign was positive again, and 20 c.c. of fluid were obtained by L.P., 40 c.c. by C.P., 30 c.c. of A.M.S. were given intra-cisternally and 10 c.c. of 2½% streptocide intramuscularly.

On the 29th the dose of streptocide was increased to 5 grms. each day and by the 30th she was definitely improving though her fever still ranged from 100-102°. Cistern punctures were discontinued after September 1st, though the cerebrospinal fluid still remained turbid.

On the 4th of September her fever began to abate and on the 6th her fluid was clear for the first time. She now complained of right sided weakness, and was found to have developed a right hemiparesis with a right extensor plantar response and slight spasticity of right arm and leg. The right arm became weak about 12 hours before the leg.

Streptocide was reduced to 4 grms. a day on the 7th and as her fever was settling and she was slowly improving it was again reduced to 3 grms. a day on the 16th, 2 grms. a day on the 25th and was stopped on the 27th. On the 29th she had fever 101.4°, and the drug was given again in a dose of 3 grms. a day. On the 29th the dose was increased to 5 grms. a day and she slowly settled again. Her fluid which had been clear become turbid again during this relapse, but no meningococci were found in it. She was kept on streptocide 5 grms. a day for 13 days, and then 4 grms. a day for the next 10 days. She improved steadily during this time, and was discharged on the 67th day of her convalescence. She then showed a trace of right sided weakness, but was able to walk quite well unaided, and complained of no other symptoms.

Summary. This moderately severe case illustrates the danger of using streptocide in too low a dosage. During her first week in hospital the woman had none of the drug by night, and that the dose was insufficient was shown by the promptitude with which she relapsed when the drug was prematurely discontinued. It seems probable that bolder treatment from the start would have prevented both her "block" and her right hemiparesis. Her cerebrospinal fluid took 15 days to become clear, another fact which shows that how necessary it is to maintain a constant high

level of streptocide by giving the drug 4 hourly day and night. During her 64 days in hospital she received 168.5 c.c. of A.M.S. intrathecally, 120 c.c. intra-cisternally, 20 c.c. intravenously and 8.5 c.c. intramuscularly; a total of 317 c.c. She was also given 270 c.c. of 2½% of streptocide intramuscularly and 250 grms. of streptocide by mouth.

CASE 2A.

Leung Shui. Male. aet. 17. Admitted 17. IX. 38.

History. The brother gave the following history. The illness began suddenly 6 days ago with intense headache and fever. There was no rigor or chill at onset. The patient's neck became stiff and painful about the 3rd day of the disease and on the 3rd day he vomited twice after Chinese medicine. He has been only partially conscious, and unable to swallow or answer questions for the last 2 days. He has had no coryza or pharyngitis nor have eyes or ears been involved during the illness.

Condition on admission. On admission the man's temperature was 100°, pulse 130 and respiration rate 38. He was delirious and restless, his face was bloated and congested and his conjunctivae were injected. Head retraction and nuchal rigidity were obvious and his attitude was one of generalised flexion. Tâche was readily elicited and Kernig's sign was bilaterally positive. The cranial nerves were normal and pupils were equal and active. Knee and ankle jerks were lost; plantar responses were flexor. The heart and lung sounds were normal.

Course and Treatment. He was lumbar punctured under evipan, and turbid fluid was obtained at a pressure of 300+. Meningococci were found in smears from the deposit. 40 c.c. of cerebro spinal fluid were drained, and 30 c.c. of A.M.S. were given intrathecally, 23 c.c. intravenously and 15 c.c. intramuscularly. No streptocide was given until 9 hours later as we were then testing the hypothesis that streptocide delayed the production by the body of antibodies, and should therefore be given some hours after the initial dose of serum. The outcome was not happy. 1 grm. of streptocide was given by mouth at midnight. Next day his condition was unchanged. L.P. under evipan yielded turbid fluid under a pressure of 300+. 30 c.c. were drained and 25 c.c. of A.M.S. were given intrathecally, 15 c.c. intramuscularly. 4 grms. of streptocide were given throughout the day at 8, 12, 4 and 8.

On the 19th he was conscious and could carry out simple requests. Both knee jerks and the right ankle jerk had returned, but the other signs were unchanged. He was lumbar punctured under evipan and 42 c.c. of turbid cerebrospinal fluid were drained. Queckenstedt's sign was positive as it had been from the start. 30 c.c. of A.M.S. were given intrathecally.

The man's heart sounds were soft and tictac in rhythm, and cardatone was ordered 4 hourly. He was also given 10 c.c. of 2½% streptocide intramuscularly. Streptocide was continued by mouth and he received 5 grms. throughout the day. His temperature rose to 103°, his pulse to 156 and he died suddenly early the next morning.

Summary. This man apparently died of myocardial failure, which supervened despite treatment. He had not responded well to adequate dosage with serum, but although streptocide was not pushed in this case one cannot feel that that alone accounted for his death. Unfortunately no autopsy could be made, so the condition of the myocardium was not ascertained.

He was given during his three days in hospital 85 c.c. of A.M.S. intrathecally, 23 c.c. of A.M.S. intravenously and 30 c.c. of A.M.S. intramuscularly. He also had 11 grms. of streptocide by mouth and 10 c.c. of 2½% solution of streptocide intramuscularly.

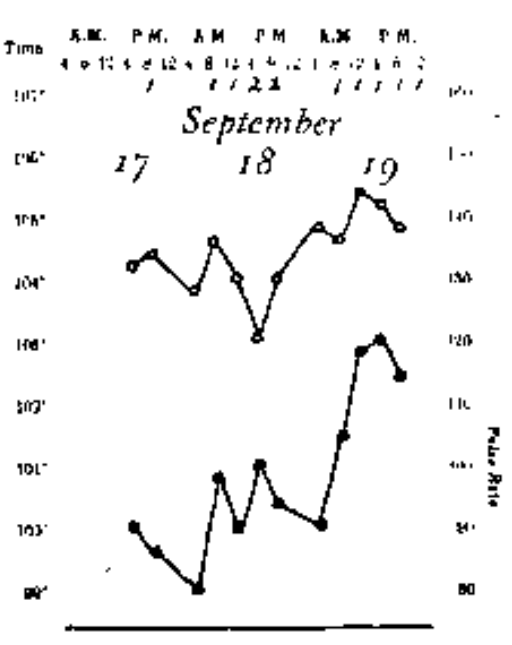


Figure 2.

Temperature and pulse chart of Leung Shui, case 2A; for explanation of chart see page 7, figure 1.

CASE 3A.

Fok Fook Seng. aet. 32. Male. Admitted 4. X. 38.

History. The man gave the following history. His illness began 8 days ago without a chill or rigor. He suddenly became feverish and had a severe frontal headache but did not vomit. This persisted for 3 or 4 days and his neck then became stiff and painful. He had no skin eruptions or petechiae, nor was there any photophobia. He had no coryza or sore throat at onset.

Condition on admission. On admission his temperature was 99°, pulse 120, respiration 24. He lay curled up on one side and wandered in speech. Head retraction and nuchal tenderness were marked. There was moderate cervical "soldering," and slight opisthotonos. No tâche was elicited nor was the belly scaphoid, but Kernig's and Brudzinski's signs were positive on both sides.

Pupils were slightly unequal and reacted sluggishly to direct light. Cranial nerves were normal. Knee and ankle jerks were present and equal and plantar responses were flexor. Sphincters were normal. The lungs, heart and belly showed no abnormality and no petechiae were found.

Course and Treatment. L.P. was done at once under local anaesthesia. Turbid fluid was obtained under a pressure of 170, and 22 c.c. were drained. Queckenstedt's sign was positive. Meningococci were found in smears made from the cerebro spinal fluid, and 20 c.c. of A.M.S. were given intrathecally. 20 c.c. were also given intravenously together with 5 c.c. of soluseptasine. 10 c.c. of 2½% solution of streptocide were given intramuscularly, and 0.5 grm. of soluseptasine was ordered 2 hourly by mouth.

The next day his temperature had fallen to normal. His signs were unchanged and he still wandered in speech. L.P. yielded 30 c.c. of turbid fluid which still contained meningococci. Queckenstedt's sign was negative. 30 c.c. of A.M.S. were given intrathecally and a further 10 c.c. of 2½% solution of streptocide intramuscularly. Treatment with 5 grms. of streptocide by mouth in the 24 hours was begun that evening as the supply of soluseptasine was exhausted.

On the 6th he was still very disoriented, his signs were unchanged and he continued to show low grade irregular fever. L.P. yielded 32 c.c. of turbid fluid and Queckenstedt's sign was doubtful. No meningococci were found. 25 c.c. of A.M.S. were given intrathecally and

treatment with streptocide by mouth was continued. 10 c.c. of 2½% solution of streptocide were given intramuscularly.

On the 7th head retraction and headache were still marked, and L.P. only yielded 5 c.c. of fluid. Queckenstedt's sign was negative, so cistern puncture was performed. The fluid was turbid but pressure was only 30. Subarachnoid lavage was carried out with 50 c.c. of normal saline and free drainage from the cistern was established. 30 c.c. of A.M.S. were given by the lumbar needle after 60 c.c. of fluid had been drained from the cistern. 10 c.c. of 2½% solution of streptocide were given intramuscularly.

On the 8th the man was rational. L.P. yielded fluid very slowly and 20 c.c. of A.M.S. were given intrathecally. The cerebrospinal fluid was by now clearing a little.

During the next three days head retraction lessened and the man's condition improved, although head movements were still limited and Kernig's sign was positive on both sides. Persistent frontal headache continued and lasted up to the 20th. At this period of the disease both optic discs were found to be blurred, but no measurable swelling was detected.

By the 14th the cerebrospinal fluid was clear, but did not become so permanently until the 20th. Streptocide was reduced to 4 grms. in the 24 hours from the 14th onwards, and no serum was given after the 17th. From the 17th of October to his discharge on the 1st of November the man remained symptom free. His papillitis subsided during his last week in hospital. Streptocide was reduced to 3 grms. in 24 hours from the 25th and Kernig's sign was negative by the 26th. He was seen on November 30th, fifteen days after his discharge and was then perfectly normal.

Summary. The response to treatment was slow, probably because streptocide was not given *every* 4 hours. The man was one of the earliest cases to be treated with large doses of streptocide which accounts for his being given only 5 grms. in the 24 hours. It is interesting to note that the cerebrospinal fluid pressure was much higher in the 3rd week of the disease when it averaged 300 than in the first week when it averaged 120. This fact may account for the persistence of the frontal headache and the late occurrence of papillitis.

Figure 3

Temperature and pulse chart of Poh Freang Seng, case 3A; for explanation of chart see page 71, figure 1.

During his month in hospital he was lumbar punctured 24 times and cistern punctured once. He received 20 c.c. of A.M.S. intravenously and 187.5 c.c. intrathecally. He also had 5 c.c. soluseptasine intravenously and 5 grms. by mouth as well as 50 c.c. of 2½% solution of streptocide intramuscularly and 111 grms. of streptocide by mouth.

CASE 4A.

In Kwai Ying. act. 12. Female. Admitted 20. X. 38.

History. The child's mother said she had been ill for four days. The onset was sudden with rigor, fever and vomiting. Frontal headache was marked at onset and had persisted.

Her neck became stiff 24 hours after onset and the head became retracted during the last two days. There were no skin rashes or eruptions. The eyes and ears were not involved. Sphincters and swallowing were not impaired. The child became delirious two nights ago.

Condition on admission. The child's temperature on admission was 100.6°, pulse 108 and respirations 24. She rambled in speech but was able to answer questions with an effort. She lay curled up on her side in the gun-hammer attitude. The head was retracted and neck muscles were tender on pressure. The cranial nerves and nervous system showed no abnormalities. The pupils were semidilated, equal and active. A tâche was readily elicited and Kernig's and Brudzinski's signs were positive.

Course and Treatment. Lumbar puncture was performed at once and turbid yellow fluid was obtained under a pressure of 230. 35 c.c. were drained and meningococci were demonstrated in smears from the deposit.

30 c.c. of A.M.S. were given intrathecally and 5 c.c. of a 2½% solution of streptocide intramuscularly. Streptocide 1 grm. was ordered by mouth in a dose of 5 grms. every 24 hours.

Next day head retraction and nuchal tenderness were more marked than on admission. L.P. yielded 65-70 c.c. of turbid fluid, and 20 c.c. of A.M.S. were given intrathecally. Serum was given intrathecally for the next five days as well as streptocide 5 grms. by mouth every 24 hours.

By the 23rd the head retraction was lessening and the cerebrospinal fluid was clearing, and by the 26th the child had no symptoms and her cerebrospinal fluid was clear. From that time on her recovery was uneventful,

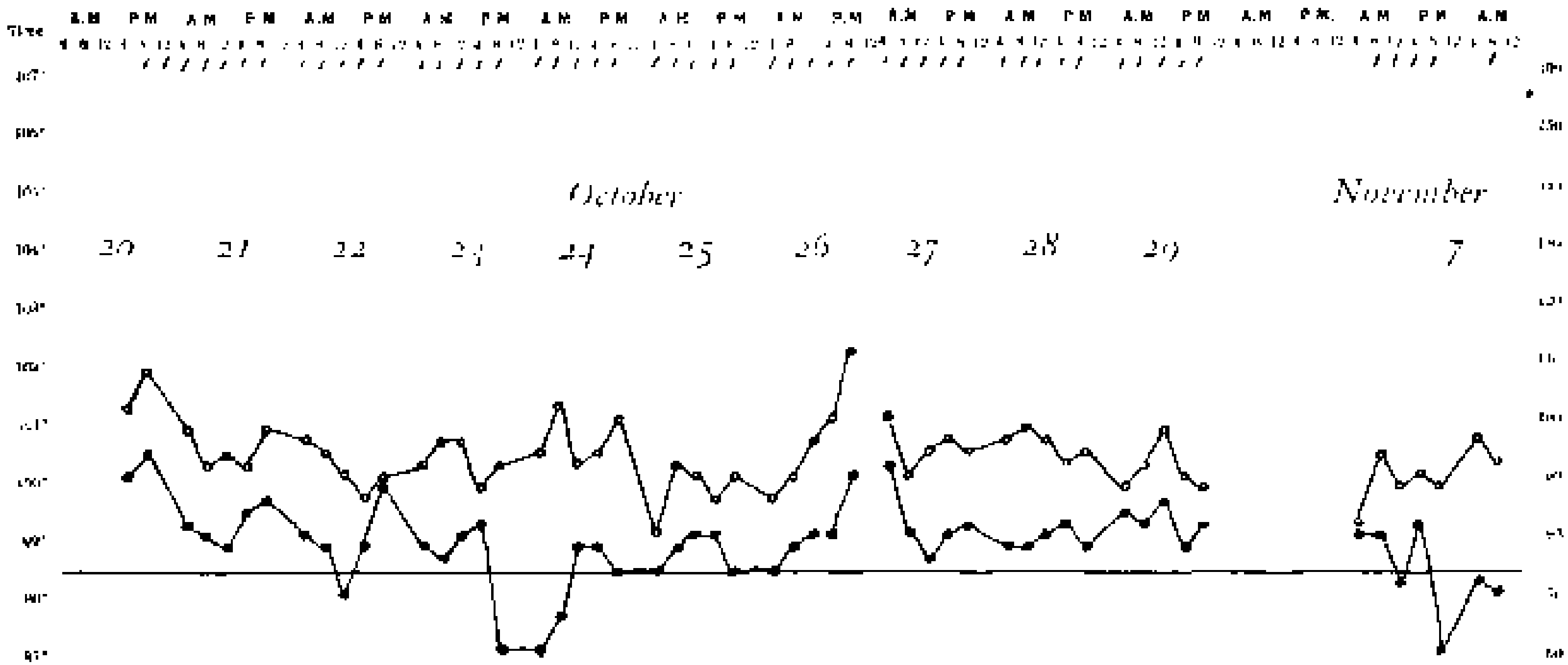


Figure 2.

Temperature and pulse chart of Dr. Kwai Ying, case 4A; for explanation of chart see page 7, figure 1.

and the cerebrospinal fluid obtained by L.P. on November 8th, when she was discharged from hospital, showed no organisms or increase of cells or globulin.

She was seen a fortnight later on the 23rd of November and showed no signs or symptoms.

Summary. This child during her 20 days in hospital had 165 c.c. of A.M.S. intrathecally, 89 grms of streptocide by mouth and 5 c.c. of 2½% solution of streptocide intramuscularly. She was lumbar punctured 15 times, but meningococci were never found in the cerebrospinal fluid after the first 24 hours of treatment. The cerebrospinal fluid was clear by the seventh day. The course of the disease was entirely without incident and it is probable that she would have recovered with an even smaller total amount of serum. She gained 3½ lbs. in weight during her convalescence and at no time did she show any toxic symptoms attributable to streptocide poisoning. The case illustrates well how smooth the course of the disease may be when a combination of streptocide and serum is used in treatment. The charts show the amounts of the drugs used and the times when they were given.

CASE 5A.

Tsui Kun Shuen. Male, aet. 16. Admitted 26. X, 38.

History. The boy's grandmother gave the following history. He had a rigor and complained of feeling cold at 11.00 p.m. on the 25th of October. He also vomited. Next morning he had a sore throat and a cold but went to school. His grandmother found him in bed that afternoon semiconscious and feverish. She noticed that purple spots were coming out on his body and thigh. He vomited again that evening and was sent in to hospital at midnight on the 26th.

Condition on admission. On admission his temperature was 100°, pulse 124 and respiration 32. He was flushed, wandering in speech and very restless. His face was a little bloated, lips were slightly cyanosed, and he was completely disoriented in time and space. He showed slight head retraction and marked tenderness on pressure over the deep cervical muscles. The right pupil was larger than the left and both reacted sluggishly to direct light. The cranial nerves were normal. The right knee jerk was obtained but the other tendon reflexes were lost. The abdominal reflexes were active, plantar responses were flexor and sphincters were said to be normal.

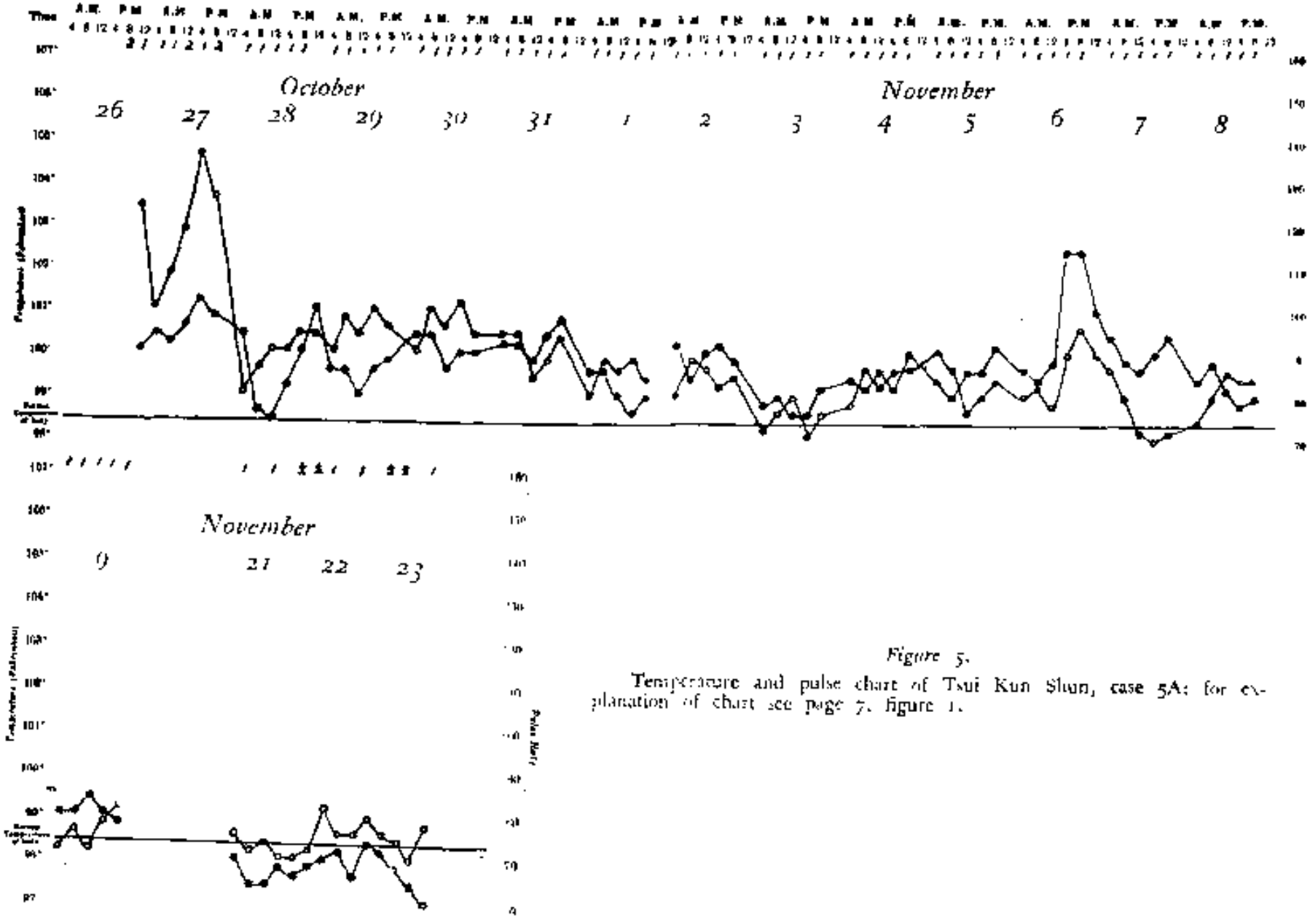


Figure 5.

Temperature and pulse chart of Tsui Kun Shun, case 5A: for explanation of chart see page 7, figure 1.

Kernig's and Brudzinski's signs were positive, but râche was only elicited with difficulty. There were numerous pinpoint haemorrhages in the lower palpebral conjunctiva on both sides, and a small subconjunctival ecchymosis at 11.00 a.m. in the right eye. There were numerous petechiae on the wrists, forearms, buttocks, thighs and legs. They were purple in colour and irregular in outline; they ranged in size from 1 to 4 m.m. in diameter and did not fade on pressure. Fundi and tympana were normal.

Course and Treatment.

L.P. was performed under local anaesthesia, and yielded exceedingly turbid fluid under a pressure of 300+. Meningococci were found in smears from this fluid but none were found in later specimens of the cerebrospinal fluid. Queckenstedt's sign was doubtfully positive and it was feared that block might already have become established. 55 c.c. of fluid were drained and 30 c.c. of A.M.S. were given intrathecally, 15 c.c intravenously, 2 grms. of streptocide were given by mouth and from then on 1 grm. 4 hourly was ordered.

Next day his condition was unchanged. He was still very restless and restraint was necessary. Evipan had to be given for the L.P. which yielded 60 c.c. of fluid. 30 c.c. of A.M.S. were given intrathecally, and 2 grms. of streptocide were given by nasal tube as he refused to swallow and a further 2 grms. of streptocide were given by nasal tube that evening as he again refused to swallow.

On the 28th he had regained consciousness and could answer questions. Head retraction had now disappeared but the other signs were unchanged. The cerebrospinal fluid was still very turbid and flowed slowly, but Queckenstedt's sign was positive. 30 c.c. were drained and 25 c.c. A.M.S. were given intrathecally, 12 c.c. intravenously, 10 c.c. of 2½% streptocide were given intramuscularly, and as he could now swallow 1 grm. of streptocide was continued 4 hourly.

On the 29th he was much improved and was perfectly rational. Head movements were freer and his petechiae were beginning to turn brown. His cerebrospinal fluid was becoming less turbid, and was clear by the 31st. The lid haemorrhages had disappeared by the 1st November and the petechiae were fast fading. He continued to have low-grade irregular fever until the 8th of November, although he had been reading each day since the 2nd.

He complained of slight joint pains and his fever rose to 102.4° on the 6th, a reaction probably due to serum.

He complained of diplopia on looking to the right on the 8th of November but nothing was found on examination to account for this. At this stage of convalescence he was "feeling very tired" and was toneless and apathetic. His lips were bluish but not more so than on admission. Anorexia was pronounced, and he had obviously lost weight since admission. On the 10th he was able to sit up, and his streptocide was reduced to 4 grms. in the 24 hours. By the 12th he was able to walk unaided and from that time on his convalescence was rapid and uneventful and he began to put on weight during his third week in hospital.

Summary. Conclusions. This boy responded well to combined treatment with streptocide and serum. It is probable that he was given more serum than was necessary, and this may have accounted for his mild serum reaction on November the 6th. Towards the end of his second week in hospital he showed signs of mild streptocide poisoning, but it never became necessary to reduce the dose of the drug suddenly. His cerebrospinal fluid became sterile in 24 hours and was clear 6 days after admission.

During his 29 days in hospital he was given 192 c.c. A.M.S. intrathecally and 27 c.c. intravenously. He also had 40 c.c. of 2½% streptocide solution by intramuscular injection and 122 grms. by mouth. Cerebrospinal fluid obtained by L.P. three days before his discharge was normal. He was seen again 19 days after his discharge, on December 12th, and said he had been perfectly well since leaving hospital. Examination showed no physical signs in any system, and he was sent back to school the next day.

CASE 5A.

Wong Put Chai. æt. 10. Male. Admitted 3. XI. 38.

History. The mother gave the following history. The child became feverish and vomited at 5.00 p.m. on the 1st of November. He complained of frontal headache. Next day his neck was stiff and his head became retracted. His back was painful on pressure, he slept poorly and was delirious at night. He coughed up much mucus from the nasopharynx during the first two days of his illness, but had no obvious coryza. He had no involve-

ment of eyes or ears, nor were there any skin rashes or petechiae.

Condition on admission. His temperature on admission was 101.2°, pulse 128 and respirations 36. He lay curled up on one side in the gun-hammer position, his skin was hot and dry, his face flushed. He wandered in speech and was so restless that restraint was necessary. He could only swallow with difficulty and was unable to answer questions.

Head retraction and nuchal tenderness were marked. There was "soldering" of the cervical vertebrae and opisthotonos. Tâche was readily elicited and Kernig's and Brudzinski's signs were positive. The pupils were unequal, the right being greater than the left, and fixed to direct light. The cranial nerves showed no abnormality. The left ankle jerk was obtained, but the other tendon reflexes and abdominal reflexes were absent. Plantar responses were flexor. The heart and lungs sounds were normal. The belly was slightly scaphoid.

Course and Treatment. Lumbar puncture was done at once under ether. The fluid was turbid and under a pressure of 300+ but Queckenstedt's sign was positive. Meningococci were found in smears from the fluid. 40 c.c. were drained and 30 c.c. of A.M.S. were given intrathecally, 20 c.c. intravenously, 2 grms. of streptocide were given by mouth. His respiration rate rose to 52 within 1 hour of the exhibition of serum but atropine and adrenalin overcame this dyspnoea. Streptocide was continued in doses of 1 grm. 4 hourly.

