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EPIDEMIC PNEUMOCOCCAL MENINGITIS IN HONG KONG. 1938—1939.

by

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Pneumococcal infections were not unduly common in Hong Kong during 1938. 483 cases of cerebrospinal meningitis were seen either in the mortuaries or in hospital during 1938, whereas only two proved cases of pneumococcal meningitis were observed during the same period at the Infectious Diseases Hospital, one of them in June, the other in November. Bar diagram 1 illustrates the monthly incidence of meningococcal meningitis during the year and bar diagram 2 shows the monthly incidence of pneumococcal meningitis during 1938 and January 1939. Diagram 3 shows the daily incidence, as far as could be judged, of primary cases of pneumococcal meningitis admitted to hospital during January 1939 and of cases of the same disease which occurred in hospital during that month.

The sudden marked increase of pneumococcal meningitis in January is strikingly shown in diagram 2, and this paper describes the cases as they occurred. They are described in detail because no record can be found of a similar outbreak of pneumococcal meningitis. Another point which makes Group B profoundly interesting is that every case in the group was recovering from proved meningococcal meningitis when attacked by pneumococcal meningitis.

INTRODUCTION.

At the end of December 1938, the incidence of primary pneumococcal meningitis in the colony suddenly increased, and at the same time pneumococcal meningitis appeared in the Infectious Diseases Hospital as a secondary infection on January 1st and spread through one ward on the ground floor in a manner which suggested transmission by droplet infection from a carrier.

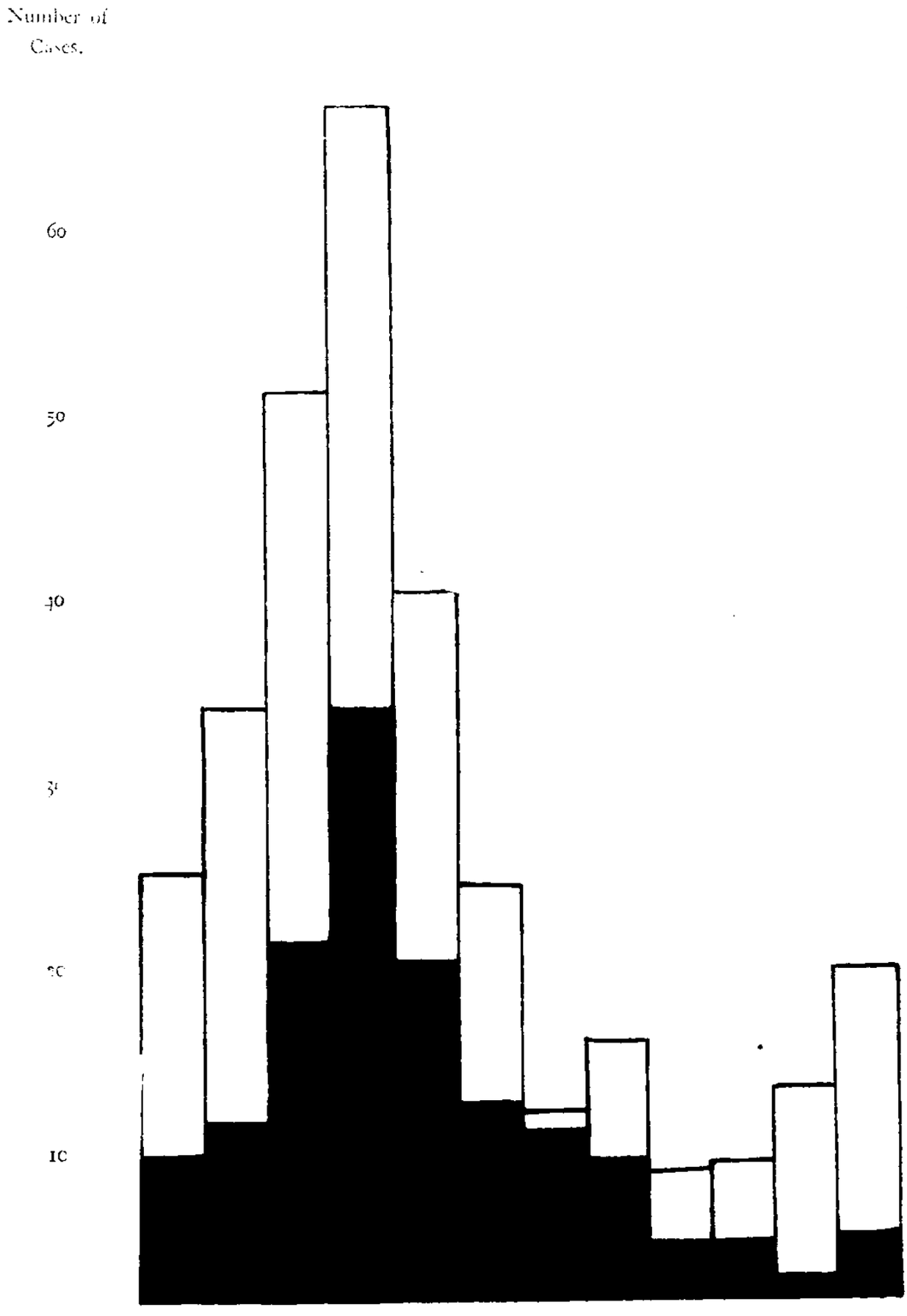


DIAGRAM 1

Monthly Incidence of Meningococcal Meningitis in 1938.

DIAGRAM II

Monthly Incidence of Pneumococcal Meningitis, 1938—1939.

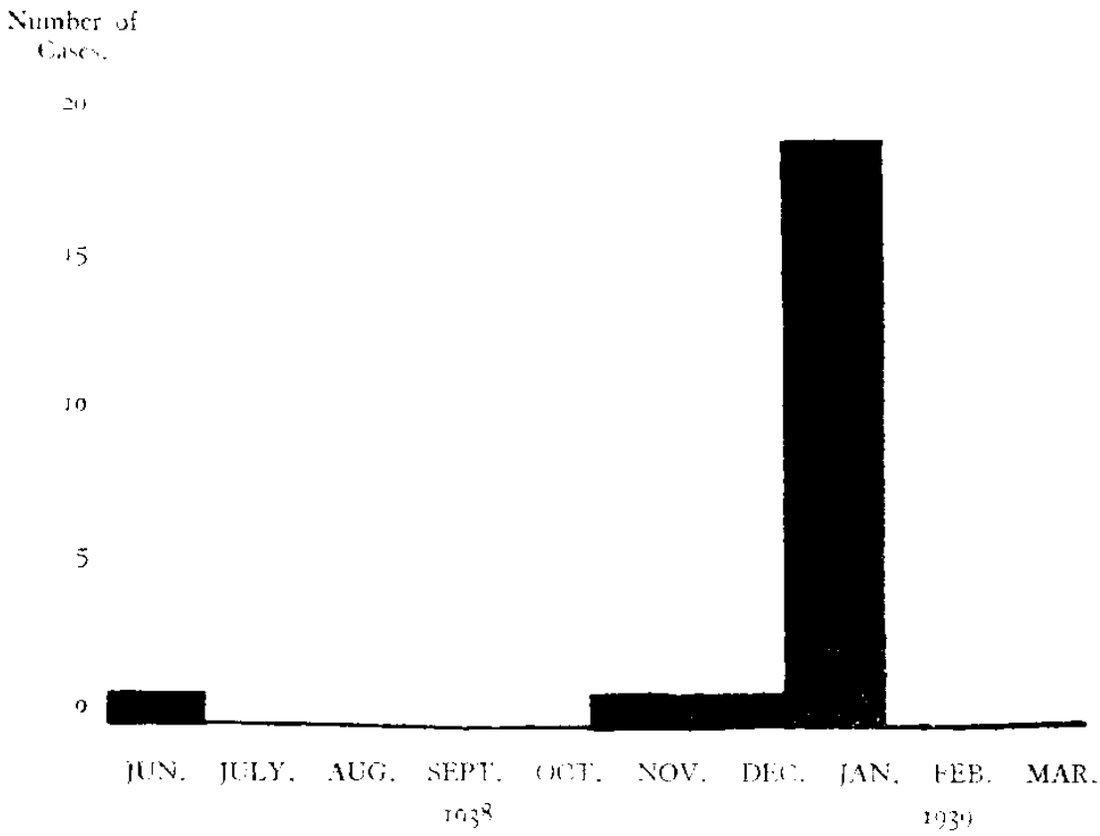
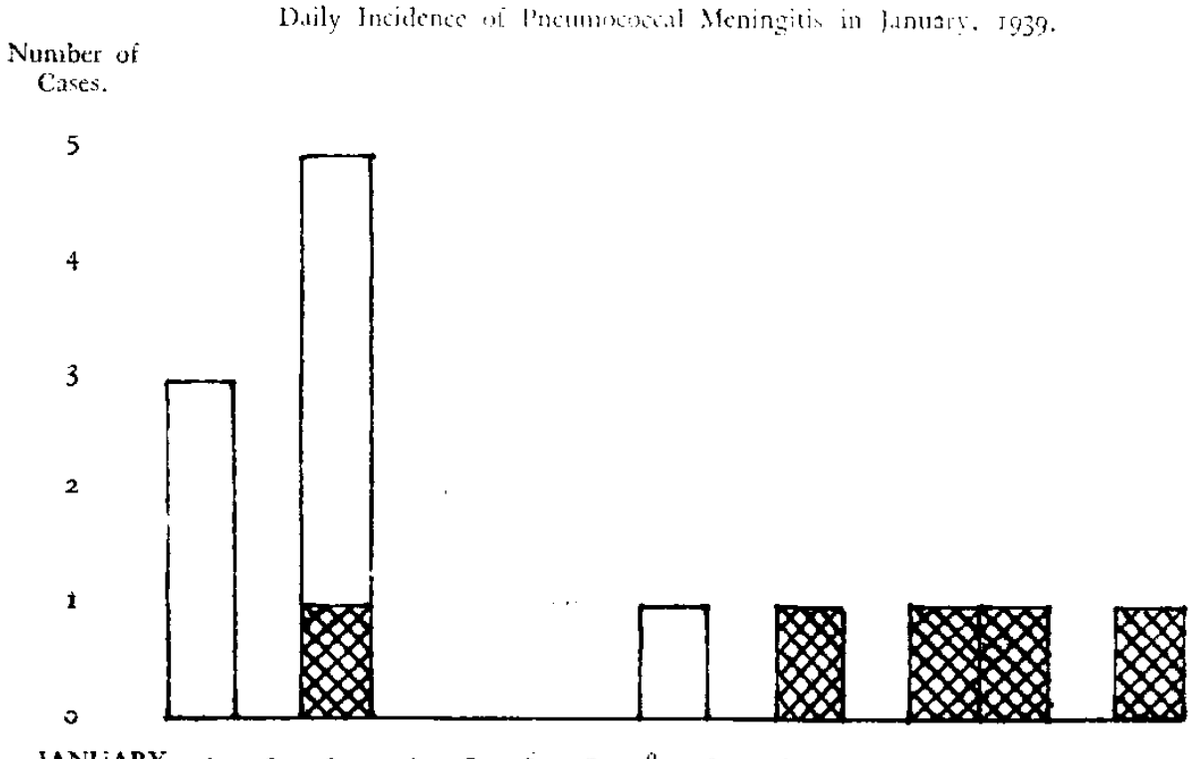


DIAGRAM III

Daily Incidence of Pneumococcal Meningitis in January, 1939.



□=Cases Secondary to Meningococcal Meningitis,

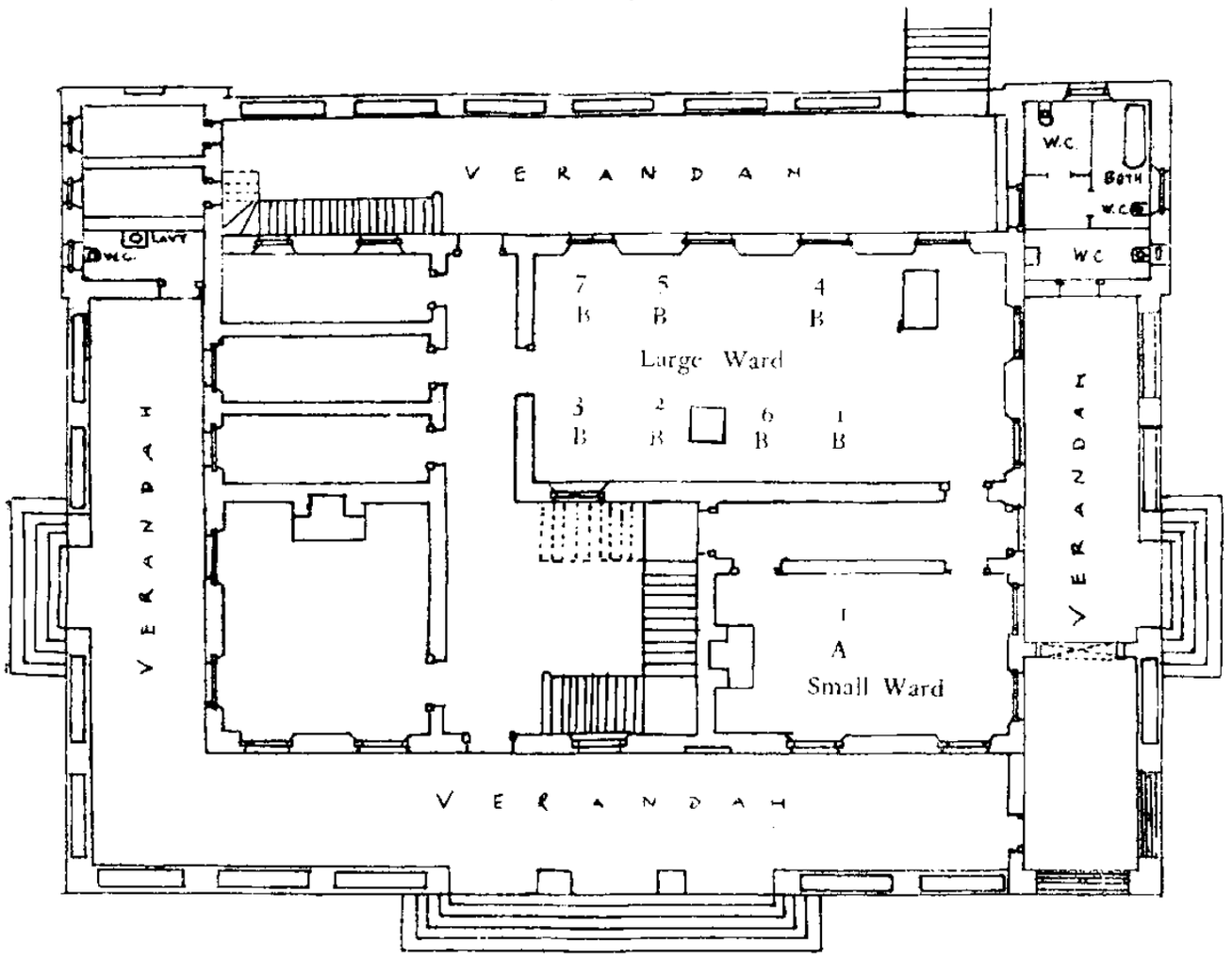
Each cross-hatched rectangle represents one case of primary pneumogocal meningitis.

A woman aged 30 was sent into the Infectious Diseases Hospital on December 30th from one of the Chinese hospitals, as a probable case of meningococcal meningitis. She was delirious, restless and noisy on admission, and no history was obtainable. She showed the signs characteristic of meningitis, and lumbar puncture yielded highly turbid fluid under great pressure. 35 c.c. were drained and the woman was treated provisionally with anti-meningococcal serum and streptocide as a case of meningococcal meningitis. Her condition deteriorated rapidly and she died 24 hours after admission without having made the least response to treatment. Pneumococci were found in her cerebrospinal fluid, but unfortunately they were not typed. At autopsy the whole of the vertex of the brain was plastered with thick, creamy, pale yellow pus and there was a marked degree of purulent infiltration at the base. Both lateral ventricles also contained purulent fluid. The whole brain surface was studded with innumerable pin-point haemorrhages, a finding which pointed strongly to a pneumococcal rather than a meningococcal infection. This case was nursed in the smaller of the two downstair wards and never came into immediate contact with the inmates of the larger ward though she was tended by the same nurses and amahs.

A case of cholera was admitted to the Infectious Diseases Hospital on January 1st, 1939. He developed signs of a typical left basal lobar pneumonia on January 2nd, 1939, and died on the 5th. At autopsy the bases of both lungs were found to be coated with a shaggy layer of fibrino-purulent material ⅛" thick, swabs from which yielded a growth of pneumococci. Pneumococci were also obtained by culture of exudate from the cut surface of the lung and on typing they were found to belong to Group IV. It is unlikely that this man can have played any part in disseminating the pneumococci which produced pneumococcal meningitis in the hospital at this time, for these reasons: first, he was nursed upstairs in a ward remote from the downstairs ward in which the first pneumococcal meningitis developed; second, he was nursed by attendants who were not working in the affected downstairs ward; and third, the first child to develop pneumococcal meningitis in the course of recovery from meningococcal meningitis had headache and nausea on December 31st and fever of 102.4° at noon on January 1st the date of this man's admission to hospital. It is, therefore, inconceivable that there could have been any causal relation between the two cases.

Diagram IV enables the spatial relationships of the wards on the ground floor and first floor of the hospital to be visualised clearly, and they also show how the outbreak was confined, at first at any rate, to the larger of the two wards on the ground floor. An attempt was made to isolate as far as possible all proved cases of pneumococcal meningitis as soon as it became known that we were,

DIAGRAM IV



GROUND FLOOR PLAN

Plan to show arrangement and dimensions of the wards on the ground floor of the Infectious Diseases Hospital. The dimensions of the first floor wards, are the same. Each large ward is 42 feet long, 22 feet wide and 15 feet high. Each small ward is 25 feet long, 13 feet wide and 15 feet high. The beds were six feet or more apart in all wards.

The serial numbers show the positions of the beds occupied by the patients denoted. The hollow rectangles indicate beds occupied by patients who escaped pneumococcal meningitis.

in fact, dealing with that type of meningitis, but as cases of primary pneumococcal meningitis were being admitted throughout the first two weeks of January, 1939 early isolation was impracticable in many instances owing to the absence of diagnostic criteria between pneumococcal and meningococcal meningitis.

All members of the staff had throat swabs taken during the first week in January 1939, and all swabs save two were negative for pneumococci. Of the two positives, one was a nurse who had begun duty at the Infectious Diseases Hospital on January 2nd and had therefore arrived after pneumococcal meningitis had appeared in the

hospital: the other was a ward-boy who had been on duty at the Infectious Diseases Hospital for several months. He had had no sore throats nor had he complained of illness of any sort during his period at the Infectious Diseases Hospital. Most unfortunately it was not possible to get typing done when his throat swab was found to be positive. From January 8th onward all members of the staff entering meningitis wards were masked.

For descriptive purposes the cases of pneumococcal meningitis observed from December 30th, 1938 may be divided into two groups, A and B; the first A, consisting of cases of primary pneumococcal meningitis, the second B of cases of meningococcal meningitis which developed pneumococcal meningitis during the course of the primary meningococcal infection. Two patients have been included in the first group who were clinically almost certainly meningococcal to start with, as they responded to anti-meningococcal treatment, but later developed signs suggestive of a secondary infection which was proved to be pneumococcal. Group A consists of six cases, group B of thirteen. Pneumococci were isolated from the cerebrospinal fluid during life in all cases in Group A, whereas in three of the cases in Group B pneumococci were only found on examination of pus and brain smears obtained at autopsy. Meningococci had been demonstrated in every cases in Group B during life.

The following tables show the relevant bacteriological points in the cases in each group.

Clinically there appear to be no very satisfactory criteria enabling one to diagnose meningococcal from primary pneumococcal meningitis with certainty. The onset is sudden in both conditions and in the absence of concomitant pneumococcal infections is of no help in differentiating the two. Some importance is to be attached to the presence of labial or facial herpes, and case 4A was considered on admission to be probably meningococcal, first because she had well marked labial herpes, second because responded in the expected manner to a line of treatment known to be efficacious in proved meningococcal meningitis. (Wilkinson 1939 B). Her chart shows clearly how well she responded to streptocide and anti-meningococcal serum. On the 10th, that is two days after admission, she was sitting up and asking could she not go home as she was perfectly well. On the 13th it was abundantly clear either that she had relapsed or had developed an intercurrent infection. Treatment with M. and B. 693 was begun at midday the same day, and pneumococci were reported in her cerebrospinal fluid the same evening. She had, in all, 32 grms. of the drug in six days and perished miserably in spite of it.

Case 2A, although never proved bacteriologically to be meningococcal, could scarcely have been pneumococcal from the day of her admission on the 14th of December. Her chart shows how she

PRIMARY PNEUMOCOCCAL MENINGITIS.

Group A.

TABLE I.

No.	*Sex, Age*	*Date admission*	*Date Pneumococci found*	*Date of death Type Pneumococci*	*Treatment*
1A	F. 30	30.12.38.	30.12.38.	31.12.38. Untyped	Streptocide A.M.S.
2A	F. 11	14.12.38.	7. 1.39.	13. 1.39. Type II	M. and B. 693 18 grms.
3A	M. 14	10. 1.39.	11. 1.39.	16. 1.39. Type II	M. and B. 693 21 grms.
4A	F. 11	8. 1.39.	13. 1.39.	19. 1.39. Type II	M. and B. 693 32 grms.
5A	M. 28	12. 1.39.	14. 1.39.	14. 1.39. Type II	Streptocide A.M.S.
6A	M. 60	15. 1.39.	15. 1.39.	16. 1.39. Type I	Streptocide

A.M.S. = Anti-meningococcal serum.

PNEUMOCOCCAL MENINGITIS FOLLOWING ON MENINGOCOCCAL MENINGITIS.

Group B.

TABLE II.

No.	*Sex, Age*	*Date of Admission*	*Meningococci found in C.S.F.*	*Pneumococci found in C.S.F.*	*Type of Pneumo-coccus*	*Pneumococci found in life or P.M.*	*Treatment*
1B	F. 10	9.12.38.	9.12.38.	4.1.39.	II	D. 5.1.39. L.	Streptocide A.M.S. No. M. & B. 693
2B	F. 9	19. 9.38.	20. 9.38.	5.1.39.	IV	D. 7.1.39. L.	Streptocide A.M.S. No. M. & B. 693
3B	F. 13	4.12.38.	4.12.38.	2.1.39.	IV	D.10.1.39.	Streptocide A.M.S. M. & B. 693 25 grms.
4B	M. 8	20.12.38.	20.12.38.	6.1.39.	II	D.11.1.39. L.	Streptocide A.M.S. M. & B. 693 20 grms.
5B	F. 25	15.12.38.	15.12.38.	4.1.39.	II	D. 3.1.39. L.	Streptocide A.M.S. No. M. & B. 693
6B	F. 9	16.12.38.	16.12.38.	5.1.39.	II	D.12.1.39. L.	Streptocide A.M.S. M. & B. 693 20 grms.
7B	F. 20	15.12.38.	15.12.38.	3.1.39.	II	D.12.1.39. L.	Streptocide A.M.S. M. & B. 693 25 grms.
8B	M. 26	22.12.38.	22.12.38.	6.1.39.	Untyped	D. 7.1.39. L.	Streptocide A.M.S. No. M. & B. 693
9B	M. 23	3. 1.39.	4. 1.39.	10.1.39.	II	D.10.1.39. P.M.	Streptocide A.M.S. No. M. & B. 693
10B	M. 19	8. 1.39.	8. 1.39.	11.1.39.	II	D.12.1.39. L.	Streptocide A.M.S. No. M. & B. 693
11B	M. 9	23.12.38.	24.12.38.	10.1.39.	II	D.10.1.39. P.M.	Streptocide A.M.S. No. M. & B. 693
12B	F. 12	10. 1.39.	10. 1.39.	14.1.39.	IV	D.16.1.39. L.	Streptocide A.M.S. No. M. & B. 693
13B	M. 9	23.12.38.	23.12.38.	14.1.39.	II	D.12.1.39. P.M.	Streptocide A.M.S. No. M. & B. 693

A.M.S. = Anti-meningococcal serum. D = died.
L = Pneumococci found during life and P.M. found after death.

responded to anti-meningococcal therapy and also how she suddenly developed a secondary infection ultimately proved to be pneumococcal on January 3rd. Pneumococci were first reported on the 7th and treatment with M. and B. 693 was begun on the 9th. Once again the drug failed to modify the course of the infection in the slightest degree and it was discontinued on the 12th when it was obvious that the child was in articulo mortis. The drug could not be given earlier in this case owing to the very small amount available.

I comment on these two cases first because they were both admitted to hospital as cases of meningococcal meningitis and they both responded to anti-meningococcal treatment. They are included in group A simply for lack of bacteriological evidence proving them to be meningococcal. Before dealing with the cases in the two groups in detail, it must be made clear first, that all through January 1939 the hospital was receiving cases of meningitis almost daily, and that in most of these cases it was impossible to tell on clinical grounds alone whether the patient was suffering from primary pneumococcal or meningococcal meningitis; second, it must be remembered that supplies of the drug M. and B. 693 only became available on the 7th of January and then in small quantity only. Three of the cases in group A, 2A, 3A and 4A received M. and B. 693 and four of the cases in group B, 3B, 4B, 6B and 7B were treated with the drug.

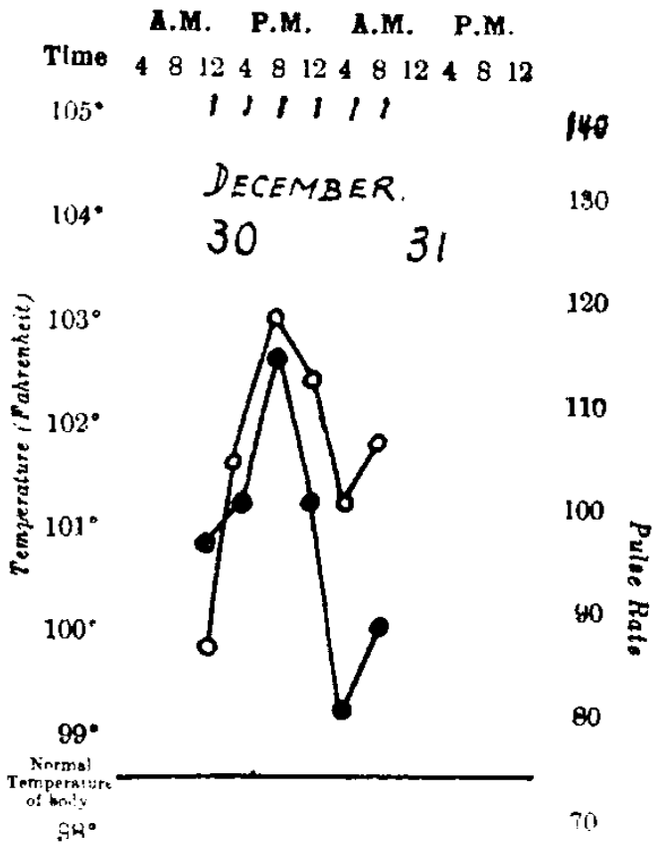
GROUP A. CASE HISTORIES.

PRIMARY PNEUMOCOCCAL MENINGITIS.

CASE 1A.

Cheng Lui, female, 30. This patient was sent into hospital on 30.12.38. She was restless, noisy and disoriented on admission and no history was obtainable from her. Her father stated that her illness had begun four days before admission with a chill but no rigor. She had slight fever and severe headache and aching of the whole body. There was no nausea or vomiting. On the third day of her illness her arms became cramped but no neck stiffness was noted. The same night she became unconscious and was admitted to the Kwong Wah Hospital. She had had no coryza, sore throat or cough, nor had there been any involvement of eyes, ears or skin. In addition to the classical signs of meningitis she showed a partial right third nerve paresis. The heart and lung sounds were normal.

Lumbar puncture was performed immediately under local anaesthesia and greenish yellow turbid cerebrospinal fluid was obtained at a pressure of 300+. 35 c.c. of cerebrospinal fluid were drained and 23 c.c. of anti-meningococcal serum were given intrathecally. Pneumococci were found in smears of the fluid and on culture and she died the day after admission. She was lumbar punctured twice and received in all 43 c.c. of anti-meningococcal serum intrathecally and 6 grms. of streptocide by mouth. M. and B. 693 was not then available and as the diagnosis was only established one hour before her death it could not have been of much use. At autopsy the whole vertex of the brain as well as the base was covered with thick, creamy, pale yellow pus, and this uniform layer of pus was studded with innumerable pin point haemorrhages. There was also pus in both ventricles. The heart, lungs, upper respiratory passages and abdominal viscera showed no naked eye abnormalities. Pneumococci were found in all preparations made from brain and cerebrospinal fluid at autopsy but were not typed.



Case 1A

Figure 1. Hollow circles show pulse rate, solid circles temperature. Figures under time line show grammes of streptocide administered.

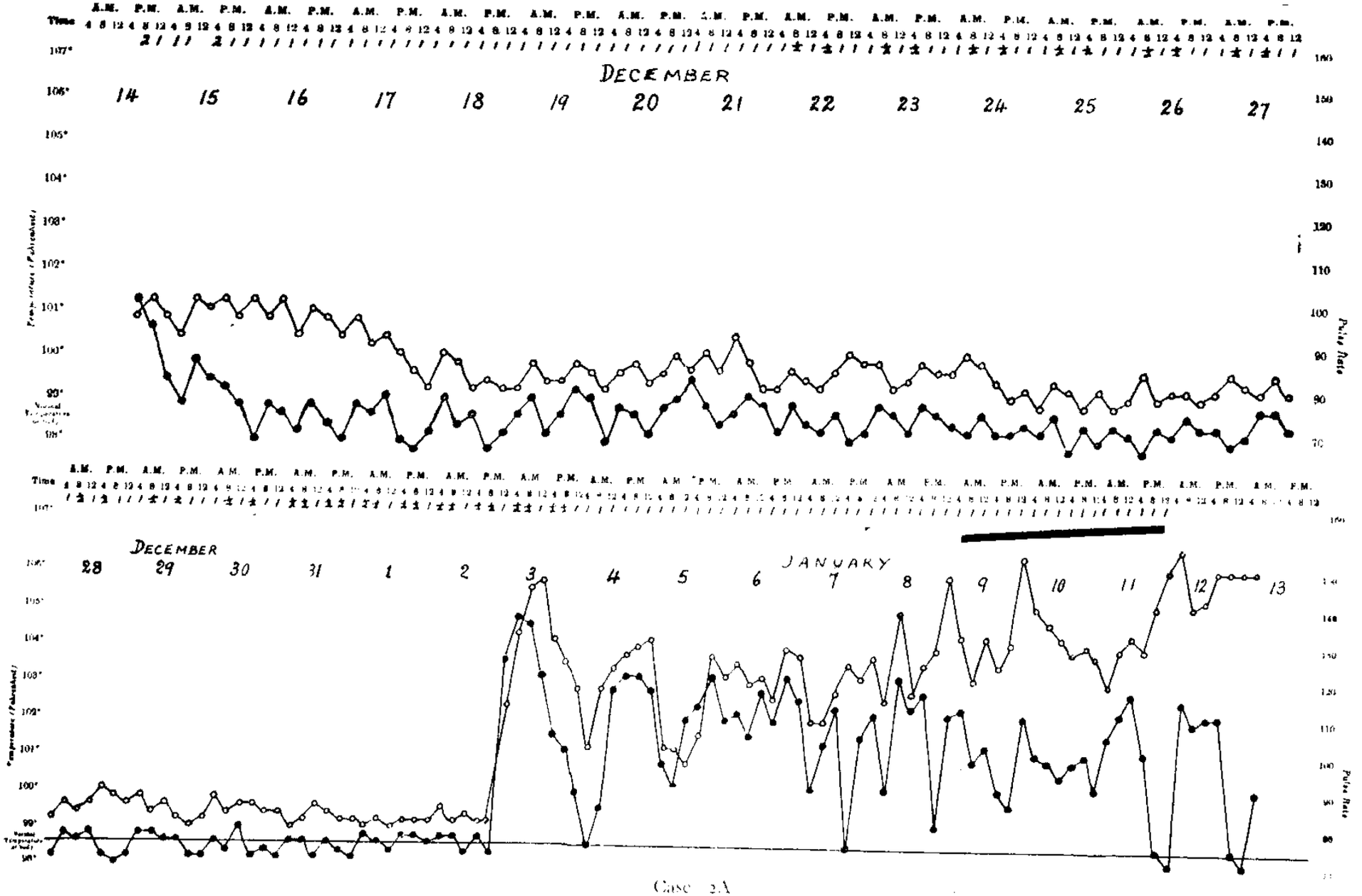
CASE 2A.

Lam Kwan, female 11. This child was admitted on the 14th of December 1938 having then been ill six days. Her father said the illness began suddenly with a rigor, fever and vomiting. Hyperaesthesia and nausea were marked but the child did not complain of headache.

On admission the child was restless and noisy but not unconscious. Tâche, head retraction and a positive Kernig's sign on both sides were noted. Lumbar puncture under ether yielded a turbid fluid under a pressure of 100. No serum was given but streptocide 6 grms. in 24 hours was ordered. Meningococci were never demonstrated in the fluid, but the child responded rapidly and well to this treatment, and by the 20th her cerebrospinal fluid was clear. By the end of the month her fluid was normal and she appeared to be perfectly well.

On the 3rd of January her temperature, which had been normal for 12 days, rose to 103.4°, her cerebrospinal fluid was found to be turbid again and her signs of meningitis recurred. A meningococcal relapse was feared and she was given 20 c.c. of anti-meningococcal serum intrathecally, her streptocide being increased from 4 to 6 gms. in the 24 hours. The serum was not repeated, pneumococci were found in the cerebrospinal fluid on the 7th and treatment with M. and B. 693, 1 grm. 4 hourly was begun on the 9th. A dose of 18 grms. in three days affected the course of the meningitis not one iota, and the drug was discontinued on the day preceding death. At no time during this terminal phase did she develop any lung signs, and a throat swab taken on 9.1.39. showed neither diphtheria bacilli nor pneumococci.

If we assume that the rise of temperature on the 3rd of January was caused by the onset of a pneumococcal meningitis, then it must be admitted that the disease ran its course for six days before M. and B. 693 was tried, and possibly this delay accounted for the inefficacy of the drug in this case. (McAlpine, Thomas 1939).



Case 2A

Figure 2. For explanation of chart, see Figure 1.

Solid black bar shows time during which M and B 693 was given. Figures above bar indicate dose in grammes.

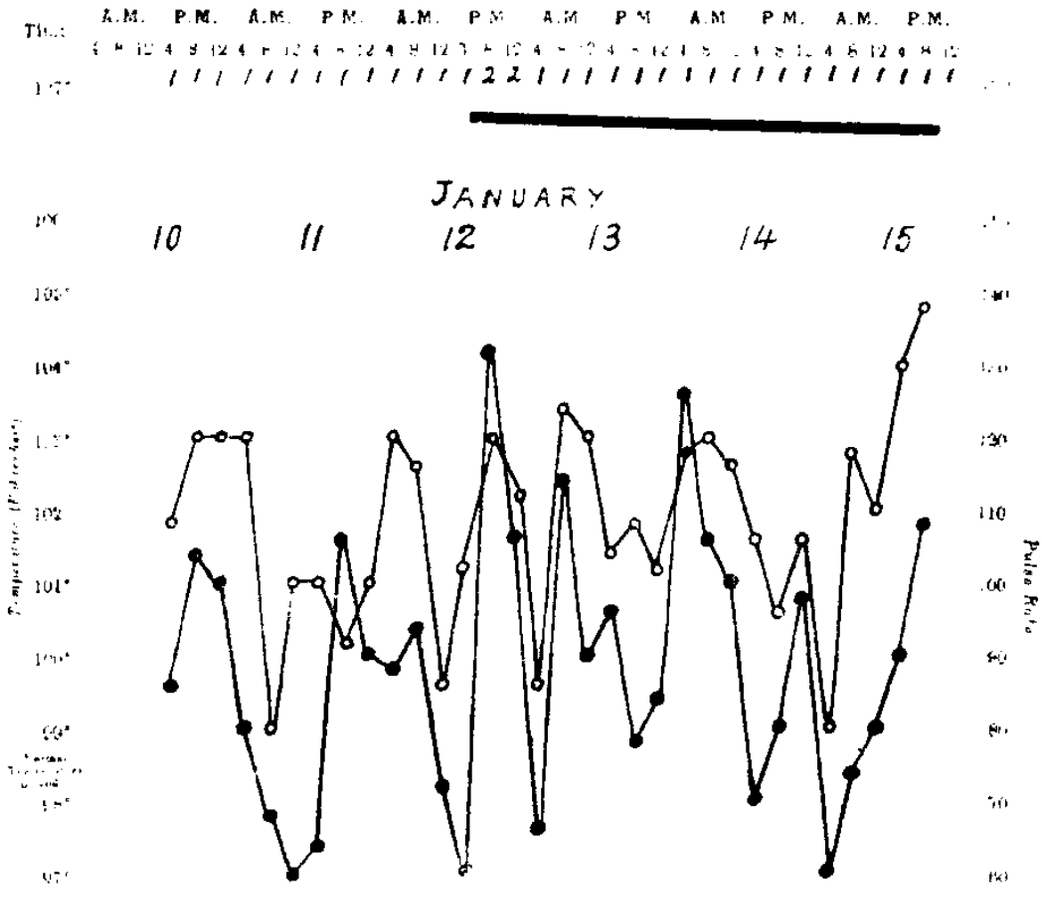
At autopsy the vertex and base of the brain were coated with thick greenish yellow pus. No haemorrhages were noted. The heart, lungs, and upper respiratory passages were normal naked eye, but the liver showed numerous small rounded foci of necrosis. The spleen was enlarged and hard, but a blood examination on 5.1.39. had shown no malarial parasites. Pneumococci were obtained from preparations made from brain pus at autopsy and were found to be Type II.

She received in all 136 grms. of streptocide by mouth and 5 c.c. of a 2½ solution intramuscularly. She was given 18 grms. of M. and B. 693 from 9.1.39. to 11.1.39., 1 grm. being given 4 hourly day and night. The drug caused slight cyanosis and nausea but no vomiting. Her charts show clearly how well she responded to the treatment of her initial infection presumed to be meningococcal, how sudden the onset of her second infection was and how signally treatment failed to influence it.

CASE 3A.

Yeung Ah Ying, female, 14. The child was admitted on the 10th of January 1939 and was lumbar punctured within half an hour of admission.

Her mother stated that she had been taken ill at 5.0 a.m. on the 9th with a rigor, fever and vomiting. Headache was slight at first but increased during the day, and in the afternoon of the 9th the child's neck became stiff. She complained of deafness and head movements and walking were found to be painful. Her mother said she had had a cold for five days preceding the onset of the illness and that her throat had been sore for five days. The three other children at home were all well.



Case 3A

Figure 3. For explanation of chart, see Figure 1.

On admission the child showed head retraction, nuchal rigidity and tenderness and a positive Kernig's sign on both sides. No tâche was elicited. Fundi and tympana were normal. The fauces were injected, but a throat swab yielded negative results. Examination of the central nervous system showed double extensor plantar responses and exaggerated knee jerks.

The cerebrospinal fluid on admission was turbid and yellow in colour, but its pressure was only 160. 20 c.c. were drained and 20 c.c. of anti-meningococcal serum were given intrathecally as well as 9 c.c. intravenously.

Treatment with streptocide 1 grm. 6 hourly and intrathecal anti-meningococcal serum daily was continued on the 11th and 12th, and when pneumococci were reported on the 12th, M. and B. 693 was at once substituted for streptocide and serum treatment was discontinued. This means that M. and B. 693 was exhibited 87 hours after the onset of the disease. The drug was given 4 hourly, 2 grms. being given for the first two doses and 1 grm. 4 hourly throughout the remainder of the disease.

The cerebrospinal fluid became daily more turbid during treatment with M. and B. 693, of which the child received in all 21 grms. in just over three days. The drug exercised not the slightest influence on the course of the disease or on the presence of pneumococci in the cerebrospinal fluid. Pneumococci were reported in the fluid on the 12th, 13th, 14th and 15th, despite the fact that relatively heavy doses of M. and B. 693 were begun late on the 12th. The organisms in this case were Type II.

The child complained neither of nausea nor vomiting during her treatment with M. and B. 693 so that the drug was obviously retained. If the drug is capable of curing pneumococcal meningitis which has been established for 72 hours before treatment is begun there is no a priori reason why it should not do so after 87 hours. The child's condition was moderately good on the 12th, her heart and her lungs showed no gross abnormalities throughout and she was not profoundly toxaemic when M. and B. 693 was first exhibited.

She received in all 23 grms. of streptocide and 21 grms. of M. and B. 693 by mouth, 63 c.c. of anti-meningococcal serum intrathecally and 9 c.c. of anti-meningococcal serum intravenously. She was lumbar punctured in all six times.

CASE 4A.

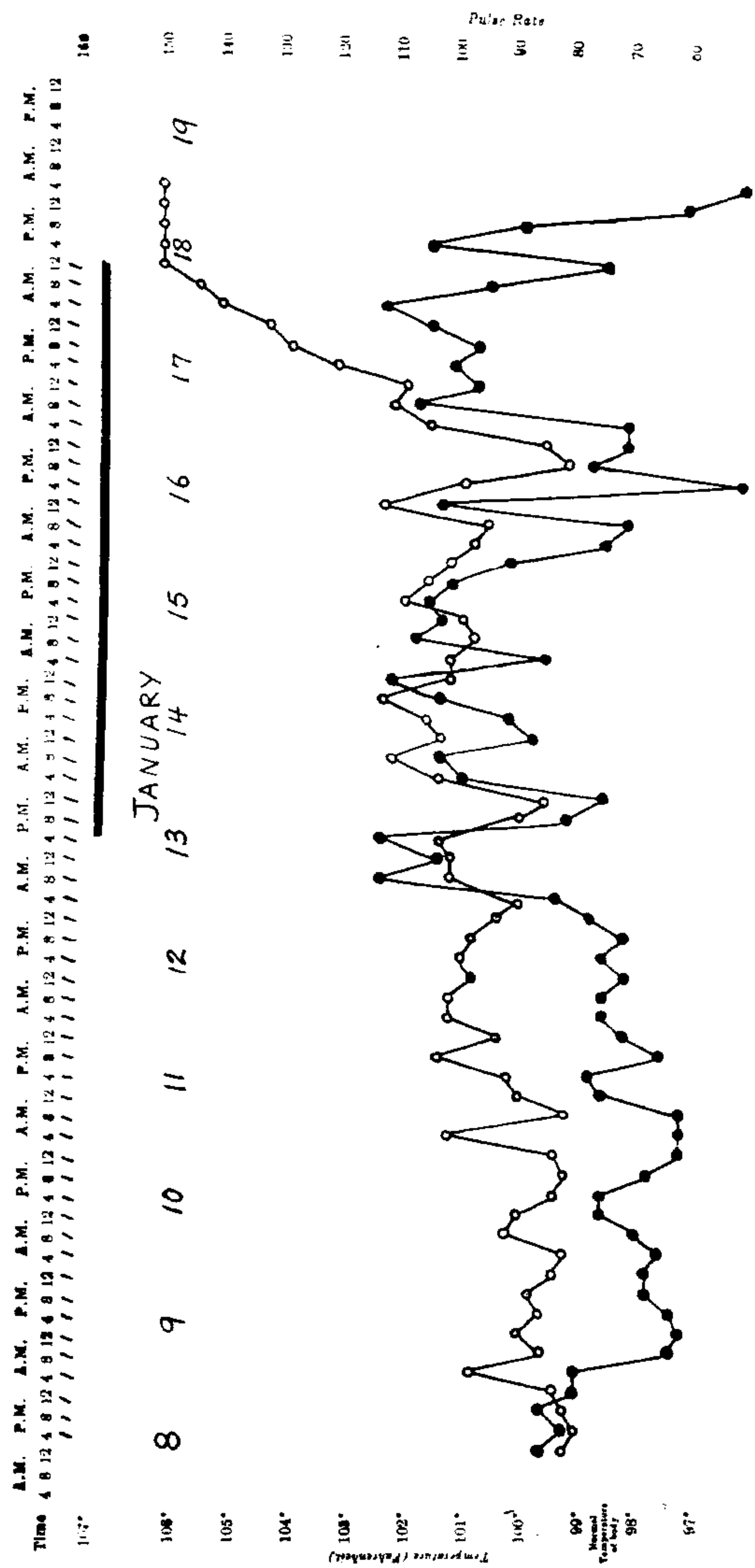
Wong Lin Tai, female, 11. The child was admitted on the 8th of January 1939 and the following history was given by the parents. Her illness began on January 5th with headache and severe vomiting. She had no rigor and was not noticed to be feverish. Her neck became stiff on the third day of the illness. There was no involvement of eyes, skin or throat nor had she had a cough.

On admission her temperature was 99.4°, pulse 80 and respirations 28. She lay in the dorsal decubitus and showed slight head retraction and nuchal tenderness, cervical "soldering" and a bilateral positive Kernig's sign. Labial herpes was noted at the left angle of the mouth. Tâche was not elicited. The cranial nerves and deep reflexes were normal, and the fundi clear. The left tympanum was normal, the right ear contained wax. Heart and lung sounds were normal and the throat was not injected.

Lumbar puncture on the 8th yielded whitish-yellow turbid cerebrospinal fluid. 35 c.c. were drained and 25 c.c. of anti-meningococcal serum given intrathecally. Meningococci were not demonstrated in the fluid, but the case was held to be clinically meningococcal and the child was nursed in an empty upstairs ward to which case 3A was admitted two days later. She responded well to combined therapy with streptocide and anti-meningococcal serum and by the 12th her cerebrospinal fluid was clear and she was clamouring to be allowed to go home.

On the 13th her temperature suddenly rose to 102.2°, a secondary infection was suspected and treatment with M. and B. 693 was begun at 1.0 p.m. The child was given 2 grms. at 1.0 p.m., a further 2 grms. at 5.0 p.m. and from then on 1 grm. 4 hourly. She had in all 32 grms. of the drug in five days, and it did absolutely nothing to check the course of the disease or sterilise the cerebrospinal fluid. On the 12th her fluid was colourless and clear, it showed no increase of cells and no meningococci or other organisms. Globulin was slightly increased. On the 13th pneumococci and pus cells were reported in the cerebrospinal fluid and pneumococci were found daily in the cerebrospinal fluid until the child's death on the 19th.

There seems little doubt that in this case the child developed her pneumococcal meningitis while in hospital. She was admitted as a case of meningococcal meningitis and responded well to treatment. Patients suffering from pneumococcal meningitis do not sit up in bed three days after admission asking to go home and saying there



Case 4A

Figure 4. For explanation of chart, see Figures 1 & 2.

The doses of M & B 693 given at 1.0 p.m. and 5.0 p.m. on 13th January should read 2 gms. and not 1.

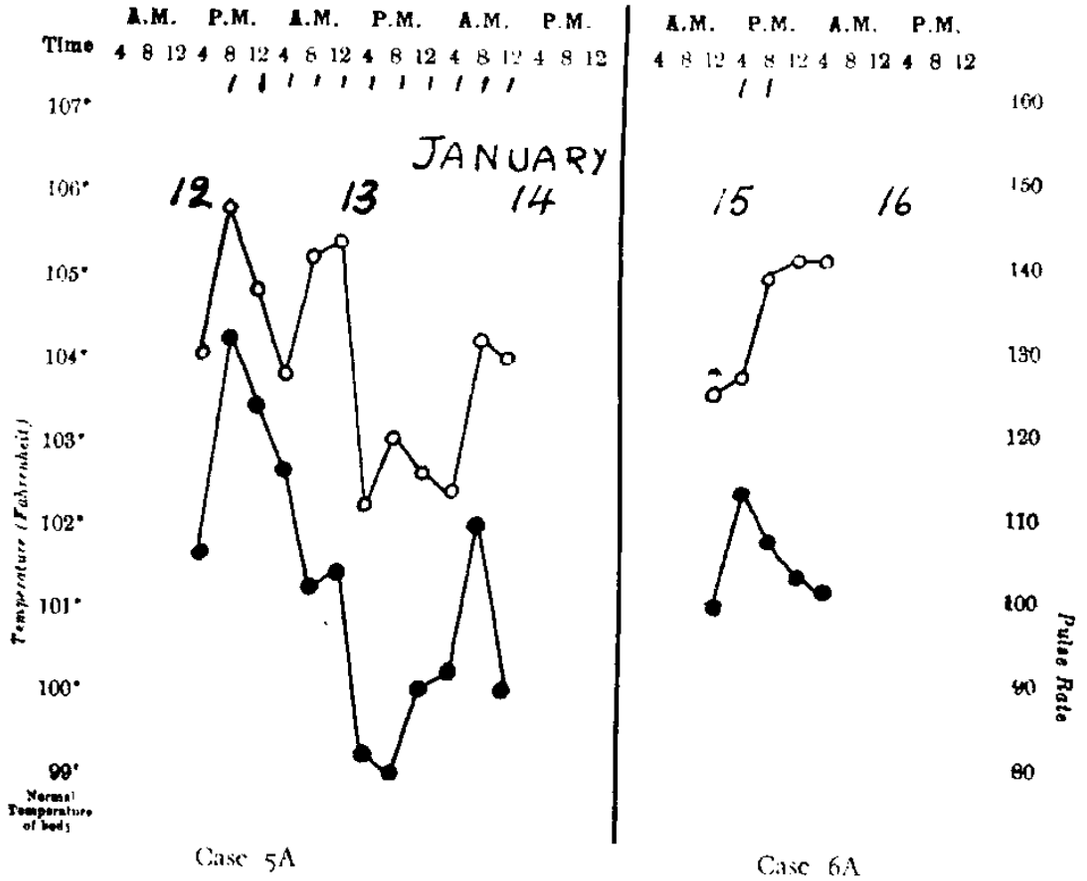
is nothing the matter with them: children suffering from proved meningococcal meningitis frequently do if they have been treated adequately. M. and B. 693 was exhibited within eight hours of the onset of this secondary and fatal pneumococcal infection, and it was conspicuously useless in aborting it. She neither vomited nor became cyanosed while taking M. and B. 693, nor did she show any heart or lung signs throughout the illness.

At autopsy the vertex and base of the brain were coated with thick yellowish green pus, swabs of which yielded pneumococci, Type II, on culture. The upper respiratory passages, lungs, heart and abdominal viscera showed no gross naked eye changes at postmortem examination.

CASE 5A.

Lee Hiu Chi, male, 30. This man was admitted unconscious on the 12th of January 1939, with a history of having had a rigor a fortnight ago and continuous fever since then. His temperature on admission was 101.4°, his pulse 128 and his respirations 26. His lips were slightly cyanosed, and he showed head retraction and nuchal tenderness on pressure. Kernig's sign was positive on both sides, but no herpes or petechiae were noted. It was impossible to obtain a view of the man's throat. The pupils were unequal, irregular and fixed to direct light, the knee jerks were exaggerated and he showed both patellar and ankle clonus. The fundi and tympana were normal.

His heart sounds were clear and the heart was not enlarged. There were signs of oedema at both lung bases. He was lumbar punctured under evipan and turbid greenish-yellow fluid under a pressure of 210 was obtained. 45 c.c. were drained and 30 c.c. of anti-meningococcal serum were given intrathecally. Streptocide 1 grm. 4 hourly was ordered by mouth. On the following day his fever abated to 99° but he was still delirious and needed restraint. His pulmonary and other signs were unchanged. The turbidity of his fluid increased steadily and he died on the 14th. Pneumococci were reported in the fluid on the 13th and 14th, but the quantity of M. and B. 693 available was not enough to allow the drug to be tried in this case.



Case 5A Case 6A

Figure 5. For explanation of chart, see figures 1 & 2.

At autopsy the vertex and base of the brain were sheeted with creamy yellow pus, and Type II pneumococci were isolated from swabs taken at autopsy. His lungs showed basal oedema and congestion but no consolidation. During his forty-four hours in hospital he had 11 grms. of streptocide and 70 c.c. of anti-meningococcal serum. He was lumbar punctured three times only.

CASE 6A.

Chan Yan Cheung, male, 60. This man was admitted unconscious on the 15th of January 1939, and no history was obtainable, though he is said to have been moribund on admission. Unfortunately no detailed notes exist about his condition on admission or his lung signs, but lumbar puncture which was performed on admission yielded turbid purulent fluid containing pneumococci. He died 20 hours after admission.

At autopsy the vertex and base of the brain were covered with greenish-yellow pus and the lateral ventricles were found to contain purulent fluid. The left ventricle of the heart was dilated and the myocardium was soft, but no valvular lesions were present. The upper lobe of the right lung was in a state of red hepatisation and its pleural surface was covered with fibrino-purulent exudate. Excised portions of this lobe sank in water. Swabs from brain and lung yielded Type I pneumococci on culture.

In considering the cases in Group B, it is most interesting to deal with them, as far as possible, in the chronological order in which they developed their secondary infection. In cases 1B, 2B, 3B, 4B, 5B, 6B, and 7B the charts show clearly how sudden the onset of this secondary infection was. In the remaining cases in the group, it is difficult, if not impossible, to fix the exact time at which the pneumococcal infection supervened.

GROUP B. CASE HISTORIES.

MENINGOCOCCAL COMPLICATED BY INTERCURRENT PNEUMOCOCCAL MENINGITIS.

CASE 1B.