On the 4th he was still restless and delirous, but able to swallow. His signs were unchanged, except that a cluster of herpetic vesicles had appeared at both corners of the mouth. 50-60 c.c. of turbid cerebrospinal fluid were drained but 30 c.c. of A.M.S. given intrathecally. Meningococci were still present in smears. Streptocide was continued by mouth, 1 grm. 4 hourly. The child was extremely noisy and restless all the afternoon and could only be controlled by rectal paraldehyde. A further lumbar puncture was done at 6.00 p.m. and cistern puncture was also performed. Subarachnoid lavage was carried out with normal saline, because Queckenstedt's sign had become negative. Plantar responses were now both extensor, and he was doubly incontinent.

On the 5th physical signs were unchanged, save that a left 6th cranial nerve paresis had now developed and the left eye was divergent. The cerebrospinal fluid was slightly

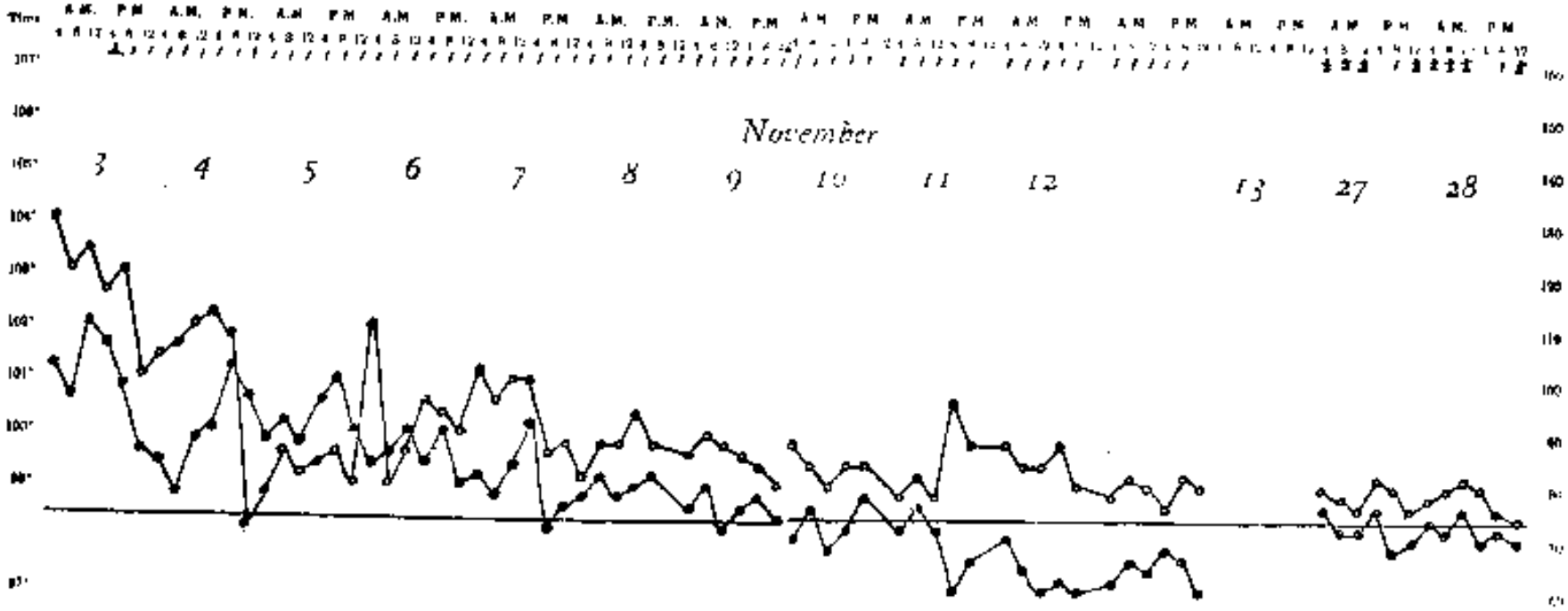


Figure 6

Temperature and pulse chart of Wm. g Pa. Chas. case 6A; for explanation of chart see page 7, figure 1.

clearer and the pressure was lower. 180. No meningococci were noted in the fluid. 30 c.c. of A.M.S. were given intrathecally. That evening he was able to recognise people, but was still restless and noisy. Lumbar puncture was done at 7.00 p.m. and 30 c.c. of fluid drained slowly.

On the 6th he was beginning to improve and could ask for drinks. The plantar responses were now flexor and the cerebrospinal fluid was deep yellow and clearer. He was still incontinent of urine.

Next day he was able to answer simple questions and head retraction was very much less. The left squint had disappeared. Despite his improvement he still needed ether for his lumbar puncture and was still incontinent of urine. Head retraction became much more marked after giving ether.

By the 8th he was able to talk rationally, and by the 10th he could sit up in bed. No A.M.S. was given after the 10th and streptocide was now reduced from 6 to 5 grms. in the 24 hours.

On the 11th his fluid was clear and pale yellow. Kernig's sign was still marked and there was still considerable generalised rigidity.

By the 14th the cerebrospinal fluid was clear and colourless and streptocide was reduced to 4 grms. in the 24 hours, and by the 15th head movements were free. He was still a little rigid in walking. From this time until his discharge on the 29th he remained free of symptoms. His last lumbar puncture on the 25th yielded normal cerebrospinal fluid. Kernig's sign was still positive but painless when he was discharged.

Summary and Conclusions.

The child was 27 days in hospital. During this time he was lumbar punctured 19 times, cistern punctured once. He was given 20 c.c. of A.M.S. intravenously and 175 c.c. of A.M.S. intrathecally. He received in all 113 grms. of streptocide by mouth. At no time did he show any signs of streptocide poisoning. His weight increased during treatment from 41 lbs. on the 7th, five days after admission, to 49 lbs. on the 27th, two days before discharge.

That the illness was fairly severe is shown by the fact that he developed a 6th nerve paresis during the first few days in hospital. The case illustrates well the value of a combination of streptocide and serum in treatment.

CASE 7A.

Choi San Wan. aet. 4. Female. Admitted 5. XI. 38.

History. The father gave the following history. The child became ill 6 days ago. She had no rigor, chill or convulsion, but suddenly developed fever and complained of pain in the head. She vomited twice and had persistent nausea. Hyperaesthesia was marked and the child cried out when she was touched or picked up.

On the 2nd day of the illness the neck became stiff and head retraction began to appear. She had been delirious at night for the past 4 nights, but swallowing and speech had not been impaired. She had had no eye or ear symptoms, nor had there been any skin rashes or sphincter disturbances.

Condition on admission. On admission the child's temperature was 99.8°, pulse 128 and respirations 24. She lay curled up on her side and head retraction was marked. Anterior and posterior head movements were limited but lateral movement was free. No nuchal tenderness was demonstrable. The cervical vertebrae were "soldered." Tache could be elicited but the belly was not scaphoid. Kernig's sign was positive on both sides. The right pupil was fractionally greater than the left, and both reacted through a small range to direct light. There was no strabismus and the cranial nerves were normal. The knee and ankle jerks and the abdominal reflexes were absent; plantar responses could not be obtained. None of the other 8 children at home had been affected in any way, and the patient had shown no catarrhal symptoms or sore throat at onset.

Course and Treatment. Lumbar puncture was done at once under ether as the child was restless and frightened. Turbid cerebrospinal fluid was obtained under a pressure of 300 , and 45 c.c. were drained. Meningococci were found in smears, and as the fluid was very thick the lumbar theca was washed out with 10 c.c. of warm normal saline. 30 c.c. of A.M.S. were given intrathecally and 10 c.c. intramuscularly. 1 grm. of streptocide was given immediately by mouth, and 4 hours later 0.5 grm. was begun 4 hourly. Next day signs were unchanged. Lumbar puncture was done under ether, and 50 c.c. of turbid fluid were obtained under pressure. 30 c.c. of A.M.S. were given intrathecally, and streptocide was continued as before.

On the 7th fever still persisted though the head retraction was lessening a little. L.P. under ether yielded

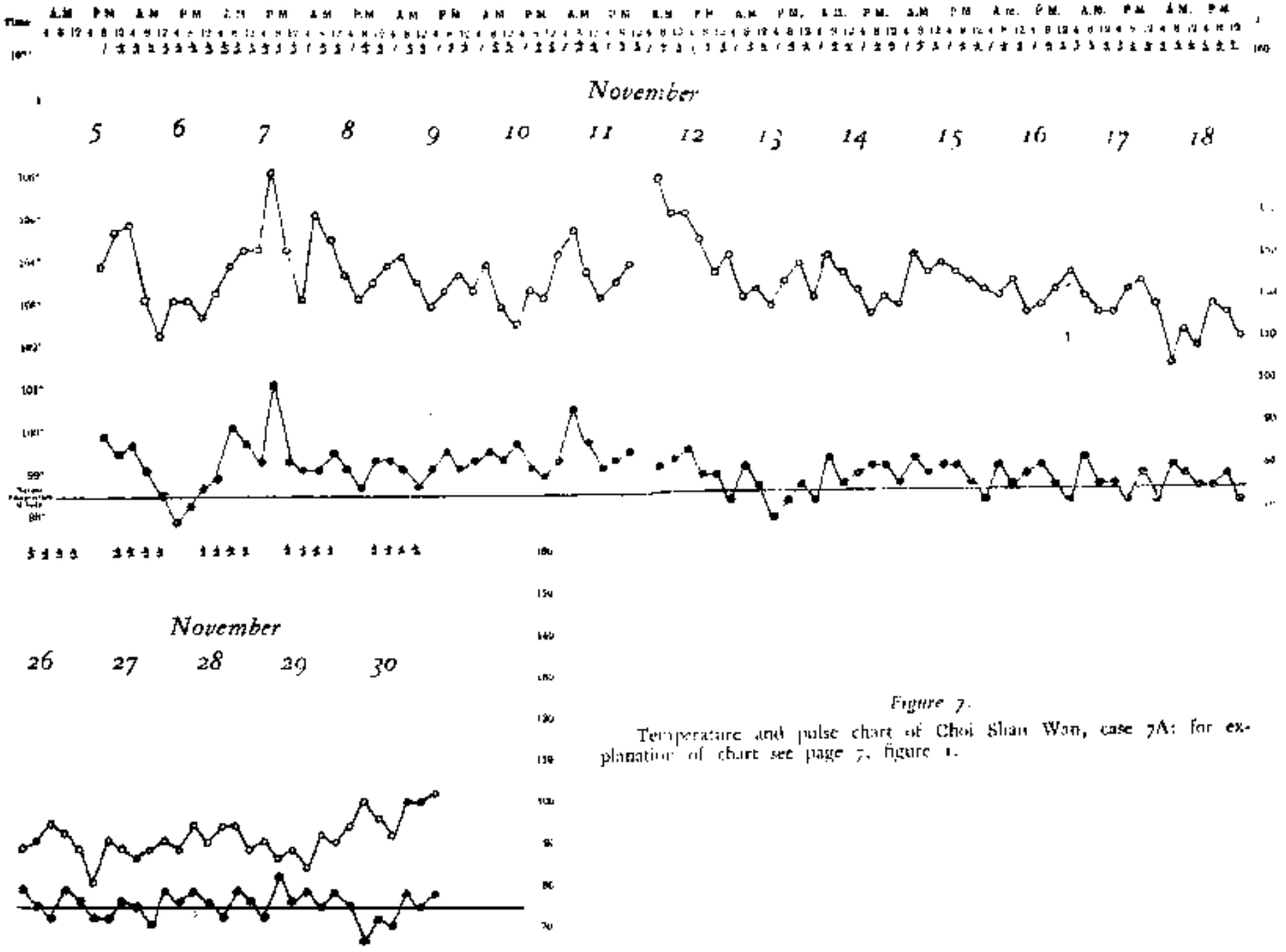


Figure 7.

Temperature and pulse chart of Choi Shan Wan, case 7A: for explanation of chart see page 7, figure 1.

40 c.c. of turbid fluid, and Queckenstedt's sign was still positive as it had been from the beginning. 25 c.c. of A.M.S. were given intrathecally. The child was still very restless, and tooth grinding and Biot's type of respiration were both noted when she was asleep.

On the 8th her fever had abated but signs were unchanged. The fluid was still very turbid but drained freely and there was no sign of block. 22 c.c. of A.M.S. were given intrathecally.

During the next three days the child's condition improved slowly and no A.M.S. was given after the 10th. Her fever had abated to 99°, but her general rigidity remained very marked and she was unable to sit up by herself. Her fluid was clear on the 13th, but became turbid again on the 14th, and did not become permanently clear till the 23rd. Streptocide which had been given in a dosage of 3 grms. in the 24 hours during the first two days of the illness had been raised to 4 grms. in 24 hours on the 8th and was kept at that level until the 17th. From the 17th to the 25th she received 3 grms. daily, from the 26th to the 30th of November, when she was discharged, 2 grms. daily. She showed no signs of streptocide poisoning at any time. By the 17th she was able to sit propped up but was still very rigid and could not walk. Hot baths daily helped in lessening the rigidity and by the 27th she was able to sit up by herself and to play with her toys. Her cerebrospinal fluid was clear on the 29th, and on the 30th her father took her home against advice.

Summary. The child spent 26 days in hospital and was then taken home by her father against advice. During her stay in hospital she was lumbar punctured 22 times and received in all 153 c.c. of A.M.S. intrathecally, 10 c.c. A.M.S. intramuscularly and 80.5 grms. of streptocide by mouth.

As her charts show her temperature was long in settling down, and the fact that her temperature rose to 99.2° the day before her parents took her home shows clearly that there was a strong possibility of relapse occurring. The persistence of her generalised rigidity and the long time the cerebrospinal fluid took to clear were also striking features of the case, and may have been due to insufficient streptocide.

CASE 8A.

Lau Yun. Female. aet. 6. Admitted 5. XII. 38.

History. The child's aunt said that the illness began suddenly with fever on the 2nd. On the following day the child complained of frontal headache and vomited twice. She had two convulsions that night and was delirious. On the 4th her head became retracted and she complained of stiff neck. Photophobia was also noticed at this stage. No skin rashes or petechiae were noted and the sphincters were not involved.

Condition on admission. The child's temperature was 100° on admission, pulse 124, respiration 26. She lay curled up on her side. Head retraction and nuchal tenderness were marked, and cervical "soldering" was easily demonstrable. No tâche could be elicited, nor was the belly scaphoid. The pupils were fractionally unequal but reacted to light. The cranial nerves were normal. Knee and ankle jerks were absent and the right plantar was extensor, the left flexor. Kernig's sign was positive on both sides. The heart and lung sounds were normal, but respirations were jerky and irregular.

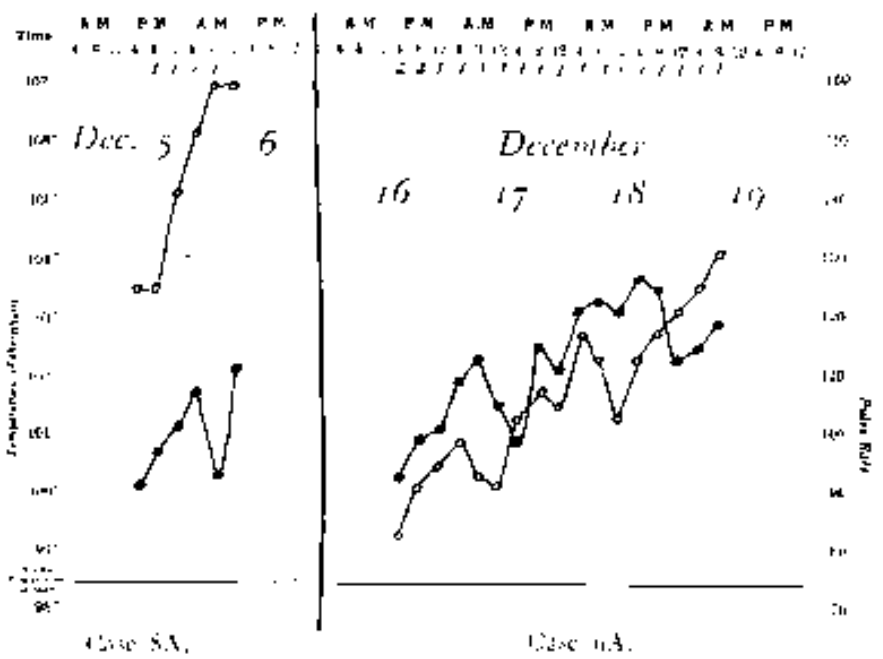
Course and Treatment. Lumbar puncture was performed under local anaesthesia and turbid fluid was obtained under a pressure of 110. Both extra and intracellular meningococci were demonstrated in abundance in smears made from the deposit. Queckenstedt's sign was positive. 25 c.c. of fluid were drained and 23 c.c. of A.M.S. were given intrathecally. 1 grm. of streptocide by mouth was ordered 4 hourly. Next day the child's condition had deteriorated slightly and cerebrospinal fluid flowed very slowly from the lumbar theca when puncture was done. The pressure was unreadable and Queckenstedt's phenomenon was doubtful. It was clear that block was becoming established and cistern puncture was done but was dry. Only 12 c.c. of fluid were obtained from the lumbar needle.

The child's pulse rate was by now 150 and continued to rise. She died early in the afternoon, never having been fully conscious since admission.

Summary. The child lived just over 24 hours from admission. During that time she was lumbar punctured twice and received 23 c.c. of A.M.S. intrathecally and 4 grms. of streptocide by mouth. The finding of both extra and intracellular meningococci showed that the infection was

a massive one and the treatment given was not nearly vigorous enough to overcome it. Block became established on the 5th and last day of the disease.

At autopsy the brain was bright red and the cortex was soft. All the superficial vessels were intensely engorged, and all the main vessels were sheathed with pus. No basal matting was found.



Case 8A. Case 9A.

Figure 3

On the left is the temperature and pulse chart of Lau Yun, case 8A and on the right that of Diu Kan, case 9A: for explanation of chart see page 7, figure 1.

CASE 9A.

Diu Kan. Female. aet. 26. Admitted 16, XII. 38.

Patient was admitted on 16, XII, 38 from Fanling Refugee Camp. No history was obtainable as she was only partially conscious and could not answer questions.

Condition on admission. On admission her temperature was 100.2°, respirations 26 to the minute, pulse 80. She showed a slight degree of head retraction and marked tenderness of posterir cervical muscles on pressure. Head movements were limited and the cervical vertebrae "soldered." Tâche was easily elicited and both Kernig's and Brudzinski's signs were present. The pupils were unequal, the right being 3 mm. in diameter, the left 2 mm. The left pupil did not react to light, but the right reacted sluggishly. The conjunctivae were injected. The fundi and tympana were normal and there was no squint. Plantar responses were flexor and the only tendon reflex present was the right ankle jerk. Heart sounds were clear in all areas and the

heart was not enlarged to percussion. Both lungs were oedematous and rhonchi and sibili could be heard all over both sides of the chest. The alae nasi were in action and the respiration rate on admission was 26. Neither herpes nor petechiae were noted.

Course and Treatment. Lumbar puncture was done immediately, and yielded turbid fluid under a pressure of 300+. Queckenstedt's sign was positive. Numerous meningococci were found in smears but culture yielded no growth. 30 c.c. were drained and 20 c.c. A.M.S. was given intrathecally, 10 c.c. intravenously. 2 grms. of streptocide were given by nasal tube as the patient was unable to swallow and a further 2 grms. were given by the same route 4 hours later. From then on 1 grm. was given 4 hourly by mouth. The bladder which was distended was emptied by catheter and atropine gr.1/100 was given hypodermically to control the bronchitis and pulmonary oedema. On the 17th the patient was able to swallow and to answer simple questions, but her pulmonary condition had not improved, and there were signs of incipient consolidation at the right base. Temperature was now 102°. Lumbar puncture yielded a fluid which was still under great pressure and still very turbid. 30 c.c. were drained and 20 c.c. A.M.S. were given intrathecally. Streptocide by mouth was continued, and an expectorant mixture ordered. Patient was kept in the Fowler position. Catheterisation was still essential each day. On the 18th tubular breath sounds were heard all over the right base and the apex beat was ½" outside the nipple line in the 5th space. 55 c.c. of cerebrospinal fluid were drained and 20 c.c. A.M.S. given intrathecally.

Death occurred early on the 19th. At autopsy the brain showed marked by hyperaemia and softening of the cortex. Some free pus was found in the left lateral ventricle and the 4th ventricle and there was a small amount of purulent exudate in and around the circle of Willis. The right lung showed consolidation of the middle and lower lobes and excised portions of these lobes sank in water. There were no other noteworthy changes.

Conclusions. During her 64 hours in hospital the patient received altogether 60 c.c. of A.M.S. intrathecally and 10 c.c. intravenously. 19 grammes of streptocide had been given in all, 5 by nasal tube and 14 by mouth. The appended chart helps to show how the pneumonic process overshadowed the meningitis towards the end. It is fair to

assume from the lung signs on admission that the woman had been ill for some days before being sent in to hospital. It is equally fair to say that it was her pneumonia rather than her meningitis which killed her.

GROUP B. TREATED WITH STREPTOCIDE.

CASE 13.

Koo Fung. aet. 15. Female. Admitted 16. XI. 38.

History. The girl's mother gave the following history. Her daughter became suddenly ill 6 days ago with a rigor, fever and repeated vomiting. She complained of severe frontal headache at onset, and nausea persisted. Her neck became stiff on the 3rd day of the illness, but she noticed no head retraction. She also developed some herpetic vesicles on both lips 3 days ago. She had had no squint or impairment of vision. There had been no deafness and she had had no petechiae. She had been delirious for the last two nights. Her bowels had been costive throughout the illness. She had a sore throat at onset, but no coryza.

Condition on admission. The girl was rational and cooperative though her temperature on admission was 103°, her pulse 100 and her respiration 24.

She lay curled up on her side and a slight degree of head retraction and generalised flexion were obvious. There was tenderness on pressure over the posterior cervical muscles. Kernig's and Brudzinski's signs were positive and there was definite "soldering" of the cervical vertebrae. She had clusters of herpetic vesicles on both lips on the chin, on the left ½ of the soft palate and on the left tonsil. She had a marked tonsillitis and nasopharyngitis and the nasopharynx contained much mucopus, which she was continually coughing up. Pupils were equal and active. The fundi and tympana were normal and the cranial nerves were unimpaired. The ankle jerks were absent, but plantar responses were flexor and knee jerks could be obtained.

Course and Treatment. The heart sounds were clear but there were a few sibili at both bases. Liver and spleen were not palpable. She was lumbar punctured at once, and 20 c.c. of whitish yellow, turbid fluid were drained. The pressure was only 160 and Queckenstedt's sign was positive. Meningococci were found in smears of the deposit. She was given 10 c.c. of 2½% solution of streptocide intramuscularly, 2 grms. of streptocide by mouth and a further 2 grms. 4 hours later. From then on she received 1 grm. 4 hourly

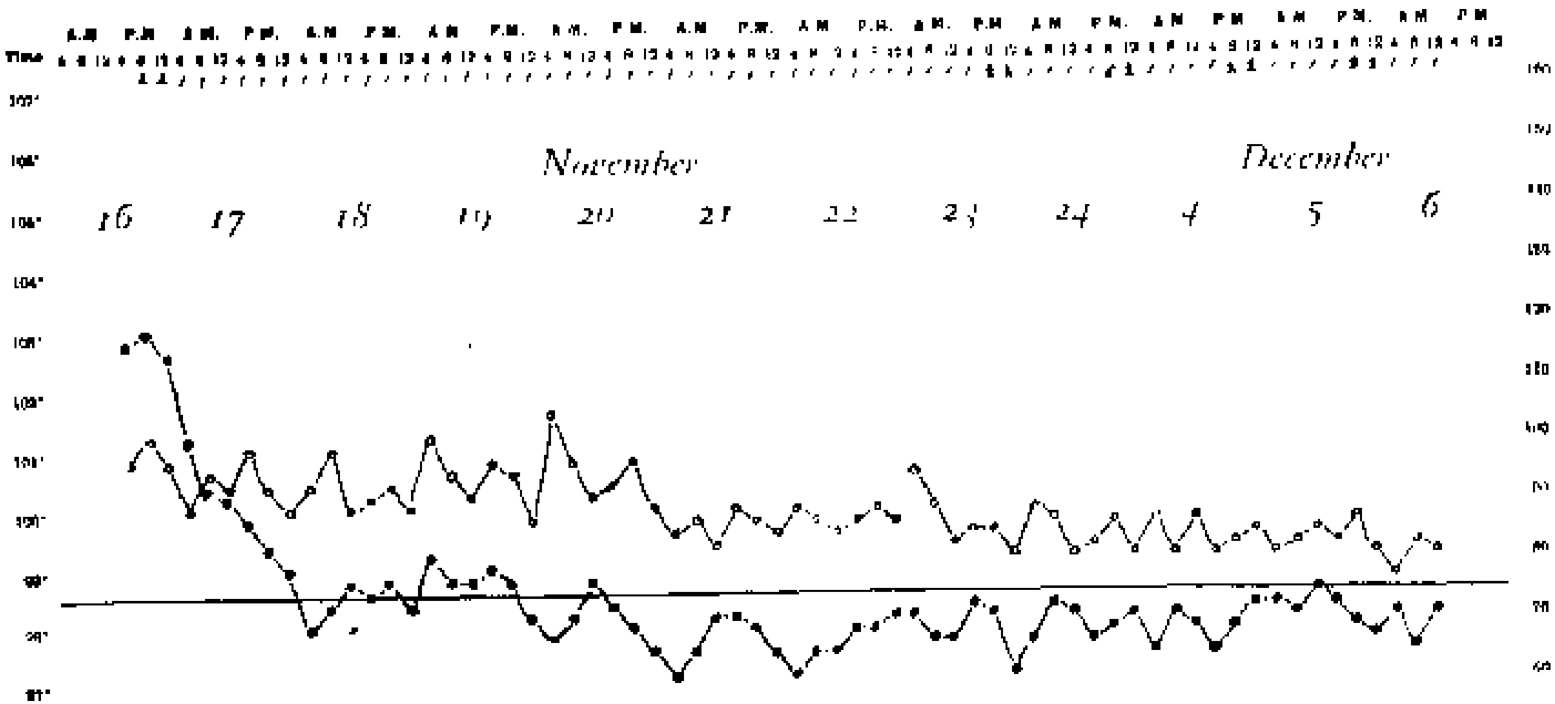


Figure 3.

Temperature and pulse chart of Koo Foong, case 11; for explanation see page 7, figure 3.

day and night. On the 17th she said her headache had gone. A new patch of herpes had appeared on the right cheek. Lumbar puncture yielded highly turbid fluid under a pressure of 260. 25 c.c. were drained. No meningococci were found in this or succeeding specimens of fluid.

On the 18th she was afebrile and could sit up in bed. The fluid was clear and pale yellow in colour by the 21st, that is to say within 6 days of beginning treatment and by this time she could walk though stiffly, and her pharyngtis and cough had completely disappeared. Kernig's sign was still strongly positive but head movements were free and painless in all directions. Her streptocide was reduced from 6 to 5 grms. in the 24 hours on the 24th. By the 25th she was walking without a trace of rigidity and from that time until her discharge from hospital she complained of no symptoms.