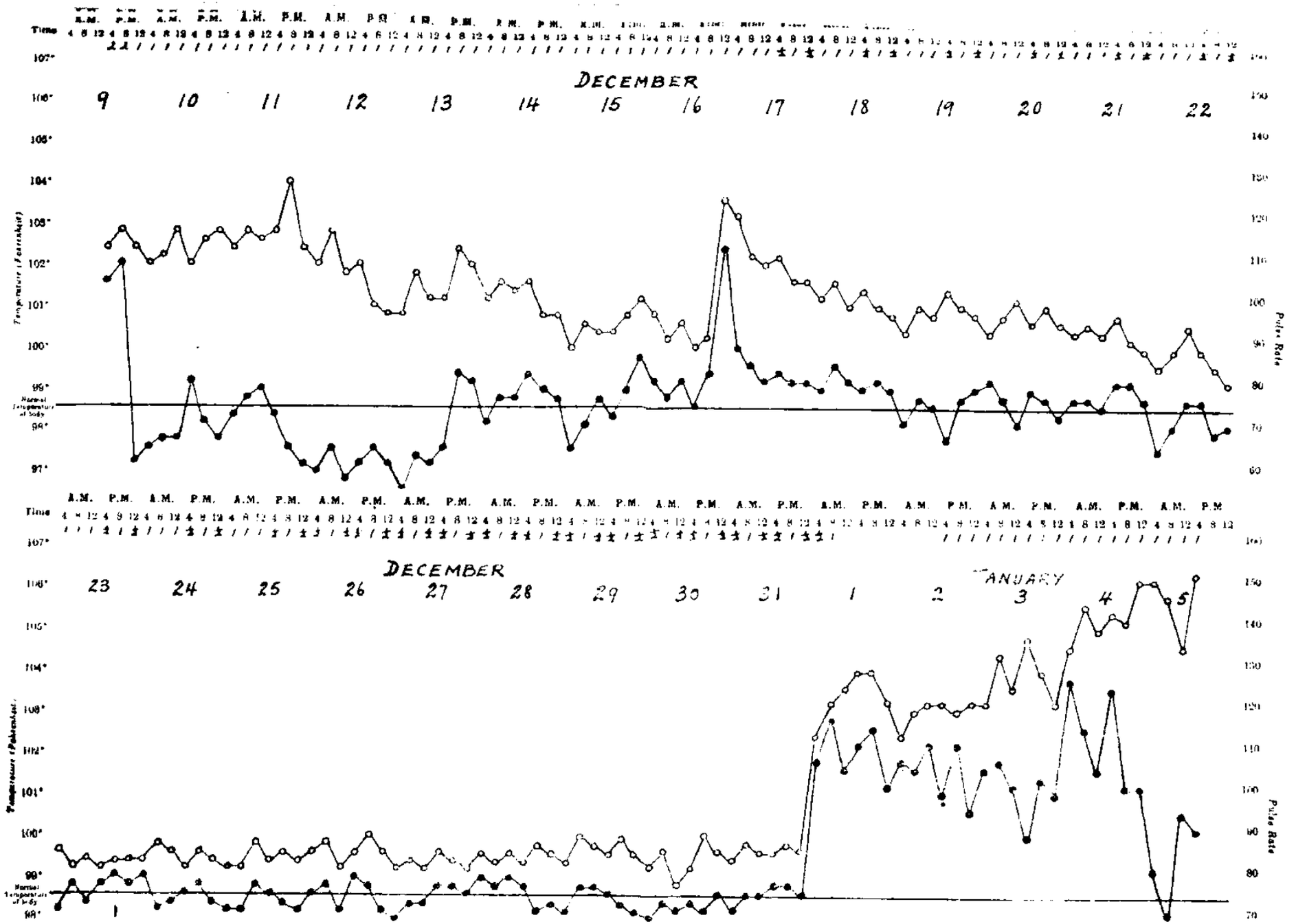
Ma Po King, female, 10.—The child was admitted on the 9th of December 1938 with a temperature of 101.8°, pulse 112 and respiration 22. She was very restless and delirious.

Her mother stated that her illness had begun two days before on the 7th, with a rigor, fever, vomiting, nausea and frontal headache. She also complained of aching in the limbs and deafness of both ears. She had three attacks of diarrhoea on the first day of the illness. Stiff neck was complained of on the third day, but the child had had no petechiae, herpes or photophobia.

On examination head retraction and nuchal tenderness were marked, and cervical "soldering" and tâche were readily demonstrable. Kernig's sign was positive on both sides and the pupils were unequal and sluggish to light. The knee and ankle jerks were absent and the child was doubly incontinent. The heart and lung sounds were normal.

Lumbar puncture yielded turbid yellow fluid under a pressure of 300+ Meningococci were found in smears. 40 c.c. of fluid were drained, 23 c.c. of anti-meningococcal serum were given intrathecally and 18 c.c. intramuscularly. 10 c.c. 2½% streptocide solution were injected intramuscularly and 2 grms. of streptocide given by mouth.

Treatment with serum and streptocide 1 grm. 4 hourly was continued and although the child had to be tube fed for the first three days, she slowly improved and by the 13th could recognise people. She had a mild serum reaction on the 17th, but her cerebrospinal fluid was clear by then and she was afebrile. By the 26th she was able to walk, though stiffly, and an uneventful convalescence seemed assured.



Case 1B

Figure 6. For explanation of chart, see Figure 1.

On the 31st her cerebrospinal fluid was normal in all respects, and she was free from symptoms. Early on the 1st of January her temperature rose to 101.6°, and lumbar puncture on the 2nd yielded 70 c.c. of turbid cerebrospinal fluid. No organisms were noted however until the 4th when pneumococci were reported. Meanwhile she had been treated unavailingly as a meningococcal relapse with intrathecal anti meningococcal serum and streptocide. No M. and B. 603 was to be procured at this time in Hong Kong and the child perished of pneumococcal meningitis on January 5th. At no time during her terminal illness did she show any lung or heart signs nor was her throat affected.

An autopsy was made on the 6th and the vertex and base of the brain were found sheeted with thick creamy yellow yellow pus. Preparations made from this pus yielded Type II pneumococci on culture. The upper respiratory passages, lungs heart, and abdominal viscera presented no noteworthy changes on naked eye examination.

This child was the first patient to develop a secondary and fatal infection, ultimately proved to be pneumococcal, while recovering from meningococcal meningitis. She had been nursed throughout in the large ward downstairs and was thought to have recovered from her meningococcal meningitis. Two other children in the same ward, cases 2B and 3B, developed fever on the same day. Her charts show the dosage of the various drugs she received, and her case makes it abundantly clear that streptocide has little or no effect on Type II pneumococci.

CASE 2B.

Poon Ho, female, 9. This child was admitted to hospital on September 10th 1938, after an illness which had lasted eight days. Her aunt said the child had had a rigor, fever and headache at onset followed immediately by stiff neck. She also vomited "continuously" for four days, and showed generalised hyperaesthesia. Neither skin, ears, nor eyes were involved nor had she been delirious. The onset was not preceded by coryza, cough or sore throat.

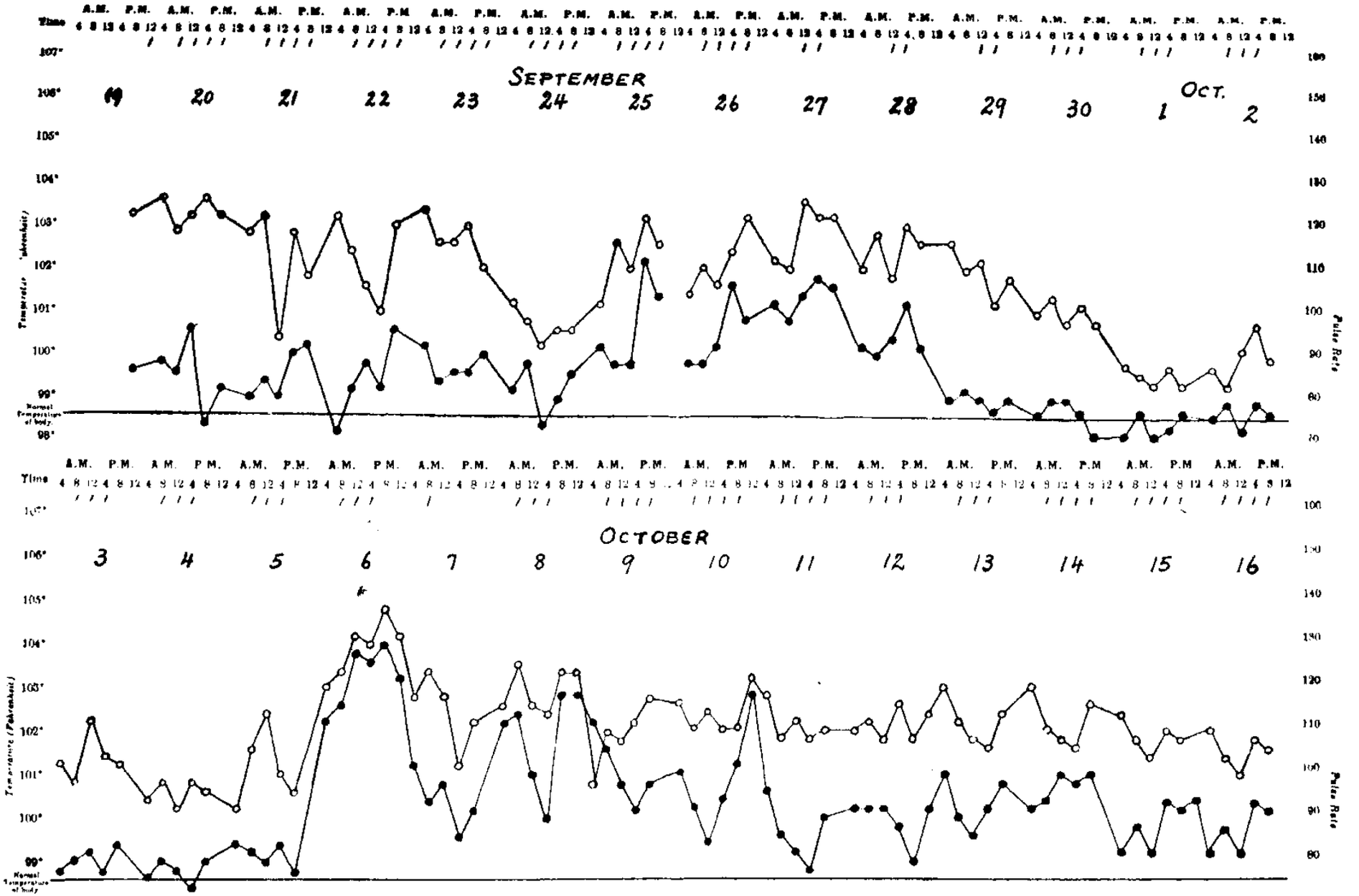
On admission her temperature was 99.4°, pulse 103 and respiration 28. She was flushed and lay in the lateral decubitus. Head retraction and nuchal tenderness and rigidity were marked and tâche was readily elicited. The belly was scaphoid and enlarged mesenteric glands were palpable, but liver and spleen could not be felt. The heart and lung sounds were normal. Kernig's and Brudzinski's signs were positive on both sides and the knee and ankle jerks were absent. The pupils were semi-dilated and fixed to light, but the fundi and tympana were normal.

Lumbar puncture under ether yielded greyish-yellow turbid fluid under a pressure of 125. Queckenstedt's sign was positive. 35 c.c. of fluid were drained and meningococci were found in smears. 25 c.c. of anti-meningococcal serum were given intrathecally and 13 c.c. intramuscularly; 1 grm. of streptocide was given by mouth on the evening of admission and thence forward the drug was given in a dose of 1 grm. 4 hourly throughout the day. Serum was continued intrathecally for seven days after admission and the child's temperature was normal and her fluid clear by the 29th.

She relapsed on the 6th of October, her temperature rising from normal to 102°, and her streptocide which had been reduced to 3 grms. daily on the 30th was increased to 4 grms. again on the 9th. Her disease ran a protracted course and by the fifth week it was evident that the child was developing an internal hydrocephalus. She emaciated rapidly, lost interest in her surroundings, never spoke except when spoken to and lay motionless hour after hour gazing at the ceiling. Her skin became dry and harsh and the only thing which roused her from her lethargy was the arrival of a meal, for she ate ravenously throughout.

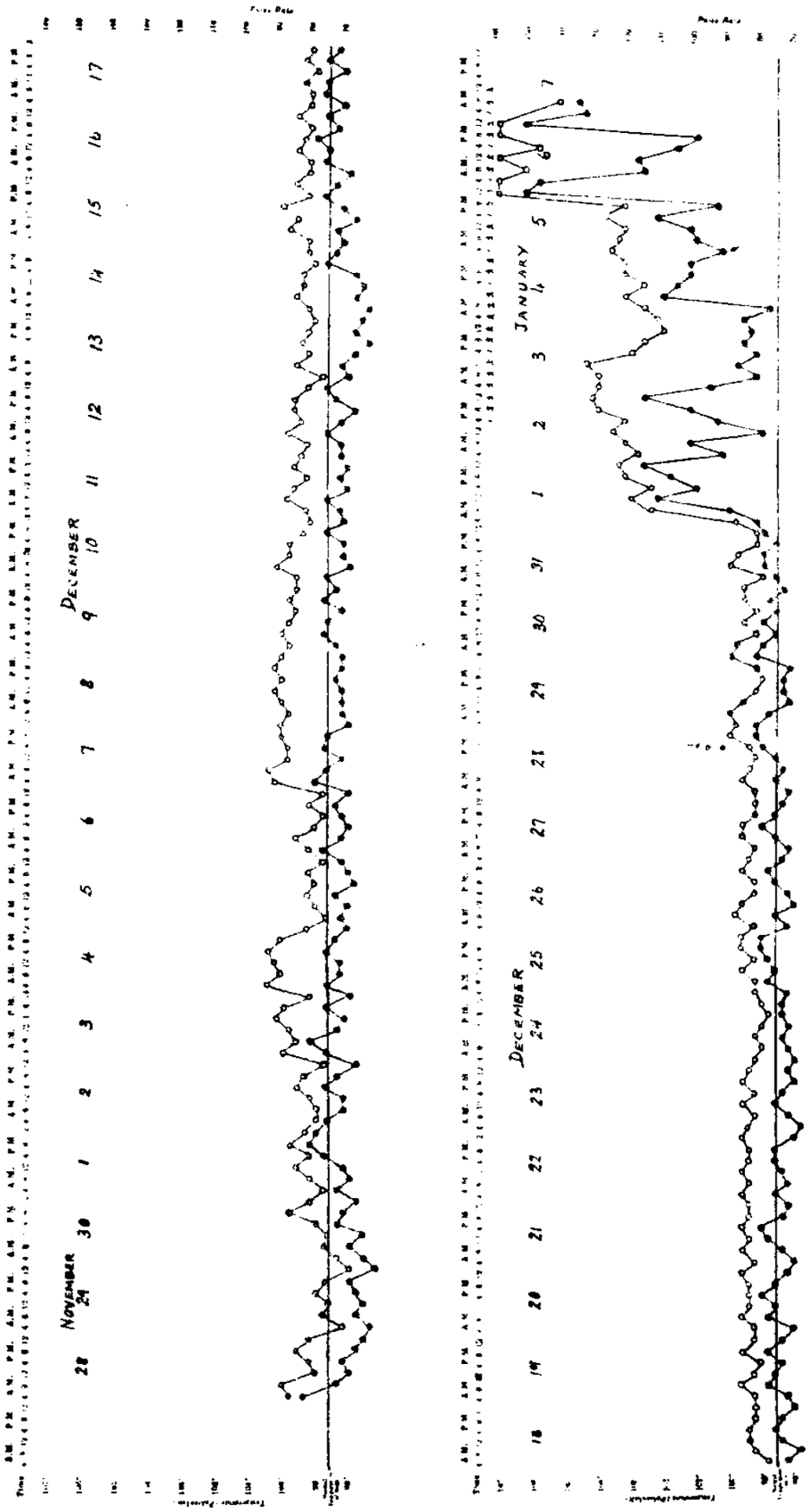
She was by now afebrile and at the beginning of November she began to put on weight and even became able to walk again. Streptocide was discontinued entirely on the 14th of November and throughout December the child's condition improved steadily. It was noted however, that her manner had become a little facile and that some mental deterioration had occurred.

She had two slight rises of temperature in December, one to 99.2° on the 1st, the second to 99.8° on the 28th, and these "spikes" suggested that the original meningococcal infection had not been completely eradicated. Her cerebrospinal fluid



Case 2B

Figure 7. For explanation of chart, see Figure 1.



Case 2b

Figure 7. For explanation of chart, see Figure 1.

was clear and colourless by this time, and showed no changes beyond a slight increase in globulin and the presence of a few mononuclear cells.

On the 29th of December she complained of "backache" and her temperature rose to 99.8°. These symptoms abated and cerebrospinal fluid obtained on the 31st of December was clear colourless and organism free. It showed "an increase of globulin and the presence of mononuclear cells."

The child developed fever on the 1st of January and by noon her temperature was 102°. The cerebrospinal fluid was turbid and yellow and remained so despite treatment which was, not unnaturally, directed against a supposed meningococcal relapse.

Pneumococci were reported in the cerebrospinal fluid on January 5th, and no further anti-meningococcal serum was given after that date. Streptocide was not pushed, as it appears to have little or no effect on pneumococcal infections and the child perished early on the 7th of January. She had shown no evidence of infection of lungs, ears, throat or peritoneum throughout the terminal phase of her illness and at autopsy the only noteworthy changes found were in the nervous system.

The vertex and base of the brain were sheeted with creamy yellow pus and a marked degree of internal hydrocephalus was found on both sides of the brain. Both lateral ventricles contained a considerable quantity of purulent fluid, and swabs made from this fluid and the pus sheeting the brain yielded a growth of Group IV pneumococci. The upper respiratory passages, lungs, heart and abdominal viscera showed no noteworthy naked eye changes.

Specific anti-pneumococcal therapy with M. and B. 693 could not be attempted in this case as the drug was not available but the prognosis, even from the meningococcal point of view, was considered poor as there was evidence to show that the original meningococcal ependymitis had never been effectively dealt with. The post-mortem findings tended to show that this view was correct, as an internal hydrocephalus had obviously been present for some weeks.

CASE 3B.

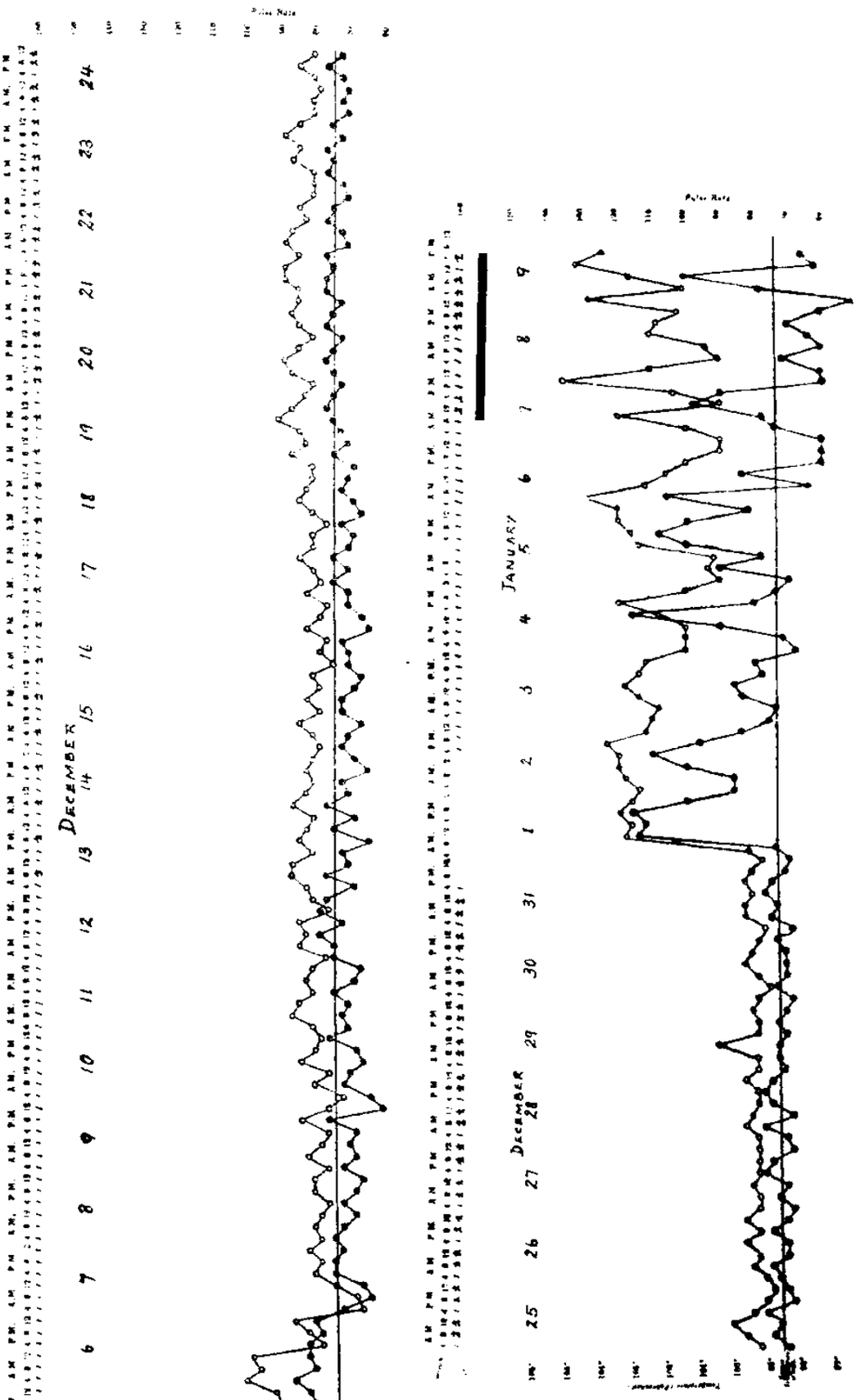
Cheung Luk Mui, female, 13. This child is perhaps the most interesting of the cases who developed a proved pneumococcal meningitis in the course of recovery from proved meningococcal meningitis.

She was admitted on the evening of December 4th 1938, and her grandmother gave the following history. The child had been suddenly taken ill on the 1st of December with a rigor, fever, headache and vomiting. She did not complain of a stiff neck nor was head retraction noted. She was delirious at night after the first two days of her illness, but at no time had she had symptoms referable to eyes or ears, or petechiae or herpes.

On admission her temperature was 99.6°, pulse 100 and respirations 22. Her head was markedly retracted and nuchal and spinal tenderness were demonstrable. Cervical "soldering" and generalised hyperaesthesia were obvious and Kernig's sign was positive on both sides.

The fundi and tympana were normal, the left plantar response was extensor and the knee jerks were diminished. The pupils were irregular and eccentric but they reacted to light. The throat was clear and the heart and lung sounds normal.

Lumbar puncture performed on December 5th yielded greenish yellow turbid fluid under a pressure of 300. 20 c.c. were drained but no serum was given. Meningococci were found in smears made from the fluid. The child was given 2 grms. of streptocide by mouth on admission and 2 grms. four hours later. From then on she was given 1 grm. four hourly day and night but no further punctures were made, no serum was given, and it was decided to treat the case without anti-meningococcal serum or daily lumbar punctures. The initial dose of streptocide was maintained until the 6th, by which time she was afebrile and free from symptoms though she had developed labial herpes on the 6th. It was then reduced to 5 grms. in the 24 hours. On the 20th by which time she was up and running about, her streptocide was reduced to 4 grms. every 24 hours. On the 23rd a second lumbar



Case 3B

Figure 8. For explanation of chart, see Figures 1 & 2.

puncture was made and the cerebrospinal fluid was found to be clear and colourless. It showed neither increase of cells nor of globulin, nor were any organisms detected in it, and the manometric reading was 150.

She remained free from symptoms until the 31st of December when she complained of headache, nausea and vomiting. She had no fever nor signs of meningitis, and these symptoms were tentatively ascribed to overdosage with streptocide. The dose was reduced from 4 to 3 grms. every 24 hours, and lumbar puncture was immediately performed. The fluid was clear and colourless and showed only a few mononuclear cells.

On the 1st of January her fever rose to 102.6° and lumbar puncture on the 2nd yielded a highly turbid fluid which contained pneumococci. Pneumococci were not actually proved and reported to be present until some days later, and in the meantime it was held, not unreasonably, that the condition was a meningococcal relapse and it was treated as such, 86 c.c. of anti-meningococcal serum being given intrathecally during the next four days, together with 1 grm. of streptocide by mouth every four hours. These measures availed nothing.

M. and B. 693 arrived in the colony on the 7th of January and treatment with it was begun at once. 2 grms. were given by mouth at 11.0 a.m. and this dose was repeated 4 hours later, 1 grm. every four hours being given by mouth from then onwards. She received in all 14.5 grms. of the drug in two days, and beyond making her cyanosed and nauseated the drug had no action at all. She vomited only once on the 8th and there is no question that most of the M. and B. 693 was retained. Pneumococci remained present in the cerebrospinal fluid to the end, and the child died on the 10th, her cerebrospinal fluid during the last three days of life having been creamy yellow pus. No definite signs of pulmonary or cardiac involvement were made out during the course of the disease, nor was the throat affected.

At autopsy both the vertex and the base of the brain were coated with thick yellow pus, the cortical vessels were engorged and outlined with pus, and the lateral ventricles contained purulent fluid. There was no internal hydrocephalus. The upper respiratory passages, lungs and heart showed no gross naked eye changes and the abdominal viscera were unremarkable. Swabs from the brain pus yielded a culture of Group IV pneumococci.