The fluid was slightly turbid on the 29th of November, but there was no fever or anything to suggest a recrudescence of infection. Her final fluid taken on December 3rd was clear and colourless, showed no organisms or increase of cells and had only a slight increase of globulin.

Summary and Conclusions. This was a mild case of the disease which responded readily to treatment. The rapidity with which the fever declined after treatment was begun, and the complete absence of toxic signs are noteworthy. It is also interesting to note that a new patch of herpes appeared during the first 18 hours of treatment with streptocide.

Her stay in hospital lasted 21 days, her whole illness 27. During her time in hospital she was lumbar punctured 13 times and had 10 c.c. of 2½% streptocide solution intramuscularly and 109 grms. of streptocide by mouth.

CASE 28

To So. Female. aet. 10. Admitted: 20.XI.38.

History The history given by the father was that the child had been taken ill seven days ago. The onset was sudden with a chill followed by fever. The child vomited several times and complained of frontal headache, but no stiffness or pain was noted until the fourth or fifth day of the disease. She was then taken to a Chinese hospital and transferred to the Infectious Diseases Hospital after lumbar puncture. She had been delirious at night for the preceding three nights but had had no difficulty in swallowing or speaking. Photophobia and slight deafness became evident on the

4th day and had persisted since then. No petechiae, no herpes. Coryza and nasopharyngitis were marked at the onset of the disease, but though the child coughed up much mucus she had had no sore throat.

Condition on admission.

On admission her temperature was 99.6°, respirations 22 and pulse 112. Generalised flexion was obvious. The head was slightly retracted, there was definite tenderness of posterior cervical muscles and head movements were limited in all directions. Cervical vertebrae were "soldered." There was marked photophobia but no tâche. The belly was not scaphoid. Kernig's and Brudzinski's signs were positive on both sides.

The throat showed no abnormality. The fundi and tympana were normal and the cranial nerves showed no impairment. Pupils were equal and active to direct light. The deep tendon reflexes were all slightly exaggerated, plantars were flexor and abdominals absent. There was a tendency to patellar clonus on both sides. The sphincters were normal. The child was a little over-talkative but was intelligent and cooperative.

Course and Treatment.

Lumbar puncture was done under local anaesthesia. Fluid was very turbid, yellowish white in colour and under a pressure of 300. Meningococci were found in smears from the deposit. Queckenstedt's sign was positive. 30 c.c. were drained and no serum was given. 2 grms. of sterptocide were given by mouth and this dose was repeated 4 hours later. From then on 1 grm. was given by mouth 4 hourly day and night.

On the 21st the child was delirious and so restless that she could only be kept in bed with the aid of a jacket. Her condition was not as good as on admission. Head retraction and Kernig's sign were both marked, and the cerebrospinal fluid was still under pressure and slightly more turbid than at the first puncture. 45 c.c. were drained, and meningococci were still present. 5 c.c. of 2½% solution of streptocide were given intramuscularly and 1 grm. by mouth was continued 4 hourly.

On the 22nd the child was much quieter and not quite rational in speech. The nuchal rigidity was less and she complained of no headache or pain. Lips were a little cyanosed. The cerebrospinal fluid was still turbid and did not flow very freely. 25 c.c. were drained and smears showed no meningococci.

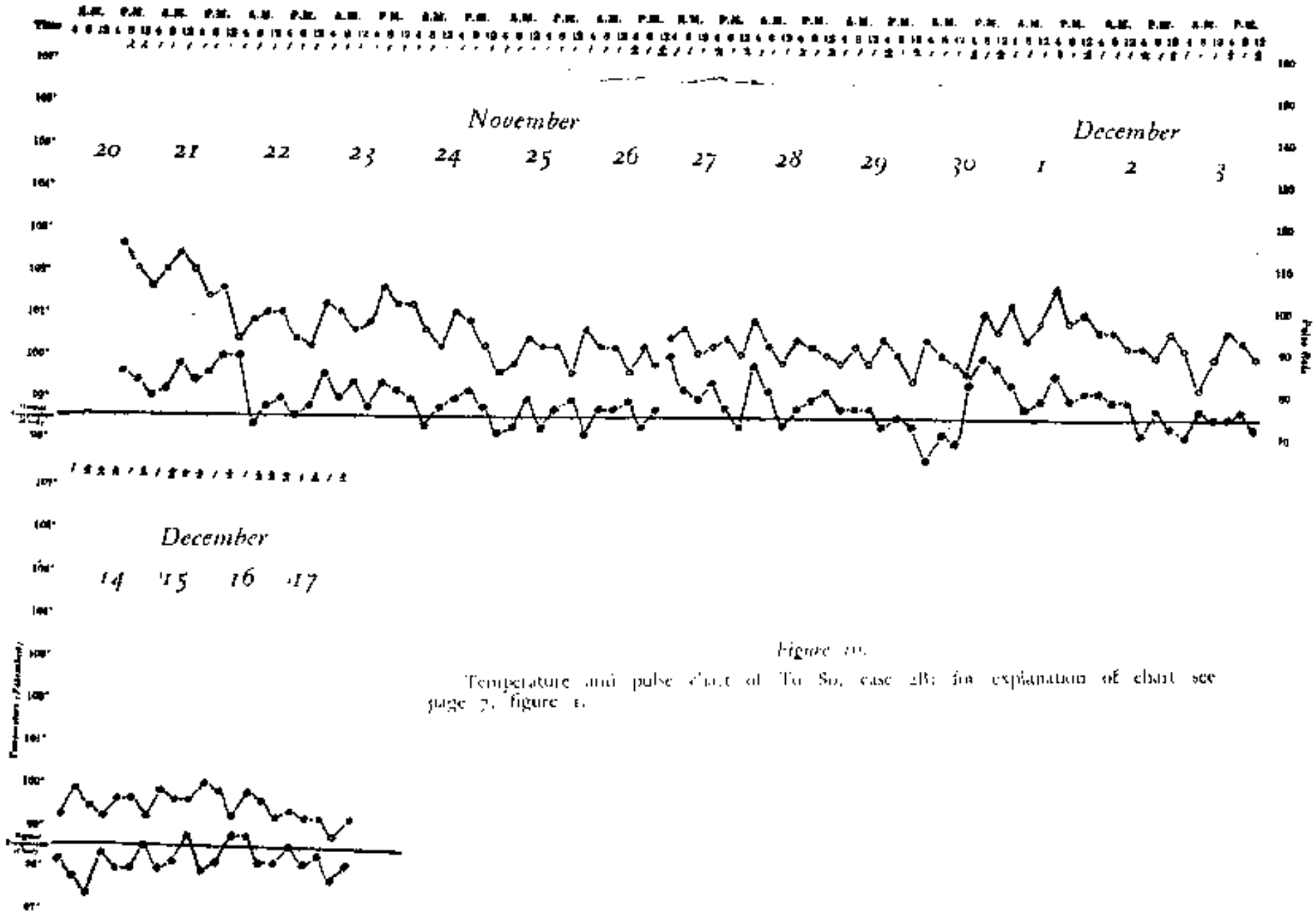


Figure 10.

Temperature and pulse chart of To So, case 2B; for explanation of chart see page 7, figure 1.

On the 23rd the child was drowsy and had to be roused to answer questions. She was rational but disliked being disturbed, and her face and nails were slightly cyanosed. The cerebrospinal fluid was still turbid, but flowed readily and 35 c.c. were drained. As she was not taking fluids well by mouth half a pint of 5% glucose saline was ordered per rectum and a further 5 c.c. of 2½% solution of streptocide were given intramuscularly. She vomited in her afternoon and brought up one living ascaris.

On the 24th she was still drowsy and slightly cyanosed. Her face was also a little bloated but she was more cheerful and was taking fluids well by mouth. Head retraction was still obvious but the cerebrospinal fluid was by now definitely clearing.

On the 25th the cyanosis had disappeared although the child was still drowsy and complained of nausea. She vomited once again but was afebrile. Retraction was lessening and the cerebrospinal fluid was rapidly clearing.

On the 26th she could sit up by herself, although generalised rigidity was marked and there was still some head retraction. The cerebrospinal fluid was now clear and pale yellow in colour, and the 24 hourly dose of streptocide was therefore reduced from 6 to 5 grms. Kernig's sign was still positive.

By the 28th the child was running about and eating a full diet. She had developed a slight lumbar pad but had no oedema elsewhere nor was there any albuminuria. The cerebrospinal fluid was clear but Kernig's sign was still positive on both sides. Temperature "spiked" to 99.8° on the 30th but from that time on the child showed no symptoms.

By the 2nd of December the child had full range of head movements and Kernig's sign was negative. The cerebrospinal fluid was clear and colourless on the 3rd. The central nervous system and fundi were normal.

On the 12th her 24 hourly dose of streptocide was reduced from 5 to 4 grms. and she continued to take that dosage until her discharge on the 17th. Her final lumbar puncture done on the 17th yielded clear colourless fluid which showed no organisms, no increase of cells and no increase of globulin.

Summary, and The child was in hospital for 28 days, during which time she was lumbar punctured fourteen times and was given

Conclusions. 135.5 grms. of streptocide by mouth and 10 c.c. of 2½% solution of streptocide intramuscularly. She showed definite though mild signs of streptocide poisoning during the first week of treatment, but they were not serious enough to demand an immediate reduction in dosage. The cerebrospinal fluid took seven days to become clear. This case illustrates well the apparent latent period which is so often noticed between the institution of treatment with streptocide and the appearance of clinical signs of improvement. The period was about 72 hours in this case, and it may have been prolonged because the child was having too heavy a dose of the drug to begin with. During her last three weeks in hospital the child gained 3 lbs. in weight and showed a great deal more than the normal physiological activity of a child of her age. She was seen again on December 30th a fortnight after her discharge, and she had remained perfectly well since leaving hospital. Examination revealed no abnormality in any system other than slightly exaggerated knee and ankle jerks.

CASE 38.

Tsui Shun. Male. aet. 22. Admitted 28. XI. 38.

History This man was a refugee from Shektung who arrived in Hong Kong after a hazardous and fatiguing journey on foot on November 22nd. On the 24th he had a rigor and developed fever. His head ached violently and he vomited three times. There was no antecedent coryza or sore throat. Diplopia was noticed on the second day of the illness but he had no photophobia or stiffness of neck, nor was he delirious at night.

Condition on admission. The man's attitude was one of generalised flexion. He showed slight head retraction and definite limitation of head movements in all directions. The cervical vertebrae showed "soldering" and there was tenderness on pressure over posterior cervical muscles. Tâche was present, but no petechiae and no herpes were noted. Kernig's and Brudzinski's signs were positive. The fundi and tympana were normal, and there was no cranial nerve abnormality. The left knee jerk was absent, but other reflexes were normal. Throat was normal and the man was conscious, rational and co-operative.

Course and Treatment. Lumbar puncture yielded a highly turbid greenish yellow fluid under a pressure of 300. Meningococci were found in smears. 65 c.c. were drained and no serum was given. 2 grms. of streptocide were given by mouth and repeated four hours later, and after that 1 grm. was given by mouth

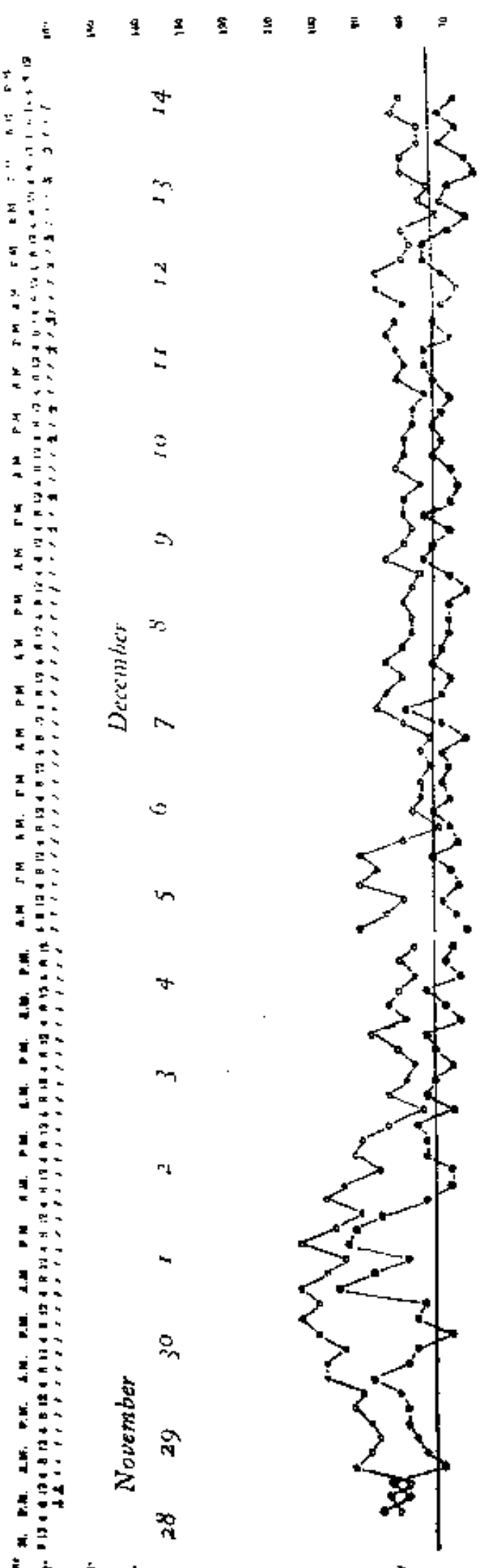


Figure 11

Temperature and pulse chart of Tsai Shun, case 38; for explanation of chart see page 7, figure 1.

4 hourly, day and night. He showed low grade irregular fever during the first four days of treatment, but from that day on his temperature never rose above 99°. Lumbar puncture was performed daily for the first six days. Meningococci were demonstrated in the cerebrospinal fluid up to the 30th of November, but none were found after that date. The cerebrospinal fluid was clear by December 3rd, that is by the sixth day in hospital.

He was given 6 grms. of streptocide every 24 hours during his first ten days in hospital, and the dose was then reduced to 5 grms. every 24 hours for the remainder of his stay. He was very drowsy and tired on November 29th and 30th and had to be roused to answer questions. He developed labial herpes on December 1st. His drowsiness had disappeared by December 3rd, and on the 4th he was able to stand and walk a little, but was still very rigid. From that date on he got up daily and by the 9th was free from symptoms.

He was discharged, at his own request and against advice, on the 17th day after admission. He was then afebrile and symptomless. There was still some rigidity of hamstrings on eliciting Kernig's sign, but the test was painless.

Summary and Conclusions. This patient was in hospital seventeen days and during that time had 91.5 grms. of streptocide by mouth. He was lumbar punctured nine times only.

The attack was a mild one and appeared to yield readily to treatment with streptocide alone. He has been seen three times since his discharge from hospital, on the 17th, the 22nd and the 29th of December. On no occasion did he complain of symptoms, but on the 17th he was found to have a marked papillitis. Both discs were red and their edges were completely obliterated. There was no loss of visual acuity or headache. The man was lumbar punctured on the 22nd as the condition remained unchanged. The cerebrospinal fluid was normal but under slightly raised pressure, 200. On the 29th the papillitis was subsiding and he was still free from symptoms.

CASE 4B.

Wong Kiu. aet. 15. Female. Admitted 4. XII, 38.

History. The mother stated that the child's illness began seven days ago with fear, headache and vomiting. There was no rigor or chill. Nausea persisted throughout the disease. On the second day of the illness the neck became stiff

and head retraction set in. The child was delirious and restless both day and night and had some difficulty in swallowing. She was also incontinent of urine and the bowels were constipated. Photophobia, slight in degree, was noticed from the third day. The ears were not involved and there were no skin rashes or petechiae. Onset was not marked by coryza, sore throat or pharyngitis.

C.O.A. Temperature on admission 99°, pulse 90, respirations 22. The child lay curled up on her side in the gun-hammer attitude. She was partially unconscious and was unable to answer questions or swallow. Head retraction and nuchal tenderness were marked and "soldering" of the cervical vertebrae was demonstrable. Tâche was readily elicited and Kernig's sign was positive on both sides. The pupils were slightly irregular, fractionally unequal and fixed to light. The cranial nerves showed no abnormalities and the knee and ankle jerks were present on both sides. Plantar responses were extensor and the abdominal reflexes were absent. Neither liver nor spleen were palpable. The heart sounds were soft but clear and there were moist sounds at the base of each lung. A few doubtful petechiae were noted about the knees on either side.

Course and Treatment. Lumbar puncture was immediately performed under local anaesthesia. Dry taps, demonstrated by the injection of saline, were obtained in lumbar spaces 1-2, 2-3, and 3-4. Cistern puncture yielded a few c.c. of greyish white turbid fluid under a pressure of 210. Meningococci were demonstrated in smears of the cisternal fluid.

Treatment was begun at once with streptocide and 2 grms. were given by nasal tube and followed up in 4 hours with another 2 grms. After that 1 grm. was given 4 hourly by nasal tube.

The next day the child's condition was unchanged. The lumbar theca still yielded no fluid, and cisternal puncture produced 12 c.c. only. Streptocide was given 4 hourly day and night, but on the third day the child's pulse rose to 140 and she perished a few hours later, never having regained consciousness.

Summary. The child was in hospital three days and during that time was given 17 grms. of streptocide by nasal tube. It was obvious from the dry taps obtained on admission that "block" was already established and that the prognosis was poor. In this type of case streptocide cannot be expected to be of great value, and it is doubtful if any

line of treatment avails much in cases which come in late and "blocked." One cannot feel that the prognosis would have been improved by the exhibition of serum as well as streptocide. Indeed, it would have been difficult to give anything like adequate quantities of serum, at any rate intrathecally.

CASE 58.

Tang Woon Tai. Female. aet. 6. Admitted 9, XII. 38.

History. The father stated that the child was taken ill on December 7th in Kam Tin Refugee Camp. She became feverish but had no rigor or chill. On the 8th she had a convulsion and a rigor, but did not vomit. The fever continued and on the 9th head retraction was noted and the child was taken to a Chinese hospital which sent it on to Kennedy Town. No photophobia and no petechiae were noted during the illness.

Condition on admission. The child had on admission a temperature of 98°, pulse 120 and respiration 26. The attitude was one of generalised flexion. There was slight head retraction and definite tenderness on pressure over the posterior cervical muscles. Cervical "soldering" was demonstrable.

The pupils were approximately equal and were fixed to direct light. There was slight ptosis of the right upper lid. Photophobia was present but the fundi were normal. The other cranial nerves were normal. The knee jerks and abdominal reflexes were absent, but the ankle jerks were present and the plantar responses were flexor.

Kernig's and Brudzinski's signs were positive and the spine was rigid throughout its length. Lips were a little cyanosed but there were no haemorrhages anywhere. The heart and lung sounds were normal.

Course and Treatment. Lumbar puncture was done under local anaesthesia on the 10th. The fluid was excessively turbid and was under a pressure of 200. Queckenstedt's sign was positive. 20 c.c. were drained and meningococci were demonstrated in films of the pus.

The child was given 1 grm. of streptocide 4 hourly. On the following day the child was unable to swallow and was only partially conscious. Fluid feeds and streptocide were given by nasal tube. Lumbar puncture yielded a fluid just as turbid as that obtained on the day of admission. It was under a pressure of only 20. 28 c.c. were drained and Queckenstedt's sign was positive.

On the 12th the child was still only partially conscious, but she was able to swallow. She was very drowsy

and the lips were a little cyanosed. A right corneal ulcer had appeared during the night. Streptocide was reduced to 5 grms. a day. Lumbar puncture yielded a fluid slightly less turbid than on the preceding day. The pressure was less than 10 and although Queckenstedt's sign appeared to be positive, sitting the child up made no difference to the manometer level. The fluid now showed no meningococci. Head retraction and neck tenderness were still marked and Kernig's sign was still strongly positive. On the 13th the child was very drowsy and the face was slate coloured. The lips and nails were only slightly cyanosed. The temperature was subnormal and although the child could still swallow she had not the energy to ask for things. The dosage of streptocide was reduced to 3 grms. daily in doses of 0.5 grm. every 4 hours and some blood was taken and citrated for spectroscopy which failed to reveal the presence of any abnormal derivative of haemoglobin. The cerebrospinal fluid was by now clearing rapidly, but the pressure remained very low. By the 14th block appeared to have become established. Only 10 c.c. of cerebrospinal fluid were obtained on lumbar puncture. Queckenstedt's sign was negative but the fluid was clear. The child's pulse became uncountable at noon and she died the same evening.

Summary and Conclusions. The child was lumbar punctured five times and was given 24 grms. of streptocide by mouth, and 10 c.c. of a 2½% solution of streptocide intramuscularly. It seems clear that she received too much streptocide and died before she had developed enough immunity to tide her over the infection. One cannot but feel that she would probably have responded better to a blend of streptocide and serum, and the case illustrates the dangers inherent in relying on streptocide alone when antisera are available.

CASE 6B.

Chan Chau. Female aet. 46. Admitted 12.XII.38.

History. This woman was admitted partially unconscious and no history was obtainable.

Condition on admission. Her temperature on admission was 102.6°, respiration rate 26, pulse 96. She was exceedingly restless, noisy and delirious and had to be restrained with a jacket. The face was flushed and a little bloated. There was no squint but the pupils were small, slightly irregular and fixed to direct light. There was slight head retraction, slight tenderness on deep pressure over splenius capitis, and definite limitation of head movements. Cervical vertebrae

showed "soldering." Kernig's sign was positive on both sides, but no tâche could be elicited, nor were any petechiae or herpetic vesicles noted.

The tendon and abdominal reflexes were absent, the plantar responses flexor. The heart sounds were imperceptible and both lungs showed signs of advanced pulmonary oedema.

Course and Treatment. Atropine gr. 1/75 was given hypodermically and lumbar puncture was done under local anaesthesia. The pressure was less than 50. The fluid was excessively turbid and 35-40 c.c. were drained. No meningococci were found in smears from the cerebrospinal fluid. Queckenstedt's sign was positive. As the woman was unable to swallow, 2 grms. of streptocide were given at once by nasal tube and this dose was repeated in the same way in 3 hours time. The woman died 4 hours after admission to hospital.

Summary and Conclusions. It was abundantly clear that this woman was moribund on admission, and it was equally clear that any line of treatment would have been ineffectual.

Autopsy. The brain was hyperaemic and soft. The convolutions were flattened, and the superficial cortical vessels were engorged, the cortical veins being surrounded by pus. There was no basal matting or internal hydrocephalus.

Lungs. Both lungs were markedly congested and oedematous. There were no areas of actual consolidation.

Heart. There were numerous subendocardial petechial haemorrhages in the left ventricle.

Kidneys. There were a few pinpoint haemorrhages in the pelvis of the left kidney.

Meningococci were demonstrated in smears of pus taken from the cortex and ventricular fluid. The woman's two daughters, Mak Kin aged 15 and Mak Kin Tai aged 12, were admitted to hospital on the 13th and 16th of December respectively. Both were suffering from meningitis and although meningococci were demonstrated in Mak Kin Tai's fluid only, there can be little doubt that the child admitted on the 13th was also suffering from the same form of meningitis.

CASE 7B.

Sun Choi. Female. aet. 8. Admitted 16.XII.38.

The child was sent in from Fanling Refugee Camp on 16.XII.38, and on admission was unable to speak or swallow and no history was obtainable.

Condition on admission. She lay in the gun-hammer position. The head was slightly retracted, neck was stiff and painful on pressure, and cervical vertebrae were "soldered." Left internal strabismus. Pupils unequal and fixed to light, left sided iritis. Tâche present, but not marked. Kernig's sign present on both sides. The knee and ankle jerks were exaggerated on both sides, the plantar responses were extensor, the abdominal reflexes absent. The child was doubly incontinent. The heart and lung sounds were normal. Temperature on admission 101.4°, pulse 94, respirations 26.

Course and Treatment. Lumbar puncture yielded turbid whitish yellow fluid under very low pressure in which numerous meningococci were detected. Queckenstedt's sign was negative. 25 c.c. of cerebrospinal fluid were drained. Streptocide 1 grm. was given 4 hourly with fluid feeds by nasal tube.

By the morning of the 17th the temperature had risen to 105°. Lumbar puncture yielded 40 c.c. of turbid fluid under a pressure of 90. Queckenstedt's sign was weakly positive. Slight stertor was noted and the child died in the afternoon 22 hours after admission.

Conclusions. At autopsy the brain was hyperaemic and the cortex soft. There were streaks of pus along the course of the intensely engorged cortical veins. There was no hydrocephalus nor was there pus in any of the ventricles. There was a considerable amount of purulent exudate all over the base of the brain and it was most marked round the circle of Willis. The lungs were both markedly oedematous, and the heart muscle was grey in colour and soft.

The child lived only 22 hours in hospital. On admission she showed evidence of block, and had obviously been ill for some days. She received in all 6 grms. of streptocide by nasal tube although it was clear that streptocide alone would have little chance of bringing about recovery, but it is doubtful if the exhibition of A.M.S. would have improved the child's chance.

CASE 8B.

Yeong Choy Kow. Aet. 25. Female. Admitted 23, XII, 38.

History. The patient was picked up unconscious at one of the refugee camps and was immediately transferred to hos-

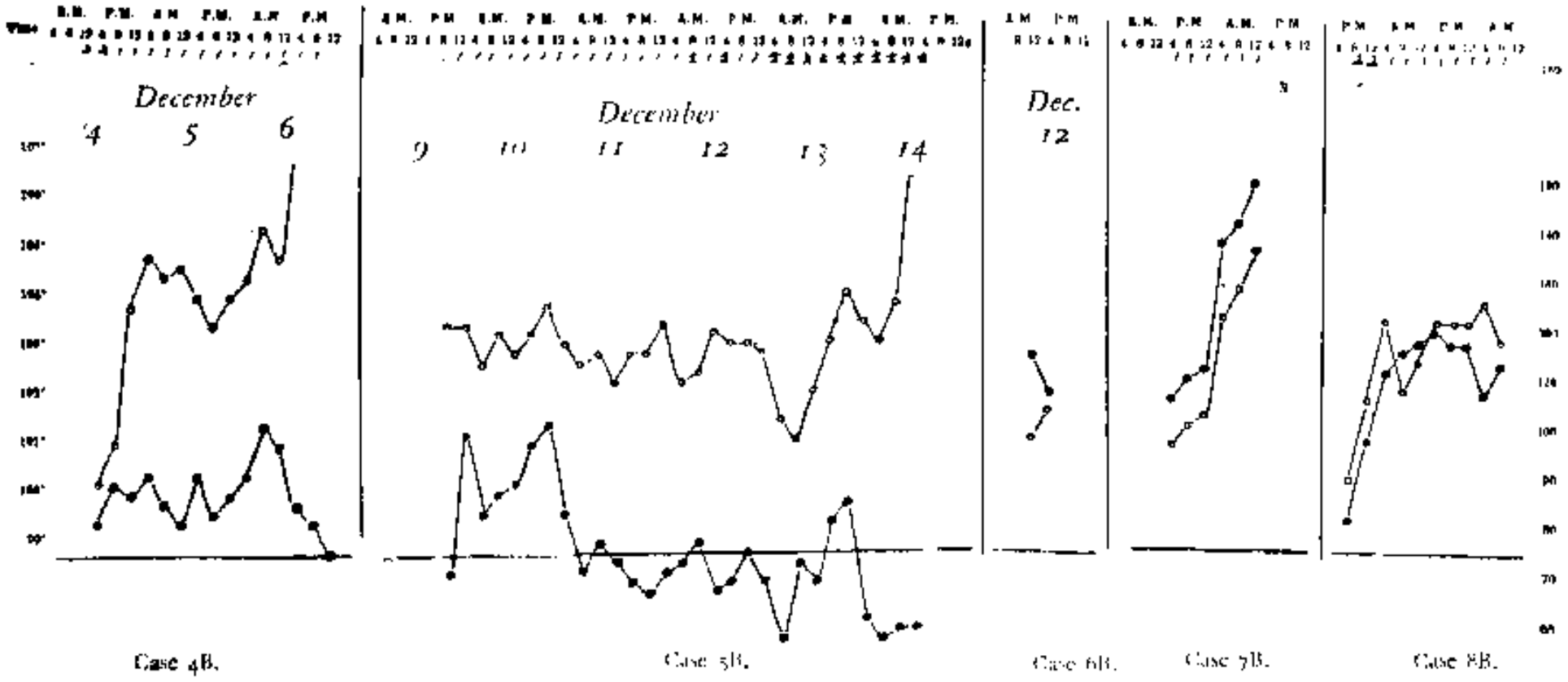


Figure 12

Temperature and pulse charts from left to right of Wong Kin, case 4B; Tang Woon Tai, 5B; Chan Chan, case 6B; Sun Choy, case 7B; and Yeong Choy Kow, case 8B; for explanation of chart see page 7, Figure 1.