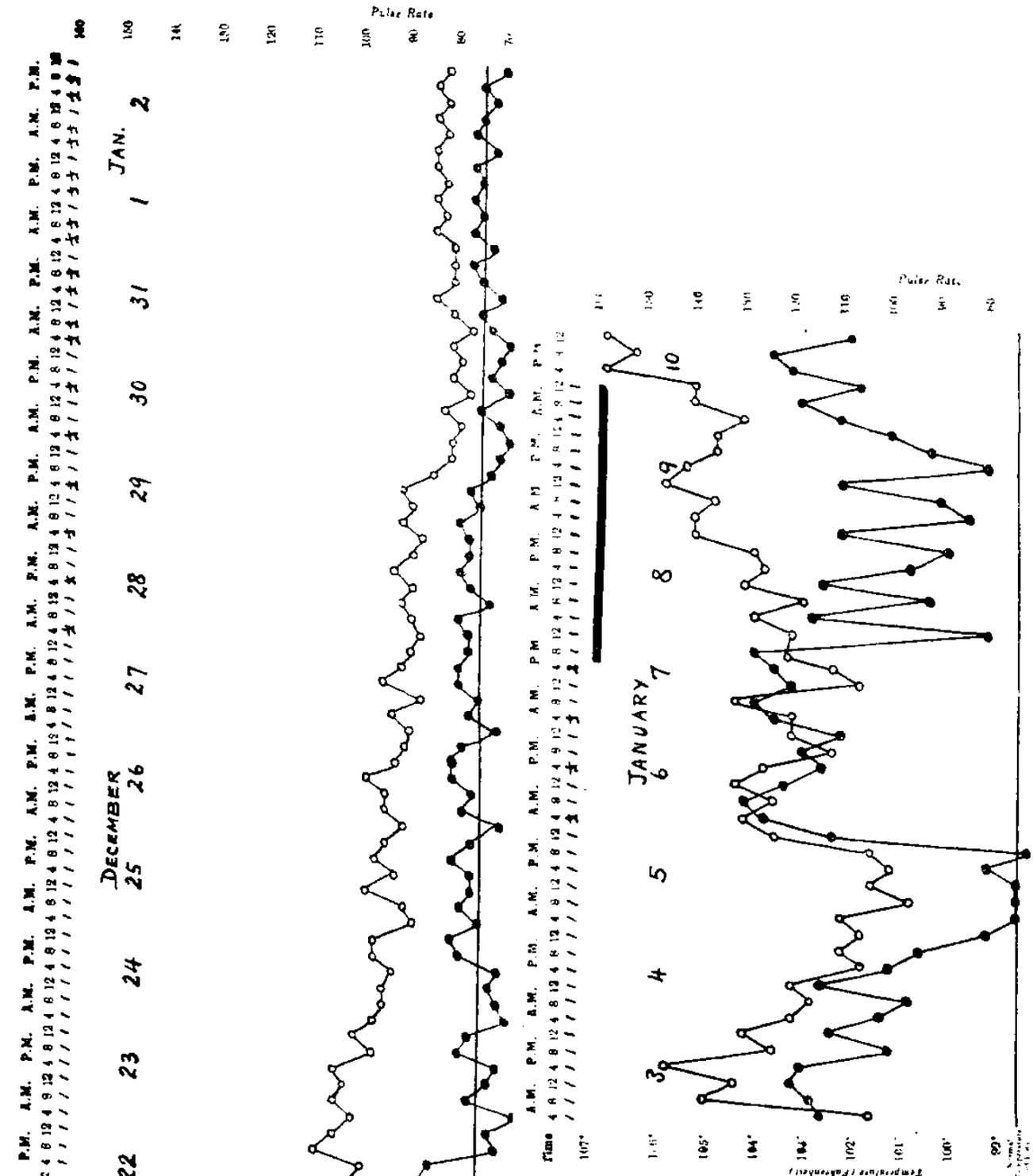
This child was the third patient to develop a pneumococcal meningitis in the hospital, and it will be noted that the initial symptoms of this secondary infection probably appeared on the 31st of December. M. and B. 693 was not obtainable here until January the 7th and assuming that her pneumococcal meningitis began on January 1st she went untreated with M. and B. 693 for six and a half days. This delay may account for the complete failure of the drug to influence the course of the disease, but if this be so then it must be admitted that its action in pneumococcal differs very materially from its action in meningococcal meningitis, for in the latter condition it acts just as promptly and potently as streptocide.

CASE 4B.

Fong Chun Kwan, male, 8. Refugee. This child was admitted on the 20th of December, 1938. His mother said he had been taken ill on December 18th at 11.0 a.m. with a rigor, fever, vomiting and frontal headache. Nausea persisted and later in the day his neck became stiff and painful, and he also complained of backache. On the 19th he became semi-conscious and delirious. He had had no skin, eye or ear involvement, nor had there been any antecedent catarrh or sore throat.

On admission the child's temperature was 98.6°, pulse 90 and respiration rate 22. He lay in the gun-hammer attitude and showed slight head retraction and nuchal tenderness. Cervical "soldering" was marked but no tâche was elicited nor was the belly scaphoid.

The pupils were equal and active, the cranial nerves normal. The throat was clear. All the deep reflexes were present and the plantar responses were flexor but Kernig's sign was positive on both sides. There was no photophobia and the lung sounds were normal. The heart was not enlarged but a systolic murmur was heard at the apex. Neither liver nor spleen was palpable.



Case 4B

Figure 9. For explanation of chart, see Figure 1 & 2.

Lumbar puncture yielded turbid fluid, which contained meningococci under a pressure of 180. 40 c.c. were drained and the child was given 5 c.c. of a 2½% solution of streptocide intramuscularly and 1 grm. of streptocide by mouth. No anti-meningococcal serum was given and during his first week in hospital he received 6 grms. of streptocide by mouth every 24 hours. At the end of this time he was able to sit up although he was markedly prostrated as a result of this intensive treatment with streptocide. By the 30th his cerebrospinal fluid was clear and on the 2nd of January he was playing with his toys, and had been afebrile for a week.

On the 3rd of January his temperature rose suddenly to 102.4° and his fluid was found to be turbid again. A meningococcal relapse was feared and the child was given anti-meningococcal serum intrathecally for the next three days and his dose of streptocide was raised from 4 to 5 grms. a day again.

Gram positive diplococci were reported in his cerebrospinal fluid on the 5th and treatment with M. and B. 693 was begun at 11.0 a.m. on the 7th, that is to say 107 hours after he had presumably developed pneumococcal meningitis. 2 grms. of the drug were given by mouth as an initial dose, and thereafter 1 grm. was given 4 hourly. In all, he received 20 grms. of M. and B. 693 in three days. Pneumococci were reported in his fluid on the 7th, 8th, 9th and 10th, and they were also found in preparations made at autopsy from brain pus and lateral ventricle fluid. On grouping they were found to be Type II.

The child's condition deteriorated steadily while he was receiving M. and B. 693 and by the 9th he was definitely cyanosed. He did not vomit at any time nor did he show any signs of lung or heart involvement. On the 10th he was unable to swallow, and as he was obviously dying, M. and B. 693 was discontinued at midday.

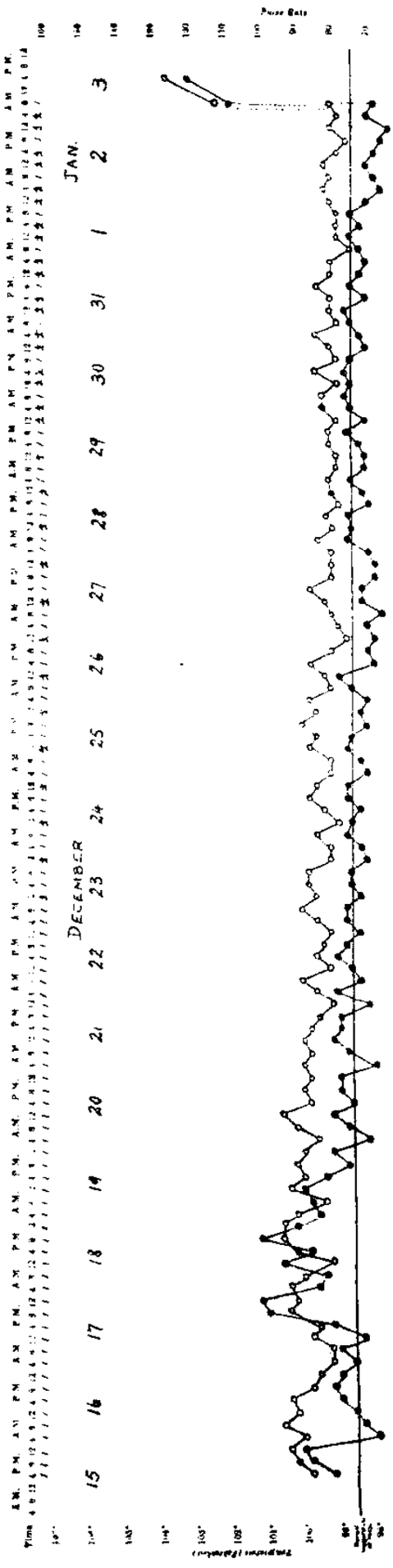
At autopsy the whole of the vertex and base of the brain were found sheeted with thick gelatinous yellow pus. Both lateral ventricles contained turbid purulent fluid, but neither was dilated and Type II pneumococci were grown from this fluid. The upper respiratory passages, lungs, heart and abdominal viscera showed no gross naked eye abnormalities.

While admitting that the drug was, of necessity, given late in the course of the disease, it is only fair to say that its action in aborting this pneumococcal infection was precisely nil.

CASE 5B.

Tan Yun Kin, female, 26. Refugee. This patient was admitted on December 16th, 1938. She said her illness had begun suddenly on the 12th with a rigor, fever, headache and vomiting while she was travelling from Nam Tao to Hong Kong. She had a "cold" for a few days before onset but no sore throat or coryza. Throughout the illness there had been no involvement of eyes, ears or skin, and tenderness of the neck and head retraction only appeared on the morning of admission. She was delivered of a full time living child on the 13th.

On admission her temperature was 99°, pulse rate 86 and repirations 22. She lay curled up on her side and showed slight head retraction and nuchal tenderness. Cervical "soldering" and tâche were demonstrable. The fundi and tympana were normal, but the pupils were small irregular and unequal. There was slight left sided facial weakness and the nasopharynx was congested and contained much mucopus. The left knee jerk and both ankle jerks were absent. Kernig's sign was positive on both sides. The heart and lung sounds were normal. The bladder was distended and had to be catheterised at once. Lumbar puncture yielded highly turbid fluid under a pressure 300. Meningococci were found in it, and 50 c.c. were drained. 20 c.c. of anti-meningococcal serum were given intrathecally, 12 c.c. intravenously and 1 grm. of streptocide was ordered 4 hourly by mouth. She improved rapidly and by the 20th her pain had disappeared and she could move her head freely in all directions. She became afebrile on the 21st and remained free from symptoms throughout the next week, although her cerebrospinal fluid still remained slightly turbid. The turbidity persisted up to the 29th, but on the 2nd of January her fluid was found clear for the first time. She was now getting up and walking about each day and her streptocide had been reduced to 4 grms. in the 24 hours. Beyond a slight blurring of disc edges on both sides, first noted on the 30th, and a mildly positive Kernig's sign on both sides she seemed to have recovered completely.



Case 5B

Figure 10. For explanation of chart, see Figure 1.

On the 3rd of January her temperature rose suddenly to 102°, and she was found sitting up in bed rubbing her gums and crying out because of intense pain in her lower jaw at 10.0 a.m. She was rolling about with pain and salivating freely but no local lesion could be made out to account for this condition. Lumbar puncture yielded a highly turbid fluid under pressure; 52 c.c. were drained and 30 c.c. of anti-meningococcal serum given intrathecally after desensitisation had been carried out. Her meningeal signs had all returned but there were no abnormal findings in the other systems. She died suddenly at midday.

At autopsy both the vertex and the base of the brain were found to be sheeted with greenish yellow pus, and purulent fluid was found in both lateral ventricles although there was no internal hydrocephalus and no change was found to account for the pain in the jaw. The lungs showed a mild degree of basal oedema only, but the upper respiratory passages appeared normal. The heart and abdominal viscera were normal to the naked eye.

Both the last specimen of cerebrospinal fluid and the swabs taken at autopsy yielded Type II pneumococci on culture. No specific anti-pneumococcal treatment was attempted in this case, first because she died so suddenly, second because the necessary drug was not available.

CASE 6B.

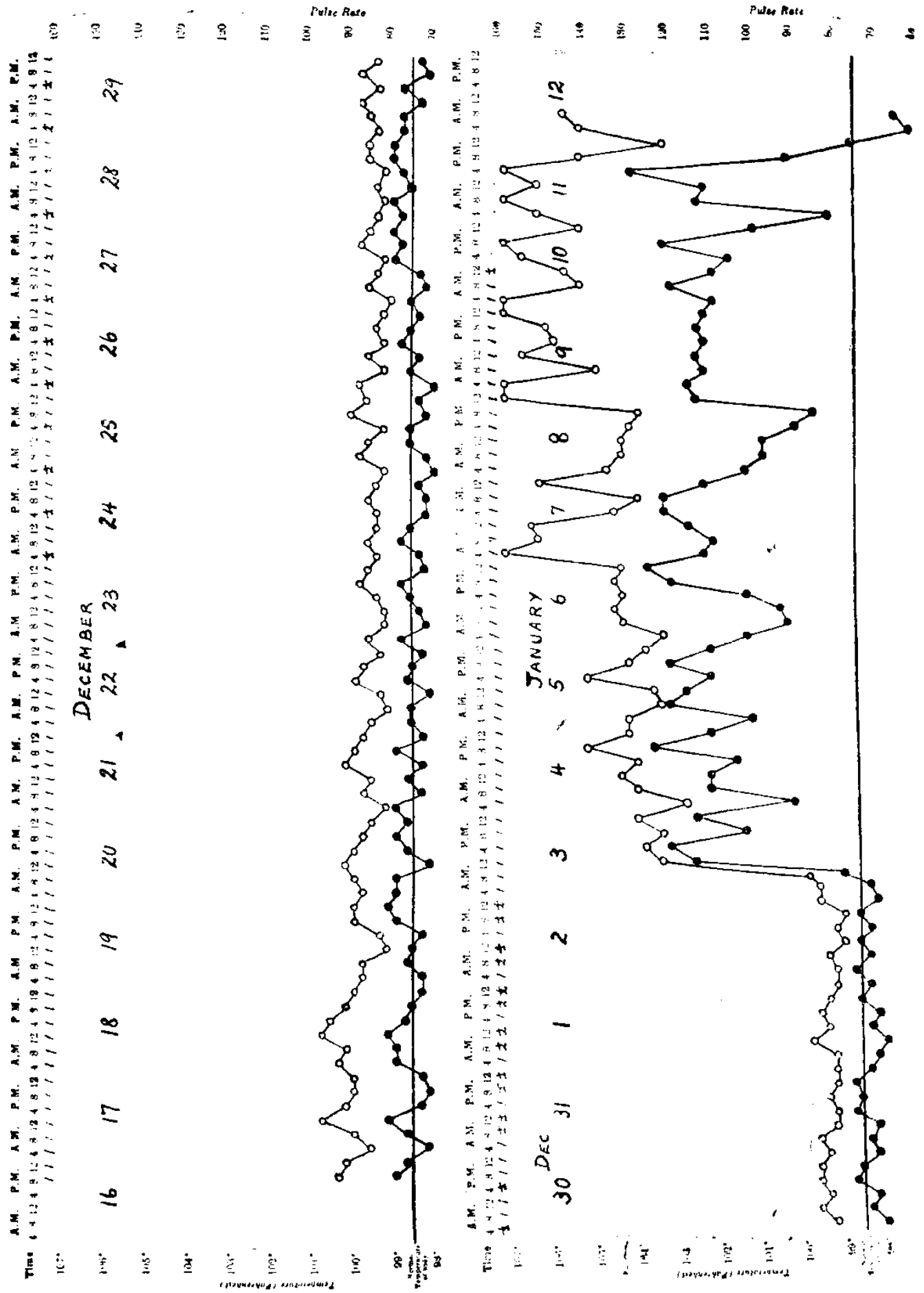
Mak Kin Tai, female, 9. This child was admitted on the 16th of December 1938. She had been living in the Ping Shan Refugee Camp for some weeks before her illness, and although she was conscious and co-operative on admission she was too young to give an accurate history.

Her temperature was 98.8°, pulse 92 and respirations 24 when she reached hospital. She showed a slight degree of cervical "soldering" and nuchal tenderness but no head retraction. Tâche could be elicited and Kernig's sign was positive on both sides. The pupils were unequal and the right eye showed a subacute iritis. The fundi and cranial nerves were normal, but both knee and ankle jerks were absent. The heart and lungs showed no abnormality and the throat was clear.

Lumbar puncture under ether yielded turbid blood-stained fluid, which was not under great pressure but contained meningococci. 40 c.c. were drained and 25 c.c. of anti-meningococcal serum were given intrathecally. 1 grm. of streptocide was ordered 4 hourly by mouth. The child was given intrathecal anti-meningococcal serum on the next two days and 6 grms. of streptocide daily during her first week in hospital. At the end of this time her cerebrospinal fluid was clear and she was afebrile and free from symptoms. She convalesced uneventfully until the 3rd of January.

On January 3rd she complained of headache and her temperature rose to 102.6°. Her meningeal signs reappeared and her cerebrospinal fluid was found to be turbid again. As a meningococcal relapse was feared she was desensitised and her streptocide was increased to 6 grms. in the 24 hours again. On the 4th she was given anti-meningococcal serum intrathecally but developed an "asthmatic" reaction after 12 c.c. had been given, despite desensitisation on the previous day. Her respiration rate rose to 60 and remained between 40 and 60 for the rest of her illness. Adrenalin, atropine and torantil all proved ineffective in overcoming this reaction, and on the 5th her face had become blotchy and a little bloated.

Streptocide was continued in a dose of 1 grm. 4 hourly until the 7th. On the 7th, in view of the fact that there were other cases of pneumococcal meningitis in the hospital and that fine consonating crepitations had been heard at the base of the right lung on the 6th, it was decided to treat the child as a case of pneumococcal meningitis. M. and B. 693 was begun at 11.0 a.m. and 1 grm. of the drug was given 4 hourly from that time until two days before the child died. The dose was reduced to 0.5 grm. 4 hourly on the 10th and the drug was discontinued altogether on the 11th. The child received in all 20 grms. of M. and B. 693. Her cerebrospinal fluid cleared on the 8th but become turbid again on the 9th and remained so to the end. On the 8th fine crepitations were heard at the base of the left lung, but they had disappeared by the 9th. The lung signs were never marked and never dominated the picture.



Case 6B

Figure 11. For explanation of chart, see Figures 1 & 2.

Pneumococci were cultured from the cerebrospinal fluid on the 8th, and remained present in the fluid until death occurred on the 12th. They were also recovered from autopsy preparations and were found to be Type II.

At autopsy the brain was found to be coated with thick yellow pus. The lungs showed nothing noteworthy beyond a mild degree of basal congestion and oedema. They both floated in water as a whole and when sectioned. The heart was normal. The kidneys and liver both showed some fatty degeneration but the other abdominal viscera were normal naked eye.

This child perforce had to wait 85 hours after developing pneumococcal meningitis before receiving M. and B. 693, but even so the drug proved to be singularly ineffective. She is the only case in group B who showed any lung signs at all during the terminal phase of her illness, and autopsy revealed nothing remotely resembling pneumococcal lobar pneumonia. Her blood showed no malarial parasites throughout the illness and a throat swab taken on the 8th was negative for pneumococci.

CASE 7B.

Chan Shan Ho, female, 20. Refugee. This woman was admitted to hospital on December 15th, 1938. She said she had been taken suddenly ill on December 9th, with a rigor, fever, frontal headache and several attacks of vomiting. Headache and nausea persisted and she also suffered from backache and pains in the limbs. Giddiness and constipation were early symptoms, but at no time during her illness had she had any stiffness of neck or limitation of head movements, nor had she complained of her eyes or ears.

She had been doubly incontinent since the second day of the illness but had had no photophobia or skin eruption. The onset was not preceded by coryza, catarrh or sore throat.

She was vaccinated on November 23rd and showed the scars of two apparently primary "takes" on her left arm when admitted.

On arrival at hospital her temperature was 100°, pulse 96 and respiration 20. She lay curled up in the left lateral decubitus and showed slight head retraction. No nuchal tenderness could be made out but tâche was readily elicited. She had a cluster of herpetic vesicles at the right corner of the mouth and a sub-acute right iritis. The left pupil and fundus were normal. The tympana and cranial nerves were normal, but Kernig's sign was positive on both sides and the knee jerks were absent. No abnormality was detected in the heart, lungs or belly.

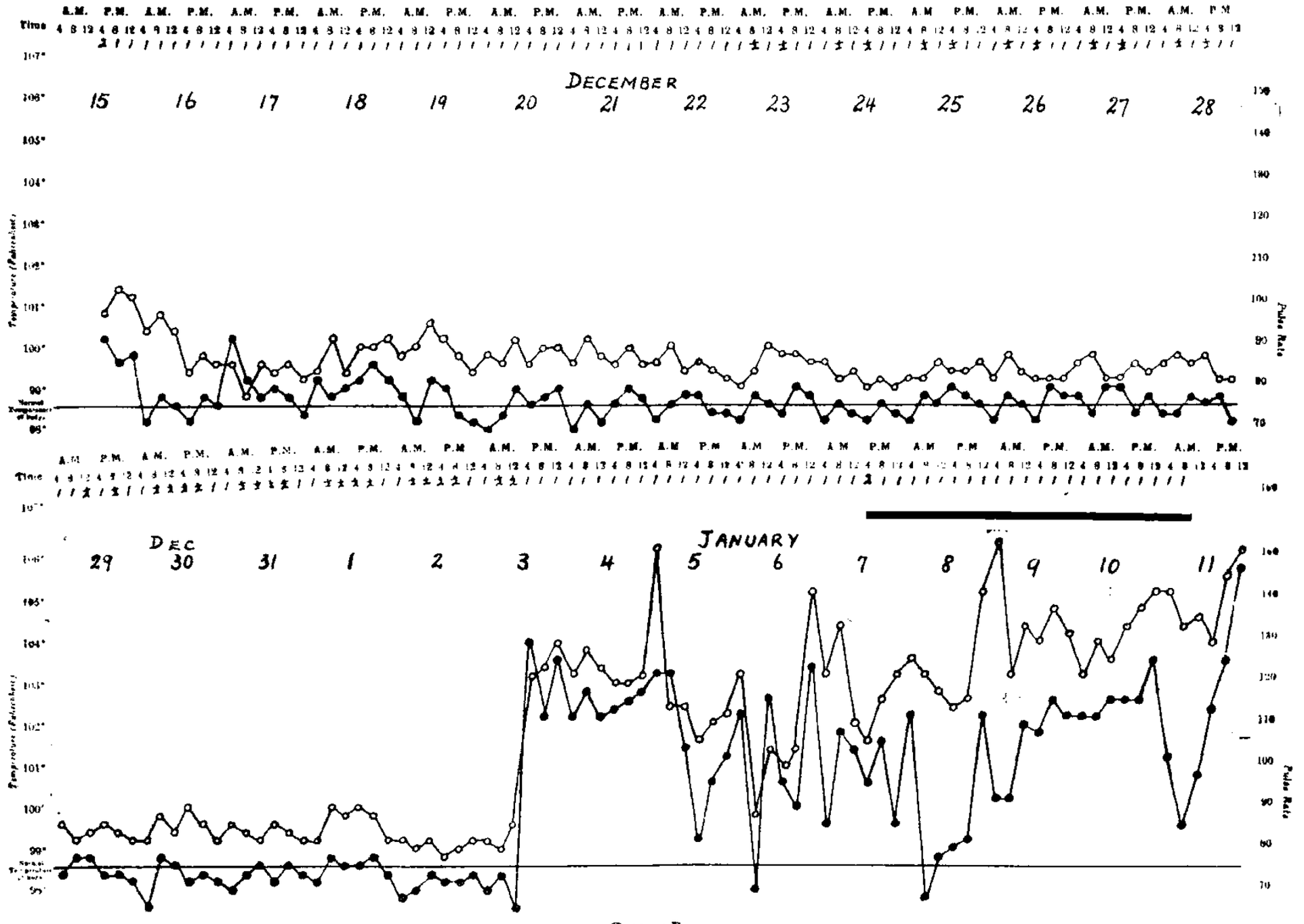
Lumbar puncture yielded a highly turbid fluid under a pressure of 190. 40 c.c. were drained, and meningococci were demonstrated in the fluid. Two grammes of streptocide were given by mouth and 1 grm. 4 hourly was ordered after this initial dose. No serum was given, but the patient was kept on 6 grms. of streptocide daily for the first week in hospital.

By the 23rd she had neither symptoms nor signs, but the cerebrospinal fluid was still a little turbid. By the 27th her fluid was pale yellow and clear and she was afebrile and able to get up each day. The right iritis had cleared up and she was feeling perfectly well and was gaining weight.

On the 2nd of January her cerebrospinal fluid which was clear and colourless, showed only a few mononuclear cells and a slight increase of globulin.

On the 3rd of January she complained of headache and her temperature at 4.0 p.m. was found to be 103.8°. Her streptocide was increased from 4 to 6 grms. in the 24 hours and she was given 10 c.c. of a 2½% solution of the drug intramuscularly. Her cerebrospinal fluid was clear but contained Gram positive cocci. On the 4th her cerebrospinal fluid was found to be turbid again and 45 c.c. were drained. No serum was given, as the condition was thought to be probably pneumococcal in view of the bacteriological findings.

Treatment with M. and B. 693 was given as soon as possible at 11.0 a.m. on the 7th when 2 grms. of the drug were given by mouth. 1 grm. was given 4 hourly after this initial dose, and the patient received in all 25 grms. by mouth. Gram positive diplococci, which were proved to be Type II pneumococci, were reported in her cerebrospinal fluid on the 3rd, 4th, 5th, 6th, 7th, 8th, 9th and 10th of January.


DECEMBER
15 16 17 18 19 20 21 22 23 24 25 26 27 28
JANUARY
DEC
29 30 31 1 2 3 4 5 6 7 8 9 10 11
Time
Temperature (Fahrenheit)
Pulse Rate

Case 7B

Beyond making her very cyanosed the drug effected no changes in her condition. Her condition deteriorated steadily and after the 8th she became delirious and semi-conscious. She did not vomit, but on the 10th she developed a paresis of the right 3rd nerve and some difficulty in swallowing. Spinal block was also noted on the 10th, although cistern puncture on the 9th had yielded a fluid distinctly less turbid than that obtained by lumbar puncture. She perished early on the 12th and at no time were adventitious or abnormal sounds heard in the lungs during this terminal phase.

At autopsy the vessels on the vertex of the brain were found to be engorged and outlined with pus. There was an abundant purulent exudate all over the base of the brain and the right lateral ventricle contained purulent fluid. The upper respiratory passages, lungs and heart showed no gross naked eye change and spleen and intestine were macroscopically normal. Both the liver and kidneys appeared to have undergone some fatty degeneration.

Type II pneumococci were isolated from material procured at autopsy as well as from cultures made from the cerebrospinal fluid during life.

96 hours unavoidably elapsed between the onset of symptoms due to her secondary pneumococcal meningitis and the exhibition of M. and B. 693. This case shows strikingly the marked difference between the action of streptocide on meningococci and M. and B. 693, on pneumococci in the same person. The girl is admitted after an illness which has lasted 192 hours (8 days) and is proved to be due to meningococci. She is treated with streptocide alone and responds rapidly and well to this treatment. She then develops a pneumococcal infection which of necessity remains untreated for 96 hours. M. and B. 693 given 100 hours earlier than streptocide fails to modify the course of this infection in the slightest degree.

CASE 8B.

Cheung Na, male, 26. This man was admitted to hospital on the 22nd of December, 1938. On admission his temperature was 101.2°, pulse 110 and respiration rate 22. He was delirious and was unable to give a coherent account of his illness.