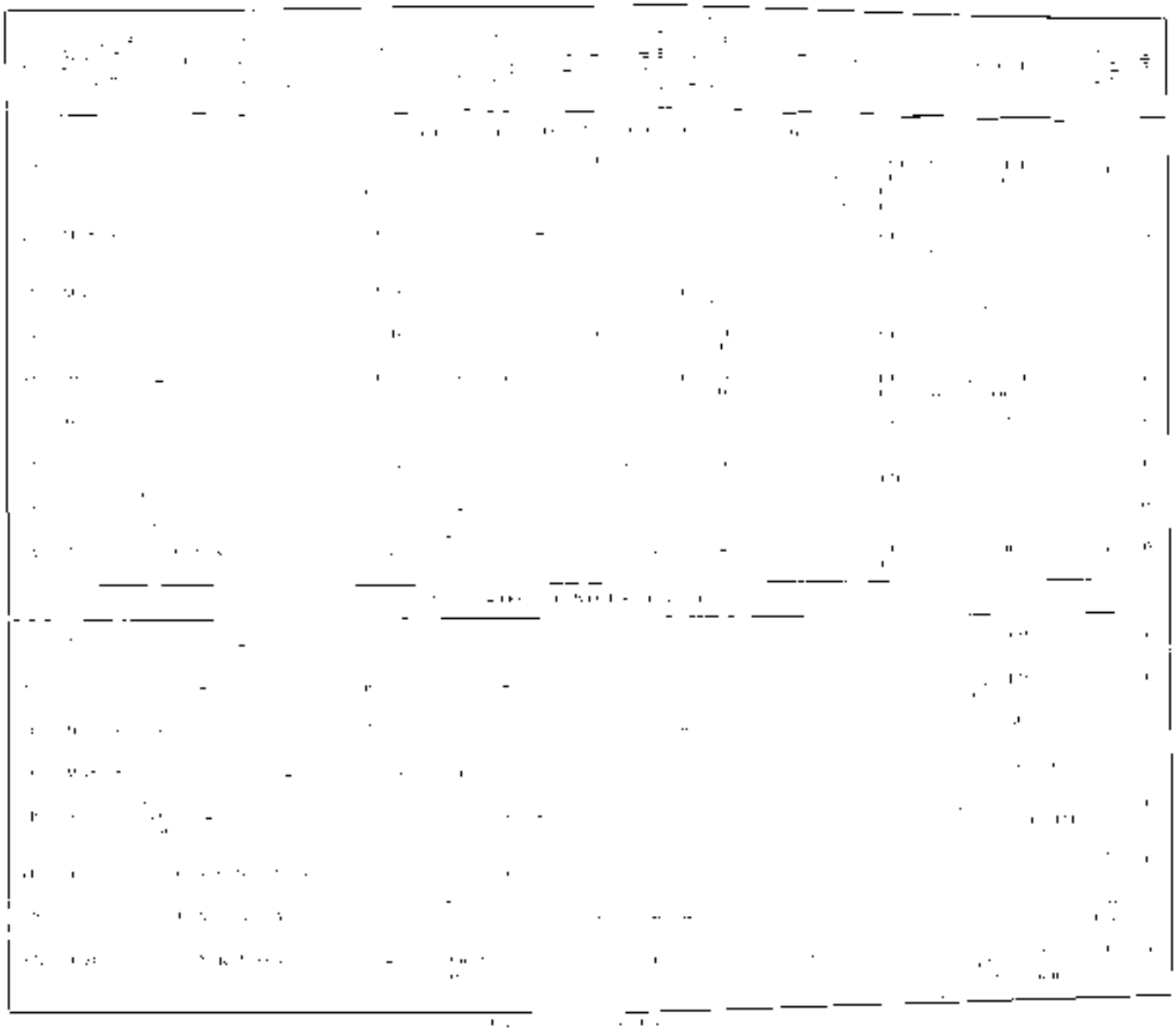
pital. No relatives were available and the history of onset was unobtainable.

Condition on admission. The woman was unconscious and unable to swallow on admission. Her temperature was 99°, pulse 86 and respiration rate 24. There was no head retraction, but nuchal rigidity and tenderness were marked. The left pupil was slightly greater than the right, and both reacted sluggishly to light. A slight left internal strabismus and left sided facial paresis were noted. Both Kernig's and Brudzinski's signs were positive, and the ankle jerks were absent. Plantar responses were flexor. The tongue was coated and the throat contained much frothy mucus. The heart sounds were normal but bronchitic signs were found at both bases. Tâche was elicited with ease.

Course and Treatment. Lumbar puncture was performed at once and yielded turbid yellow fluid which was not under pressure. 25 c.c. were drained and intracellular meningococci were demonstrated. Queckenstedt's phenomenon was positive, but in view of the pulmonary involvement the prognosis was considered grave. Treatment with streptocide was begun by giving 2 grms. by nasal tube and 10 c.c. of 2½% streptocide intramuscularly. The 2 grms. were repeated 4 hours later, and from then on 1 grm. was given 4 hourly. Next day her condition was deteriorating rapidly. A slight expiratory grunt was noted, the respiration rate was rising and it was abundantly clear that a pneumonic process was beginning to gain the upper hand. Lumbar puncture yielded no fluid and a dry tap was demonstrated by the introduction of three needles. Cistern puncture yielded turbid fluid under great pressure, and 45 c.c. were drained from the cistern after which 5 c.c. of 2½% streptocide solution in 10 c.c. of normal saline were given intracisternally. No meningococci were found in the cisternal fluid. On the 25th it was obvious that a bilateral basal pneumonia was about to end the patient's life. Lumbar puncture yielded fluid under pressure, and 45 c.c. were drained. Streptocide was continued by nasal tube although no hopes were entertained of recovery. The woman was by now doubly incontinent and respiration was becoming very embarrassed. She died at noon.

Summary and Conclusions. It was impossible to find out how long this patient had been ill before admission, but it was evident from her pulmonary condition on admission that her illness had already lasted several days. Experience goes to show that

Table to show mean ranks of the cases in each region and class



cases admitted with established pulmonary complications usually perish and it seems unlikely that serum by any route would have availed aught in this case. The fact that lumbar puncture was dry on the day after her admission also tends to show that the disease had lasted for some days, and makes it probable that on admission she already had a considerable degree of basal "matting."

SUMMARY.

1. Seventeen cases of meningococcal meingitis are described, and their treatment is given in some detail.
2. Eight of these cases were treated with streptocide alone, nine with streptocide and antimeningococcal serum.
3. No selection was made but alternate cases were put on streptocide and streptocide combined with serum.
4. The mortality rate of the series on combined treatment was 33.3%, of the series on streptocide alone 62.5%.
5. Little importance is to be attached to these figures but evidence is adduced to show why and how combined therapy affords a better chance of recovery than treatment with streptocide alone.

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NOTES ON A CASE OF TRANSPOSITION OF VISCERA.

by

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INTRODUCTION AND MATERIAL.

The specimen to be described is derived from the body of a male infant which came to autopsy at the Victoria Mortuary, Hong Kong, on April 11th, 1938.

The child was large and well nourished but cyanosed; its age is sufficiently indicated by the fact that the umbilical cord was not yet separated.

The autopsy had progressed to some considerable extent before the abnormal disposition of the viscera was observed. It is due to this circumstance that some details of anatomical structure have been undetermined. This is not to be regretted, because comparatively seldom are bodies which present this abnormality completely dissected. Transposition of viscera is noted not very infrequently in various journals, and usually no more than the most obvious of the phenomena of transposition are recorded, every effort being directed to the preservation of the specimen entire. In this case, however, we have been able to make fairly complete notes of the arrangement of the abdominal and thoracic viscera, as the whole specimen was unsuitable for preservation.

Pneumonic patches were observed in both lungs and bronchopneumonia was established as the cause of death and perhaps of the cyanosis. The thoracic and abdominal cavities were laid open freely, the body was preserved in weak formalin solution in the Department of Anatomy and detailed examination was deferred until the specimen was hardened.

GENERAL DESCRIPTION.

A general view of the thoracic and abdominal viscera from the front is shown in Figure 1.

In the thorax the heart seems to occupy a median position and each lung seems to have a cardiac notch in which the heart is partly exposed.

The right lung seems to have three lobes and the left two. In anticipation of later description it may be stated that a deep notch in the interior part of the right lung gives a spurious appearance of a middle lobe, and it is the left lung which has three lobes. The apex of the heart is on the right side.

That part of the heart which is visible from the front is ventricle. The interventricular sulcus is not to be seen; the line on the drawing represents a conspicuous anterior ventricular artery. This ventricle is that from which the pulmonary artery arises and for that reason may perhaps be suitably called the pulmonary ventricle.

The drawing shows the abdominal organs in a somewhat diagrammatic fashion. The bulk of the liver lies on the left side and the falciform ligament with the ligamentum teres also lie somewhat to the left. The gall-bladder lies to the left and when the colon and the small intestine have been displaced only a little downwards and to the right, the foramen of Winslow may be seen with its opening directed towards the left.

This finding in itself suggests some abnormal feature in the colon or of its mesentery. The hepatic, or in this case, the left flexure of the colon makes less contact with the liver than would be expected, and seems to be low and rather medially placed.

The stomach is small and contracted. Of the great omentum no more was present than a few fat laden shreds of peritoneum attached to the greater curvature of the stomach near the pyloric and (not represented in Figure I).

The stomach is attached to the liver by a gastro-hepatic ligament and to the right flexure and the medial part of the colon by a ligament which had best, perhaps, be called gastro-colic.

The right flexure of the colon projects well to the front and though attached to the posterior abdominal wall by a mesocolon, is unusually mobile.

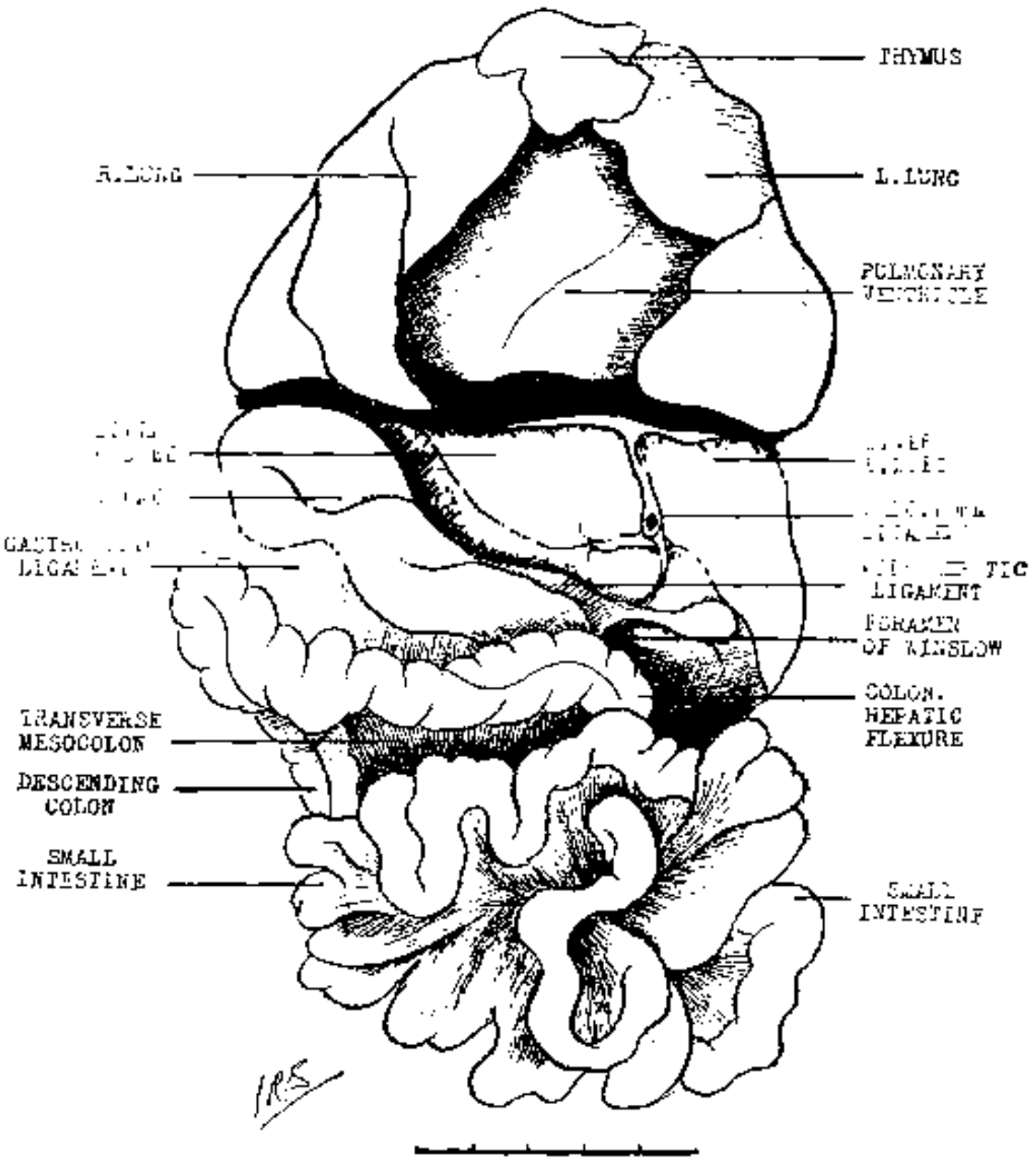


Figure 1

General view of the abdominal and thoracic viscera from the front.

The right lung gives an appearance of the trilobed condition by reason of a very deep anterior fissure in the upper lobe. The inferior lobe of the left lung cannot be seen from the front.

The arrangement of the lungs in front of the heart is nearly symmetrical; each lung has a cardiac notch. The chamber of the heart seen from the front is that from which the pulmonary artery arises.

The oblique line on the front of the heart represents an anterior ventricular artery, not the interventricular sulcus.

The left lobe of the liver is the more bulky and bears the gall bladder. Below the gall bladder can be seen the opening of the foramen of Winslow; the colon does not extend so far to the left as might be expected.

The stomach lies to the right side and is connected by a gastrohepatic ligament to the liver, and by a gastro-colic ligament to the colon. The great omentum is almost entirely lacking except for a few shreds attached to the greater curvature of the stomach near the pyloric end and not represented in this drawing.

The colon lies more to the right than might be expected and the right (splenic) flexure is by much the longer and more mobile, lies at a higher level and is further removed from the mid-line than the left (hepatic) flexure.

The mesentery of the small intestine is short and median in position.

The scale at the bottom is in centimetres.

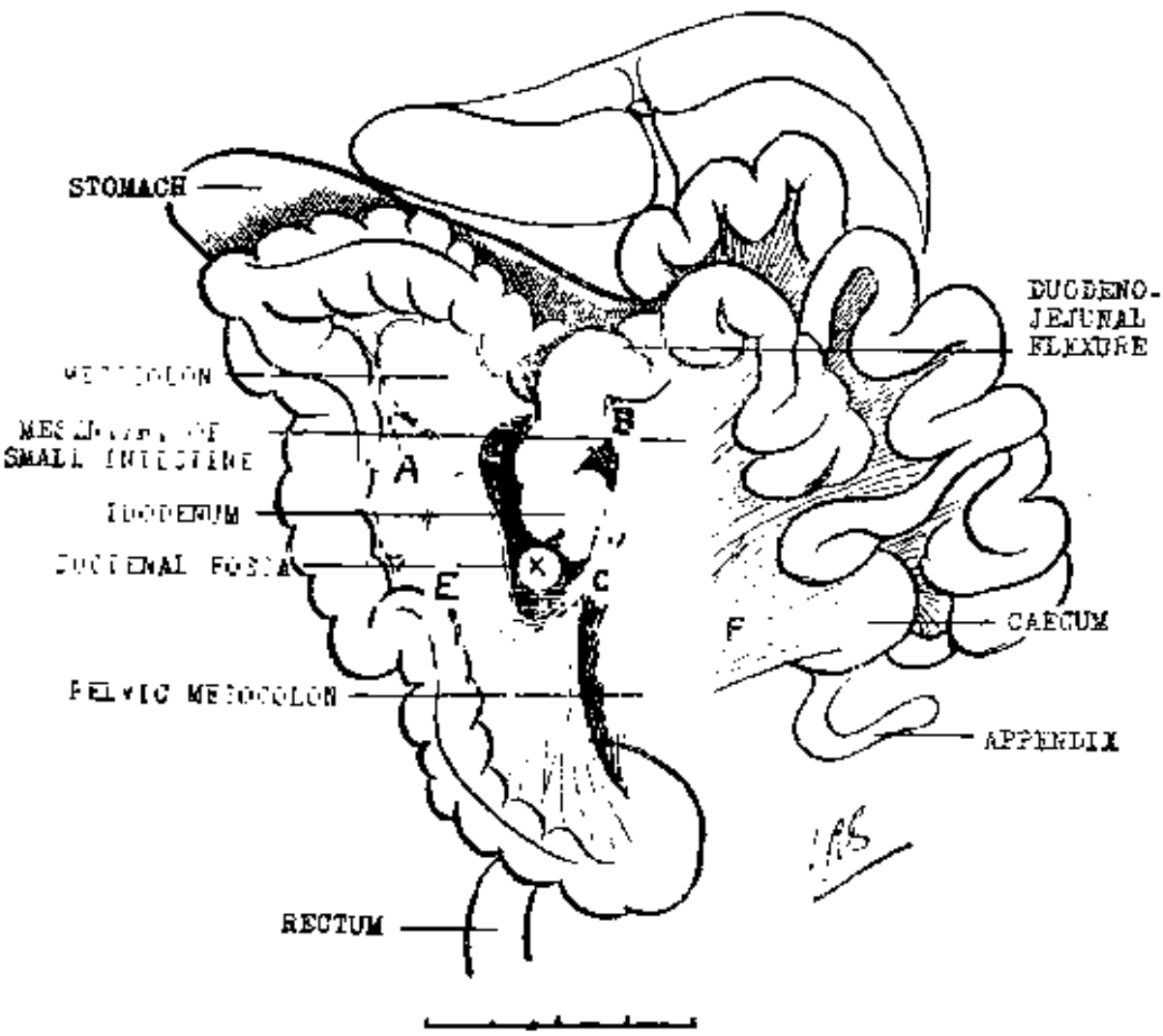


Figure II

In this drawing of the abdominal contents the small intestine has been drawn upwards and to the left over the front of the left colon.

This procedure exposes the right colon, the lower part of the duodenum and the mesentery of the small intestine.

The attachment of the right end of the mesocolon to the posterior abdominal wall is shown again at A. E is a point of attachment of the pelvic mesocolon.

E and C are both on the pelvic mesocolon, which from C to F is fused with the mesentery of the small intestine. This same mesentery C—F is shown in Figure III as the attachment of the left mesocolon.

The structure C—F is composed of the blending of three mesenteries, viz:—the pelvic mesocolon, the mesentery of the small intestine and the left mesocolon. This structure we call the "conjoined mesentery."

B is a point of attachment of the mesentery of the small intestine just below the duodeno-jejunal flexure on the front of the left or descending part of the duodenal loop. This point is very close to the point D shown in Figure III, but above C the mesentery of the small intestine is quite separate from the left mesocolon.

The point X lies in the right duodenal fossa, which contains the right or ascending part of the duodenal loop quite mobile within it. This fossa is separated from the left duodenal fossa by the conjoined mesentery at C.

The blood vessels shown on the right of the duodenal fossa going towards the right colic flexure are branches of the inferior mesenteric artery.

The scale at the bottom represents centimetres.

Following the colon downwards from the right flexure we observe the descending colon, obscured from anterior view by the coils of the small intestine in Figure I, but shown in greater extent in Figure II.

The small intestine is considerably simplified in the drawing; it lies mainly in the lower part of the abdomen except on the left side where it makes an extensive contact with the liver. This contact is certainly more extensive than would be expected, and seems to be a consequence of recession of the left flexure of the colon from the liver.

The small intestine is attached by a medially and vertically placed mesentery of which the details will follow in their place.

THE PERITONEAL ATTACHMENTS OF THE STOMACH.

In the virtual absence of the great omentum the arrangement of the peritoneum round the stomach is more easily examined than usual. Traced to the right and backwards the gastro-colic ligament becomes wider from above downwards, and attaches the fundus of the stomach from above to the right flexure of the colon. This in its turn is attached by the mesocolon to the posterior abdominal wall.

The spleen lies in the expanse of peritoneum which lies between the right and inferior edge of the stomach, i.e. the greater curvature, and the right colon. This peritoneal sheet we have referred to above as the gastro-colic ligament.

That part of the gastro-colic ligament that lies in front of the spleen and between it and the stomach is obviously the gastro-splenic ligament, and that part which lies behind the spleen corresponds to the lieno-renal ligament of ordinary description. The lieno-renal ligament however should extend from the spleen to the front of the kidney and contain part of the body and the tail of the pancreas; but reference to Figure IV will show that these conditions do not apply. Therefore, the whole sheet of peritoneum which extends from the greater curvature of the stomach to the dorsal abdominal wall would better be called the dorsal mesogastrium. The continuity of this dorsal mesogastrium above and below the spleen and above and below the pancreas is normal but is more easily appreciated than is usual on account of the absence of the great omentum.

An unusual mobility was noted in the fundus of the stomach, the spleen and the right colon.

The *omental bursa* or the lesser peritoneal sac was examined very easily by passing a probe to the right through the widely patent foramen of Winslow. This cavity lies behind the gastro-hepatic ligament and the gastro-colic ligament, and above the mesocolon, all of which structures widen towards the right and narrow towards the left in the region of the pylorus. The cavity is bounded on the right by the gastro-splenic ligament, the spleen and the dorsal mesogastrium.

THE CONJOINED MESENTERY.

The arrangement of the several mesenteries was found to be highly unusual and somewhat complicated. Certain features have already received notice. In general it may be stated that the left or ascending colon, the small intestine and the pelvic colon have their separate mesenteries, whose attachments cross the abdominal cavity at different levels and inclinations but fuse below the duodenum and the left kidney.

Reference to Figure II will show the reader that the mesentery of the small intestine extends from B to C in front of the descending horn of the duodenum and at C makes a connection with the mesentery E-C-F. E-C seems to be part of the pelvic mesocolon and C-F a continuation of the mesentery of the small intestine towards the ileo-caecal region.

Figure III. shows that when the intestines both small and large are pressed down and to the right, the large intestine has atrachments somewhat similar to those described above.

The mesocolon ceases to be transverse at D: thence it is attached along D-C to the front of the duodenum and also joins a mesentery which lies nearly transversely and also extends to the ileo-caecal region on the left side.

The points C & F on Figure III. are the same as those so distinguished in Figure II. Between C and F the two mesenteries are blended and form what we call a "conjoined mesentery."

The conjoined mesentery traced to the left gives place to the meso-caecum F-G, and traced to the right makes a union with the pelvic mesocolon.

It must be understood that the mesentery D-C shown in Figure III. is quite separate from B-C shown in Figure II, though the two lie very close together.

The arrangement is shown in more schematic fashion in Figure IV. and an analysis is made of the arrangement in Figure VI.

For the present it may be stated that low on the left side and lying transversely there is a conjoined mesentery which is made up by contributions from each of the following:—

(*a*) The mesentery of the large intestine.

(*b*) The mesentery of the small intestine.

(*c*) The pelvic mesocolon.

THE PERITONEAL ATTACHMENTS OF THE COLON.

The right colic flexure is prominent and very mobile; its peritoneal attachments have already been described.

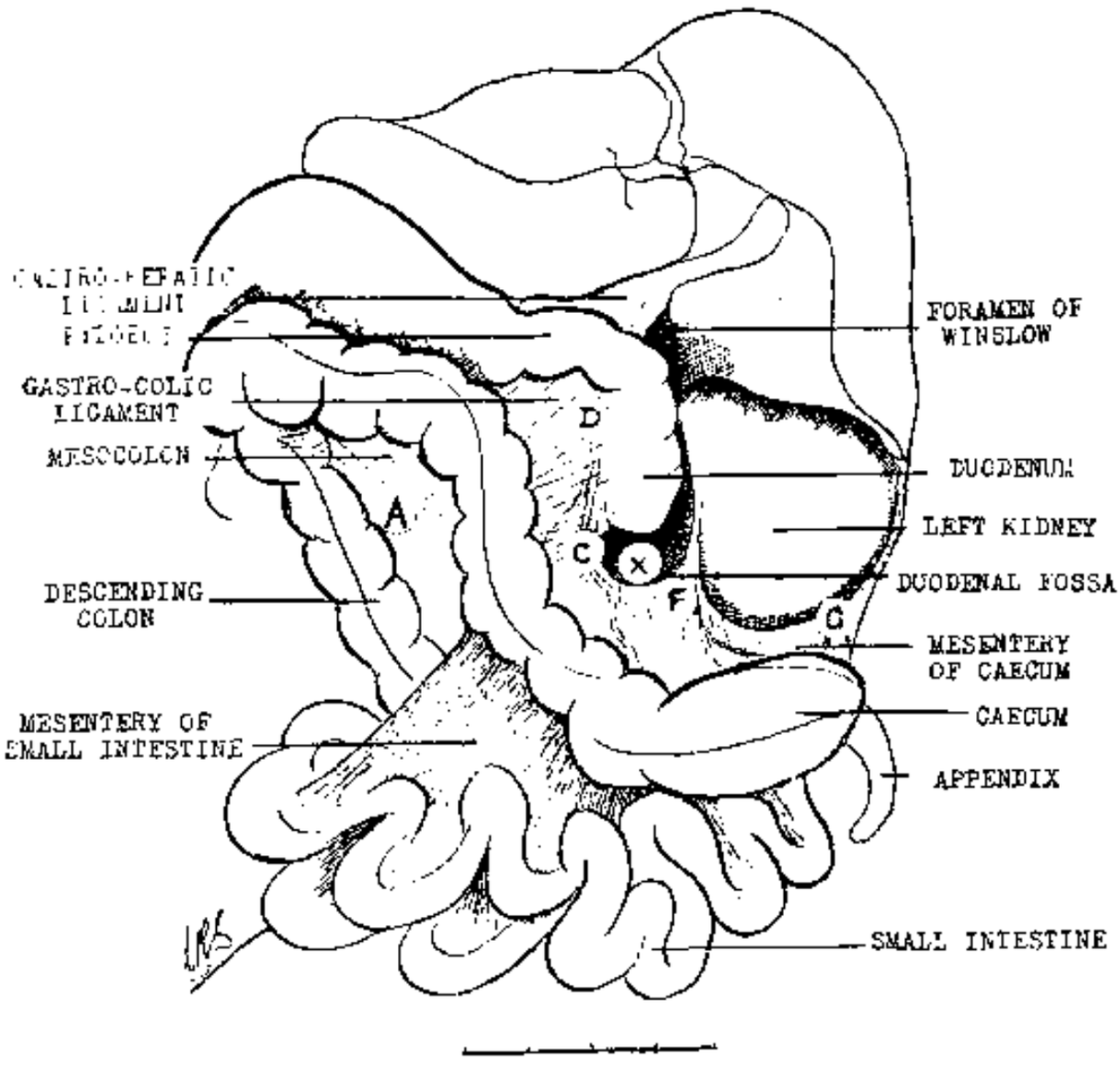


Figure III

This drawing gives a view of the abdominal contents after the left colon and the small intestine with its mesentery have been drawn downwards and to the right.

The right colon is seen attached to the stomach by a gastro-colic ligament (see Figure I) and to the posterior abdominal wall at the point marked "A" by a mesocolon.

The left part of the mesocolon has an attachment to the front of the duodenum, D—C, which is almost vertical before it turns to the left transversely below the left kidney.

The points C, F and G are all on the posterior abdominal wall.

The points C and F, as will be shown in the next Figure, correspond to a blending of the mesentery of the small intestine and the pelvic mesocolon i.e. the conjoined mesentery. The point G though continuous with this blended mesentery is on the mesocaecum.

The point X lies in the left duodenal fossa which is virtually made up by the blended mesenteries referred to above. In this fossa the duodenum is partly mobile.

Two strands from the conjoined mesentery at F and F_1 pass to the posterior abdominal wall medial and lateral to the left kidney and form a fossa in which the left kidney is lodged.

Blood vessels shown at D and C are branches of the superior mesenteric artery.

The scale at the bottom represents centimetres.

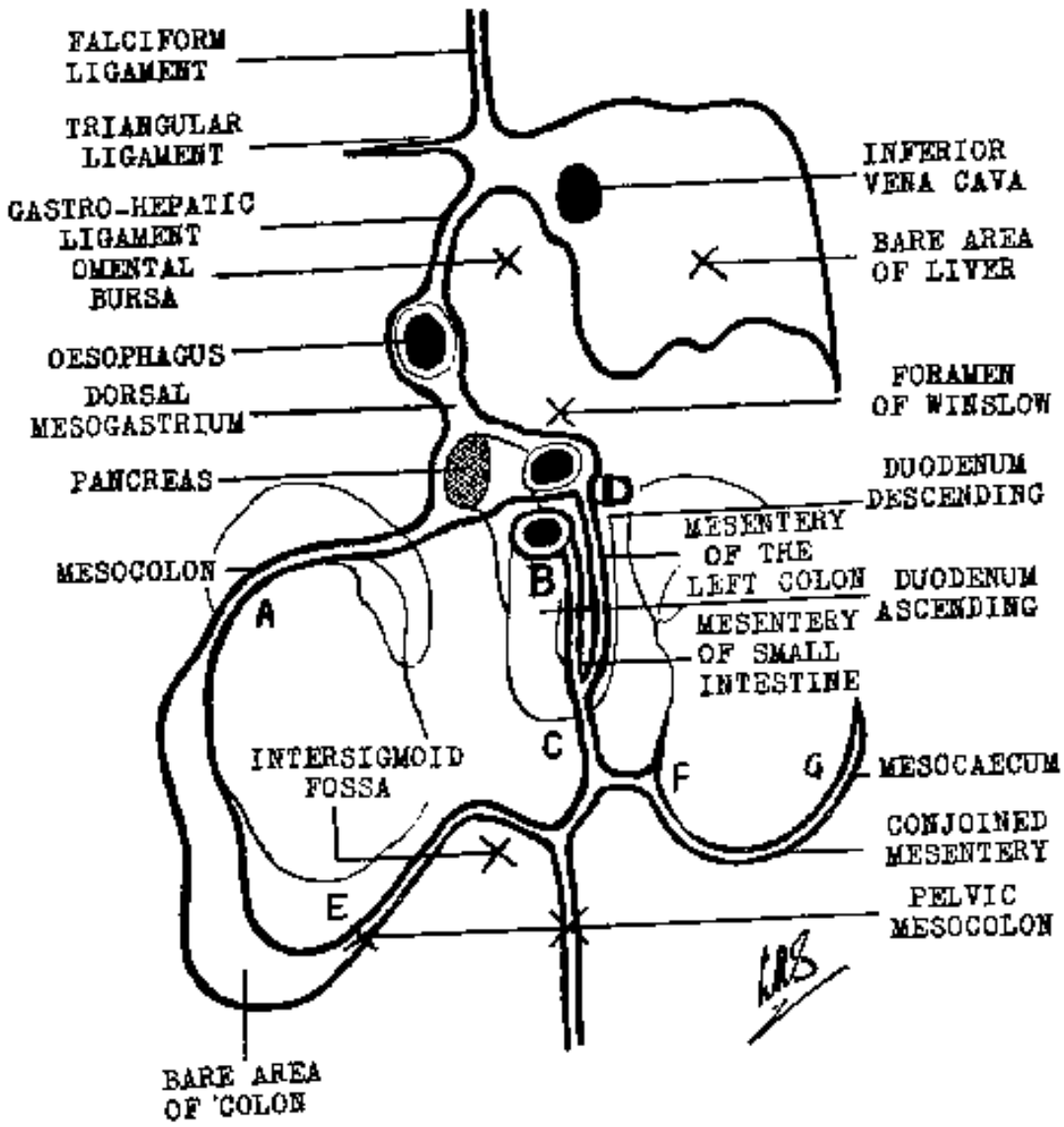


Figure IV

This Figure shows the arrangement of the peritoneum in diagrammatic fashion. It is supposed that the abdominal viscera have been removed by cutting peritoneum where that membrane has attached them to the posterior abdominal wall. The cut edges of peritoneum which result from this process are shown in heavy black lines.

The outlines of the kidneys, supra-renal glands, the duodenum and the pancreas are indicated by lighter lining.

Where the peritoneal coverings of organs diverge from each other on the posterior abdominal wall larger "bare areas" appear, but for the most part cut edges of peritoneum are parallel and separated by a minimal space.

The oesophagus is at a lower level than would be expected, also, the pancreas is cut nearer the middle line than one would expect, and this finding is in harmony with the unusual mobility of both pancreas and spleen.

The right mesocolon is attached at A, at a rather low level. When traced to the left its attachment turns at D abruptly down in the front of the left or descending part of the duodenal loop, lying alongside the mesentery of the small intestine which is attached below B. The mesentery of the large intestine blends with that of the small intestine at C; below C the conjoined mesentery lies vertically, separates the two duodenal fossæ (see Figures II and III), and turns left.

Below this again the pelvic mesocolon fuses with the conjoined mesentery between C and F. G is the mesocaecum, the left extremity of the "Conjoined mesentery."

After the small intestine has been pressed away towards the left, as in Figure II, the attachment of the mesocolon can be seen at A.

The right or descending colon is fixed to the posterior abdominal wall directly, i.e. without the intervention of a mesentery, to the lateral side of the right kidney as far down as E.

The pelvic colon becomes mobile at the point E in Figure II below the lower pole of the right kidney. From this point the mesocolon has a transverse attachment as far as the middle of the body at C. Thence the pelvic mesocolon makes a connection with the conjoined mesentery, C-F. Downwards, the pelvic mesocolon is continued into the pelvis in a tolerably normal manner.

Figure II shows at C a small but prominent peritoneal band which passes from the pelvic mesocolon to the mesentery of the small intestine in front of the left horn of the duodenal loop. This band seems to have a share in the production of a well marked intersigmoid fossa (see Figure IV), and contains a branch of the inferior mesenteric artery.

The right flexure of the colon when traced to the left is found to be attached by a mesentery which takes a transverse and then a descending course. The mesocolon crosses the summit of the duodenum and here at D in Figure III the transverse mesocolon must be considered to end, in correspondence with the pylorus and left extremity of the omental bursa.

From the summit of the duodenum at the point D the attachment of the mesocolon descends almost vertically on the front of the left or descending horn of the duodenal loop. Below the lower pole of the duodenum it has a transverse course C-F below the lower pole of the left kidney and ends in the mesocaecum G. Below the duodenum at C it has become an element in the conjoined mesentery C-F.

There is no definite flexure in the colon between the right flexure and the caecum. The left colon, physiologically one presumes the ascending colon, lies loose in front of the left kidney and makes a contact with the liver. Because the mesentery of the left colon is shorter than the mesentery of the small intestine, the small intestine is enabled to ride up from the right in front of the left colon and make a contact with the liver above and anterior to that of the colon. Figure II shows the mesentery of the small intestine to be the longer, and the two contact areas on the liver are shown in Figure VII. It is because the left part of the mesocolon is short and medially placed that the foramen of Winslow is so easily seen from the front, as in Figure I and Figure III, when the very mobile small intestine is moved only a little downwards from its free contact with the left lobe of the liver.

THE PERITONEAL ATTACHMENTS OF THE SMALL INTESTINE.

Figure II shows the mesentery of the small intestine attached from the summit of the duodenum at B just below the mesocolon to a point below the lower pole of the duodenum at C where it blends with the pelvic meso-colon. This attachment is to the front of the left or descending part of the duodenum and lies immediately to the right of the attachment of the mesentery of the left colon with a gutter between the two mesenteries. The vertical part gives place to a transverse part C-F which is the conjoined mesentery.

The vertical attachment of the mesentery of the small intestine measures about 3.5 cm, but the small intestine is very mobile.

Figure IV shows the arrangements of the mesenteries in a purely diagrammatic fashion.

The Right Duodenal Fossa.

A view of the right duodenal fossa is shown in Figure II. It will be seen that this fossa, which measures about 2 cm. in length and 1 cm. in breadth, lies below the transverse colon and is defined by sharp peritoneal folds of which one is medial and the other right lateral. The small circle marked X in the Figure lies within the fossa.

The medial or left boundary is formed by the mesentery of the small intestine attached to the front of the descending part of the duodenum, marked B-C in Figure II. In particular, the left boundary of the fossa is emphasized by the peritoneal strand which passes upwards from the pelvic mesocolon to the mesentery of the small intestine, and which has already received notice.

The lateral fold is slightly raised and contains a branch of the inferior mesenteric artery which is passing upwards to the supply of the right flexure of the colon.

In the upper border of the fossa lies the inferior mesenteric vein, which is taking a transverse course to join the splenic near the midplane of the body.

In the back of the fossa lies the abdominal aorta, and in the cavity of the fossa lies the ascending part of the loop of the duodenum which has a surprising degree of mobility.

The Left Duodenal Fossa.

Figure III shows the left duodenal fossa which measures about 3 cm. in length and 1.5 cm. in breadth. Its borders do not permit of the same precision of definition as do those of the right fossa, because it is continuous with the furrow between the left kidney and the left horn of the duodenal loop, and extends up to the entrance of the foramen of Winslow.

Medially the fossa is bounded by the mesocolon D-C, which contains branches of the superior mesenteric artery which probably correspond to the ileo-colic artery.

Laterally, it is bounded by a peritoneal band which stretches up from the conjoined mesentery to the medial edge of the left kidney, and below C by the conjoined mesentery C-F.

The inferior crescentic edge of the fossa also contains branches of the superior mesenteric artery passing to the caecum and the ileo-colic junction.

The left part of the duodenum is fairly mobile in this fossa, which is separated from the right fossa below C by the conjoined mesentery, and above C by the mesoduodenum, which is indicated in Figure V.

The peritoneal attachments of the duodenum.

It has been remarked already that the duodenum lacks its normal broad posterior attachment, and has its own mesentery, the meso-duodenum.

The mesoduodenum separates the two duodenal fossae, and at a lower level gives place to the conjoined mesentery. The vertical length of the mesoduodenum is no more than 1.5 cm.

The duodenum consists of no more than a simple "U" loop of gut, and may be said to lie in the basal part of the mesentery of the small gut. It does not contain the head of the pancreas which is not clearly differentiated into head, neck and body. Medially the pancreas presents a blunt end which projects downwards and lies close to the duodenum but is free from it.

The superior mesenteric artery lies between the blunt medial end of the pancreas and the mesoduodenum.

COMMENTS ON THE ARRANGEMENT OF THE PERITONEUM.

Figure IV shows in diagrammatic form the arrangement of the peritoneum in relation to the various parts of the intestinal tract and other abdominal organs.

The drawing is made as if the intestinal tract had been removed by cutting through the peritoneum wherever that membrane fastened the organs to the posterior abdominal wall. The heavy black lines represent cut edges of peritoneum. For the most part these lines lie very close to each other, but in places such as the "bare areas" behind the colon and above the liver these lines are widely separated. The kidneys, suprarenal glands, duodenum and pancreas are shown in fine lining.

The peritoneal connections of the liver are much as would be expected with transposition, with a large "bare area," falciform and triangular ligaments.

It is to be noted that the peritoneal layers which enclose the omental bursa on the right side are attached nearer to the mid-line than would be expected. This finding is reflected in the lengthening of the gastrohepatic ligament, and in the extra mobility of the stomach, spleen and pancreas, and may be interpreted as imperfect fixation of the dorsal mesogastrium to the dorsal abdominal wall.

There is an extraperitoneal area around the medial part of the pancreas which gives place to a mesentery for the colon on either side.

To take the right side first: the mesocolon extends to the right of the kidney at a rather low level, and by separation of its layers below A gives place to a "bare area" behind the descending colon. This, of course, implies the fixation of that part of the colon.

Followed downwards the peritoneal layers close up again as the pelvic colon becomes mobile near E, and form the pelvic mesocolon which extends to the left. Near the mid-line, as we have seen, the pelvic mesocolon on the one hand is continued into the pelvis in a normal manner, and, on the other hand, it passes to the left to effect a fusion with the conjoined mesentery at C. Traced further to the left this conjoined mesentery fuses with the mesocaecum, F-G.

On the left side, below the foramen of Winslow at D, the attachment of the mesocolon descends abruptly on the left of the mesentery of the small intestine, also being attached to the front of the left or descending duodenum and thence to the region of the left kidney, and into the conglomeration of mesenteries, which we have called the "conjoined mesentery," C-F.

Figure V shows essentially the same features with only this difference: the duodenum has been removed and the mesoduodenum is revealed. The arrangement of the letters is the same in both diagrams.

The presence of a mesoduodenum represents the persistence of a structure which ordinarily disappears at a very early stage indeed.

The arrangements of the mesenteries in other respects can perhaps be interpreted in terms of defective rotation of the intestinal loop, and of secondary fusion in places.

Besides the mesoduodenum, the following mesenteries are concerned:

(1) the mesentery of the left colon,

(2) the mesentery of the small intestine,

(3) the pelvic mesocolon.

All of these, we have seen, make a contribution to the conjoined mesentery.

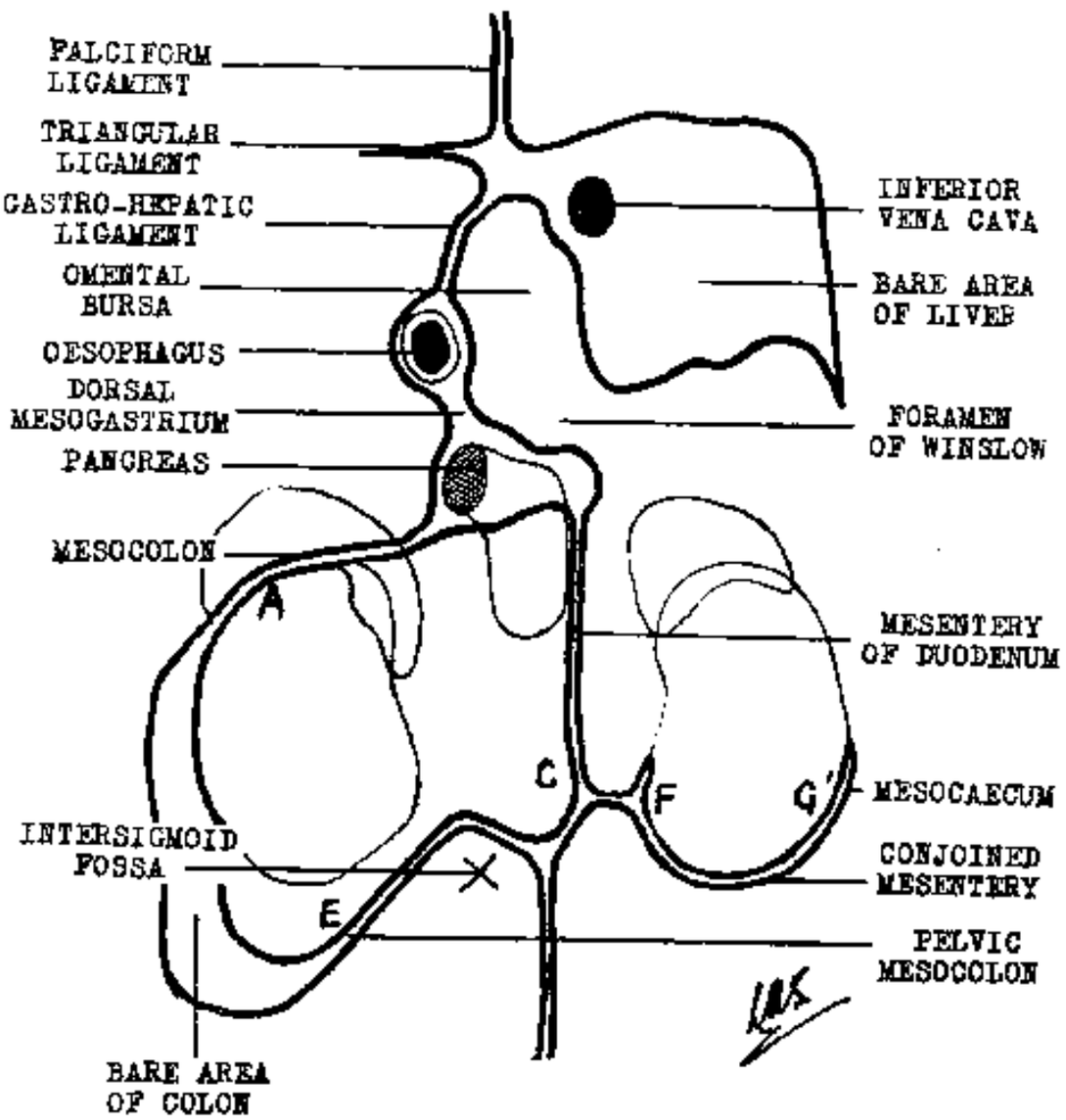


Figure V

This figure closely resembles Figure IV in presenting the same main features, but with this difference. The duodenum has been removed. The duodenum was attached to the posterior abdominal wall by a simple, but short, mesentery from its left or descending part.

The head of the pancreas lies to the right of this mesentery.

In its lower part, below the inferior pole of the duodenum, the mesentery of the duodenum is blended with the conjoined mesentery C-F.

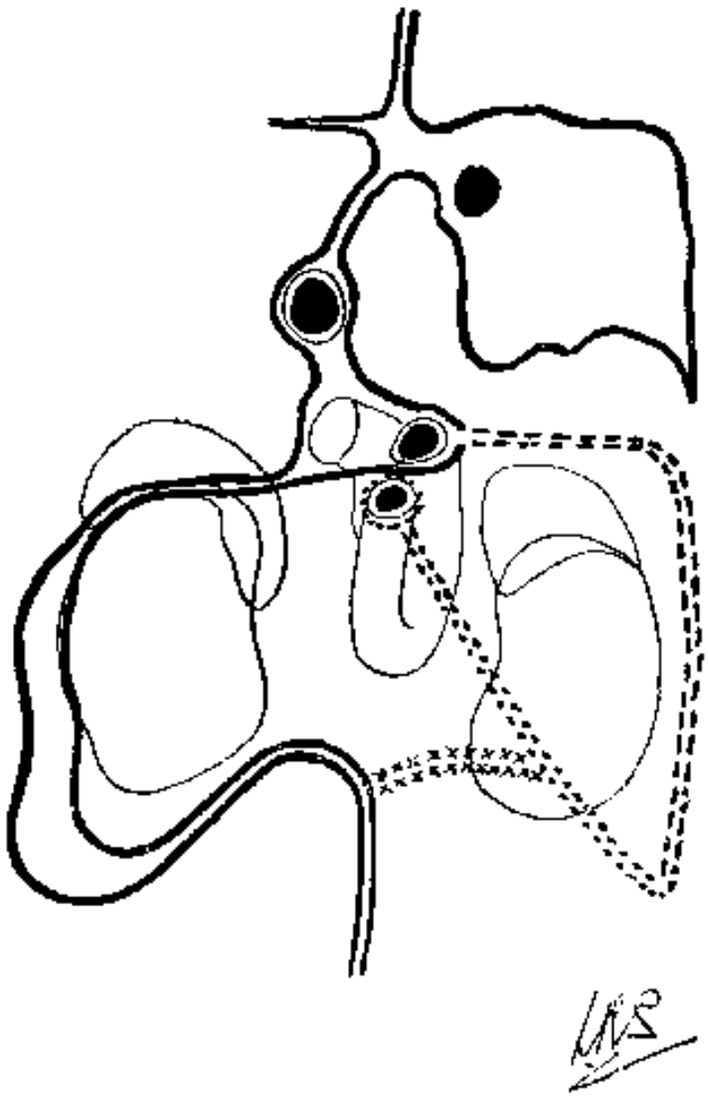


Figure VI

This figure resembles the last two in some respects. It presents the appearances that would be expected after the peritoneum of the various organs had been cut if the organs had been perfectly transposed. The various mesenteries have been distinguished by different methods of lining and an analysis of the complex arrangement of the mesentery shown in Figures IV and V is given.

The mesocolon of the left side and the mesentery of the ascending colon are shown in interrupted line at a much higher level and much further to the left than in the other figures. The mesentery of the ascending colon is usually lost and that organ gains a broad attachment to the posterior abdominal wall. At its lower end this mesocolon becomes continuous with the mesentery of the small intestine, which is distinguished in this figure by dotted lines as it extends from the duodenum near the duodeno-jejunal flexure to the region of the ileo-caecal junction.

The connection between the pelvic mesocolon and the mesentery of the small intestine is shown by small crosses.

It is supposed that the rotation of the intestinal loop was deficient and the left colon came to lie near the middle line in front of the duodenum very close to the mesentery of the small intestine. Below the duodenum these two fused and gave rise to the conjoined mesentery.

The mesentery marked by crosses between the pelvic mesocolon and the mesentery of the small intestine is thought to be the result of fusion of those two structures consequent upon the close proximity thrust upon them by the crowding of mesenteries near the mid-line.

Retention of the mesentery of the caecum and ascending colon, here the left colon, is unusual but not rare. In Figure VI this mesentery is distinguished by interrupted black lining.

It is supposed that in this figure we are viewing the features of the specimen as they might be expected in the presence of complete transposition. The mesocolon has now an attachment extending well out to the flank, and then vertically downwards. The vertical mesentery, i.e. that of the ascending colon is normally lost, but it blends at its lower end with the mesentery of the small intestine.

The dotted mesentery is that of the small intestine, again viewed in the position we might expect if no grosser abnormality than complete transposition were present.

It might be supposed that the course of events had been somewhat as follows.

Rotation of the primitive intestinal loop was imperfect, and these consequences ensued.

The left colon failed to rise so high on the left side as would be expected, and the mesocolon came to lie at a remarkably low level by the duodenum and thence in a transverse line below the left kidney.

Perhaps in consequence of this last event, the mesentery of the small intestine also came to occupy a position more medial in its upper part and more transverse in its lower part than would have been expected.

These two mesenteries must have been in close proximity and below the duodenum and thence to the left have become secondarily fused into the conjoined mesentery.

The extension of the pelvic mesocolon to the left and fusion with the conjoined mesentery may be the result of secondary fusion between the pelvic mesocolon and the mesentery of the small intestine, when this was thrust downwards and medially towards the pelvis. The part of the pelvic mesocolon which is normal is shown in the Figure by heavy continuous black, and the part which is the result of secondary fusion by small crosses.

The reader is invited to compare the plan set out in Figure VI with those in Figures IV and V.

What caused this defective rotation of the intestinal loop is a matter of conjecture. It may, however, be supposed that the mobility of the duodenum and its defective development may have had a large share in the procedure.

The rotation of the primitive intestinal loop takes place round a fixed duodenum. Rotation round an unsteady axis, the mobile duodenum, might well be a factor in the imperfect effect that has followed.

The peritoneal arrangements above the duodenum are also unusual. The fixation of the pancreas is scanty as compared with the normal. The œsophagus occupies a very low position, and the right or lateral wall of the omental bursa lies nearer to the middle than normal. This has already been the subject of comment, and is no doubt related to the unusual mobility of the stomach, right colon and pancreas.

It seems reasonable to suppose that defective development and fixation of the duodenum may lie at the root of these abnormalities.

The pancreas, though of unusual form, is not obviously underdeveloped whereas the duodenum is of very simple form.

THE BLOOD VESSELS OF THE INTESTINE.

The superior and inferior mesenteric arteries are both present, and in spite of transposition of the viscera have a normal relation to each other, i.e. the superior mesenteric artery arises at a higher level from the abdominal aorta than does the inferior mesenteric.

The Superior Mesenteric Artery arises from the abdominal aorta between the medial end of the pancreas and the mesoduodenum, about 6 mm. above the origin of the renal arteries.

This artery supplies the whole of the small intestine, the medial part of the colon, the left colon, the cæcum and the appendix.

The Inferior Mesenteric Artery arises 8 mm. above the bifurcation of the abdominal aorta, i.e. about 2.4 cm. below the origin of the superior mesenteric artery. The artery supplies the right flexure of the colon, the right and the descending colon. The coeliac artery arises 6 mm. above the superior mesenteric just above the upper border of the pancreas.

The *Hepatic Artery* is given off from the coeliac almost at once and enters the gastro-hepatic ligament, in which it lies to the right of the common bile duct and to the right and the front of the portal vein.