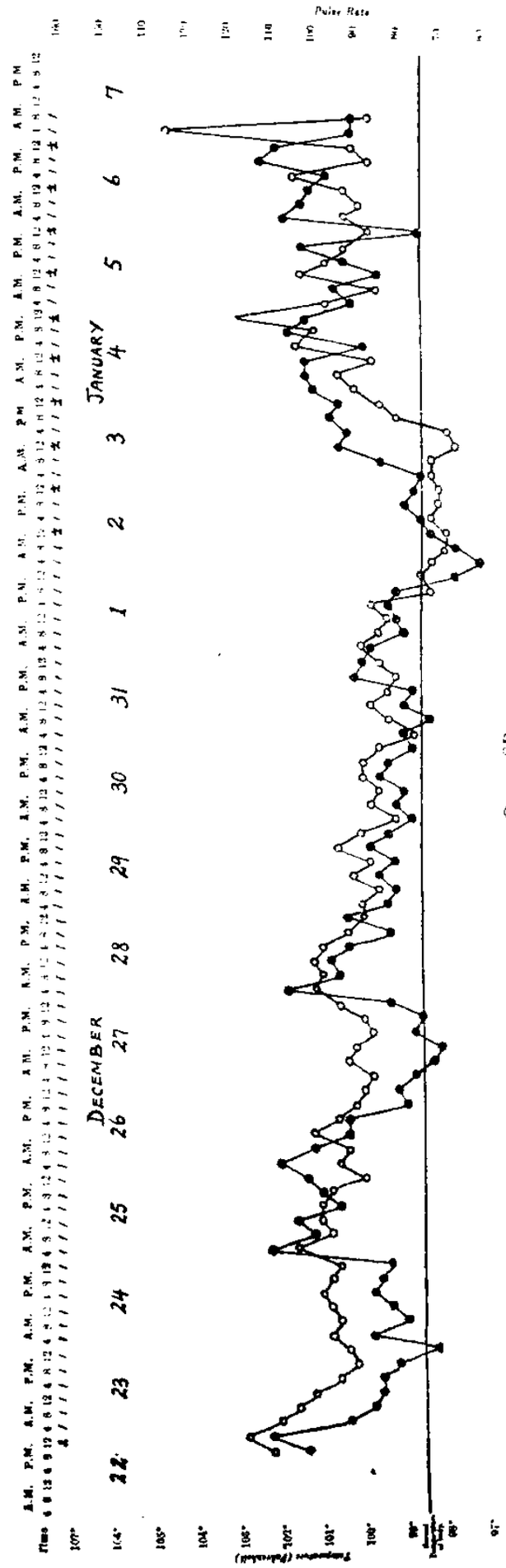
He showed slight head retraction and nuchal tenderness, and cervical "soldering" was demonstrable. He had no tâche but Kernig's sign was positive on both sides. His pupils were eccentric, unequal and fixed to light. His fundi, tympana and cranial nerves were normal. Knee and ankle jerks were exaggerated but plantar responses were flexor and no clonus was elicited. No abnormalities were detected in the heart, lungs or belly.

Lumbar puncture was impracticable as the lumbar region of the spine was bruised and oedematous as a result of earlier attempts at puncture. Cistern puncture was performed under evipan anaesthesia and highly turbid fluid was obtained under a pressure of 210. Meningococci were found in the fluid of which 65 c.c. were drained. 23 c.c. of anti-meningococcal serum were given intracisternally and 18 c.c. intravenously; 2 grms. of streptocide were given by mouth and 10 c.c. of a 2½% solution of streptocide were given intramuscularly.

Despite intensive treatment with streptocide and anti-meningococcal serum he did not respond readily. Streptocide was continued in a dose of 6 grms. every 24 hours for the first ten days of his illness but he never became completely afebrile. Meningococci were reported in his cerebrospinal fluid on the 28th of December and the fluid was still very turbid. Lumbar puncture became practicable during the second week of his illness, and his fluid on the 3rd of January was clear and colourless and showed only a few mononuclear cells and a slight increase of globulin. His temperature did not rise above 99° on that day, but on the 2nd it rose to 100.6° and he continued to have low grade fever till his death on the 7th.

Between the 2nd and the 7th his fluid became turbid and yellow and pneumococci were demonstrated in it on the 5th. These pneumococci were unfortunately not typed. He died before the arrival of M. and B. 693. At no time during his illness did he show symptoms suggestive of a pneumococcal infection of lungs, throat or ears.

At autopsy the brain was found uniformly sheeted with thick yellow pus and both lateral ventricles contained purulent fluid. The heart and lungs and upper respiratory passages showed no naked eye changes, but both liver and kidneys showed naked eye evidence of mild fatty degeneration. Pneumococci were found in preparations made at autopsy, and on culture of post mortem material, but they were not typed.



Case 8B

Figure 13. For explanation of chart, see Figure 1.

CASE 9B.

Yuen Ying Lee, male, 23. This patient was admitted on the 3rd of January, 1939. He was delirious on admission and unable to co-operate, but the following history was obtained from his wife. His illness began suddenly on December 29th with a shivering attack and fever followed by headache. He complained of nausea but did not vomit. His neck became stiff on the second day of the illness, but he had no visual or auditory symptoms. He became delirious on the third day of the illness. His wife stated that he had had no antecedent coryza or sore throat.

On admission his temperature was 98.8°, pulse 86 and respiration rate 22. He showed slight head retraction and nuchal tenderness and some degree of cervical "soldering" was demonstrable. The pupils were equal and active and there were no reflex abnormalities. Tâche was not elicited but he was found to have a cluster of herpetic vesicles at the left corner of the mouth. Kernig's and Brudzinski's signs were both positive. The heart, lungs and belly showed no abnormalities. Lumbar puncture yielded a turbid fluid under low pressure. Queckenstedt's sign was doubtfully positive and it was feared that block was impending. 25 c.c. of fluid were drained and meningococci were demonstrated in smears. 20 c.c. of anti-meningococcal serum were given intrathecally, 10 c.c. intravenously, and 2 grms. of streptocide were given by mouth. This dose was repeated in 4 hours time and from then on the man was given 1 grm. 4 hourly. Serum was continued intrathecally for the first week, and 6 grms. of streptocide were given by mouth every 24 hours. He also had two intramuscular injections of 10 c.c. of 2½% solution of streptocide, one on the second and the other on the third day in hospital. Universal myoclonic twitches were noted on the 5th and 6th but on the 7th he was afebrile and on the 8th his fluid was clear. He was, however, still incontinent of urine. On the evening of the 8th his temperature rose suddenly to 103.4° and his condition deteriorated rapidly. His cerebrospinal fluid became purulent again and signs of meningitis returned in full force. He died on the 10th, and at autopsy the vertex of the brain was found to be covered with creamy yellow pus, which was splashed here and there with recent haemorrhages ranging in size from 1 to 3 cms. in diameter. The heart muscle was soft and showed a few minute subepicardial haemorrhages. The lungs, upper respiratory passages and abdominal viscera showed no naked eye abnormality.

He had shown no symptoms suggestive of throat, ear or lung involvement during his illness, and pneumococci were never isolated from the cerebrospinal fluid during life. Type II pneumococci were obtained on culture of fluid taken from the lateral ventricles at autopsy. No specific anti-pneumococcal therapy was carried out in this case.

CASE 10B.

Ng Tung, male, 19. This man was extremely ill when admitted to hospital on January the 8th, 1939, and he was unable to give a detailed history although he insisted that he had been ill for sixteen days before admission.

His temperature was 100.6°, pulse 96 and respiration rate 22, when he reached hospital. He lay curled up in an attitude of generalised flexion and showed well marked head retraction, nuchal tenderness and cervical "soldering." Tâche was slight and no herpes or petechiae were noted. The pupils were equal and active and the fundi and tympana normal. His cranial nerves showed no abnormalities, the knee and ankle jerks were brisk and the plantar responses flexor. Kernig's and Brudzinski's signs were positive on both sides. The heart, lungs and belly appeared to be normal. Lumbar puncture yielded greyish-yellow fluid under a pressure 159. Meningococci were demonstrated in the cerebrospinal fluid of which 50 c.c. were drained. 20 c.c. of anti-meningococcal serum were given intrathecally, 10 c.c. intravenously and 1 grm. of streptocide by mouth was ordered 4 hourly. Despite active treatment the man's condition deteriorated and on the 10th he was coughing a little and a few non-consonating medium crepitations were heard at the lung bases. He had also become doubly incontinent, and the cerebrospinal fluid was still very turbid. No sputum was obtainable but a throat swab taken on the 10th was negative for pneumococci.

Pneumococci were found in the fluid obtained by lumbar puncture on the 11th and his temperature rose to 103.6° that afternoon. He died a few hours later and an autopsy was made on the 12th. He had had no specific anti-pneumococcal treatment during this illness.

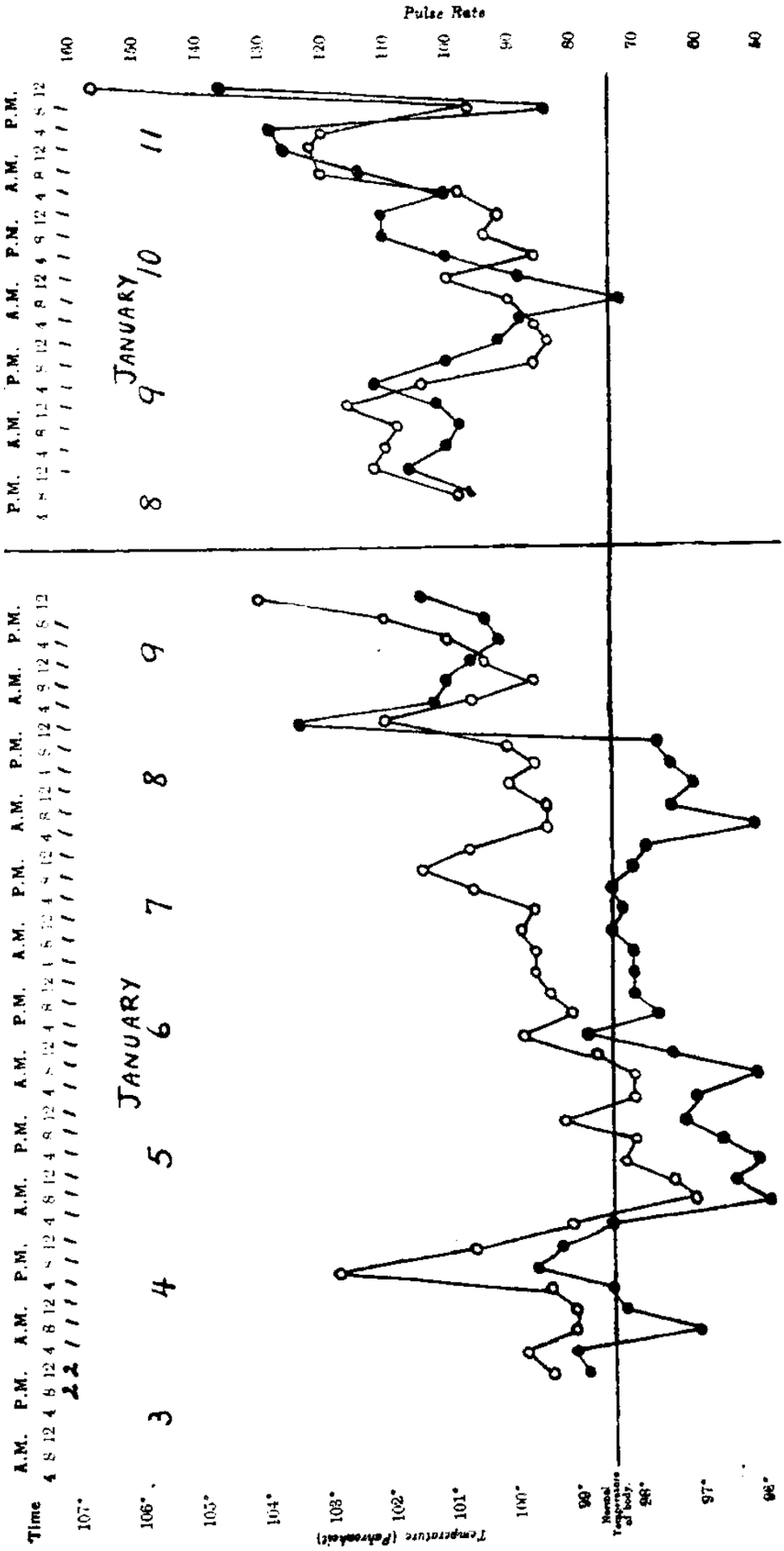


Figure 14. For explanation of chart, see Figure 1.

The vertex and base of the brain were both thickly sheeted with creamy yellow pus, and both lateral ventricles showed a mild degree of internal hydrocephalus. The heart showed no naked eye abnormality and the lungs showed merely a mild degree of basal congestion and oedema. The spleen was soft, congested and enlarged, but the intestines showed no gross changes. The parenchyma of the liver showed numerous small round greyish areas about the side of a hemp seed.

Type II pneumococci were isolated from preparations made from brain pus and lateral ventricle fluid.

CASE 11B.

Sie Kei, male, 9. This child was admitted to hospital from one of the refugee camps on December the 23rd, 1938. He was unable to give a lucid account of the early stages of his illness, but his grandmother told the following story. On the 18th he had suddenly become feverish and had vomited repeatedly. He complained of frontal headache and backache but had no rigor. He also said that all his limbs were "numb." Labial herpes appeared on the third day of the illness, but he made no complaint of stiff neck nor had he any eye or ear symptoms. The bowels were obstinately constipated throughout. He had had a sore throat for a few days before onset but no coryza or cough. He had been vaccinated about twenty days before the onset of the illness, and showed one modified take when he arrived at hospital.

On admission his temperature was 99.4°, pulse 98 and respiration 22. He lay in the gun-hammer attitude and showed marked head retraction, nuchal tenderness and cervical "soldering." There were clusters of herpetic vesicles at the right labial commissure but no petechiae. The pupils were slightly unequal and reacted sluggishly to direct light. The fundi and tympana cranial nerves were normal, but the nasopharynx was markedly congested. Kernig's and Brudzinski's signs were positive on both sides, the knee jerks and the right ankle jerk were absent, and the plantar responses extensor, but no abnormalities were detected on examination of the heart, lungs and belly.

Lumbar puncture yielded slightly turbid blood-stained fluid under a pressure of less than 20. Queckenstedt's sign was positive and meningococci were demonstrated in the fluid but no serum was given. 10 c.c. of a 2½% solution of streptocide were injected intramuscularly and 1 grm. of streptocide was ordered 4 hourly by mouth.

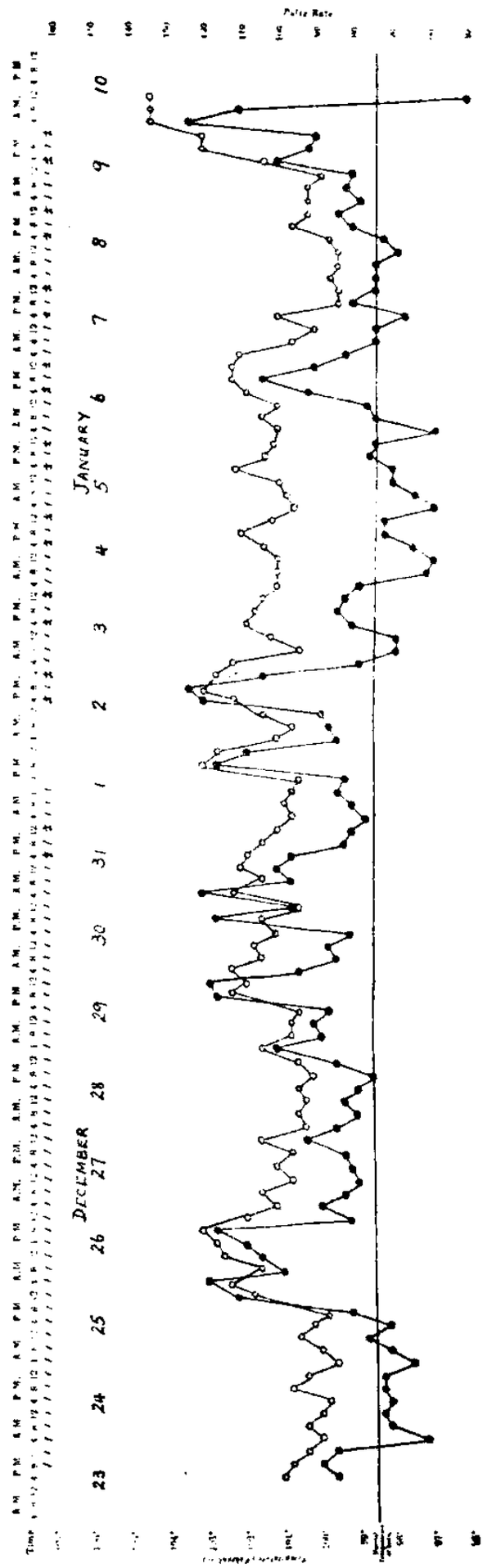
By the 25th he was afebrile and rational and his cerebrospinal fluid was practically clear. He had a rigor that evening and his meningeal signs became intensified again. His blood was examined for malarial parasites with negative results. His fever persisted irregularly and on the 2nd of January it was decided to give anti-meningococcal serum as his response to streptocide alone seemed to be unsatisfactory. He appeared to respond to serum at first as the fever abated during the next three days, but his temperature rose again on the 6th day to 101.6° and finally to 103.4° on the 10th, the day of his death. Pneumococci were never found in his cerebrospinal fluid during life nor did he show any symptoms suggestive of a pneumococcal infection, and it is difficult to tell either from his history or his charts at what precise point he developed his pneumococcal meningitis.

At autopsy a moderate amount of pus was found all over the vertex of the brain and there was also a small amount of purulent exudate at the base, but no internal hydrocephalus was noted. Culture of material obtained from the brain yielded a growth of Type II pneumococci. The lungs and heart showed no naked eye abnormalities and there were no gross changes in any of the abdominal viscera. No specific anti-pneumococcal treatment was attempted in this case.

CASE 12B.

Lin Lin Tai, female, 12. This child was admitted to hospital on the 10th of January, 1939. She said she was taken ill on the 8th with a rigor followed by fever, frontal headache and vomiting. Her neck had been stiff and painful since the onset and she had had backache since the 9th. Her left eye became painful on the 9th and she noticed she could not see clearly with it.

On admission her temperature was 97.4°, pulse 76 and respiration rate 20. She lay curled up on her side, and showed marked head retraction and nuchal tenderness and some cervical "soldering." Her right pupil was circular and active to light,

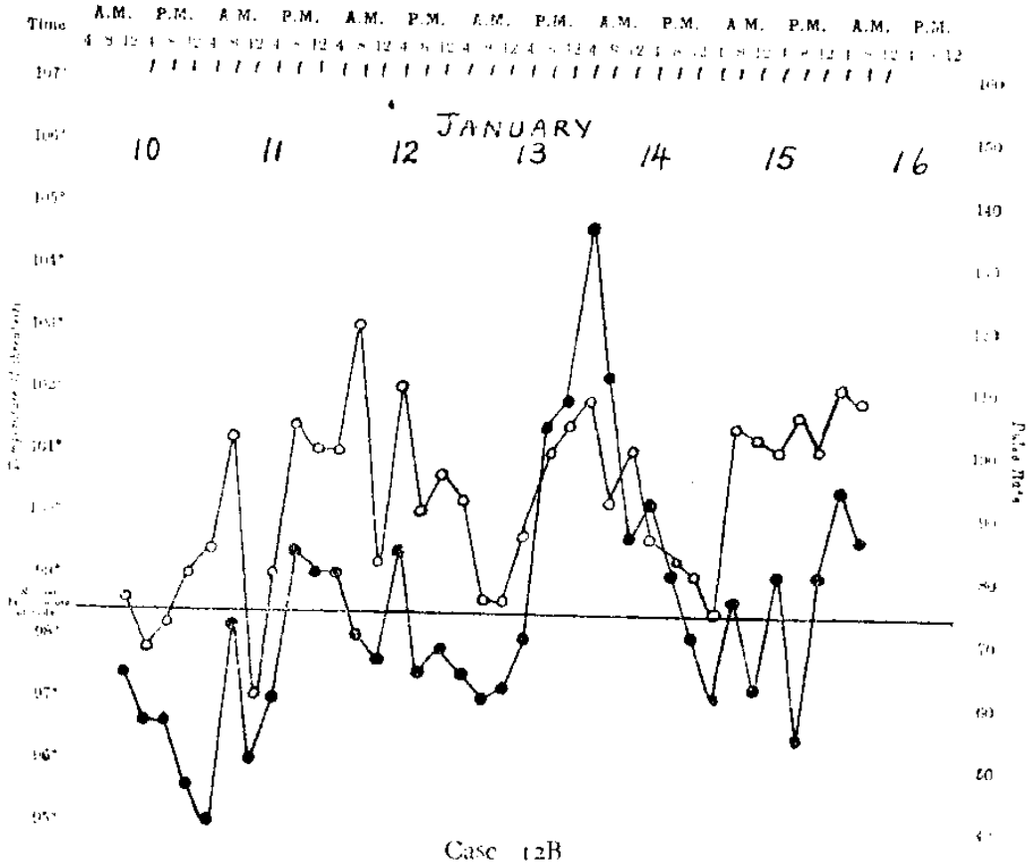


Case 11B

Figure 15. For explanation of chart, see Figure 1.

her left iris was inflamed and the media were opaque. Both tympana and the right fundus were normal. The cranial nerves showed no abnormality but knee and ankle jerks were absent and the right plantar response was extensor. Kernig's and Brudzinski's signs were positive on both sides. The lungs, heart and belly showed no signs of involvement.

Lumbar puncture yielded slightly turbid fluid under a pressure of 300. 35 c.c. were drained and meningococci were found. 20 c.c. of anti-meningococcal serum were given intrathecally and 1 grm. of streptocide was ordered 4 hourly by mouth. This combined treatment was continued for the next five days, but the child did not respond well. Meningococci were still present in the fluid on the 11th and headache and rigidity persisted. The turbidity of the fluid persisted and early on the 14th her temperature rose to 104.6°. From then on her condition deteriorated rapidly and she died on the 16th. On the 17th pneumococci were reported in the fluids obtained on the 13th, 14th, 15th and 16th. Presumably her pneumococcal meningitis began with the rise of temperature which occurred on the 14th.



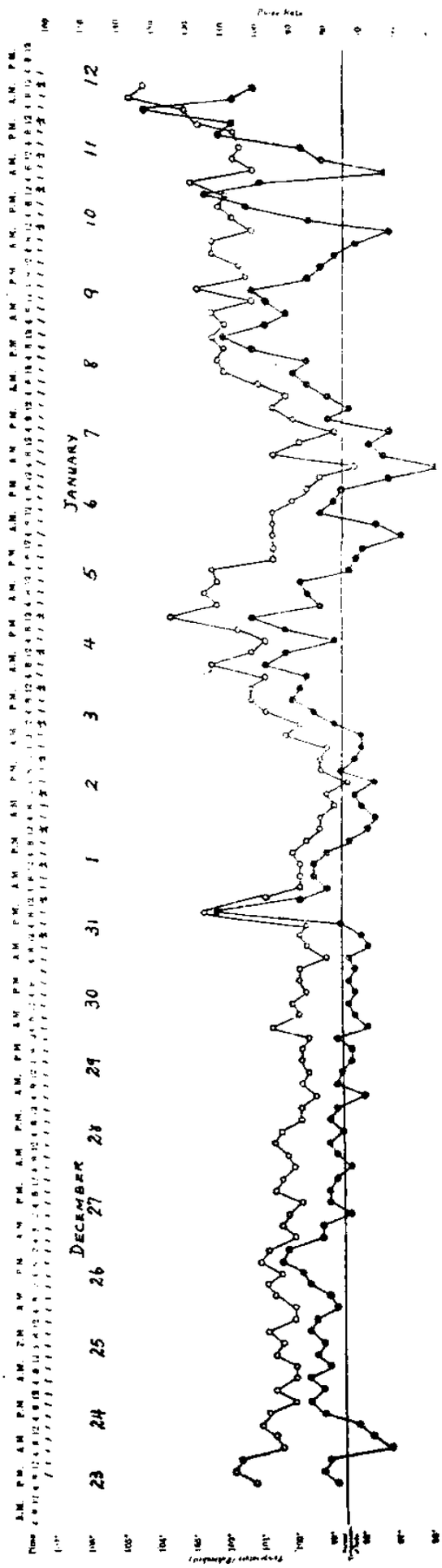
Case 12B

Figure 16. For explanation of chart, see Figure 1.

At no time throughout the illness were any signs of lung involvement detected. At autopsy the cerebral convolutions were found to be flattened and the sulci were obliterated. The whole of the vertex and base of the brain were covered with purulent exudate which was most abundant along the the course of the vessels. Preparations from the lateral ventricle fluid yielded Group IV pneumococci on culture. The lungs, upper respiratory passages, heart and abdominal viscera showed no gross naked eye changes. No treatment with M. and B. 693 was attempted in this case, first because so little of the drug was available and second because bacteriological proof of the pneumococcal nature of the final condition was not forthcoming till after the death of the child.

CASE 13B.

Wong Sui Luen, male, 9. This child was admitted to hospital on December the 23rd, 1938, and his mother gave the following history. On the 20th the child had had a rigor followed by fever, vomiting and severe frontal headache. He did



Case 13b

Figure 17. For explanation of chart, see Figure 1

not complain of photophobia but on the second day of the illness his neck became stiff and on the third his head became retracted. He showed no petechiae or herpes, but his mother had noted a discharge from his right ear which was not present, so she said, before the illness.

He had had a cough and cold just before the onset of this illness and had complained of slight sore throat. He had also been vaccinated about three weeks ago but without result. On admission his temperature was 98.6°, pulse 100 and respiration rate 22. He lay in the gun-hammer position and showed marked head retraction and nuchal tenderness. Tâche was not elicited nor were herpes or petechiae noted. The pupils were equal and circular and reacted sluggishly to direct light. The fundi and left tympanum were normal; the right external auditory meatus contained pus. The cranial nerves showed no other abnormalities and all deep reflexes were obtained. The child had a slight cough but lungs, heart and belly showed no physical signs of disease.

Lumbar puncture was carried out at once, and 45 c.c. of turbid fluid containing meningococci were obtained under a pressure of 130. 20 c.c. of anti-meningococcal serum were given intrathecally and 1 grm. of streptocide by mouth was ordered 4 hourly. Local treatment was applied to the discharging right ear, but as staphylococci only were grown from the pus, it was thought unlikely to have played an important part in the aetiology of the condition. The child responded to combined treatment, and by the 30th his fluid had became clear. He was afebrile and his appetite was improving. His right ear was now dry and a recent perforation could be seen in the right tympanum. He had a mild febrile reaction on the 31st but this was interpreted as a serum reaction. On the 4th he complained of left sided frontal headache and had slight fever. On the 5th his cerebrospinal fluid became turbid again. Signs of meningitis returned slowly, he had some epistaxis from the left nostril on the 7th and the succeeding three days, and on the 8th he developed irregular fever and began to waste rapidly. It was clear that neither streptocide nor anti-meningococcal serum were having the least influence on the meningitic process. He showed no physical signs in lungs, heart or throat nor was his cerebrospinal fluid markedly turbid. The irregular fever and wasting continued until the 12th, the day of his death.

At autopsy the brain was sheeted all over the vertex with pale yellow thin pus, and there was a considerable amount of exudate at the base. There was no internal hydrocephalus, but Type II pneumococci were cultured from swabs made from lateral ventricle fluid.

The right tympanic cavity was filled with purulent grumous matter and a perforation was found in the right tympanum. There appeared to be no direct connection between this process and the meningitis, but unfortunately no swabs were taken from the tympanic pus.

The heart, lungs and upper respiratory passages were normal, but the spleen was enlarged and engorged. The kidneys and liver were also congested, and the mesenteric glands were enlarged, congested and soft.

DISCUSSION.