The *Splenic Artery* accompanies the splenic vein to the spleen. The vein receives the inferior mesenteric vein as a tributary, and presents no special features.

The arrangement of the renal arteries and veins, of the abdominal aorta and of the inferior vena cava exhibit the features of perfect transposition. The inferior vena cave lies immediately behind the left horn of the duodenal loop and the abdominal aorta on its right.

THE ABDOMINAL ORGANS.

The Pancreas.

The pancreas shows no differentiation into head, neck and body. Medially the pancreas has a blunt end which is bent downwards at

an angle. It is not located in the loop of the duodenum, but lies entirely to the right of the descending horn of the duodenal loop and of mesoduodenum.

The superior mesenteric artery lies between the pancreas and the duodenum, as has already been remarked. The ducts of the pancreas, of which there are two in this specimen, lie behind the peritoneum and in front of the root of the superior mesenteric artery.

The Duodenum.

The duodenum, as we have seen, consists of a simple "U" loop with a descending horn on the left measuring 3.1 cm. in length, and an ascending horn on the right measuring 2.8 cm. The two horns of the loop lie very closely side by side: the greatest width of the organ is no more than 1.4 cm.

The whole duodenum is covered by peritoneum and is fairly mobile.

On the right side of the descending duodenum, at about its middle, the common bile duct and the main pancreatic duct enter. Internal examination shows a well marked papilla, the ampulla of Vater, and 10 mm. above this an accessory duct makes its entry which is also marked by a papilla.

The Spleen.

The spleen measures 4 cm. from the upper to the lower pole and 3.2 cm. transversely at the hilum. The anterior border is heavily incised by a double notch. Figure VII shows at about the level of the hilum a transversely lying tongue-like process which makes an inferior wall to a fossa whose superior wall is formed by the liver. In this fossa lies the fundus of the stomach, and the same tongue partly separates the stomach from the right flexure of the colon. Transversely in the length of this tongue the spleen measures 5.1 cm.

Figure VII shows lateral and medial views of the spleen. On the lateral surface is to be seen an area distinguished by even stippling which makes a contact with a corresponding area of the visceral surface of the liver. Below this is an area which makes contact with the diaphragm and through this with certain of the lower ribs which leave oblique grooves on the organs.

The medial view shows a long transverse hilum containing the splenic artery lying between contact areas for the stomach above, and for the right flexure of the colon below. A patch of peritoneum and part of the left wall of the omental bursa, i.e. dorsal mesogastrium, is adherent just below the hilum. There is no area for contact with the tail of the pancreas.

The Liver.

The organ presents no features of special interest except in the contacts it makes on its inferior or visceral surface. This surface is shown in Figure VII. The spleen makes a contact impression at the pole of the right lobe. To the left of the splenic impression is a groove for the oesophagus and the area for the stomach which is mostly concave except for the omental tubercle just to the right of the gastro hepatic ligament.

The spleen and the oesophagus were in fact almost touching as these liver contact areas suggest. There is a deep portal fissure which contains the usual structures, not shown in detail in this figure, but exhibiting reversal. There is a large pyloric area near the fossa for the gall bladder which may perhaps have been encroached upon by the small intestine. The small intestine makes a large contact in front of that of the colon, as has been remarked before. The area for the colon is transverse and inconspicuous compared with that for contact with the left kidney. A reversed extra-peritoneal area is present which has no special features of interest.

The Oesophagus.

The oesophagus has a rather long intra-abdominal course which measures 1.5 cm. It is attached to the liver by an extension of gastro-hepatic ligament, and to the dorsal wall by the dorsal mesogastrium.

The Kidneys.

The kidneys lie both at very much the same level. The right is the larger. In both the renal vein, renal artery and ureter occupy normal relations to each other in the renal pelvis.

The length, breadth at the hilum and thickness of the two kidneys are as follows:—

Right	4.6 cm.	3.1 cm.	2.5 cm.
Left	4.1 cm.	2.8 cm.	2.0 cm.

THE DIAPHRAGM.

The form of the diaphragm seems normal on first inspection, though its left cupola is higher than the right, as would be expected in the presence of a left sided liver. Asymmetry is very marked when the details of its structure are examined.

The tendinous part of the diaphragm is arranged in three main leaflets, of which the middle transmits the inferior vena cava. The left leaflet is quite twice as large as the right. No abnormality such as hernia was found.

Asymmetry of the diaphragm is most marked in the arrangement of its musculature.

The left crus is estimated to be of over four times the bulk of the right. The left crus lies to the left of the abdominal aorta and in part behind the inferior vena cava. It is crossed transversely in front by the left renal artery and the right renal vein. Also, it lies posterior to the omental bursa, and almost makes a contact with the caudate lobe of the liver.

Most of the musculature of the medial part of the diaphragm is derived from the left crus; the right crus makes only a meagre contribution.

Muscle fibres decussate around the oesophagus in a normal way, but are all traceable to the left crus.

It proved impossible to report on the receptaculum chyli and the origin of the vena azygos.

THE LUNGS.

It has already been noted that the left lung has three lobes and the right only two, in spite of the appearance of extra lobulation shown in Figure I.

From anterior view the lower lobe of the left side is entirely hidden. The cardiac notch is in the left middle lobe.

A posterior view of the lungs is shown in Figure VII.

Separation of the upper and middle lobes on the left side is not quite complete; these lobes are linked between the points X-X by a bridge of lung tissue near the anterior border, and very close to the surface.

Figure VII

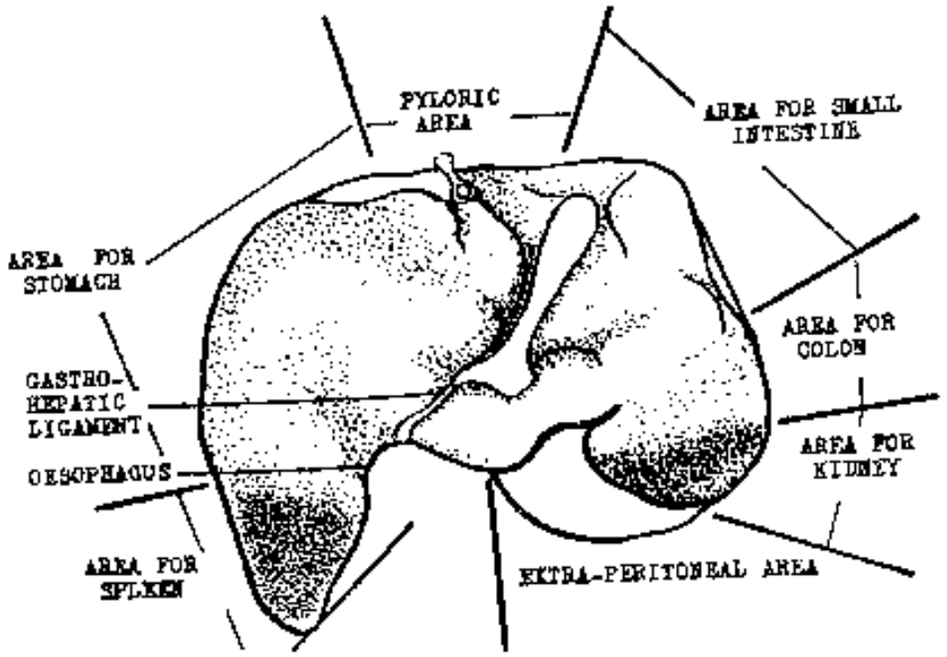
This figure presents drawings of the lungs, the spleen and the liver, each being drawn to the same scale.

The Liver is shown from the visceral aspect and the various contact areas are indicated. The right of the drawing as the reader sees it is the left side of the body. Apart from transposition of the whole organ and consequently of the various contacts the most conspicuous abnormalities are the small and transverse area for the colon and the large area for the small intestine.

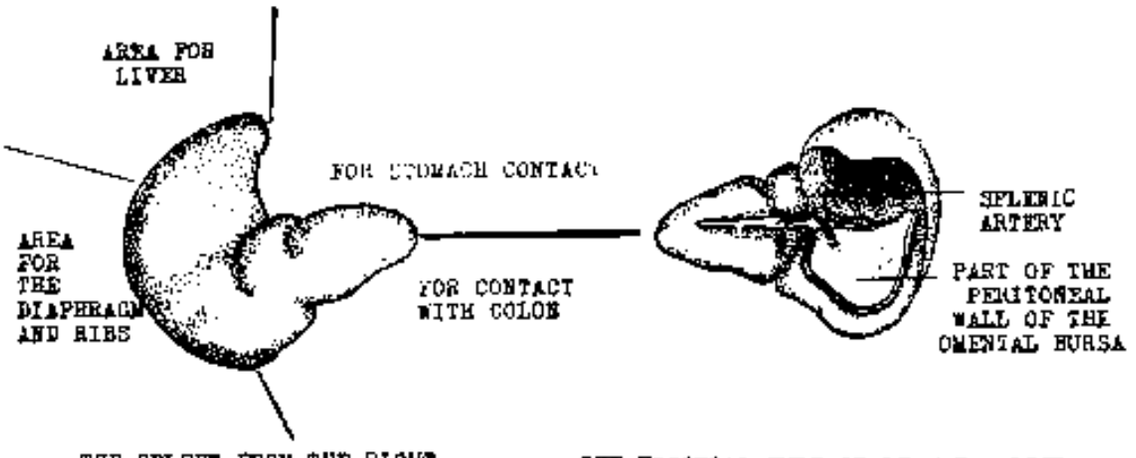
The Spleen is shown in the middle from two points of view. On the reader's left is the right lateral aspect showing the tongue like process which partly separates the fundus of the stomach and the right flexure of the colon. Two contact areas are shown, an upper area for the liver, and a lower area for contact with the diaphragm and the ribs. These two areas are separated by slight ridge.

On the reader's right is the visceral surface of the spleen. The cut gastro splenic ligament is shown which separates the gastric from the colic area. A part of the splenic artery is shown at the hilium. Below the hilium is a peritoneum covered area which corresponds to the left wall of the omental bursa—the dorsal mesogastrium. There are no contact areas for the pancreas or for the kidney.

The *Lungs* are viewed mainly from the posterior aspect. The two lungs still attached to the trachea by the two bronchi have been laid with their mediastinal surfaces on a flat plate. The right lung is to the right as the reader views it; two main lobes can be seen, the upper one with a deep cleft in its anterior edge and spreading to the posterior. The left lung has three lobes, of which only the upper and middle can be seen from the front (see Figure I). The upper and the middle lobes are connected by lung tissues in the anterior part of the fissure between them. Separation of the left lung into three lobes, therefore, is incomplete but more nearly complete at the hinder end of the fissures than at the front.

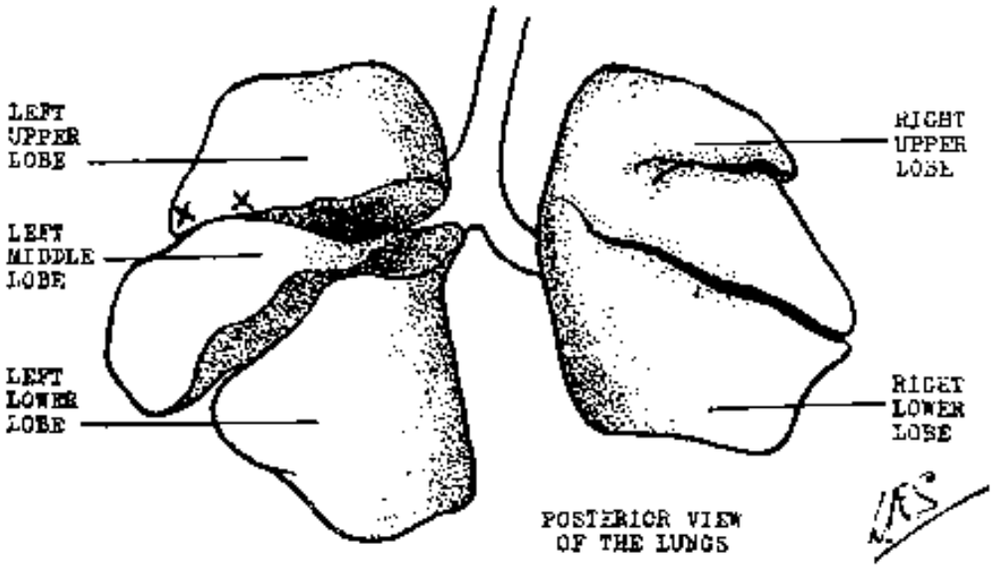


THE VISCERAL SURFACE OF THE LIVER



THE SPLEEN FROM THE RIGHT

THE VISCERAL SURFACE OF THE SPLEEN



POSTERIOR VIEW OF THE LUNGS

The deep fissuring of the right upper lobe extends to the side and back. It has been noted that the left lower lobe was invisible from the front, though this may perhaps be an effect of pressure exerted by an enlarged and abnormal heart, rather than evidence of imperfect development of the left lower lobe.

It seems then that the right lung conveys a distinct suggestion of the trilobed condition and the left lung is trilobed but with imperfect separation of the lobes.

In fact, on neither side has a perfect trilobed or a perfect bilobed lung developed; it is almost as if nature had hesitated in her processes, and for a while were uncertain on which side the trilobed lung were to be developed, and finally, after a tentative experiment, decided on the left side.

THE HEART.

The great blood vessels show complete transposition. The superior vena cava lies before and above the left lung root, where it is joined by the vena azygos major. Its other tributaries are the two innominate veins, which show complete transposition, and certain thymic and thyroid veins.

The aortic arch arises from the middle of the heart and passes over the right lung root. Its branches from left to right are innominate, right common carotid and right subclavian.

The pulmonary artery arises from the summit of the heart in front of the origin of the aorta. From the summit of the pulmonary artery at its bifurcation is a patent ductus arteriosus. This vessel measures only about 2 mm. in length and joins the under part of the arch of the aorta.

The pulmonary artery is the largest of these vessels, the aorta and the ductus arteriosus are about the same size and both are much smaller than the first.

In general form the heart presents an upper surface which is approximately hemispherical; its base is almost horizontal and flat. The organ lies very nearly in the mid-line of the body; it projects to the right in a rounded extremity which is rather less obtuse than the rounded extremity on the left. At its summit are found the stems of

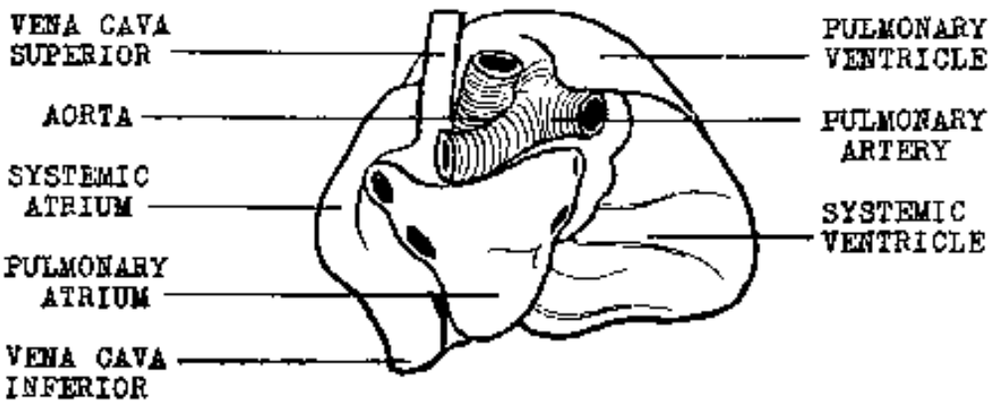
Figure VIII

This Figure presents three outline drawings of the heart to show the arrangement of the different heart chambers and of the great arterial and venous trunks.

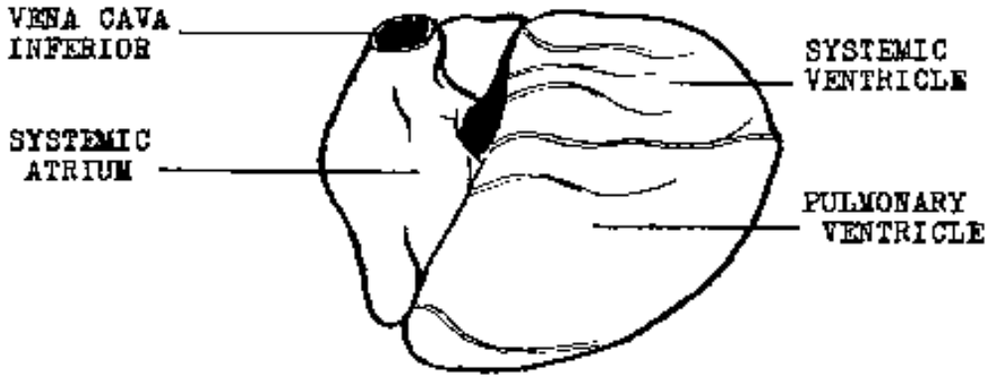
The great proportional size of the pulmonary ventricle is a very conspicuous feature. This chamber of the heart appears in all three views and its bulk is so great that the superior and the inferior views are not very dissimilar.

The pulmonary ventricle occupies almost all the anterior view as is shown in Figure I.

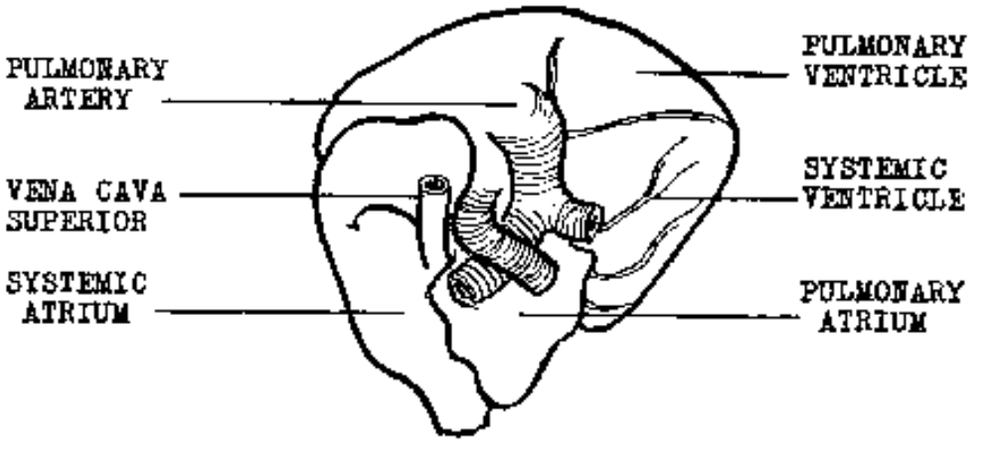
The scale at the bottom represents centimetres.



POSTERIOR VIEW OF HEART



INFERIOR VIEW OF HEART



SUPERIOR VIEW OF HEART

the pulmonary artery, of the aorta and of the superior vena cava. A conspicuous feature of the base is the inferior vena cava, which projects downwards in a funnel form and alone breaks the even flatness of the inferior surface.

The measurements of the heart are as follows:—transverse 62 mm, antero-posterior 48 mm. and vertical 43 mm. The volume is estimated at 65 cc.

Figure VIII presents three views of the heart, posterior, inferior and superior, set out in that order from above downwards. In these three drawings the right of the drawing is the right of the body and vice versa.

In all these drawings we have adopted a nomenclature for the heart chambers which is unusual, but it is hoped neither irrational nor difficult to understand.

That chamber from which the pulmonary artery arises is called the "pulmonary ventricle," and that from which the aorta arises the "systemic ventricle." In the same way, that chamber which collects blood from the body generally and receives the two venae cavae we call the "systemic atrium," and that which collects blood from the lungs by way of the pulmonary veins the "pulmonary atrium."

The superior view shows that the whole of the front of the heart is composed of pulmonary ventricle, as was remarked earlier and shown in Figure I. A proportionately very small systemic ventricle is indicated. The pulmonary artery is decidedly larger than the aorta; its stem arises in the middle of the heart in front of the root of the aorta. The aorta arises from a deep recess in the summit of the heart, between the pulmonary artery on its right and the appendix of the systemic atrium and the superior vena cava on its left.

The inferior vena cava is larger than the superior, and the systemic atrium larger than the pulmonary.

The interventricular groove is sufficiently shown in Figure VIII by the interventricular branch of the right coronary artery. The posterior view of the heart also shown in Figure VIII is not very different from the superior view. It is included in this series chiefly because it gives further emphasis to the disproportion that exists in the relative sizes of the two ventricles. Even in posterior view the pulmonary ventricle occupies almost as much of the norma as does the systemic ventricle.

The inferior view shows the same preponderance of the pulmonary ventricle. It also shows the coronary sinus directed backwards in the auriculo-ventricular groove towards the systemic atrium.

Transposition of the heart chambers and of the great vessels is, in fact, complete, but the arrangement is obscured by the great size of the pulmonary ventricle. The ventricles are anterior and posterior rather than left and right. The one ventricle, the pulmonary, takes

up the whole of the anterior view of the heart and the posterior and the inferior normae are shared by the three other chambers and a good part of the pulmonary ventricle as well.

These appearances and disproportions led us to expect some gross abnormality in addition to the patent ductus arteriosus which was present.

The Dissection of the Heart.

The aorta and the pulmonary arteries were cut along their length in order to expose the aortic and the pulmonary valves. Both the aortic and the pulmonary arteries are furnished with three sinuses and three semilunar cusps.

In the pulmonary artery the sinuses lie thus—two anterior and one posterior. In the aorta they are—one anterior and two posterior.

The Coronary Arteries.

The coronary arteries may be quite properly described as right and left. The left coronary artery arises from the anterior aortic sinus and the right from the right posterior sinus.

These arteries are directed right and left very early, so we use this terminology without any implication of precise homology with the right or the left coronary arteries as they ordinarily occur. The *Left Coronary Artery*, which is the larger of the two, lies in the atrio-ventricular sulcus to the left of the origin of the pulmonary artery. It courses to the left and round to the back, giving branches to the front of the pulmonary ventricle, a large left marginal branch and a branch to the inferior interventricular groove (see Figure VIII inferior view).

The *Right Coronary Artery* sends one branch forward to the root of the pulmonary artery and thence to the superior interventricular groove, as is shown in the superior view in Figure VIII. A posterior branch lies in the atrio-ventricular sulcus, courses to the right backwards and downwards, giving branches to the wall of the systemic ventricle. The *Coronary Sinus* is shown in the inferior view of the heart, lying in the atrio-ventricular sulcus with a course from behind forwards to its termination in the systemic atrium.

Dissection of the Heart.

Flap-like openings were made into the four chambers of the heart in order that the interior might be examined.

The pulmonary atrium seems of rather greater capacity than the systemic, and the pulmonary ventricle is very much larger than the systemic. There is no appreciable difference in thickness between the walls of the two atria, and but little in the walls of the two ventricles. Despite the much greater capacity of the pulmonary ventricle its wall is but little the thicker.

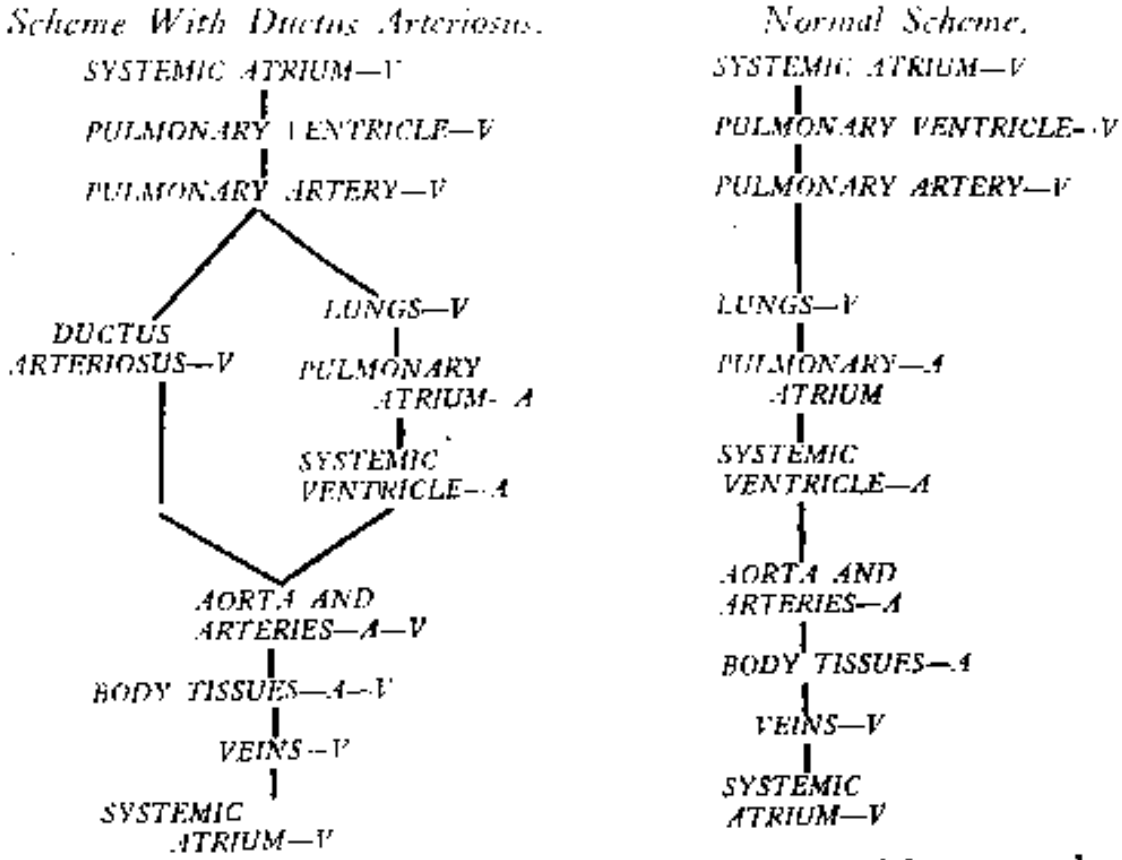
The valve directed into the pulmonary ventricle is tricuspid—its cusps being septal or posterior, superior and inferior. The valve from the pulmonary atrium to the systemic ventricle is bicuspid with anterior and posterior cusps.

No abnormality was found in the septum of the heart, either in the interatrial or in the interventricular part. The conformation of the fossa ovalis was normal, and not even here was there any discoverable inter-atrial communication.

The ductus arteriosus however is a notable abnormality connecting the pulmonary and the systemic systems and in size it is little smaller than the aorta itself.

The circulation of the blood in this individual may be supposed to have taken some such course as is represented in the scheme below, where it is compared with the normal.

We may begin with the systemic atrium receiving "V" blood from the body tissues, and follow its course and its conversion into "A" blood in the lungs. "A" and "V" will serve to briefly describe the quality of the blood in the various chambers and stages in the circulatory scheme.



It seems inevitable that, in view of the widely open ductus arteriosus and the small aorta, the tissues were supplied with blood of mixed quality, A—V. This circumstance might well explain the presence of cyanosis. The need for a very large pulmonary ventricle is also seen; it has to supply the aorta as well as the lungs.

Whether the small size of the systemic ventricle is a consequence of the diversion of blood by the ductus arteriosus, or is due to deficient development of the systemic ventricle must be uncertain, but the second seems the more probable. This ventricle would be undersized in any case, and the condition is regarded by us as representing a primary deficiency in the development of the systemic ventricle partly compensated by a patent ductus arteriosus.