That lung infections due to the pneumococcus can appear in epidemic form has been known for many years. Hirsch gives numerous accounts of epidemic outbreaks of lobar pneumonia and states that descriptions of epidemics of the disease in various European countries, with a high mortality rate, exist as early as the XVIth century. In the XVIIth century the same type of epidemic was described in Switzerland, Germany and France, and even at this early date Ravicio noted that those in contact with the disease sometimes caught it. In the XVIIIth century accounts of epidemic outbreaks of the disease appeared in Spain, England, Demark and America, as well as in the countries just mentioned. To explain these increases in the local incidence

of lobar pneumonia recourse was had to peculiarities of the soil and fluctuations of the sub-soil water. Hirsch himself believed that this form of the disease was due to a specific infections agent, and more recent observations have shown clearly that the infection can be conveyed from man to man.

But until Dochez and Avery demonstrated early in this century that 80% of cases of lobar pneumonia were caused by pneumococci Types I, II and III, it was generally held that pneumococcal infections were autogenous. This belief was based on the fact that pneumococci were found in the mouths and throats of many healthy individuals, and it was not unnaturally assumed that pneumococcal infections developed when the resistance of the individual was lowered, when the virulence of the pneumococcus was increased or when a combination of these two conditions occurred.

Dochez and his collaborators showed that it was rare to find pneumococci Types I and II in sputum from normal people, and they also observed that normal people who harboured such pneumococci in their sputum had recently been in contact with a case of pneumonia caused by an organism of the type they were carrying.

Much work has been done on the epidemiology of pneumococcal pneumonia since the publication of these findings, and considerable attention has been devoted to the problems raised by pneumococcus carriers and outbreaks of pneumococcal pneumonia in family groups and institutions. It is now universally admitted that pneumococcal pneumonia is an infective disease, the infectivity of it not being much higher than that of meningococcal meningitis, and it has been proved conclusively that the carrier rate rises rapidly in normal individuals exposed to cases of lobar pneumonia (Tilghman, Finland, 1936).

During the last decade great advance have been made in our knowledge both of the pneumococci and of the infections due to them. Thanks to the fruitful researches of Cooper and her associates the problem of the identity of the organisms belonging to Group IV has at last been solved, (Cooper *et al.* 1929, 1932) and the serological identities of the thirty-two types of pneumococci have been firmly established. (Finland, Winkler, 1934 *et al.*).

Numerous reports of epidemic outbreaks of pneumococcal infections have appeared in the German and American literature in recent years but they have been concerned for the most part with pulmonary and upper respiratory infections, and no report of an epidemic outbreak of pneumococcal meningitis similar to the one described in this paper has been found in the available literature.

Smillie and Caldwell in 1929 studied an outbreak of lobar pneumonia in a rural in South Aldbama and found that the majority of their cases were due to Group IV organisms and ran a mild course.

They noted that the disease tended to follow a "cold" and suggest that modern conditions have produced communities resistant to the relatively avirulent Group IV pneumococci but susceptible to the fixed I, II and III types. Strøm in 1931 reported an outbreak due to Type I pneumococci in an orphanage and found that 33% of contacts became carriers during the epidemic. Webster and Hughes in 1931 made observations on the carrier state in 105 adults and children and found that pneumococci Types I and II only occurred in carriers who had been in contact with cases of pneumonia due to these organisms, whereas pneumococci of Type III and Group IV tended to assume a saprophytic mode of existence in carriers and to spread more readily among people who had sinus infections.

In 1934 Viktorow, Semtzowa and Oettinger in an important paper, based on three groups of people totalling 571, were able to confirm these results regarding contacts with Type I and II pneumonias, and they were also able to show that direct transmission of Type I and II pneumococci with subsequent infection was possible. They consider that pneumonia due to Type I and II pneumococci is an exogenous infection which is about as contagious as cerebrospinal-meningitis, and they explain the outbreaks of lobar pneumonia which occur in barracks, camps, schools and ships on this hypothesis.

> "Es ist festgestellt, dass die Zerebrospinal-meningitis fast ausschliesslich durch Bazillenträger übertragen wird . . . . . und die vollkommene Aehnlichkeit der Erscheinungen, die wir beim Studium der Epidemologie der Zerebrospinal-meningitis und der Typus I and Typus II Pneumonie beobachteten, spricht für die Analogie der Verbreitung dieser Infektion."

In the same year Christie in a paper on the epidemological significance of serological types of pneumococci was able to show that organisms of Group IV showed an increased activity during epidemic prevalence of disease due to Types I and II. He found that 75% of his cases of lobar pneumonia were due to Types I and II and that 25% of convalescents became carriers for varying periods. He states that organisms of Group IV can be found as commensals in 40-60% of normal persons.

Gundel and Wallbruch gave an admirable account of an epidemic outbreak of lobar pneumonia in 1935, 19 cases occurring in a relatively self-contained community of 300 people. In this instance too, carriers played a predominant part in spreading the infection among school children and those in contact with them at home, and a high Type I carrier rate was demonstrated. The epidemic was preceded by an outbreak of "colds."

Wüstenberg in 1935 recorded a family, three members of which developed Type I pneumococcal infections, while four other members

became carriers, two of Type I, one of Type III and one of Type XXIX pneumococci.

Tilghman and Finland in 1936 reported a series of pneumococcal infections in families. In 33 groups of such infections, they were able to show that Type I and Type II infections were commonest and that 23 of the groups included 54 people ill of pneumococcal infections of homologous type. Contact with an infected case was an important factor in the production of such cases and they were able to demonstrate homologous pneumococcal infections in cases of empyema, primary meningitis, otitis media, upper respiratory infections and bronchopneumonia, which had been in contact with uncomplicated cases of lobar pneumonia. They further showed that a high homologous carrier rate occurred in the members of such families, and that such carriers developed tpye-specific homologous antibodies. In only one of these groups did they record a case of primary pneumococcal meningitis occurring as a result of contact with lobar pneumonia.

The association of pneumococci with coryza, pharyngitis and sinusitis has been stressed by some observers, and Webster and Clow were able to show a connection between the incidence of such infections and the carrier state caused by pneumococci, *H. influenzae* and *Str. haemolyticus*. One of the transient carriers of *H. influenzae* and *Str. haemolyticus* investigated by them was later found to be harbouring Type II meningococci in the nasopharynx.

These quotations from the literature will suffice to show first, the marked differences between pneumococci of different types, second, the infectivity, low but definite, of Type I and Type II pneumococci and third the possibility of spread of Type I and Type II infections by carriers who have recently been in contact with infections due to these organisms.

There is, therefore, no inherent difficulty in explaining this outbreak on a carrier basis, but even if we assume that the first case was infected by the ward-boy who was found to be carrying pneumococci in his throat, it is a little difficult to explain the occurrence of some of the later cases in the same way as several of them were nursed in wards to which he had no access. It is clear from the remarks made in the Introduction to this paper that the other possibilities fall to the ground when examined closely; the carrier nurse and the choleraic man who developed lobar pneumonia arrived after the outbreak had begun; that the woman suffering from primary pneumococcal meningitis who was admitted on the 30th should have been responsible for three cross-infections within 24 hours of her admission is exceedingly unlikely as the time interval was so short. One of the most striking points about the outbreak is the fact that the first three cases of meningococcal meningitis who developed pneumococcal meningitis sickened within a few hours of one another, a circumstance which

strongly suggests that they, in common with the other occupants of the large ward downstairs, had been simultaneously exposed to the infecting organism. Parents were allowed to visit their children on December 25th in the afternoon, and it is possible that some visitor suffering from an ambulatory pneumococcal infection of the upper respiratory passages was admitted to this ward then. Such a supposition would explain the simultaneity of appearance of symptoms in the early cases and would also give a reasonable incubation period for the second infection. Gundel and Wallbruch (1935) were able to show that in several of their contact cases the incubation period lay between 4 and 6 days, a figure which agrees fairly well with that demanded by this supposition. It seems easier to explain the origin of the outbreak by some exogenous hypothesis such as this than by any of the endogenous hypotheses which have been tested, and the fact that the cases who developed a pneumococcal infection in hospital all showed Type II or Group IV pneumococci is readily explicable in this way, as carriers have been shown on occasion to harbour more than one type of pneumococcus at the same time. But even an exogenous hypothesis of this sort leaves many questions unanswered. No patient suffered from a "cold" or sore throat before contracting this secondary infection, and the throat swabs which were taken during the course of the pneumococcal meningitis were negative for pneumococci, Case 6A, the last of the primary series, was the only patient who showed evidence of lobar consolidation of a lung at autopsy and he was the only patient in either group whose pneumococci were Type I. In none of the other autopsies was evidence found to suggest the presence of a pneumococcal infection of lungs or respiratory passages, and one is therefore left to assume that in some unexplained way these patients suddenly and almost simultaneously developed a pneumococcal meningitis, presumably as the result of a pneumococcal septicaemia. That it is justifiable to regard their meninges as loci minoris resistentiae, in view of their recent meningococcal meningitis, is indisputable but it does not explain how or why these meninges were so suddenly invaded by pneumococci. It is worth pointing out here that three of the cases in Group B, 7B, 11B and 13B, had all been recently vaccinated against smallpox. 13B had apparently not taken, but the other two had well marked signs of a recent take. Investigations into the aetiology of post-vaccinal encephalitis have shown that the condition is not more likely to occur when epidemic diseases of the nervous system are prevalent, and it seems unlikely that vaccination should produce an increased susceptibility to meningeal infections when they are present in epidemic form.

On turning to the therapeutic aspect of the outbreak, the first point to attract attention is that all these cases of pneumococcal meningitis, whether primary or not, died whatever the treatment. Much interest has been aroused recently by the introduction into

medicine of the synthetic preparation M. and B. 693. (2-p-aminobenzene sulphonamido-pyridine). This drug, so it is said, has an action on pneumococci comparable to the action of sulphanilamide on haemolytic streptococci. Whitby (1938) showed experimentally that 2-p-amino-benzene sulphonamido-pyridine is active against pneumococcal infections produced by Types I, II, III, V, VII and VIII in mice, and he found it especially active against types, I, VII and VIII. He noted that the capsules after swelling and becoming crenated, disappeared, and that the toxicity of the drug for animals was low. Fleming later in 1938 was able to show experimentally that the drug retarded the growth both of pneumococci and streptococci in human blood in concentrations which could be attained in the human being, but that it had no bactericidal effect in such concentrations. He also showed that leucocytes must be present if the organisms are to be destroyed by the blood, and he found that the addition of specific immune serum to the mixture enhanced the effect of the drug. He regards the drug as being bacteriostatic rather than bactericidal and advises that it be combined with serum therapy whenever possible.

These communications on M. and B. 693 were immediately followed by attempts to assess the therapeutic value of the drug in various pneumococcal infection. A recovery from pneumococcal meningitis had been attributed to Prontosil in 1937 by Caldwell and Byrne, and in 1938 Young recorded a case of the disease in a child who recovered after a total dosage of 23 grm. of sulphanilamide. The first case of recovery from pneumococcal meningitis attributed to M. and B. 693 was recorded by Dyke and Reid in September 1938, and Dyke also reported uneventful convalescence in a case of pneumococcal septicaemia, treated with the drug. In the same month Robertson recorded a recovery from pneumococcal meningitis in a boy of 14, who was treated with M. and B. 693 from the fourth day of the disease. The response was immediate and gratifying.

Cunningham in November, 1938, published a case of recovery from pneumococcal meningitis: the patient, a woman aged 47, had suffered from sinuisitis for some years before her illness. On admission lumbar puncture yielded a fluid containing pneumococci. She was treated for thirty-six hours with sulphanilamide receiving 15 grms. of the drug, and with 50 grms. of M. and B. 693 over the next seven and a half days. The author states that it is impossible to assess the merits of the two drugs from this case. He gives a brief review of recently recorded instances of recoveries from this disease attributed to Prontosil, (and points out how unconvincing the bacteriological evidence is in many of them) mentioning Allmann's 15 year old patient who recovered from a Type III pneumococcal meningitis secondary to chronic otitis media, Frankmann and Stewart's 5 year old child who recovered from a type II meningitis and Latto's patient of 26 whose recovery from a Type I meningitis

was also attributed to Prontosil. He quotes Finland, Brown and Rauh's' work on pneumococcal meningitis treated either with sulphanilamide alone or in combination with specific serum. They examined ten cases, six of whom recovered, the youngest of the recoveries being 7, the oldest 19. The four fatal cases were due to Type XI, Type XXI, Type VII and Type IV meningitis.

Hobson and McQuaide published a paper in 1938 on the treatment of meningococcal meningitis with M. and B. 693, which is of value because in their investigation of six cases they were able to show that the passage of the drug from the gut to the blood was rapid but depended on the state of the gut. They also found that the concentration of the drug in the cerebrospinal fluid was only about half that in the blood and that efficient bacteriostasis was obtainable with a concentration of 3 mg. per 100 c. cm. in the cerebrospinal fluid.

McAlpine and Thomas reported a recovery in April 1939. Their patient, a man of 24, was treated for pneumococcal meningitis with M. and B. 693 from the third day of the illness. The man received in all 25.5 grms. of the drug spread over seven days and the highest concentration of the drug in the cerebrospinal fluid was 3 mg. per 100 c.c. They believe that 5-6 grms. of the drug daily represents the optimal dose and that it should not be necessary to waken the patient at night for it.

MacKeith and Oppenheimer in May 1939 reported five cases of pneumococcal meningitis treated with M. and B. 693 with three deaths. They stress the difficulty of making a differential diagnosis when dealing with purulent meningitis and advise the exhibition of the drug in all such cases. May (1939) recorded a death occurring in a male child whose lobar pneumonia became complicated by pneumococcal meningitis, and suggests that the earlier treatment of the lung condition with M. and B. 693 had induced 693 fastness. Raman (1939) reported a recovery from the disease in a Tamil coolie aged 34, and gave in all 80 grm. of the drug.

Various papers have also been published recently recording the action of the drug in pneumococcal lobar pneumonia, the outstanding ones being those of Evans and Gaisford (1938), Barnett (1939) and Flippin (1939).

Seven of the cases recorded in this paper were treated with M. and B. 693, and the failure to respond to the drug has been commented on in their case-histories. The following table gives the relevant data concerning dosage, time interval from onset of disease to beginning of treatment and type of pneumococcus in each case.

CASES TREATED WITH M. and B. 693.

TABLE III.

No.	Sex. Age.	Date onset pn. meningitis.	Date treatment M. and B. 693 begun.	Total M. and B. 693 dose.	Hours of Treatment.	Type or group of pneumococcus.
		Primary pneumococcal meningitis—Group A.				
2 A	F. 11	3. 1. 39.	9. 1. 39.	18 grms.	72	Type II
3 A	M. 14	9. 1. 39.	12. 1. 39.	21 grms.	76	Type II
4 A	F. 11	13. 1. 39.	13. 1. 39.	32 grms.	120	Type II
		Pneumococcal meningitis occurring during recovery from meningococcal meningitis Group B.				
3 B	F. 13	1. 1. 39.	7. 1. 39.	14.5 grms.	48	Group IV
4 B	M. 8	3. 1. 39.	7. 1. 39.	20 grms.	72	Type II
6 B	F. 9	3. 1. 39.	7. 1. 39.	20 grms.	100	Type II
7 B	F. 20	3. 1. 39.	7. 1. 39.	25 grms.	96	Type II

While it must be admitted that the time interval between the onset of the pneumococcal meningitis and the inception of treatment with M. and B. 693 was unduly long in most of these cases, this will hardly account for the complete failure of the drug in all of them. Case 4A, for example, received M. and B. 693 within a few hours of developing fever on the 13th of January. From the 8th to the 13th she had been adequately treated with streptocide and anti-meningococcal serum and had responded so well that her cerebrospinal fluid was clear and showed no increase of cells by January 12th, whereas on the 8th pus cells had been reported + +. Within eight hours of developing fever on the 13th she was given 2 grms. of M. and B. 693 by mouth. 2 grms. were given four hours later and from then on 1 grm. four hourly, until she had had in all 32 grms. of the drug. Pneumococci were reported in her fluid on the 13th, 14th, 15th, 16th, 17th and 18th and they were also obtained by culture from autopsy preparations made on the 19th. Clearly the drug had not exerted the slightest influence on this Type II pneumococcal infection.

These failures, be it noted, cannot be explained by saying that the drug was lost through vomiting because, although marked nausea was common among those taking it, vomiting was unusual, and this point has already been brought out in the case-histories. Exceedingly wide variations have been found in the concentration of the drug in the blood during its administration, and these are thought to be due to varying rates of absorption, but even so, the therapeutic effect appeared to be as good with a blood concentration of 3 mg. per 100 c.c. as with one of 18 mg. (Flippin *et. al.* 1939) Bullowa and Finland (1939) consider the clinical reports hitherto published "grossly inadequate for any evaluation" and other American observers have sounded

the same note of caution and reserve. It is, of course, possible that the heavy doses of streptocide which these patients had received before taking M. and B. 693 may in some way have impeded the action of the latter drug, but it seems unlikely on a priori grounds. It is also possible that the dosage employed was inadequate, though it will be noted that most of the patients treated with the drug were children and all of them showed toxic signs of some sort during the administration of the drug. A more likely explanation seems to be that put forward by Fleming (1939) in discussing the failure of the drug in a case of lobar pneumonia: that the patient had not developed sufficient immunity to deal with the pneumococci even after they had been subjected to a high concentration of M. and B. 693. In a word, it may be that in certain cases the pneumococci become immunised to M. and B. 693 but the patients do not develop immunity to their pneumococci. In any case, it is almost unthinkable that seven unselected cases of pneumococcal meningitis of both sexes and varying ages should all absorb this anti-pneumococcal specific so irregularly, so poorly and so slowly that they all perish of the disease just as rapidly as untreated cases. "*Credat Judaeus Apella, non ego.*" I have no explanation to give of the uniform lack of success of M. and B. 693 in this group of cases.

SUMMARY.

1. Nineteen cases of pneumococcal meningitis are described.
2. Six of these were apparently cases of primary pneumococcal meningitis.
3. Thirteen of them developed pneumococcal meningitis in the course of recovery from meningococcal meningitis.
4. The possible modes of infection in hospital of these thirteen cases are discussed.
5. The pneumococci isolated were typed in all save two cases. One meningitis was due to Type I, thirteen to Type II and three to pneumococci belonging to Group IV.
6. Treatment is discussed and a description is given of the lack of action of M. and B. 693 in seven of these cases (three primary, four secondary), despite the fact that the drug was pushed to the point of producing toxic effects in all of them.
7. Evidence is afforded by these cases that streptocide has little or no effect on the course of pneumococcal meningitis due to pneumococci of Types I and II.

Since writing this paper I have been able, thanks to the courtesy of my friend and colleague Dr. K. D. Ling, to observe the action of Dagenan in pneumococcal meningitis supervening on a pneumococcal lobar pneumonia with empyema. I say to observe its action; I should

say rather its entire lack of action, for 1 grm. of the drug 4 hourly for 36 hours altered the course of the disease not one whit. The man died and all Dagenan did was to turn him blue while he was dying.

ACKNOWLEDGMENTS.

I have to express my warmest thanks to Dr. R. Begbie of the Bacteriological Institute, Hong Kong. Without his continued encouragement and help on the bacteriological side it would not have been possible for me to collect many of the data essential for the writing of this paper. I have also to thank Dr. A. Greaves, Director of the Institute, for it was owing to his indefatigable zeal and untiring energy that we were enabled to have the various strains of pneumococci isolated typed by the Henry Lester Institute, Shanghai. Last, but by no means least, I must thank the authorities of the Henry Lester Institute for carrying out the typing and Miss K. Steers for copying my charts.

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CLINICAL REPORT OF THE DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY OF THE UNIVERSITY OF HONG KONG, 1937 and 1938.

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TABLE OF CONTENTS.

ABBREVIATIONS.

G.C.H.	Government Civil Hospital.
Q.M.H.	Queen Mary Hospital.
T.Y.H.	Tsan Yuk Hospital.
A.P.H.	Ante-Partum Haemorrhage.
A.P.V.D.	Ante-Partum Vaginal Discharge.
A.R.M.	Artificial Rupture of Membranes.
F.H.N.H.	Foetal Heart not Heard.
P.F.	Promontory Felt.
P.N.F.	Promontory not Felt.
P.R.M.	Premature Rupture of Membranes.
G.	Good.
F.	Fair.
P.	Poor.
S.B.	Still-born.
M.	Mother.
C.	Child.
A.	Alive.
D.	Dead.
C.S.	Caesarean Section.
B.W.	Baby's Weight.
P.M.	Post Mortem.
H.S.	Haemolytic Streptococcus.
D.A.A.	Discharged against Advice.
N.I.L.	Not in Labour.
I.D.I.	Induction-Delivery Interval.
M.R.	Manual Removal.

INTRODUCTION.

The years 1937 and 1938, for which a combined report is now issued, have seen considerable changes both in the personnel of the Department and in the hospital accommodation provided for the patients. Professor W. C. W. Nixon was in charge of the Department until 10th December, 1937, when he vacated the Chair and returned to England. His successor, Professor Gordon King, was not appointed until nearly a year later, and during the interim Dr. P. F. S. Court gave very able service as Acting Professor of Obstetrics and Gynaecology. The University is indebted to the Director of the Government Medical Department for his kindness in allowing the services of Dr. Court to be made available during this period. Professor Gordon King took up his duties on 12th November, 1938 and wishes to record his appreciation of the high quality of the work developed by his predecessors, Professors R. E. Tottenham and W. C. W. Nixon, and maintained by Dr. P. F. S. Court during the time that he was Head of the Department.

Great changes have taken place in the distribution of Hospital accommodation during the period under review. Up to June 1937 the Hospital work was carried out at the Tsan Yuk Hospital, with its 45 obstetrical and 15 gynaecological beds, and the Government Civil Hospital, with 16 obstetrical and 8 gynaecological beds. At the end of June 1937 all gynaecological cases were transferred to the new Queen Mary Hospital, the use of the Government Civil Hospital was discontinued, except as an Out-Patient Department, and the Tsan Yuk Hospital became exclusively a Maternity Hospital. The present position, therefore, is that 60 beds are devoted to Obstetrical work in the Tsan Yuk Hospital, and 21 beds to Gynaecological work in the new and splendidly equipped Queen Mary Hospital. Both of these Hospitals are Government institutions, and it is by the courtesy of the Director of Medical Services that the University is able to make use of the extensive clinical facilities provided by them.

The report here presented covers a period of two years and represents work carried out in three institutions, the Government Civil Hospital (G.C.H.), the Tsan Yuk Hospital (T.Y.H.) and the Queen Mary Hospital (Q.M.H.) during the years 1937 and 1938.

The writer would like to express his thanks to all who have helped him in the compilation of the report, particularly to his assistants, Dr. H. C. Ku and Dr. C. K. Quek, and to his secretary, Miss S. H. Ng.

GORDON KING.

REPORT OF THE OBSTETRICAL DEPARTMENT.

During the years 1937 and 1938 there were 4,756 admissions and 4,553 deliveries. During the first half of 1937 (from the beginning of January until the middle of June) the old Government Civil Hospital was still in use as a Maternity Hospital and cases treated there will be grouped under the heading G.C.H. (1937) in the following tables. The Tsan Yuk Hospital functioned during the whole of the two years under review, and cases dealt with in this hospital will be grouped under the headings T.Y.H. (1937) and T.Y.H. (1938).

Booked Cases:	*G.C.H. (1937)*	*T.Y.H. (1937)*	*T.Y.H. (1938)*
1. Delivered in Hospital (a) Discharged ...	35	93	126
(b) Transferred...	0	0	0
2. Baby born before arrival	0	0	0
3. Discharged undelivered	2	5	10
4. Died (a) after delivery	0	0	0
(b) undelivered	0	0	0
	37	98	136

Total of Booked Cases = 271

Emergency Cases:	*G.C.H. (1937)*	*T.Y.H. (1937)*	*T.Y.H. (1938)*
1. Delivered in Hospital (a) Discharged ...	280	1,807	2,114
(b) Transferred...	2	3	11
2. Baby born before arrival	4	10	1
3. Abortion	0	0	1
4. Discharged undelivered	12	98	120
5. Died (a) after delivery	1	6	13
(b) undelivered	0	1	1
	299	1,925	2,261

Total of Emergency Cases = 4,485

Admissions to G.C.H. (1937)	336
,, ,, T.Y.H. (1937)	2,023
,, ,, T.Y.H. (1938)	2,397
Total admissions to Clinic	4,756
Total deliveries	4,553

Of the 271 Booked Cases, 101 were Primiparae and 170 were Multiparae.

Of the 4,485 Emergency Cases, 1,456 were Primiparae and 3,029 were Multiparae.

NUMERICAL SUMMARY OF CASES DELIVERED IN HOSPITAL, ADMITTED FOR TREATMENT OR AFTER DELIVERY.

A. PRESENTATIONS.

	G.C.H. (1937)	*T.Y.H. (1937)*	*T.Y.H. (1938)*	*Total*
Persistent Occipito-Posterior	11	52	63	126
Breech	7	59	66	132
Face and Brow	1	2	3	6
Transverse	0	13	17	30
Twins	1	23	23	47
V. 1 (L.O.A.)	201	1,362	1,678	3,241
V. 2 (R.O.A.)	103	454	461	1,018

B. COMPLICATIONS.

	G.C.H. (1937)	*T.Y.H. (1937)*	*T.Y.H. (1938)*	*Total*
Antepartum Haemorrhage:				
(a) Accidental	3	1	4	8
(b) Placenta Praevia	3	18	20	41
Albuminuria with Toxic Symptoms	16	66	54	136
Eclampsia	1	5	11	17
Hydramnios	1	13	10	24
Prolapse of cord	2	6	11	19
Uterine inertia	1	7	5	13
Cardiac disease	1	6	3	10
Trial Labour	0	6	1	7
Induction of Labour	2	11	2	15
Version (a) Antenatal	0	1	2	3
(b) During Labour	1	11	11	23
Forceps	2	29	58	89
Craniotomy	1	2	1	4
Caesarean Section	1	3	4	8
Post-partum Haemorrhage	4	36	42	82
Manual Removal of Placenta	1	5	4	10
Puerperal Pyrexia	13	109	109	231
Maternal Deaths	1	7	14	22

CASES TREATED IN THE HOSPITAL BEFORE LABOUR.

Reg. No.	*Age*	*Gravida*	*Maturity*	*DISEASE*	*Result*	*No. of days in Hospital before delivery or discharge*	*REMARKS*
T.Y.H. (1937)							
BOOKED							
1342	41	9	30	Chronic Nephritis	F.	5	D.A.A.
1480	30	1	32	Oedema with toxaemia	G.	4	
EMERGENCY							
27	29	6	18	Hyperemesis Gravidarum	G.	6	
634	30	4	30	Pregnancy complicated by Mitral Stenosis...	?	11	Generalised oedema D.A.A.
1435	39	7	32	Marked oedema. Mitral Stenosis	G.	13	
1531	23	1	40	Oedema—Avitaminosis B_1	G.	7	
1533	24	1	38	Oedema. ?Avitaminosis B_1	G.	4	
1542	34	10	36	Oedema. ?Avitaminosis B_1	G.	4	
1544	26	6	34	Oedema—Avitamino-is B_1	G.	8	
1436	32	1	37	Oedema—Mitral Stenosis	G.	10	
T.Y.H. (1938)							
EMERGENCY							
948	25	1	33	Oedema of pregnancy	Improved	13	N.I.L. D.A.A.
1455	38	8	36	Cardiac disease. Oedema	D.	2	Died undelivered.
1963	29	6	37	Acute Bronchitis	Improved	23	
2019	20	2	37	Cystitis (Streptococcus, staphylococcus) ...	Improved	12	
2149	24	1	33	Acute Bronchitis	G.	6	N.I.L.

PERSISTENT OCCIPITO-POSTERIOR POSITION.

In 126 cases the Occiput did not rotate spontaneously.
13 babies were stillborn and 8 died, a mortality of 15.9%.
Two mothers died, a mortality of 1.6%.

Mode of Delivery	*No. of Cases*	*Mother* G.	*Mother* D.	*Child* G.	*Child* S.B.	*Child* D.
A. *Booked Cases:* Total 15						
Spontaneous delivery, face to pubes	7	7	—	7	—	—
Manual Rotation	1	1	—	1	—	—
Manual Rotation and Forceps	3	3	—	2	1	—
Forceps, face to pubes	1	1	—	—	1	—
Forceps and perforation, face to pubes ...	1	1	—	—	1	—
Caesarean Section	2	2	—	2	—	—
B. *Emergency Cases:* Total 111						
Spontaneous delivery, face to pubes	66	66	—	59	3	4
Manual Rotation	1	1	—	1	—	—
Manual Rotation and Forceps	33	32	1	25	4	4
Forceps, face to pubes	9	8	1	6	3	—
Caesarean Section	2	2	—	2	—	—

FACE AND BROW PRESENTATIONS.

There were 4 cases of Face and 2 of Brow Presentations.

No mother died.

5 babies were stillborn, a mortality of 83.3%.

Reg. No.	*Age*	*Gravida*	*Maturity*	*POSITION*	*TREATMENT*	*Result*		*Weight of Child*		*REMARKS*
						M.	*C.*	*lbs.*	*ozs.*	
G.C.H. (1937)										
EMERGENCY										
218	33	7	39	Brow	Bipolar Version	G.	S.B.	7	4	Presentation of cord.
T.Y.H. (1937)										
EMERGENCY										
1039	31	2	40	R.M.P.	Nil	G.	S.B.	7	0	Spontaneous rotation.
1182	36	6	40	L.M.P.	Nil	G.	G.	6	4	Spontaneous rotation.
T.Y.H. (1938)										
EMERGENCY										
765	30	6	39	R.M.P.	Manual Rotation and Forceps	G.	S.B.	7	12	
1187	31	6	37	R.M.P.	Nil	G.	S.B.	6	8	Spontaneous rotation.
1405	40	8	40	Brow	Craniotomy	G.	S.B.	6	4	Failed forceps.

SHOULDER PRESENTATIONS.

There were 30 cases of Shoulder Presentation.

No mother died.

21 babies were stillborn and 1 died, a mortality of 73·3%.

Reg. No.	*Age*	*Gravida*	*Maturity*	*Complication*	*TREATMENT*	*Result* M.	*Result* C.	*Weight of Child* lb.	*Weight of Child* oz.	*REMARKS*
T.Y.H. (1937)										
BOOKED										
1016	27	2	28	Placenta Praevia	Internal Version	G.	S.B.	2	1	Prolapsed hand and forearm. D.A.A.
EMERGENCY										
367	39	4	34	Prolapsed arm	Internal version. Traction	G.	G.	5	2	
454	31	6	40	Prolapsed arm	Internal version. Traction	G.	S.B.	7	10	
530	28	3	32	—	External Version	G.	S.B.	3	0	
661	40	9	38	Prolapsed hand	Internal Version	G.	S.B.	4	4	Extraction.
706	23	1	40	—	Manual Rotation-Forceps	G.	G.	7	8	
904	22	4	34	Pre-eclampsia	Induction of labour	G.	S.B.	2	0	Extraction. Macerated foetus.
985	28	3	34	Placenta Praevia	Bipolar Version	G.	S.B.	2	4	Foetal Monstrosity.
1295	29	2	42	Twins	Internal Version	G.	S.B.	5	0	2nd of twins.
1453	31	5	34	Placenta Praevia	Internal Version	G.	S.B.	6	0	
1568	21	2	40	Prolapsed arm	Internal Version	G.	S.B.	8	4	
1779	36	8	29	Twins	Internal Version	G.	D.	2	8	Extraction. 2nd of twins.
1949	35	6	40	Prolapse of cord	Internal Version	G.	S.B.	7	2	
T.Y.H. (1938)										
BOOKED										
483	29	4	39	Flat Pelvis	Caesarean Section	G.	G.	5	10	
927	19	1	40	—	Forceps	G.	S.B.	6	14	
2161	31	1	37	—	Manual Rotation and Forceps ...	G.	S.B.	6	8	
EMERGENCY										
2028/37	22	3	40	—	Internal Version	G.	S.B.	5	12	
186	28	4	39	—	Internal Version	G.	S.B	6	0	D.A.A.
416	25	3	36	Prolapsed arm	Internal Version	G.	S.B.	1	0	D.A.A.
740	25	2	37	—	External Version	G.	S.B.	1	0	Willett's Forceps.
857	25	3	34	Prolapsed arm	Internal Version	G.	S.B.	1	12	Impacted shoulders.
1026	28	4	25	—	Bipolar Version	G.	S.B.	1	8	
1067	32	1	38	—	Manual Rotation and Forceps ...	G.	G.	5	12	D.A.A.
1315	32	2	29	Placenta Praevia	Bipolar Version	G.	S.B.	3	8	
1384	35	6	29	Prolapsed cord & arm ...	Internal Version	G.	S.B.	4	8	Extraction.
1655	29	1	33	—	Manual Rotation and Forceps ...	G.	G.	6	0	Impacted head.
1696	22	4	40	Prolapsed cord	Manual Rotation and Forceps ...	G.	G.	7	0	
1740	28	2	40	Minor Pelvic contraction...	Manual Rotation and Forceps ...	G.	G.	6	12	
1866	32	2	27	—	Bipolar Version	G.	S.B.	1	13	Miscarriage.
2318	31	5	40	Prolapsed cord & hand ...	Internal Version	G.	G.	7	8	D.A.A.

UNCOMPLICATED BREECH DELIVERIES.

There were 114 cases.
In 76 cases the Breech was delivered spontaneously.
One mother died, a mortality of 1.3%.
24 babies were stillborn and 10 died, a mortality of 44.7%.
In 38 cases the delivery of the Breech was assisted.
No mother died.
7 babies were stillborn and 4 died, a mortality of 29%.

Reg. No.	*Age*	*Gravida*	*Maturity*	*METHOD OF DELIVERY*	*Result M.*	*Result C.*	*REMARKS*
G.C.H. (1937)							
EMERGENCY							
37	32	3	40	Assisted delivery	G.	S.B.	Extended arms brought down.
58	18	1	40	Assisted delivery	G.	G.	Arms brought down; Prolapse of cord. D.A.A.
179	35	2	37	Spontaneous delivery ...	G.	G.	Complete
220	23	2	29	Spontaneous delivery ...	G.	S.B.	Macerated foetus.
246	28	5	40	Assisted delivery	G.	D.	Extended arms and legs brought down.
290	24	1	37	Assisted delivery	G.	G.	A.P.H.
309	33	2	37	Spontaneous delivery ...	G.	G.	Extended legs.
T.Y.H. (1937)							
BOOKED							
322	20	1	41	Spontaneous delivery ...	G.	G.	Complete. Central Episiotomy.
EMERGENCY							
1605/36	23	1	40	Spontaneous delivery ...	G.	D.	
1616/36	23	3	39	Spontaneous delivery ...	G.	D.	
1622/36	20	1	40	Assisted delivery	G.	G.	Extended arms brought down.
8	22	2	40	Assisted delivery	G.	G.	Extended arms brought down.
97	19	1	40	Spontaneous delivery ...	G.	G.	Complete.
138	26	1	40	Assisted delivery	G.	G.	Arms brought down.
150	37	7	40	Spontaneous delivery ...	G.	G.	
196	37	8	35	Spontaneous delivery ...	G.	G.	Complete.
223	25	1	30	Spontaneous delivery ...	G.	S.B.	
228	42	6	40	Spontaneous delivery ...	G.	G.	
235	43	9	40	Spontaneous delivery ...	G.	G.	Complete. Pendulous Abdomen.
238	20	2	30	Spontaneous delivery ...	G.	S.B.	D.A.A.
247	29	3	31	Spontaneous delivery ...	G.	S.B.	
253	29	2	38	Spontaneous delivery ...	G.	D.	
277	21	1	40	Assisted delivery	G.	G.	P.P.H. Manual Removal of Placenta. D.A.A.
302	24	1	37	Spontaneous delivery ...	G.	G.	Central Episiotomy.
309	29	2	40	Assisted delivery	G.	G.	Legs brought down. Central Episiotomy.
107	28	1	40	Spontaneous delivery ...	G.	G.	Foot presentation.
146	36	2	40	Spontaneous delivery ...	G.	G.	Extended legs.

UNCOMPLICATED BREECH DELIVERIES.—*(Continued 1).*

Reg. No.	*Age*	*Gravida*	*Maturity*	*METHOD OF DELIVERY*	*Result M.*	*C.*	*REMARKS*
T.Y.H. (1937)							
EMERGENCY							
450	22	1	40	Assisted delivery	G.	G.	
466	19	1	40	Assisted delivery	G.	G.	Extended arms. Foot presentation.
582	28	2	30	Spontaneous delivery ...	G.	S.B.	
663	26	2	30	Assisted delivery	G.	D.	One leg and both arms extended.
704	22	1	40	Assisted delivery	G.	G.	Extended legs.
712	22	1	40	Assisted delivery	G.	G.	Extraction. Extended legs and arms.
731	30	2	30	Spontaneous delivery ...	G.	S.B.	
755	23	1	30	Spontaneous delivery ...	G.	S.B.	
804	31	4	40	Spontaneous delivery ...	G.	S.B.	Syphilis.
821	20	4	40	Spontaneous delivery ...	G.	S.B.	Malformation.
827	24	2	38	Spontaneous delivery ...	G.	G.	
852	27	4	40	Spontaneous delivery ...	G.	G.	Complete.
941	25	2	40	Assisted delivery	G.	G.	Incomplete.
980	23	1	40	Assisted delivery	G.	S.B.	Extended legs and arms.
1054	35	1	40	Spontaneous delivery ...	G.	G.	Complete.
1075	24	1	40	Spontaneous delivery ...	G.	G.	
1097	21	2	36	Assisted delivery	G.	S.B.	Extended legs and arms.
1199	23	2	34	Spontaneous delivery ...	G.	S.B.	Complete. D.A.A.
1289	27	4	40	Spontaneous delivery ...	G.	G.	Complete. D.A.A.
1290	28	2	38	Spontaneous delivery ...	G.	G.	Complete.
1308	31	7	40	Spontaneous delivery ...	G.	G.	Complete. D.A.A.
1324	33	2	40	Assisted delivery	G.	D.	Extended legs and arms. D.A.A.
1390	26	3	40	Spontaneous delivery ...	G.	G.	Incomplete.
1414	24	4	28	Spontaneous delivery ...	G.	S.B.	D.A.A.
1468	34	4	39	Spontaneous delivery ...	G.	G.	
1502	33	4	36	Spontaneous delivery ...	G.	G.	
1519	20	1	39	Spontaneous delivery ...	G.	G.	Complete.
1718	21	2	29	Spontaneous delivery ...	G.	S.B.	
1736	20	1	40	Assisted delivery	G.	D.	Extended arms.
1837	33	8	38	Spontaneous delivery ...	G.	G.	Complete.
1805	18	1	39	Spontaneous delivery ...	G.	G.	Local uterine infection. H.S.
1880	41	4	31	Spontaneous delivery ...	G.	S.B.	Complete. Pre-eclampsia.
T.Y.H. (1938)							
BOOKED							
42	23	1	40	Assisted delivery	G.	S.B.	Extended legs and arms brought down.
130	24	4	26	Spontaneous delivery ...	G.	S.B.	Miscarriage.
706	22	2	38	Assisted delivery	G.	G.	Extended legs and arms.
803	25	1	38	Spontaneous delivery ...	G.	D.	Complete.
1217	31	2	39	Spontaneous delivery ...	G.	G.	Extended legs and arms.
1838	21	1	40	Spontaneous delivery ...	G.	G.	Complete.
1931	21	2	40	Spontaneous delivery ...	G.	G.	Complete.
2212	41	4	37	Spontaneous delivery ...	G.	G.	Footling.

UNCOMPLICATED BREECH DELIVERIES.—*(Continued 2).*

Reg. No.	*Age*	*Gravida*	*Maturity*	*METHOD OF DELIVERY*	*Result M.*	*Result C.*	*REMARKS*
T.Y.H. (1938)							
EMERGENCY							
2011/37	22	1	37	Spontaneous delivery ...	G.	S.B.	Extended arms.
32	35	2	35	Spontaneous delivery ...	G.	S.B.	Complete. D.A.A.
33	38	7	38	Assisted delivery	G.	S.B.	Extended legs and arms brought down. D.A.A.
72	27	1	40	Assisted delivery	G.	G.	Extended legs and arms brought down.
112	23	1	29	Spontaneous delivery ...	G.	D.	
141	31	7	34	Spontaneous delivery ...	G.	D.	
144	24	2	36	Assisted delivery	G.	G.	Extended legs and arms.
186	21	1	40	Assisted delivery	G.	G.	Extended arms.
219	26	1	40	Assisted delivery	G.	G.	Extended arms brought down. Incomplete.
239	25	1	43	Spontaneous delivery ...	G.	G.	Complete.
318	33	6	40	Spontaneous delivery ...	G.	G.	Complete.
356	27	1	40	Assisted delivery	G.	G.	Extended legs and arms. D.A.A.
369	29	4	39	Spontaneous delivery ...	G.	G.	Complete.
450	32	9	40	Assisted delivery	G.	G.	Extended legs and arms.
479	25	3	40	Assisted delivery	G.	G.	Extended legs and arms.
492	23	1	40	Assisted delivery	G.	G.	Extended legs and arms.
504	25	1	39	Spontaneous delivery ...	G.	G.	Complete.
590	44	10	40	Assisted delivery	G.	S.B.	Extended legs and arms.
603	23	1	40	Assisted delivery	G.	G.	Extended legs and arms.
630	18	1	40	Assisted delivery	G.	G.	Extended legs and arms.
652	25	2	32	Spontaneous delivery ...	G.	S.B.	Complete.
678	28	3	40	Assisted delivery	G.	G.	Extended legs and arms.
726	36	11	32	Spontaneous delivery ...	G.	S.B.	Induction of labour.
780	35	5	40	Spontaneous delivery ...	G.	G.	Complete.
793	20	1	35	Spontaneous delivery ...	G.	D.	Extended legs and arms.
807	22	1	37	Spontaneous delivery ...	G.	D.	Extended legs. D.A.A.
820	22	2	31	Spontaneous delivery ...	G.	S.B.	
825	36	6	39	Assisted delivery	G.	S.B.	Footling.
1078	29	2	30	Spontaneous delivery ...	G.	S.B.	Complete.
1221	21	2	43	Spontaneous delivery ...	G.	G.	Footling.
1276	27	3	40	Spontaneous delivery ...	G.	D.	Complete.
1299	28	1	26	Spontaneous delivery ...	G.	S.B.	Footling. Miscarriage.
1330	26	3	37	Spontaneous delivery ...	G.	D.	
1519	21	1	31	Spontaneous delivery ...	G.	S.B.	Footling.
1583	23	3	40	Spontaneous delivery ...	G.	G.	Footling.
1730	38	9	30	Spontaneous delivery ...	G.	S.B.	Incomplete.
1808	32	7	28	Spontaneous delivery ...	G.	G.	Extended legs and arms.
1988	23	4	29	Spontaneous delivery ...	G.	S.B.	
1993	24	3	35	Spontaneous delivery ...	G.	G.	Complete.
2024	28	2	39	Spontaneous delivery ...	G.	G.	Extended legs and arms.
2035	23	1	40	Assisted delivery	G.	G.	Extended legs and arms.
2088	20	1	37	Spontaneous delivery ...	G.	G.	Complete.
2121	17	1	40	Assisted delivery	G.	G.	Extended legs and arms.
2137	28	3	40	Spontaneous delivery ...	G.	G.	Complete.
2151	28	2	40	Spontaneous delivery ...	G.	G.	Footling.
2313	22	1	40	Assisted delivery	G.	G.	Extended legs and arms.
2358	27	5	37	Spontaneous delivery ...	G.	G.	

COMPLICATED BREECH DELIVERIES (excluding Breech by Version).

There were 18 cases.

No mother died.

3 babies were stillborn and 6 died, a mortality of 50%.

Reg. No.	*Age*	*Gravida*	*Breech Maturity*	*Delivery with some other Obstetric Complication present. Complications*	*TREATMENT*	*Result M.*	*C.*	*REMARKS*
T.Y.H. (1937)								
EMERGENCY								
198	22	1	29	2nd of Twins ...	Legs and arms brought down ...	G.	D.	
823	21	1	40	1st of Twins ...	Spontaneous delivery	G.	G.	
1252	42	7	29	2nd of Twins ...	Spontaneous delivery	G.	S.B.	
1310	29	4	38	2nd of Twins ...	Spontaneous delivery	G.	D.	
1586	22	2	38	2nd of Twins ...	Spontaneous delivery	G.	D.	Syphilis.
1600	21	2	36	2nd of Twins ...	Spontaneous delivery	G.	D.	
1974	21	1	27	Placenta Praevia ...	Traction on Prolapsed leg	G.	S.B.	Complete.
T.Y.H. (1938)								
BOOKED								
965	27	5	40	2nd of Twins ...	Spontaneous delivery	G.	G.	
1423	10	1	33	2nd of Twins ...	Assisted delivery	G.	G.	Extended legs and arms.
EMERGENCY								
226	37	4	40	2nd of Twins ...	Spontaneous delivery	G.	G.	
302	32	10	38	2nd of Twins ...	Assisted delivery	G.	S.B.	Toxaemia.
391	20	1	40	2nd of Twins ...	Spontaneous delivery	G.	G.	Toxaemia.
594	29	6	30	2nd of Twins ...	Spontaneous delivery	G.	G.	
1306	31	4	27	2nd of Twins ...	Spontaneous delivery	G.	D.	
1378	30	2	39	2nd of Twins ...	Assisted delivery	G.	G.	Extended legs and arms.
1520	20	2	36	2nd of Twins ...	Spontaneous delivery	G.	G.	
1684	22	1	36	2nd of Twins ...	Spontaneous delivery	G.	G.	
1928	26	2	28	2nd of Twins ...	Spontaneous delivery	G.	D.	

TWINS (and Triplets if any).

There were 47 cases of Twins and none of Triplets.

One mother died, a mortality of 2.1%.

7 babies were stillborn and 24 died, a mortality of 33.1%.

Reg. No.	*Age*	*Gravida*	*Maturity*	*Position* 1st	*Position* 2nd	*Sex* 1st	*Sex* 2nd	*Weight* 1st lbs.	*Weight* 2nd lbs.	*Type*	*M.*	*Result* 1st	*Result* 2nd	*REMARKS*
G.C.H. (1937)														
EMERGENCY														
48	21	1	38	V1	V2	F.	F.	5. 4	5. 6	Uniovular	G.	G.	G.	
T.Y.H. (1937)														
EMERGENCY														
1685/36	26	8	40	V2	V1	F.	F.	5. 8	5. 8	Binovular	G.	G.	G.	
11	20	1	37	V2	V1	F.	F.	5.12	5.12	Binovular	G.	G.	G.	
198	22	1	29	V	B2	M.	M.	3. 0	2.12	Uniovular	G.	D.	D.	
284	22	2	40	V1	B2	F.	F.	5. 4	4.14	Uniovular	G.	G.	G.	
420	40	16	40	V1	V2	M.	F.	6. 8	5. 4	Binovular	G.	G.	G.	Prolapsed arm of 2nd baby.
560	35	3	35	V1	V2	F.	F.	5. 8	6. 0	Binovular	G.	G.	G.	
568	39	10	37	V	V	F.	F.	6.10	6. 8	Uniovular	G.	G.	G.	B.B.A.
590	21	1	34	V1	V2	F.	F.	2.14	3. 4	Uniovular	G.	D.	D.	
627	38	5	40	V1	V2	M.	M.	5. 8	6. 0	Binovular	G.	G.	G.	D.A.A.
828	21	1	40	B2	P.O.P.	F.	F.	5. 3	4. 4	Uniovular	G.	G.	D.	
1096	24	4	38	V1	V2	F.	M.	4.10	5. 0	Binovular	G.	G.	G.	D.A.A.
1252	42	7	29	V1	B1	F.	F.	2.11	4. 6	Binovular	G.	S.B.	S.B.	D.A.A.
1295	29	2	42	B.B.A.	Shoulder	M.	M.	6. 8	5. 0	Binovular	G.	G.	S.B.	
1310	29	4	33	V1	B1	M.	F.	4.12	4. 4	Binovular	G.	D.	D.	D.A.A.
1359	22	2	40	P.O.P.	V1	M.	M.	5. 8	6. 8	Binovular	G.	G.	G.	
1451	36	7	38	V2	V1	M.	M.	5. 8	6. 8	Uniovular	G.	G.	G.	D.A.A.
1530	36	7	30	V1	V1	M.	M.	5. 0	4. 8	Binovular	G.	G.	G.	
1536	22	2	38	V1	B1	M.	M.	3. 4	3. 0	Binovular	G.	D.	D.	
1580	37	8	36	V1	V2	M.	M.	5. 0	5.12	Uniovular	G.	G.	G.	
1600	21	2	36	V1	B	M.	M.	5. 8	2. 0	Uniovular	G.	G.	D.	
1619	30	2	34	V1	V1	F.	M.	1. 0	5. 1	Binovular	G.	G.	G.	
1720	21	1	40	V1	V1	F.	F.	1. 8	3. 8	Binovular	G.	G.	D.	
1779	36	9	29	V1	Shoulder	M.	F.	3.10	2. 8	Binovular	G.	D.	D.	

TWINS (and Triplets if any).—*(Continued).*

Reg. No.	*Age*	*Gravida*	*Maturity*	*Position* 1st	2nd	*Sex* 1st	2nd	*Weight* 1st lbs.	2nd lbs.	*Type*	*M.*	*Result* 1st	2nd	*REMARKS*
T.Y.H. (1938)														
BOOKED														
965	27	5	40	V1	B1	F.	F.	5.14	5. 1	Uniovular	G.	G.	G.	
1423	19	1	36	P.O.P.	B1	F.	F.	4. 2	4. 4	Uniovular	G.	G.	G.	
1761	27	1	30	V1	V1	M.	F.	5. 4	3.10	Binovular	G.	G.	G.	
EMERGENCY														
75	28	2	40	V1	V1	M.	F.	3. 0	4. 8	Binovular	G.	D.	D.	
80	29	1	30	V1	V1	F.	F.	5. 4	3. 1	Uniovular	G.	S.B.	S.B.	
226	37	4	40	V1	B3	F.	F.	6. 0	5. 4	Binovular	G.	G.	G.	D.A.A.
231	22	2	37	V1	V2	F.	F.	1. 0	4. 0	Binovular	G.	D.	D.	D.A.A.
302	32	10	33	V1	B2	F.	F.	4. 2	3. 0	Binovular	G.	D.	S.B.	D.A.A.
391	20	1	40	V1	B2	M.	M.	5. 8	5. 0	Uniovular	G.	G.	G.	
548	24	1	38	V1	V2	F.	F.	5. 4	5. 0	Binovular	G.	G.	G.	
594	29	6	39	V1	B	M.	M.	7.10	7. 8	Uniovular	G.	G.	G.	
600	24	2	40	V1	V2	M.	M.	4.10	1. 6	Uniovular	G.	G.	G.	
686	22	1	39	V1	V1	M.	M.	1.10	4. 4	Uniovular	G.	D.	G.	
1063	35	5	38	V1	P.O.P.	M.	M.	5. 0	5. 8	Binovular	G.	G.	G.	
1175	31	4	40	V1	V2	M.	M.	4.12	5. 8	Uniovular	G.	G.	G.	
1206	31	4	27	V1	B2	M.	M.	0.11	1. 0	Uniovular	G.	S.B.	D.	
1378	30	2	39	V1	B1	F.	F.	5. 2	4. 6	Uniovular	G.	G	G.	
1488	31	8	40	V1	V2	M.	M.	6.12	7. 0	Binovular	G.	G.	G.	
1502	20	2	36	V1	B2	F.	F.	4. 4	4. 2	Uniovular	G.	G.	G.	
1619	20	1	40	V1	V1	M.	M.	5. 0	3.12	Uniovular	D.	D.	D.	
1684	22	1	36	V3	B1	F.	F.	5. 8	5. 0	Binovular	G.	G.	G.	
1899	30	4	37	V1	V2	F.	F.	5. 0	5. 8	Binovular	G.	G.	G.	
1928	26	2	28	V1	B2	M.	M.	3.12	3. 0	Binovular	G.	D.	D.	Two placentae.

PROLAPSE OF CORD.

19 cases.

One mother died, a mortality of 5.3%.
11 babies were stillborn and 1 died, a mortality of 63.2%.

Reg. No.	Age	Gravida	Maturity	Size of os when diagnosed	TREATMENT	Result M.	Result C.	Complications	REMARKS
G.C.H. (1937)									
EMERGENCY									
58	18	1	40	Fully dilated ...	Nil	G.	G.	Breech	D.A.A.
246	28	5	40	Fully dilated ...	Cord pushed up ..	G.	D.	Breech	
T.Y.H. (1937)									
EMERGENCY									
73	29	4	40	Full dilatation ...	Nil	G.	G.	Nil	
1276	25	1	40	Full dilatation ...	Nil	G.	S.B.	Oedema	
							G.	Twins	
1530	36	7	30	Full dilatation ...	Nil	G.	G.		
1911	29	3	33	Full dilatation ...	Nil	G.	G.	Forceps. P.O.P.	
1949	35	6	40	½ dilated	Internal version ...	G.	S.B.	Transverse lie	
1974	24	1	27	½ dilated	Nil	G.	S.B.	Placenta Praevia	
T.Y.H. (1938)									
EMERGENCY									
2011/37	22	1	37	Full dilatation ...	Nil	G.	S.B.	Breech	
158	37	3	37	2 fingers	Nil	G.	S.B.	Placenta Praevia	D.A.A.
176	19	1	38	Full dilatation ...	Nil	G.	S.B.	Puerperal Pyrexia	D.A.A.
474	26	1	40	Full dilatation ...	Nil	D.	S.B.	P.O.P.	Manual Rotation and Forceps.
1334	35	6	39	½ dilated	Nil	G.	S.B.	Transverse	
1696	22	4	40	Full dilatation ...	Nil	G.	G.	Transverse lie	Long cord 28 inches.
1886	32	2	27	2 fingers	Nil	G.	S.B.	L. Placenta Praevia	Transverse. Bipolar version.
1993	24	3	35	Full dilatation ...	Nil	G	G.	Breech	
2090	20	1	36	Size of a dollar ...	Nil	G.	S.B.	Prolapsed hand	
2050	26	1	40	Full dilatation ...	Replaced	G.	S.B.	P.O.P.	
2318	31	5	40	Full dilatation ...	Internal version ...	G.	G.	Shoulder present	

ACCIDENTAL ANTE PARTUM HAEMORRHAGE.