GENERAL COMMENTS.

The interest in a case of transposition of viscera lies less in the mere fact of transposition than in the degree to which this transposition has been effected.

If transposition be perfect in all its details the result is no more than if the descriptions of our textbooks and the arrangements of our common experience were perfectly reversed, as if they were viewed in a mirror.

This effect is unquestionably of great interest and indeed of some practical importance; but ultimately we know of no better reason why, for example, the liver should be developed on the right side and the heart on the left side in the vast majority, than why the opposite should occur in the very small minority.

We know of no reason why the majority of individuals should be right-handed and minority left-handed; but we must suppose that a structural difference in the arrangement of brain cells lies at the root of the phenomenon. Cases of dextrocardia are detected occasionally and phenomena such as the persistence on the opposite side of structures which normally disappear during development are met with in the Dissecting Room; one might instance a left superior vena cava. Each of these examples, left-handedness, dextrocardia, a left superior vena cava is perhaps to be regarded as showing a differing degree of the same essential phenomenon. If such a phrase may be permitted it seems as if there were an occasional whim on the part of nature to produce a reversed picture and some degree of transposition.

To pursue this proposition: the interest in this case seems to lie in the suggestion that nature works less effectively in transposition: defects occur and signs of hesitancy are to be seen at times. We may instance the duodenum, pancreas, mesentery, lungs and the heart.

It is worthy of remark that though the position of the parts of the intestinal tract has been in a great measure reversed or transposed, the blood supply follows the normal pattern.

Any deviation from the normal pattern would imply an abnormality in development of the greatest moment.

The coeliac artery is the artery of the stomach-pancreas region, the superior mesenteric of the small gut and part of the large gut,

the inferior mesenteric of the hinder part of the large gut. These regions, stomach-pancreas, small gut, large gut necessarily lie in that order from the head tailward, and any alteration of that order is scarcely conceivable. But this specimen gives us some reason to think that the mechanism may be less efficient if the primitive intestinal loop rotates clockwise instead of anti-clockwise. These terms are used in their relation to the specimen and not to the observer, in the same way as the other terms used in our descriptions. The persistence of the mesoduodenum and inadequate development of the duodenum has already received comment in connection with incomplete rotation of the intestine.

Further: this faulty rotation of the gut gives us some idea of the date in embryonic history at which the defect occurred. The primitive intestinal loop undergoes much of its development outside the embryo and rotation occurs at the period when it is drawn into the abdominal cavity.

It is possible that an abnormal process of elimination of the primitive umbilical and vitelline veins are circumstances related to the development of a right heart or of a left heart, and perhaps of a left liver in place of right liver. Normally, persistence of a left umbilical

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Figure IX

This figure presents in a highly diagrammatic fashion the changes which it is supposed have taken place in order to bring about transposition of the abdominal viscera.

The middle diagram shows the primitive gut with its dorsal mesentery as if viewed from the left side. It will be noted that much of the intestine projects through the umbilical ring. The headward and tailward loops of the mid gut are represented in position.

The greater growth of the headward loop is shown by its twisting; the tailward part of the loop is distinguished by the presence of the diverticulum of the caecum, which evidently indicates the position of the colon. The blood supply shown in the coeliac artery for the stomach-pancreas region, in the superior mesenteric artery for the mid-gut.

The thick line with arrow marks is intended to show the direction of rotation of the intestine.

Return to the abdomen of the headward loop of the mid-gut to the left of the dorsal mesentery initiates a movement of the whole intestine in a direction which is clockwise relative to the embryo.

The result is shown diagrammatically in the figure on the reader's right.

In this figure the caecum lies on the left and also the termination of the mesentery. Mobile mesenteries are shown in single lining. Where mesenteries have made secondary attachments to the posterior abdominal wall they are indicated by cross lining. Thus, the original mesenteries of both the right and the left vertical parts of the colon are fixed and those parts of the gut are immobilized.

The figure on the reader's left represents the more usual arrangement of the intestine arising from a rotation which is counterclockwise relative to the embryo. Again: in this figure single lining represents mobile mesentery and cross lining mesentery fixed to the posterior abdominal wall.

Rotation Clockwise

Rotation Anti Clockwise

vein is associated with the development of a ductus venosus guiding the bulk of placental blood to the right horn of the sinus venosus. The development of a right systemic atrium and a left heart then necessarily follows. Presumably the opposite occurs in the event of a persistence of the right umbilical vein and the obliteration of the left.

It is conceivable that extra-embryonic circumstances of posture, pressure or placental formation might be deciding factors.

The arrangement of the peritoneum has been fully described and we have furnished an analysis and an explanation of the findings. In brief, they seem to be the effects of imperfect transposition, of clockwise rotation of the primitive gut, and of fusion in certain places.

Any explanation of why rotation of the gut should be clockwise instead of anticlockwise is mainly speculative.

Frazer (1931) gives an interesting account of the normal development of the intestinal tract and of the arrangement of its parts.

It seems clear that the mid-gut develops in an extra-abdominal umbilical sac. The cephalic part of the primitive gut loop: from the duodenum to the vitelline stalk (i.e. diverticulum of Meckel), exceeds in its growth the caudal part of the gut loop, from the vitelline stalk to the cloaca.

The caudal part of the gut loop is attached by a median lying septum or mesentery which is taut as compared with the loose convoluted mesentery of the cephalic part of the loop.

Return of the gut from the umbilical sac is brought about by extra-embryonic pressure, room having been made for the extra-abdominal gut by growth of the liver and expansion of the abdominal parietes.

The gut has to return by stages because the neck of the extra-abdominal sac, i.e., the umbilicus, is constricted.

The derivative of the cephalic part of the gut loop, i.e. the small intestine, returns to the abdominal cavity first. The small intestine returns on one side or the other of the dorsal mesentery of the caudal part of the loop. This is to say the small gut returns on one side or the other of the mesocolon. Normally the small gut returns on the right of the mesocolon and is pressed down to the left against the mesocolon and the colon, which when it enters the abdomen in its turn is pushed on a course of rotation around the coils of the small intestine. A given point such as the caecum occupies successive positions as follows—inferior, left superior and right, moving therefore in a direction which is anticlockwise relative to the embryo. The direction of rotation of the gut as a whole moves in correspondence.

The circumstances which determine that the small gut returns on the right of the mesocolon and therefore initiates an anticlockwise rotation of the large gut are a matter for speculation.

Frazer (1931) attaches great importance to the fixation of the duodenal loop on the right and the bulking of the pancreatic growth on the left in the dorsal mesogastrium. These events seem to be due to primary rotation of the stomach consequent upon unequal growth of the two sides of that organ. Frazer finds that asymmetry of the liver arises at a later date.

Presumably the reverse events occur in transposition of viscera. Growth of the right side of the stomach exceeds that of the left; the pancreatic mass is fixed on the right side and the duodenal loop is developed on the left. The small intestine returns to the belly cavity on the left of the mesocolon and initiates rotation, which is clockwise.

Two explanations of transposition then appear which may indeed be mutually complementary. The primary developmental abnormality is the excess growth of the stomach rudiment on the right rather than on the left side and other events follow. Alternatively the primary abnormality lies in obliteration of the left instead of the right umbilical vein, which may be an extra-embryonic circumstance. It may be remarked in this context that there are few matters on which we are less informed than on the variations, abnormalities and the pathological changes to which the placenta is subject.

A recently published account of a case of defective rotation of the gut by Carter (1939) is associated with a persistent mesoduodenum. It would be interesting to know how frequently a mobile duodenum is an accompaniment of transposition of viscera or of defective rotation of the gut.

SUMMARY.

The case described presents the features of transposition of viscera with certain special features. Both the lungs show some subdivision into three lobes, the left being more complete than the right.

The heart has an imperfectly developed systemic ventricle and the circulatory arrangements are compensated by a patent ductus arteriosus.

The omental bursa is imperfectly developed in the presence of undue mobility in the pancreas and in the dorsal mesogastrium.

Rotation of the intestine, though clockwise relative to the whole specimen, is incomplete. The left, or ascending, colon retains its mesentery, is unduly mobile and is medially placed.

A complex arrangement of the mesenteries arises from the failure of the ascending colon to find its usual posterior attachment and from fusion between the mesocolon, the mesentery of the small intestine and the pelvic mesocolon.

The duodenum has retained its mesentery, is unusually mobile and is of very simple form.

The pancreas is of simple form and unusually mobile, but has retained two ducts.

ACKNOWLEDGEMENTS.

We make our grateful acknowledgements to Dr. R. S. Begbie, who while acting as Medical Officer in Charge of the Victoria Mortuary Hong Kong, observed the great interest of the abnormality described and gave us facilities for making the observations which we have set out above.

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FRAZER, J. E. ..... 1931) *Manual of Embryology* p. 399.

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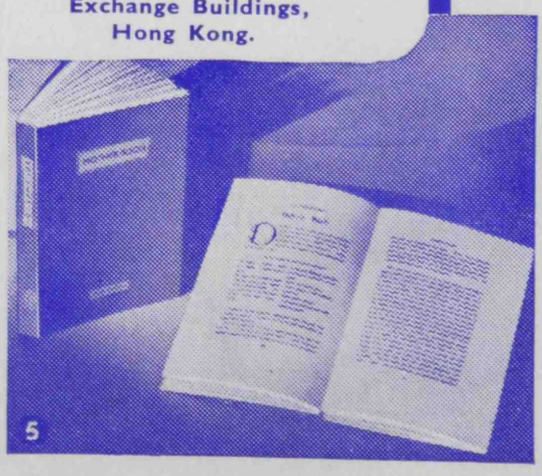

A CASE OF FRACTURE OF THE NECK OF THE RIGHT FEMUR WITH ANTERIOR DISLOCATION OF THE HEAD (ALSO FRACTURE OF THE SHAFT OF THE LEFT FEMUR) ILLUSTRATING THE IMPORTANCE OF THE RETINACULA.

by

Kenelm H. Digby and Lien Tsoong Kya.

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The patient L—C—(205/38) a Chinese billiard marker, 49 years of age had had a severe fall on his left hip, the right lower limb being caught by a door. This occurred on May 31st, 1938. An attempt was made to reduce the dislocation under anaesthesia on the same day and subsequently the patient was admitted to the University Surgical Clinic. X-ray and clinical examination showed the condition depicted in the figures 1 and 3.

The patient was placed in knee-flexion traction splints on the special fracture femur bed used in this Clinic (Digby 1926) with traction applied to both legs, and on the evening of the 1st June the right hip joint was opened through a Petersen incision. The head of the femur was not in the acetabulum and the combined heads of origin of the rectus femoris muscle and the ilio-femoral ligament had to be divided before the head could be located and spooned back into the acetabulum. A good position was obtained and the fragments fixed with a Smith Petersen fluted nail. Strict-skin-segregation and no-hand-touch techniques were followed.

It was observed that the neck of the femur with its periosteum was completely divided and the ligamentum teres was seen to be severed close to the head. A band of *retinacular* fibres still connected the head and neck though the bony parts were separated by a full inch.

It seems likely that in fractures of the neck of the femur (so-called intracapsular fracture of the neck) the nourishment of the head of the femur is chiefly carried on by the vessels in the retinacula. Very little blood is carried by the ligamentum teres, and in this case the ligamentum teres was torn across. The retinacula may be described as the reflected fibres of the capsule of the hip joint reflected longitudinally upwards, chiefly along the inferior aspect of the neck of the femur, and carrying large vessels to the red marrow and bone of the head. Figures 5, 6, 7, and 8.

Probably the synovial membrane and capsular ligament were originally attached around the head, but with the upright position of plantigrade man these became secondarily attached further away along the neck, except where they persist as the retinacula. Some superior retinacula exist and a few other strands on the front of the neck but

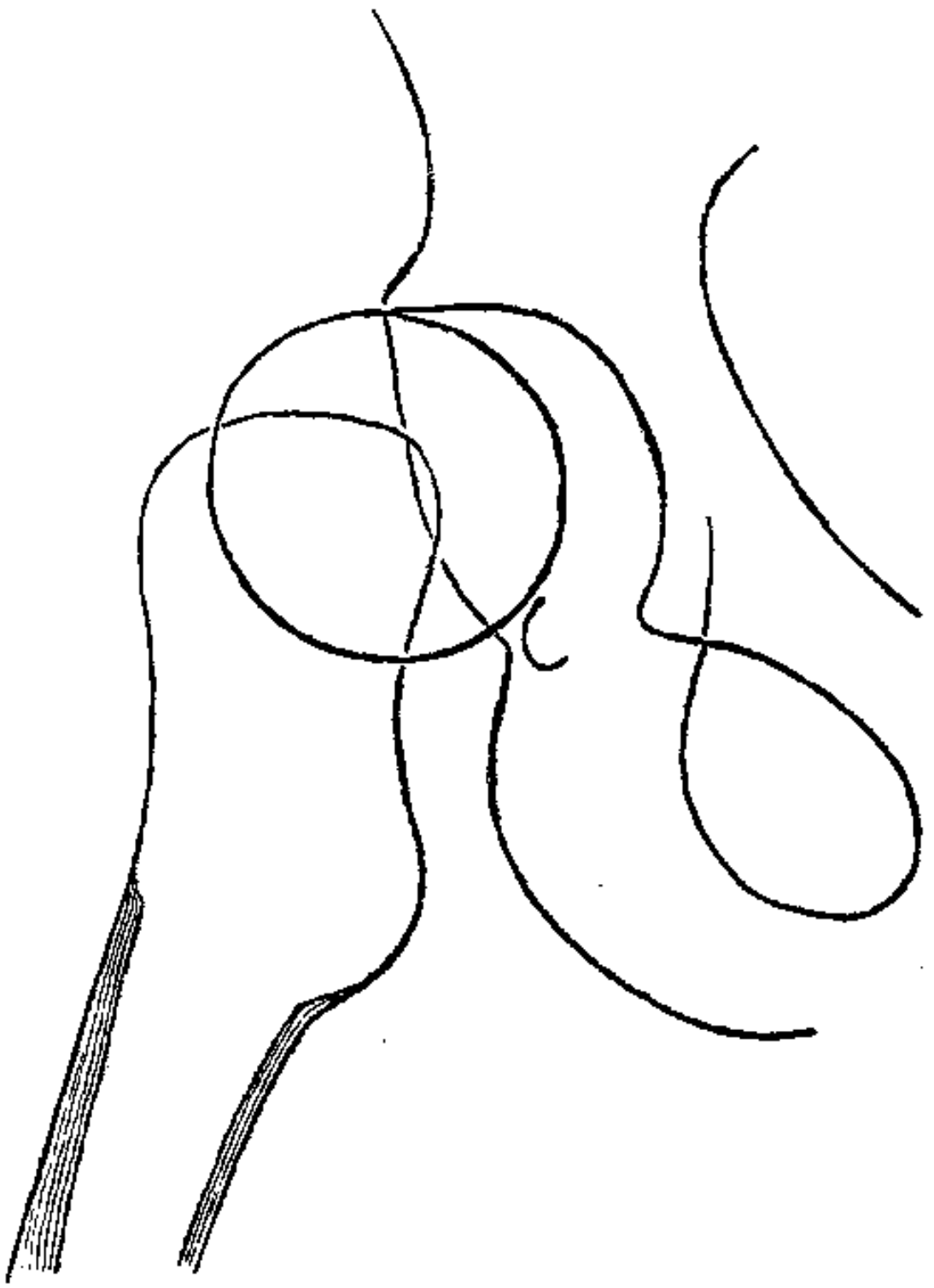

Figure 1. Tracing of x-ray film taken on 31st May, 1938. Fracture of neck of right femur with anterior dislocation of the head.

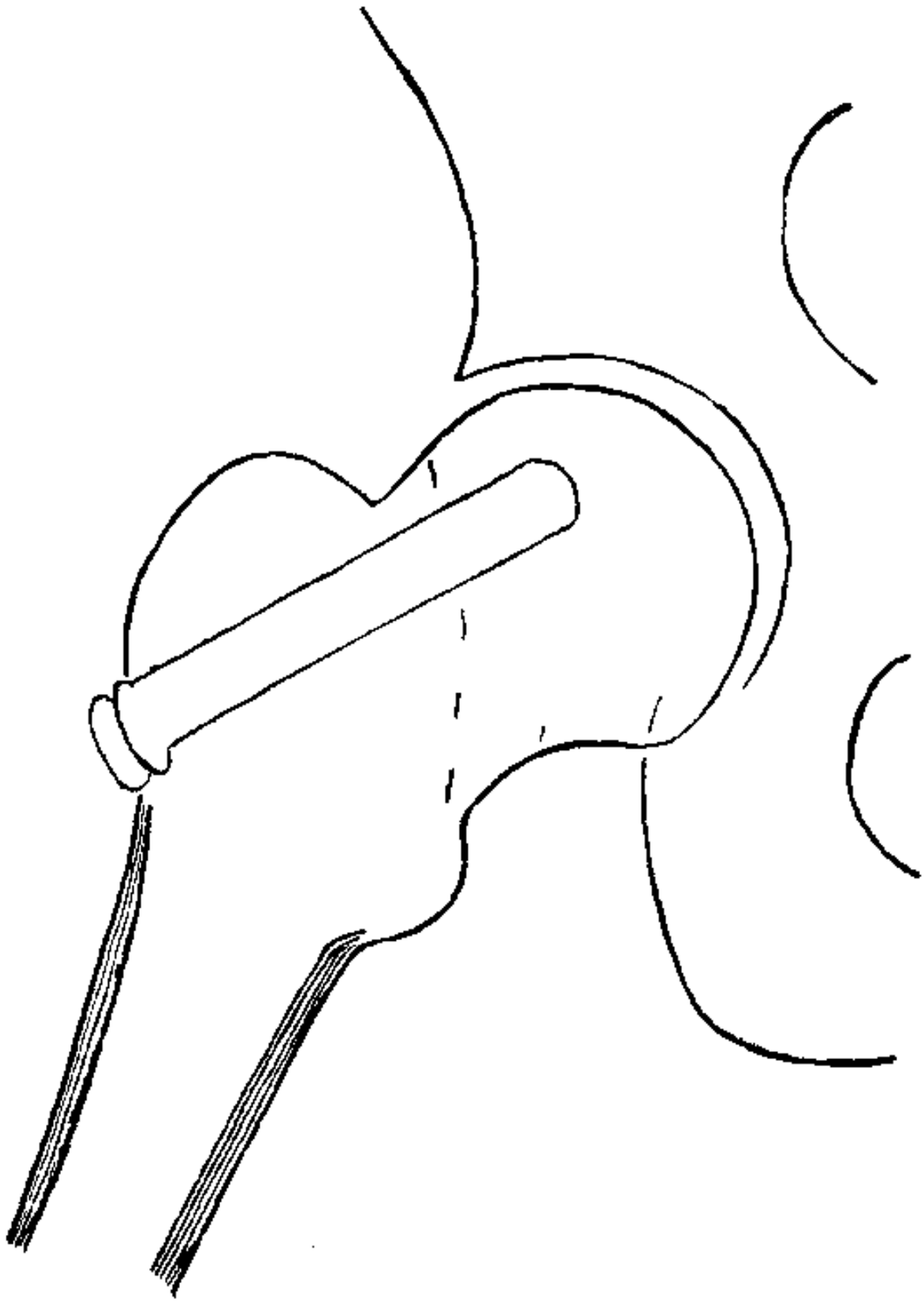

Figure 2. Tracing of x-ray of right femur taken on August 26th, 1938, showing head in acetabulum and good alignment of head and neck secured by fluted nail.

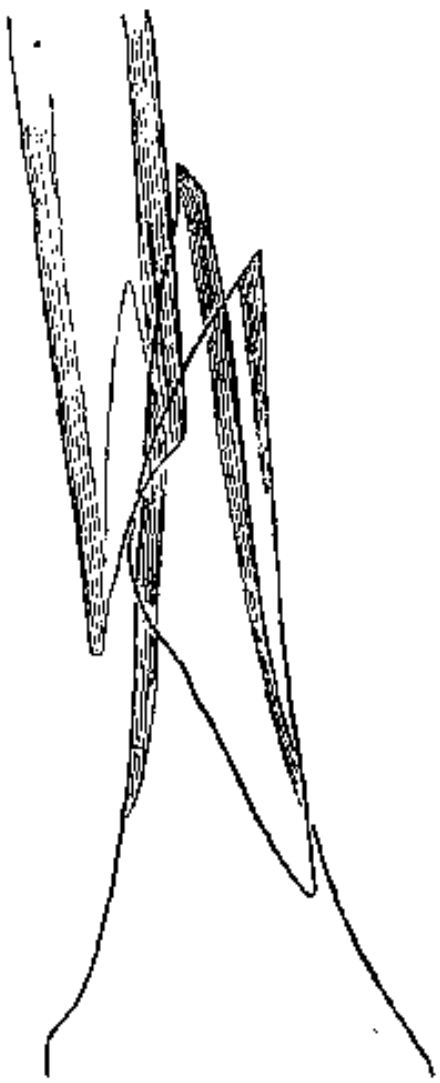

Figure 3.

Tracing of x-ray film taken 31st May showing fracture of shaft of left femur.

Figure 4.

Tracing of x-ray film taken on 1st October, 1938, showing union of the fragments of the left femur in fair position. This is the antero-posterior view—the lateral view was less satisfactory.

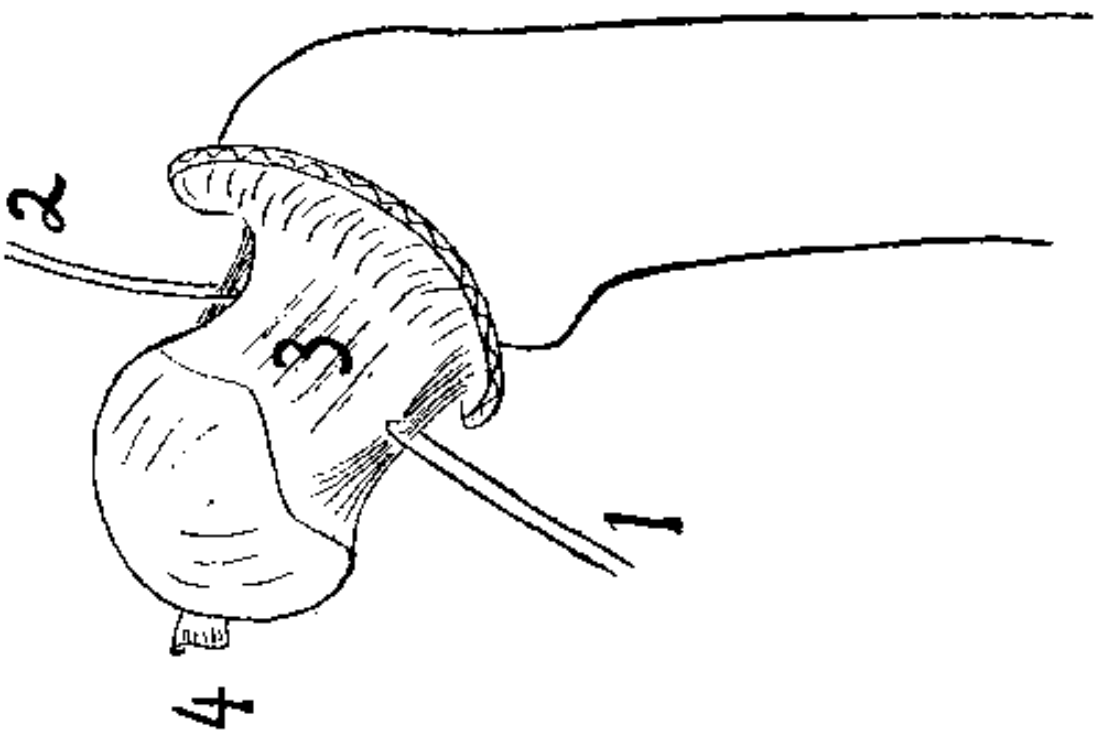

Figure 5. Sketch of front of upper end of a dissected left femur with fringe of capsular ligament attached.
1. Thread passed under the inferior retinacular fibres.
2. Thread passed under the superior retinacular fibres.
3. A few anterior retinacular vessels.
4. Ligamentum teres.

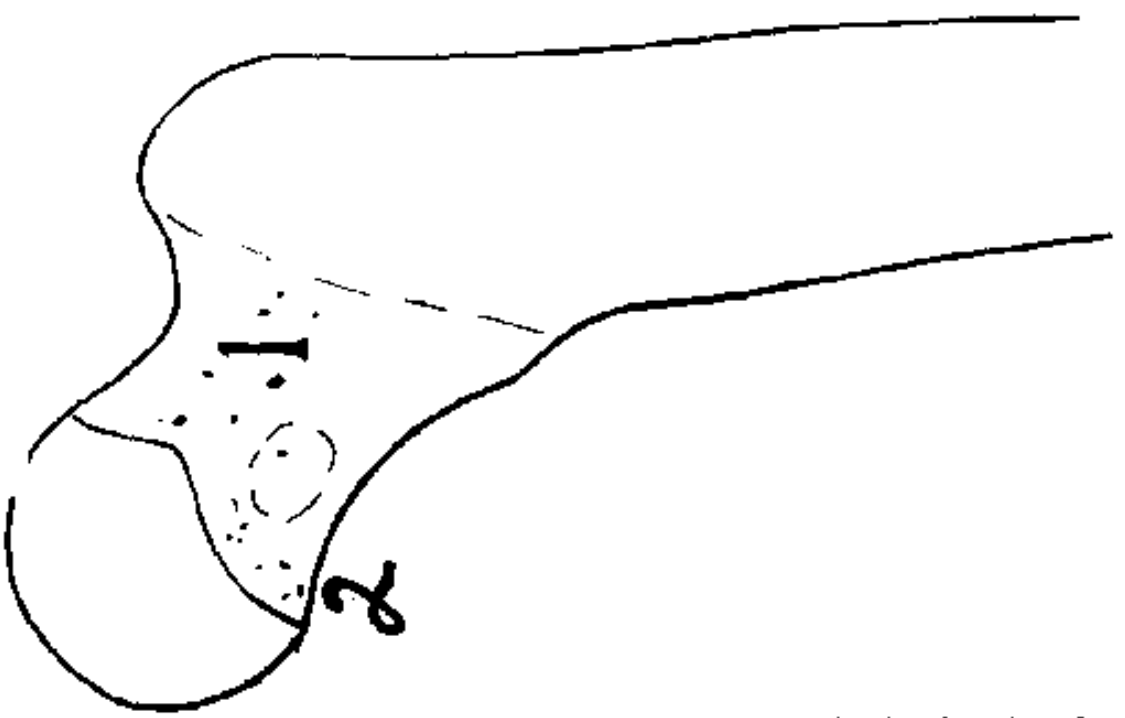

Figure 6. Sketch of front of a dry preparation of the same femur showing foramina of vessels entering the bone from the retinacula.
1. Anterior group.
2. Inferior group.

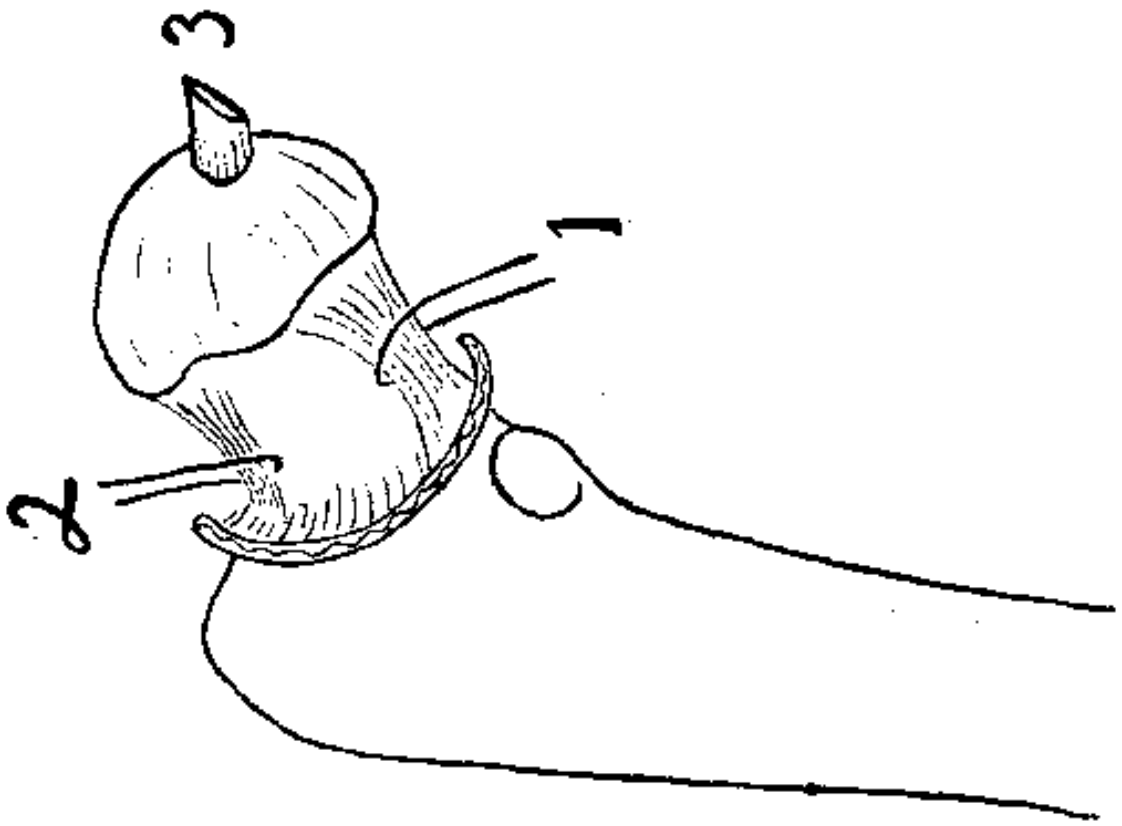

Figure 7. Sketch of back of upper end of a dissected left femur with fringe of capsular ligament attached.
1. Thread passed beneath inferior retinaculum.
2. Thread passed beneath superior retinaculum.
3. Ligamentum teres.

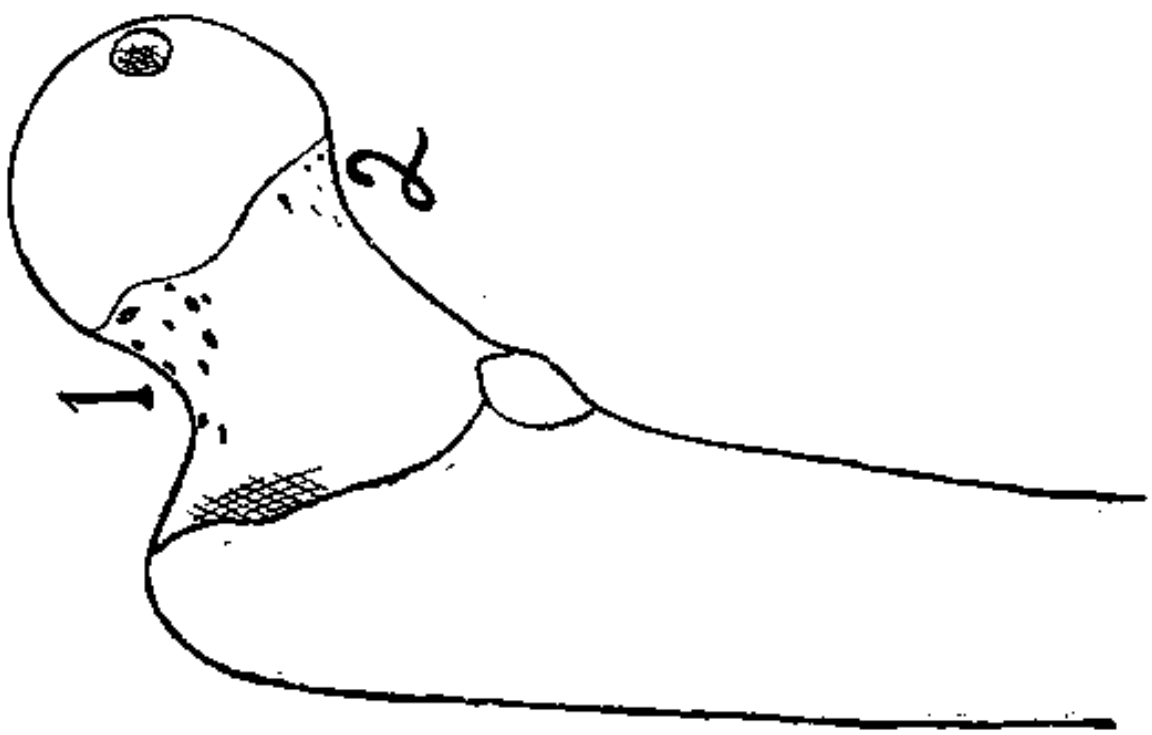

Figure 8. Sketch of back of a dry preparation of the same femur showing foramina of vessels entering the bone from the retinacula.
1. Superior group.
2. Inferior group.

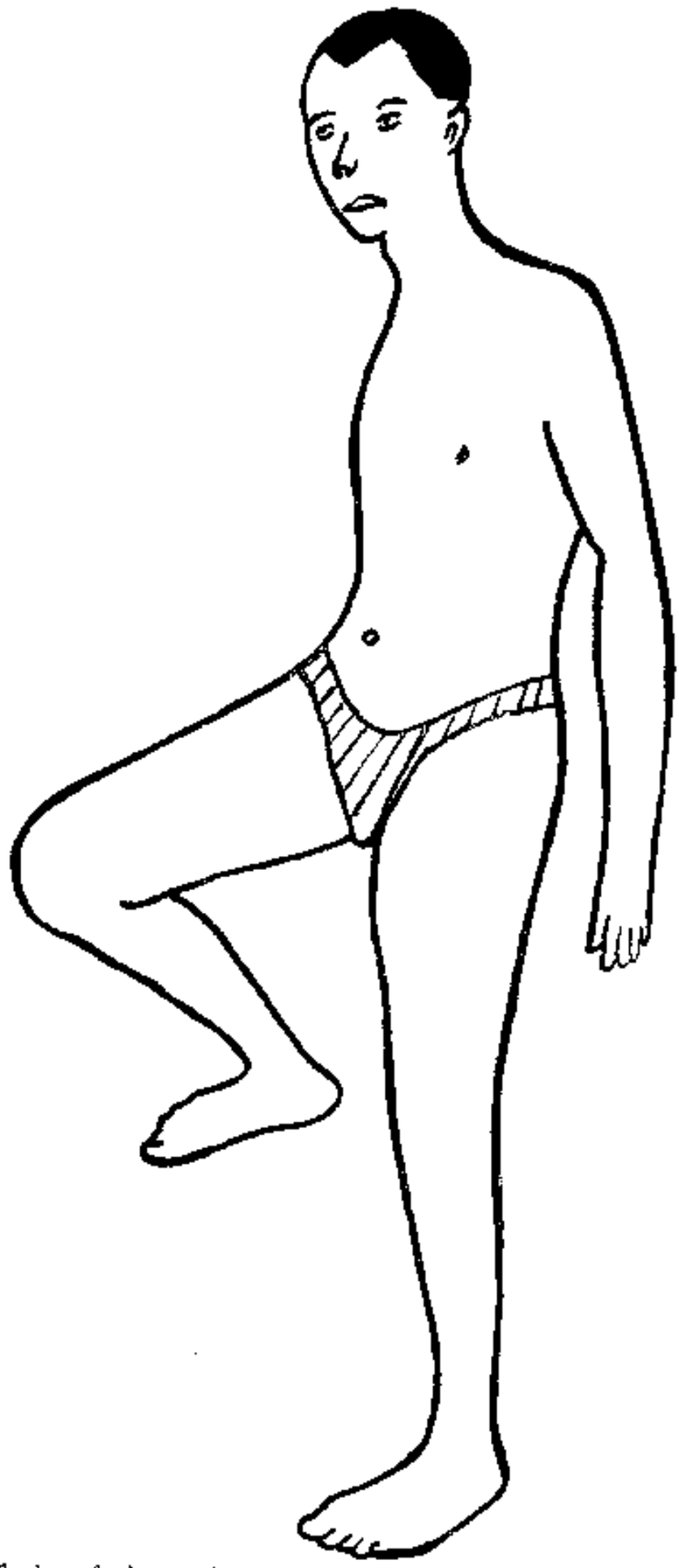

Figure 9. Tracing of photograph of patient at the end of October. He could walk, stand on one leg, and had full movements of both knee joints and fair movements of the hip joints.

these latter adhere so closely to the bone that they are torn with the fracture.

In our case reported here the patient recovered and the head of the bone neither necrosed nor underwent absorption although the neck was broken and the ligamentum teres was torn across. A good functional result was obtained as shown in the following tracings of X-rays and photographs. Figures 2, 4 and 9.

SUMMARY.

The retinacular fibres maintain the blood supply of the head after many (intracapsular) fractures of the neck of the femur, unless they too are all torn or unless the fracture occurs exactly at the junction of the head and neck.

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DIGBY, K. H. ..... (1926) .. A Flexible-Lance Traction Splint for the Lower Limb. *Surgery, Gynecology and Obstetrics*. August, p. 207-214.

A CASE OF OLD UNUNITED FRACTURE OF THE PATELLA WITH WIDE SEPARATION OF FRAGMENTS— EXCISION OF PATELLA-MUSCULO-APONEUROTIC TRANSPLANT TO CLOSE GAP.

by

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The Department of Surgery, The University, Hong Kong.

Chan Tin, U.S.C. No. 471/37, a Chinese male aged 50 was admitted on 3rd December, 1937 with inability to extend his right knee joint, following an injury four months previously.

At the end of July or at the beginning of August, patient slipped and fell on a concrete pavement one rainy day, and was taken home in a rickshaw. The knee was swollen and bruising developed.

Apart from numbness of both legs and feet for many years the patient had enjoyed good health.

On admission it was found that patient could not extend the right knee joint, and the right thigh was wasted. A fracture of the patella with wide separation of the fragments could be easily palpated. (Figure 1). The gap varied from two to three inches or more, depending on the degree of flexion of the joint. This was confirmed by X-ray examination (Figure 2).

On 3rd January, 1938, five months after the injury, the fragments were exposed by a curved lateral incision. No-hand-touch and strict-skin-segregation were of course observed.

All attempts to bring the fragments close together failed, so it was decided to excise the fragments. When these had been removed however the gap was slightly larger.

A strip of musculo-tendinous tissue 4" long, 1" broad, and ¼" thick was excised from the quadriceps, above where the upper fragment had been. This was turned upside down and one end was sutured with square stitches to the remains of the quadriceps whilst the other end was similarly sewn to the ligamentum patellae. (Figure 3).

A back splint was kept on till the 18th February, but after the 5th week, was removed from time to time for active movements.

By the 23rd of March flexion beyond a right angle was possible and the power of extension was good, but the patient was unable unaided to complete the last few degrees of extension (Figures 4 & 5).

This case is of interest chiefly because it records the survival of a large musculo-aponeurotic free transplant.

Figure 1. Tracing of photograph showing wide separation of fragments.
A—Outline of proximal fragment inked on the skin.
B—Outline of distal fragment inked on the skin.

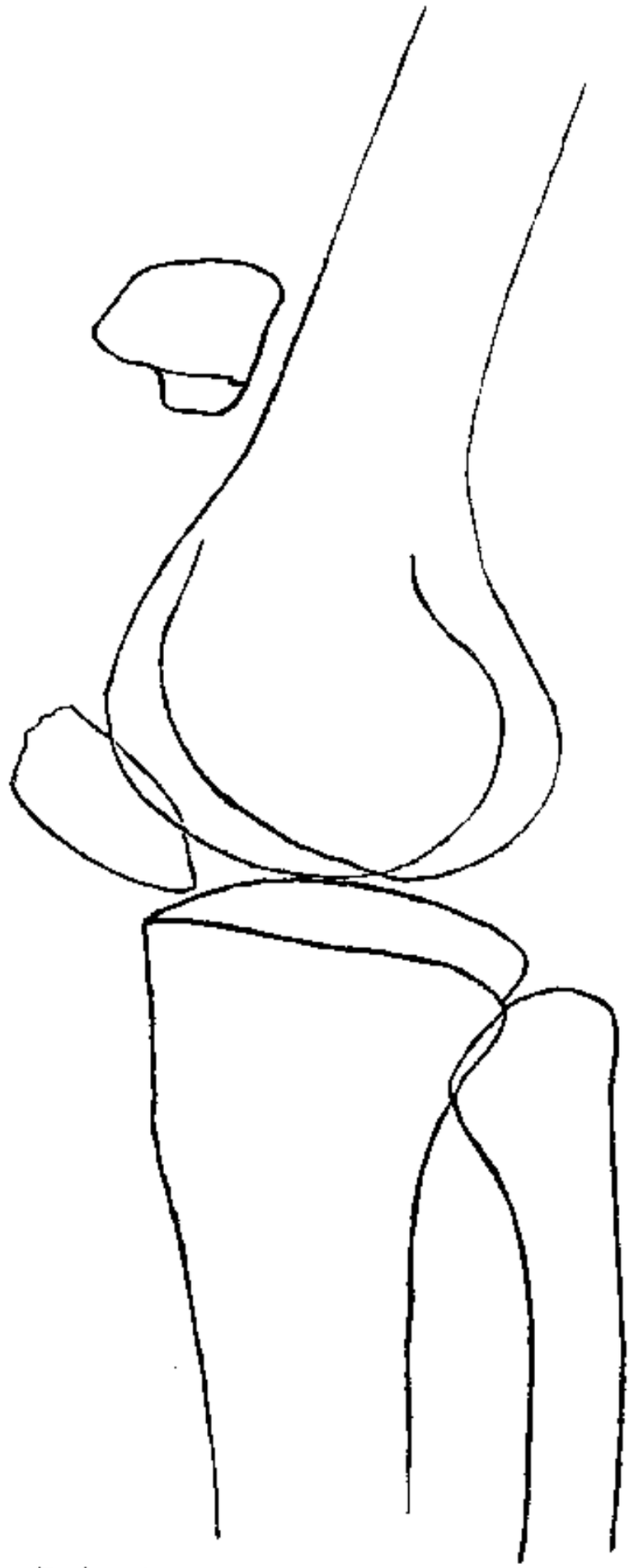


Figure 2. Tracing of x-ray film showing wide separation of fragments.

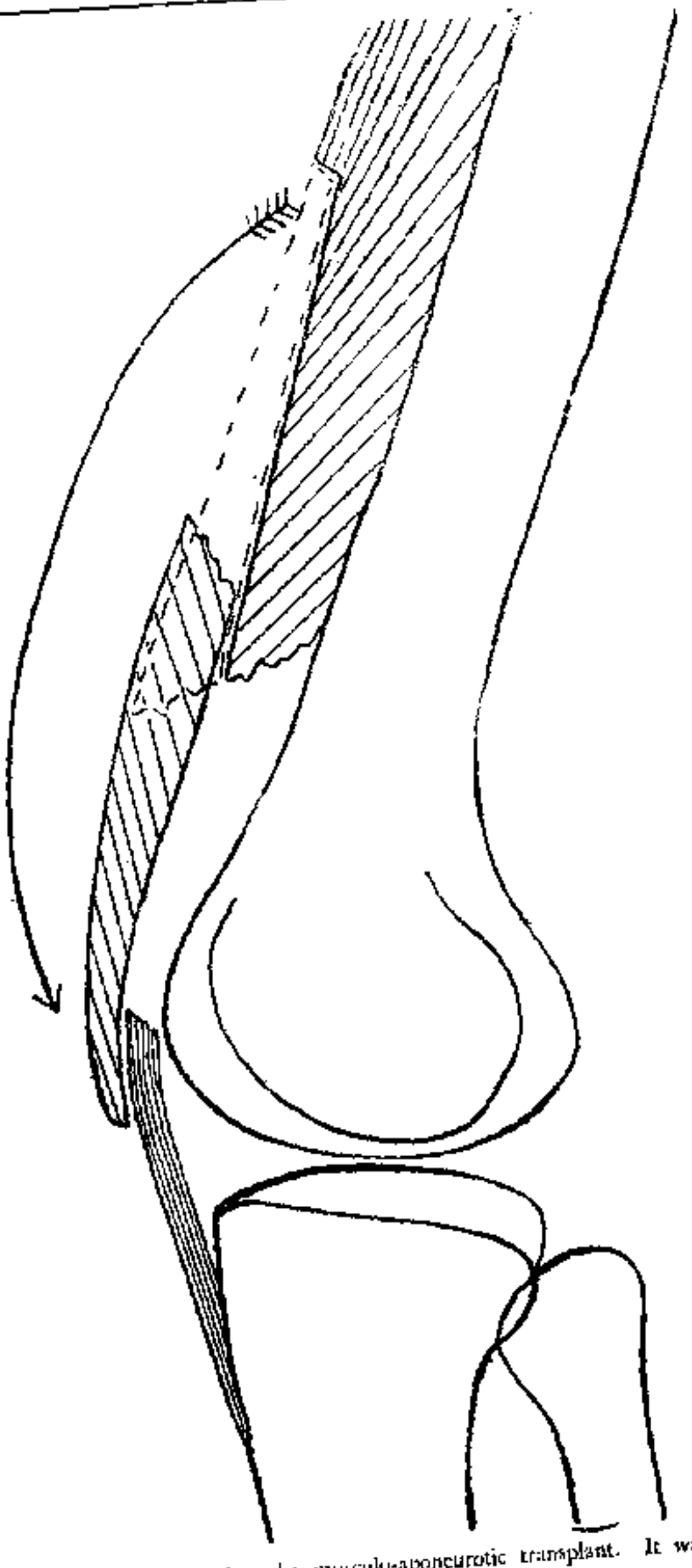
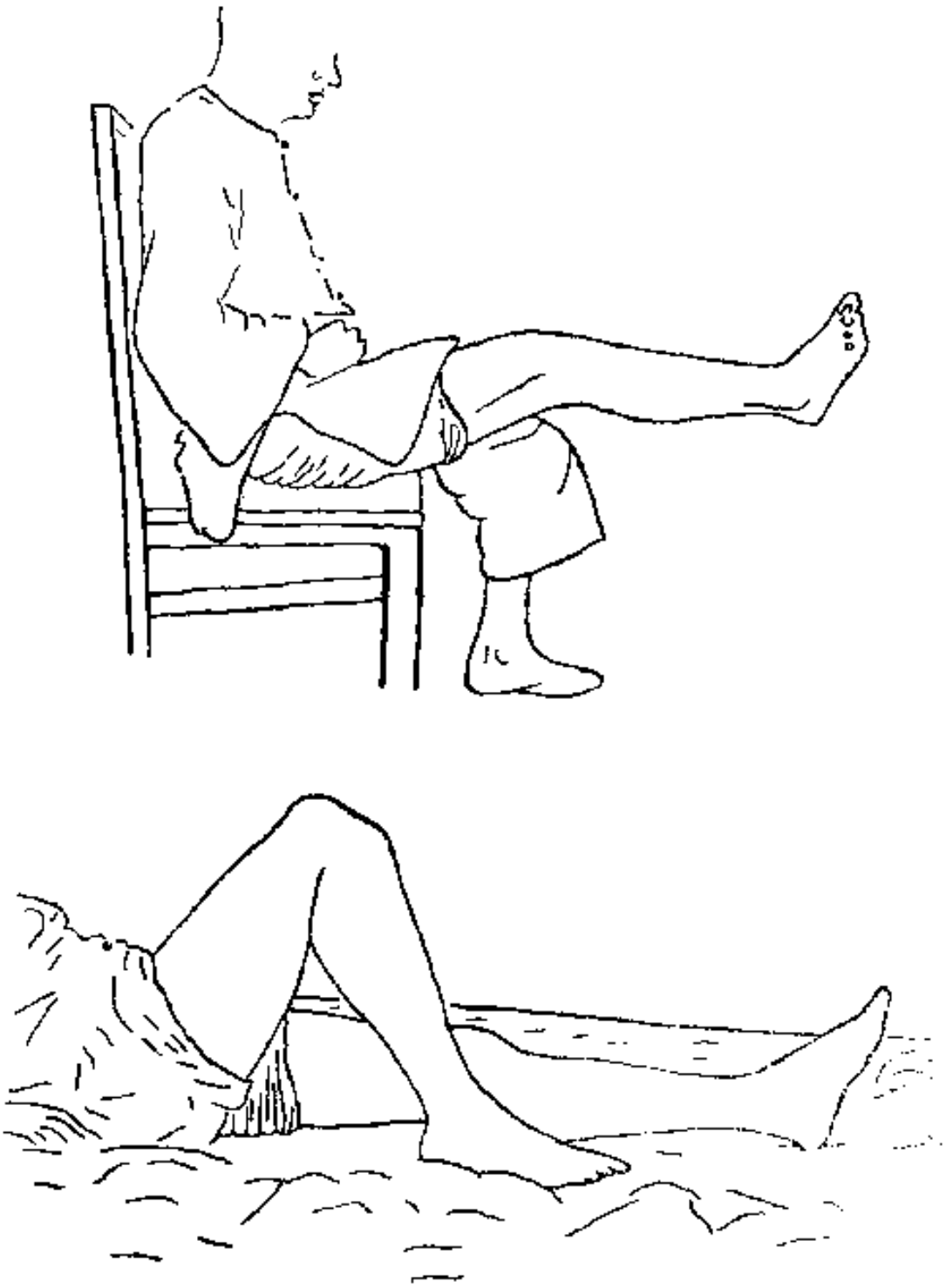


Figure 3. Diagram showing the musculo-aponeurotic transplant. It was inserted upside down as it seemed to fit better so.



Figures 4 & 5. Tracings of photographs illustrating the range of voluntary movement eleven weeks after operation.

It may be noted that in this old neglected case with wide separation of the fragments of the patella· removal of the fragments rather widened than lessened the gap. But it facilitated the attachment of the transplant.

In this case the patient could not complete the last few degrees of extension after recovery from the operation. And this draws attention to a function of the patella.

In a recent article Brooke (1937) states that "it (the patella) subserves no important function." But it *has* three important functions:

1. It protects the front of the knee joint from direct injury and penetrating wounds.

2. It provides a cartilaginous surface diminishing friction over the patellar surface of the femur.

3. It gives a better line of pull for the ligamentum patellae in the last few degrees of extension. To test the deterioration of this in a case where the patella has been removed it is necessary to compare the strength, speed, and completeness of voluntary extension from 15° of flexion *against gravity or other force* on the sound and on the affected knee.

One other point one would like to controvert. In an addendum to the above mentioned article, Hey Groves (1937) shows a drawing of a specimen supplied by Whitnall of *all* the fibres of the quadriceps passing over the front of the patella to the ligamentum patellae. In doubt of this, the writer of the present note prepared a specimen from an adult Chinese cadaver, showing that only a few fibres pass in front, the bulk of the quadriceps being inserted into the front of the upper border; and the bulk of the ligamentum patellae likewise coming from a bony attachment at the front of the lower border of the patella. (Figure 6).

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Figure 1 — Photograph of specimen showing cross-section of the sandstone block inserted into the panel.

Review of Books

"*Exercises in The Bath*," by T. R. Togna with an introduction by Sir Leonard Hill, M.B., F.R.S., LL.D. Pitman & Co., Ltd., London. Price 5/- nett.

This book of 125 pages describes, explains and elaborates the Tonga system of home gymnastics to promote health, prevent disease, and prolong life and does not contain, as might at first sight be expected, an elaborate system of vocal exercises for budding baritones or sibilant sopranos.

A book such as this, although obviously not written by a member of the medical profession, must of necessity contain a great deal of medical material including statements concerning both physiology and therapeutics, and must therefore be reviewed from that aspect as well as others.

The author is greatly impressed by the dread power of toxins, the excretion of which by the skin relieves the kidneys of a great deal of work, unpleasant work too apparently because the abnormally high concentration of toxins in the perspiration gives it in many cases an abnormally pungent odour. (p. 44). Toxins are also excreted by the lungs (p. 66), but despite these methods of escape additional to the bowels and kidneys they gradually silt up the body (p. 70), (more rapidly as age advances), impairing its function and shortening its life.

If these be the main methods of elimination of the dread toxins, then surely the exercises which cause the greater increase in respiration and sweating will result in the greater toxin elimination, but we are asked to believe the contrary from the scientific data in the appendix. The data on p. 119 lead to one conclusion only and that is exercises in the bath are more economic as far as oxygen consumption is concerned than exercises in the air. Whether that is a good or bad thing we do not know, but the author just assumes it to be every-day knowledge that it is a good thing. The lowered oxygen consumption merely means less work being done as one would expect when the muscles are rid of the necessity of supporting the limbs and body by the water; but it should be noted that any exercise, particularly type 1, see Figures 1-23, resulting in the lifting of limbs out of the water, removes this buoyancy advantage. If the author really believes in his theory the facial exercises depicted in Figures 9 and 10 should also be done under water. How these bath exercises hasten the change of lactic acid into its precursor is not explained but just stated as an axiom on p. 24.

Is there then no sense in the book at all? Of course there is; any system of regular exercises, especially performed where they cannot be overdone—and that to our mind is the real value of the bath, not that it reduces oxygen consumption but that it limits the work

done both by water buoyancy and by discomfort—is going to be of benefit. Mr. Togna and his wife have both evidently received great benefit from a discovery that has now become a fad—we wonder how much their improvements in health are due to their very wise moderation in all other things—and they have become missionaries in the cause. Certain eminent London physicians have taken an interest in them, for example Drs. Ray and Poulton, and very wisely too, for we should be always ready to accept help from any source.

The weak point of the whole book is the obvious and feeble attempt to give the whole scheme a scientific foundation which to our mind turns out to be veneer.

Sir Leonard Hill's introduction says nothing and says it quickly in 14 lines. We found only two typographical errors; "are" on page 17, 5th line from the bottom should read "is." and in Table I the normal chest measurement should obviously read "36¾" in the September 1926 column.

Acknowledgements.

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