8 cases.

No mother died.
5 babies were stillborn, a mortality of 62.5%.

Reg. No.	Age	Gravida	Maturity	Condition on Admission	Albumen	TREATMENT	Result M.	Result C.	Amount of Bleeding Concealed	Amount of Bleeding Revealed	REMARKS
G.C.H. (1937)											
EMERGENCY											
186	19	1	39	Anaemic...	½	Tight binder applied... ...	G.	S.B.	—	15 ozs.	
289	22	1	35	Oedema	¼	Sedative	G.	G.	—	3 ozs.	Toxaemic.
290	24	1	37	Slight oedema ...	Nil	Leg pulled down	G.	G.	—	10 ozs.	
T.Y.H. (1937)											
EMERGENCY											
1701	40	6	33	Slight bleeding ...	4+	General. A.R.M.	G.	S.B.	Combined	10 ozs.	D.A.A.
T.Y.H. (1938)											
EMERGENCY											
905	38	5	35	Fair	½	A.R.M.	G.	G.	—	6 ozs.	Toxaemia. D.A.A.
1287	31	4	36	Good	¼	A.R.M.. Binder applied ...	G.	S.B.	—	20 ozs.	D.A.A.
1320	37	8	30	Good	¼	Nil	G.	S.B.	20 ozs.	—	
2258	34	6	34	Anaemic...	½	A.R.M.	G.	S.B.	—	20 ozs.	D.A.A.

PLACENTA PRAEVIA.

41 cases.

In 3 cases the Placenta was Central.
In 15 cases the Placenta was Marginal.
In 23 cases the Placenta was Lateral.
One mother died, a mortality of 2.5%.
21 babies were stillborn and 9 died, a mortality of 73.2%.

Reg. No.	Age	Gravida	Maturity	Condition on Admission	Variety	Treatment	Result M.	Result C.	Amt. of Bleeding	Remarks
G.C.H. (1937)										
EMERGENCY										
215	41	13	39	Oedema	Marginal	Willett's Forceps...	G.	G.	18 ozs.	
218	33	7	39	Good	Marginal	Bipolar Version	G.	S.B.	10 ozs.	
241	29	3	38	Bleeding per Vaginam	Marginal	Willett's Forceps...	G.	D.	12 ozs.	
T.Y.H. (1937)										
BOOKED										
1016	27	2	28	Good	Marginal	Internal Version	G.	S.B.	6 ozs.	Old Tb. hip. D.A.A.
EMERGENCY										
54	30	5	33	A.P.H.	Marginal	Willett's Forceps...	G.	G.	6 ozs.	
264	45	13	24	Good	Marginal	Willett's Forceps...	G.	G.	25 ozs.	
528	25	8	10	Good	Lateral	Willett's Forceps...	G.	G.	10 ozs.	D.A.A.
607	30	1	37	Good	Lateral	Willett's Forceps...	G.	G.	10 ozs.	
655	34	6	36	Good	Marginal	Willett's Forceps...	G.	S.B.	2 ozs.	
657	21	1	34	Good	Lateral	Morphia	G.	S.B.	30 ozs.	
688	28	5	37	Fainted	Central	Willett's Forceps...	G.	D.	70 ozs.	Caesarean Section.
796	27	2	37	Good	Lateral	Willett's Forceps...	G.	G.	26 ozs.	D.A.A.
985	28	3	34	Transverse lie	Central	Bipolar Version	G.	S.B.	30 ozs.	D.A.A.
1007	38	9	33	Abdominal pain ...	Lateral	A.R.M.	G.	S.B.	50 ozs.	D.A.A.
1013	26	6	40	Good	Lateral	A.R.M.	G.	G.	20 ozs.	
1337	31	7	32	Good	Marginal	Willett's Forceps...	G.	G.	4 ozs.	D.A.A.
1398	25	2	32	Fair	Lateral	Willett's Forceps...	G.	S.B.	10 ozs.	
1453	31	5	34	Good	Lateral	Internal Version	G.	S.B.	14 ozs.	Shoulder presentation.
1535	39	9	28	Good	Marginal	Willett's Forceps...	G.	D.	10 ozs.	D.A.A.
1675	21	2	35	Good	Marginal	Willett's Forceps...	G.	S.B.	30 ozs.	
1974	24	1	27	Slight bleeding	Lateral	Traction on prolapsed legs ...	G.	S.B.	20 ozs.	Prolapsed cord.
T.Y.H. (1938)										
EMERGENCY										
2023/37	22	3	40	Good	Lateral	Internal Version	G.	S.B.	Normal	
136	28	4	39	Good	Lateral	Internal Version	G.	S.B.	20 ozs.	D.A.A.
153	37	3	37	Good	Marginal	Willett's Forceps...	G.	S.B.	80 ozs.	A.R.M. D.A.A.
154	29	6	36	Anaemic	Lateral	A.R.M.	G.	S.B.	30 ozs.	D.A.A.
266	31	6	40	Good	Marginal	Willett's Forceps...	G.	G.	30 ozs.	A.R.M. D.A.A.
360	20	1	38	Bleeding	Central	Willett's Forceps...	D.	S.B.	20 ozs.	Discharging Bartholin's abscess.
662	41	12	38	Slight oedema	Marginal	Willett's Forceps...	G.	D.	15 ozs.	A.R.M.
727	28	3	40	Slight bleeding	Lateral	Spontaneous delivery	G.	G.	12 ozs.	
746	40	3	32	Slight bleeding	Lateral	Willett's Forceps...	G.	D.	20 ozs.	A.R.M. D.A.A.
777	37	8	32	Fair	Lateral	Willett's Forceps...	G.	S.B.	10 ozs.	A.R.M.
1020	35	9	36	Good	Lateral	A.R.M.	G.	S.B.	20 ozs.	D.A.A.
1026	28	1	25	Good	Lateral	Bipolar Version	G.	S.B.	6 ozs.	
1117	30	5	35	Good	Lateral	Willett's Forceps...	G.	S.B.	20 ozs.	
1164	22	2	28	Good	Lateral	A.R.M.	G.	D.	10 ozs.	
1276	27	3	40	Good	Lateral	Traction of limbs...	G.	D.	8 ozs.	
1815	32	2	29	Good	Marginal	Bipolar Version	G.	S.B.	30 ozs.	
1480	26	3	40	Good	Marginal	Willett's Forceps...	G.	G.	30 ozs.	A.R.M.
1615	30	7	37	Marked oedema... ...	Lateral	Willett's Forceps...	G.	G.	15 ozs.	A.R.M.
1886	32	2	27	Pulmonary Tb.	Lateral	Bipolar Version	G.	S.B.	12 ozs.	
1988	26	2	28	Good	Lateral	Nil	G.	D. / D.	20 ozs.	Twins One of the 2 placentae.

PRIMARY UTERINE INERTIA.

(Arbitrary definition being the first stage of labour lasting 48 hours or more).

Reg. No.	Age	Gravida	Maturity	Position of Fœtus	Time of Rupture of Membranes	Other Obstetric Abnormalities	Int. Spin.	Int. Crist.	Ext. Conj.	D.C.	Duration of Labour 1st St.	2nd St.	Method of Delivery	Treatment Medical	Treatment Operative	Weight of Child lb.	Weight of Child oz.	Result M.	Result C.	
T.Y.H. (1937)																				
EMERGENCY																				
127	20	1	40	V3	—	P.O.P.	9½″	10″	8″	—	55½ hours	—	Forceps	Yes	Yes	7	1	G.	G.	
T.Y.H. (1937)																				
BOOKED																				
301	37	3	40	V4	28 hrs.	P.P.H.	9	10	7	—	106 hrs.	1¼ hrs.	Forceps	Yes	Yes	7	0	G.	G.	
350	24	1	42	V4	25 mins.	Trial labour	9	10	7½	—	52¾ hrs.	25 mins.	Posterior lie	Yes	No	7	2	G.	G.	
EMERGENCY																				
94	25	1	41	V3	3 hrs.	P.O.P.	9	10	7	—	118½ hrs.	2 hrs.	Forceps	Yes	Forceps	6	12	G.	G.	
177	28	1	40	V1	2¾ hrs.	Nil	9½	10½	7	—	50 5/12 hrs.	14¾ hrs.	Normal	Yes	Nil	6	2	G.	G.	
495	21	1	42	V4	11 hrs.	Eclampsia	8¾	9½	7½	—	51¼ hours	—	Forceps	Sedative	Forceps	6	6	G.	G.	
684	26	1	40	P.O.P.	—	Manual Rotation ...	9	10	7½	—	62 hours	—	Forceps	Yes	Yes	6	6	G.	D.	
818	28	1	38	V2	3¼ hrs.	Nil	9½	10½	6¾	—	55¼ hrs.	3¼ hrs.	Normal	Yes	Nil	6	1	G.	G.	
T.Y.H. (1938)																				
BOOKED																				
927	19	1	40	T.	49 hrs.	Nil	9½	10½	8½	—	53 hrs.	1 hr.	Forceps	Yes	Yes	6	14	G.	S.B.	
EMERGENCY																				
288	22	1	40	V3	7 hrs.	P.O.P.	8½	9½	7½	—	51½ hrs.	2½ hrs.	Forceps	Yes	Yes	6	0	G.	S.B.	
357	26	1	40	V2	18 hrs.	Fœtal distress ...	10	11	7	—	50 hours	—	Forceps	Yes	Yes	6	10	G.	G.	
474	26	1	40	V3	77 hrs.	Prolapsed cord ...	10	10½	7	—	74 hrs.	2 hrs.	Forceps	Yes	Yes	7	4	D.	S.B.	P.O.P.
2100	29	1	40	V3	30 hrs.	P.P.H.	9	10½	7½	—	66½ hrs.	5½ hrs.	Forceps	Yes	Yes	7	0	D.	S.B.	

CASES OF ALBUMINURIA ADMITTED FOR TREATMENT.

136 cases.

6 mothers died, a mortality of 4.4%.

25 babies were stillborn and 16 died, a mortality of 27.9%.

No. Reg.	Age	Gravida	Maturity	History of Renal Disease	Albuminuria on admission	Albuminuria on discharge	Oedema	Headache	Eye Signs	Highest Blood Pressure	No. of days in Hospital before labour or discharge	Type of labour	Result Mother	Result Child	Remarks
G.C.H. (1937)															
EMERGENCY															
11	27	2	39	Nil	†	Clear	Legs	Nil	Nil	120/64	In labour	Normal	G.	G.	
19	24	3	40	Nil	††	Clear	Legs and Abdomen ...	Nil	Nil	110/98	In labour	Normal	G.	G.	
29	22	2	29	Nil	††	Clear	Nil	Nil	Nil	100/80	In labour	Normal	G.	G.	
31	38	7	41	Nil	†††	Clear	Legs	Nil	Nil	154/100	In labour	Normal	G.	G.	
36	30	1	37	Nil	†	Clear	General	Nil	Dimness	170/110	In labour	Normal	G.	S.B.	Ankylostomiasis Avitaminosis B1
64	38	6	40	Nil	†	Clear	Legs	Nil	Nil	172/110	In labour	Normal	G.	G.	
99	33	5	35	Nil	††	Clear	General	Nil	Yes	138/98	In labour	Normal	G.	G.	Avitaminosis B1 D.A.A.
125	30	2	41	Nil	††	Clear	Legs	Nil	Nil	118/90	In labour	Normal	G.	G.	
144	22	1	40	Nil	††	Clear	Nil	Nil	Nil	122/80	In labour	Normal	G.	G.	
154	36	11	40	Nil	†	Clear	Nil	Nil	Nil	174/110	In labour	Normal	G.	G.	
170	26	1	41	Nil	Trace	Nil	General	Yes	Nil	190/110	In labour	Normal	G.	S.B.	
186	19	1	39	Nil	††	Clear	Nil	Nil	Nil	120/68	In labour	Normal	G.	S.B.	
196	27	5	40	Nil	†	Nil	Legs	Nil	Nil	124/94	In labour	Normal	G.	G.	
206	25	2	37	Nil	††	Nil	Face & Extremities...	Nil	Nil	158/108	In labour	Normal	G.	G.	Ankylostomiasis.
299	22	1	35	Nil	††	Nil	Legs & feet	Nil	Nil	162/78	In labour	Normal	G.	G.	D.A.A.
311	37	7	38	Nil	†††	Trace	Legs	Yes	Yes	168/124	In labour	Normal	G.	G.	Pre-eclampsia.
T.Y.H. (1937)															
BOOKED															
272	22	1	40	Nil	††	Clear	Legs	Nil	Nil	144/130	In labour	Normal	G.	G.	
1157	21	1	37	Nil	†	Trace	Feet-Legs	Yes	Nil	134/84	In labour	Normal	G.	G.	Pre-eclampsia. D.A.A.
1853	28	1	35	Nil	Trace	Clear	Legs	Yes	Yes	126/90	In labour	Forceps	G.	G.	
EMERGENCY															
1606/36	23	2	40	Nil	†††	Clear	Legs-Hands	Yes	Yes	112/90	In labour	Normal	G.	G.	Anaemia, D.A.A.
1626/36	30	3	40	Nil	†	Clear	Legs & Abdominal wall	Nil	Nil	175/120	In labour	Normal	G.	G.	
20	37	2	40	Nil	†	Clear	Legs	Nil	Nil	132/88	In labour	Normal	G.	D.	Central Episiotomy.
41	21	1	40	Nil	Trace	Clear	Legs	Nil	Nil	154/100	In labour	Normal	G.	G.	D.A.A.
75	33	1	40	Nil	††	Clear	Legs	Nil	Nil	150/100	In labour	Forceps	G.	G.	Subinvolution.
186	24	2	33	Nil	††	Clear	General	Nil	Nil	172/90	In labour	Normal	G.	D.	
228	42	6	40	Nil	†	Clear	Legs	Nil	Nil	138/92	In labour	Normal	G.	G.	
235	43	9	40	Nil	†	Clear	Legs & Lower abdomen	Nil	Nil	144/98	In labour	Normal	G.	G.	D.A.A.
240	25	1	40	Nil	†	Clear	Legs	Nil	Nil	158/102	In labour	Normal	G.	G.	
244	32	1	40	Nil	†	Clear	Nil	Nil	Nil	120/88	In labour	Normal	G.	G.	
251	22	1	40	Nil	††	Clear	Legs	Nil	Nil	144/110	In labour	Normal	G.	G.	
38a	41	8	32	Nil	††	—	General	Nil	Nil	176/110	In labour	Induction	D.	G.	P.P.H.
366	38	10	40	Nil	††	†	Legs	Nil	Nil	124/62	In labour	Normal	G.	G.	Haematuria, D.A.A.
414	31	1	40	Nil	†	Clear	Legs	Nil	Nil	158/90	In labour	Normal	G.	G.	
431	43	12	32	Nil	†††	—	Nil	Nil	Nil	—	In labour	Normal	D.	S.B.	On admission weak and pale, Severe A.P.H.
570	23	1	40	Nil	Clear	Clear	Legs	Yes	Yes	152/110	In labour	Normal	G.	G.	D.A.A.
629	39	4	39	Nil	††	Clear	Legs	Nil	Nil	104/140	In labour	Normal	G.	G.	Pre-eclampsia.
643	28	1	42	Nil	†††	Clear	Legs	Yes	Nil	206/120	In labour	Normal	G.	S.B.	Pre-eclampsia. Induction of labour.
678	32	7	38	Nil	†††	Trace	Generalised	Yes	Yes	200/144	In labour	Normal	G.	G.	Pre-eclampsia. Induction of labour. D.A.A.
753	23	1	30	Nil	††	Clear	Legs	Yes	Yes	178/132	In labour	Breech	G.	S.B.	Induction of labour.
788	22	1	40	Nil	Nil	Clear	Legs	Nil	Nil	156/122	In labour	Normal	G.	D.	
907	20	3	34	Nil	†††	†	Legs	Nil	Nil	134/84	In labour	Normal	G.	G.	Pre-eclampsia.
969	22	1	35	Nil	††	Clear	General	Nil	Nil	164/102	In labour	Normal	G.	S.B.	Pre-eclampsia.

CASES OF ALBUMINURIA ADMITTED FOR TREATMENT.- *(Continued).*

Reg. No.	Age	Gravida	Maturity	History of Renal Disease	Albuminuria on admission	Albuminuria on discharge	Oedema	Headache	Eye Signs	Highest Blood Pressure	No. of days in Hospital before labour or discharge	Type of labour	Result Mother	Result Child	Remarks
T.Y.H. (1937)															
EMERGENCY															
904	22	4	34	Nil	+	Clear	Legs & Abdominal wall	Yes	Nil	222/160	In labour	Shoulder presentation	G.	S.B.	Pre-eclampsia, Macerated Foetus.
951	10	6	36	Nil	+	Clear	Legs	Yes	Yes	132/80	In labour	Normal	G.	G.	Pre-eclampsia, D.A.A.
994	30	3	40	Nil	+	Clear	Legs	Nil	Nil	160/104	In labour	Normal	G.	G.	
1044	28	1	10	Nil	Nil	Nil	Legs	Yes	Yes	140/96	In labour	Normal	G.	G.	Pre-eclampsia, D.A.A.
1050	14	8	10	Yes	+	+	Legs-Feet	Nil	Nil	196/120	In labour	Normal	G.	G.	Chronic Nephritis. D.A.A.
1078	30	3	10	Nil	+	Clear	Legs	Nil	Nil	160/90	In labour	Normal	G.	G.	Pre-eclampsia.
1091	31	4	37	Nil	Trace	Trace	Legs	Yes	Yes	154/108	In labour	Normal	G.	G.	Pre-eclampsia, D.A.A.
1097	21	2	36	Nil	++	Clear	Legs	Nil	Nil	162/104	In labour	Assisted Breech...	G.	S.B.	Pre-eclampsia, D.A.A.
1118	22	2	39	Nil	Trace	Clear	Legs-Feet	Nil	Nil	148/104	In labour	Normal	G.	G.	Pre-eclampsia, D.A.A.
1138	41	6	37	Nil	++	Clear	Legs	Nil	Nil	160/44	In labour	Normal	G.	S.B.	D.A.A.
1210	39	7	39	Nil	++	Clear	Legs	Nil	Nil	144/88	In labour	Normal	G.	G.	D.A.A.
1276	25	1	40	Nil	+	Clear	Legs	Nil	Nil	160/92	In labour	Normal	G.	S.B.	D.A.A.
1295	29	2	42	Nil	++	Trace	Legs	Nil	Nil	152/102	In labour	Assisted	G.	G. S.B.	2nd of twins (1st of twins B.B.A.). D.A.A.
1274	27	3	36	Yes	+++	++	General	Nil	Nil	184/108	In labour	Normal	G.	G.	Pre-eclampsia, Chronic Nephritis.
1344	38	4	40	Nil	Trace	Clear	Legs	Nil	Nil	150/90	In labour	Normal	G.	G.	
1347	32	6	38	Nil	Trace	Clear	Legs	Nil	Nil	162/100	In labour	Normal	F.	S.B.	D.A.A.
1354	21	2	40	Nil	Clear	Clear	Legs	Nil	Nil	182/130	In labour	Normal	G.	G.	Pre-eclampsia. Induction of labour D.A.A.
1359	22	2	40	Nil	+	Clear	Legs	Nil	Nil	140/84	In labour	Normal	G.	G. G.	Twins.
1380	27	1	36	Nil	Trace	Clear	Legs	Nil	Nil	152/110	In labour	Normal	G.	G.	D.A.A.
1488	35	2	40	Nil	++	Trace	Legs-Feet	Nil	Nil	145/95	In labour	Normal	F.	G.	Pre-eclampsia. Avitaminosis B1 D.A.A.
1500	20	1	35	Nil	Clear	Clear	Legs-Feet	Nil	Nil	151/118	In labour	Normal	G.	G.	Pre-eclampsia Syphilis.
1507	23	1	31	Nil	Trace	Clear	Legs	Nil	Nil	158/90	In labour	Normal	G.	D.	D.A.A.
1528	31	2	40	Nil	Trace	Clear	Legs	Nil	Nil	152/106	In labour	Normal	G.	G.	D.A.A.
1538	25	1	10	Nil	Clear	Clear	Legs	Nil	Nil	150/90	In labour	Normal	G.	G.	Pre-eclampsia.
1549	21	1	34	Nil	Trace	Clear	Legs	Nil	Nil	154/90	In labour	Normal	G.	G.	Ascariasis. D.A.A.
1581	31	5	40	Nil	Trace	Clear	Feet, Vulva	Nil	Nil	128/84	In labour	Normal	G.	D.	Avitaminosis B1 D.A.A.
1584	31	6	34	Nil	Trace	Clear	General	Nil	Nil	165/95	In labour	Normal	G.	G.	Mitral Stenosis with Regurgitation.
1586	37	9	10	Nil	Trace	Clear	Legs	Nil	Nil	170/108	In labour	Right Cleidotomy	G.	S.B.	Pre-eclampsia.
1589	23	3	10	Nil	Trace	Clear	Legs	Nil	Nil	150/98	In labour	Normal	G.	G.	Pre-eclampsia.
1612	21	1	10	Nil	Clear	Clear	Legs-Feet	Nil	Nil	152/95	In labour	Normal	G.	G.	Pre-eclampsia.
1615	19	1	36	Nil	Trace	Clear	Legs	Nil	Nil	144/90	In labour	Normal	G.	G.	Pre-eclampsia.
1616	31	9	36	Nil	+	Clear	General	Nil	Nil	142/80	In labour	Normal	G.	G.	Pre-eclampsia.
1720	21	1	40	Nil	+	Clear	Legs	Nil	Yes	150/118	In labour	Normal	G.	G. D.	Pre-eclampsia Avitaminosis B1 Syphilis. Twins. D.A.A.
1886	27	3	38	Nil	Trace	Clear	Legs	Nil	Yes	132/92	In labour	Normal	G.	G.	Pre-eclampsia.
1889	41	1	31	Nil	++	—	General	Yes	Nil	134/94	In labour	Breech	D.	S.B.	Pre-eclampsia.
1980	26	5	40	Nil	+	Clear	Legs	Nil	Nil	180/96	In labour	Normal	G.	G.	
1994	40	8	40	Nil	Clear	Clear	Legs	Nil	Nil	186/110	In labour	Normal	G.	G.	
2002	26	1	40	Nil	+	Clear	Legs	Yes	Yes	170/110	In labour	Normal	G.	G.	
2007	20	1	36	Nil	Clear	Clear	Legs	Yes	Yes	140/98	In labour	P.O.P.	G.	G.	
2013	27	5	38	Nil	+	Clear	Legs	Nil	Nil	160/106	In labour	Normal	G.	G.	
T.Y.H. (1938)															
EMERGENCY															
1992/37	32	2	39	Nil	+	Clear	Legs	Nil	Nil	142/86	In labour	Normal	G.	G.	
75	29	2	40	Nil	++	Clear	Legs	Nil	Nil	166/112	In labour	Normal	G.	D. D.	Twins.
80	29	1	38	Nil	+	Clear	Feet-Legs	Nil	Nil	164/102	In labour	Forceps	G.	S.B. S.B.	Twins.
104	29	3	38	Nil	+	Clear	Legs	Nil	Nil	134/78	In labour	Normal	G.	G.	D.A.A.
107	35	8	38	Nil	++	—	Legs	Nil	Nil	168/94	In labour	Normal	D.	D.	Pre-eclampsia.
152	19	1	40	Nil	+	Clear	Legs	Nil	Nil	166/110	In labour	Normal	G.	G.	Pre-eclampsia.

CASES OF ALBUMINURIA ADMITTED FOR TREATMENT.—(*Continued 2*).

No. Reg.	*Age*	*Gravida*	*Maturity*	*History of Renal Disease*	*Albuminuria on admission*	*Albuminuria on discharge*	*Oedema*	*Headache*	*Eye Signs*	*Highest Blood Pressure*	*No. of days in Hospital before labour or discharge*	*Type of labour*	*Result Mother*	*Result Child*	*Remarks*
T.Y.H. (1938)															
EMERGENCY															
190	20	1	36	Nil	+	Clear	Legs-Vulva	Yes	Nil	178 128	In labour	Normal	G.	S.B.	Pre-eclampsia.
226	37	4	40	Nil	++	Clear	Legs	Nil	Nil	188 80	In labour	Normal	G.	G.	Twins D.A.A.
240	36	1	37	Nil	±	Clear	Legs	Nil	Nil	198 124	In labour	Normal	G.	G.	D.A.A.
300	38	2	36	Nil	Trace	Clear	Legs	Yes	Nil	152 100	In labour	Normal	G.	G.	
302	32	10	33	Nil	+	Clear	Legs	Nil	Nil	160 102	In labour	Spontaneous ...	G.	D.	Twins D.A.A.
313	23	1	40	Nil	+	Clear	Legs	Nil	Nil	144 100	In labour	Forceps	G.	S.B.	P.O.P. Manual Rotation D.A.A.
315	26	1	40	Nil	+	Clear	Legs	Nil	Nil	166 90	In labour	Normal	G.	G.	D.A.A.
325	23	1	37	Nil	Trace	Clear	Legs	Yes	Yes	150 110	In labour	Normal	G.	S.B.	Avitaminosis B1 D.A.A.
367	26	6	36	Nil	+	Clear	Legs	Nil	Yes	110 78	In labour	Normal	G.	G.	Avitaminosis B1.
410	25	1	40	Nil	Trace	Clear	Legs	Nil	Nil	126 68	In labour	Normal	G.	G.	
422	36	4	41	Nil	Trace	Clear	Legs	Nil	Nil	146 92	In labour	P.O.P.	G.	S.B.	
434	24	1	40	Nil	++	Clear	General	Slight	Slight	190 [illegible]	In labour	Normal	G.	S.B.	D.A.A.
466	25	1	40	Nil	+	Clear	Legs	Nil	Nil	134 80	In labour	Normal	G.	G.	D.A.A.
527	26	1	37	Nil	+	Clear	Legs	Nil	Nil	184 102	In labour	Normal	G.	G.	Hydramnios D.A.A.
590	41	10	40	Nil	+	Clear	Legs	Nil	Nil	178 94	In labour	Breech	G.	S.B.	
612	40	8	39	Nil	±	Clear	Legs	Nil	Nil	174 102	In labour	Forceps	G.	G.	P.O.P., Manual Rotation, D.A.A.
643	26	2	38	Nil	±	Clear	Legs-Feet	Nil	Slight	164 114	In labour	Normal	G.	G.	D.A.A.
529	27	2	39	Nil	Trace	Clear	Legs	Nil	Nil	154 120	In labour	Normal	G.	G.	Avitaminosis B1.
759	28	3	40	Nil	±	Clear	Legs	Yes	Nil	146 90	In labour	Normal	G.	G.	
804	18	1	33	Nil	+	Clear	Legs	Nil	Nil	140 88	In labour	Normal	G.	D.	
905	38	5	35	Nil	++	Clear	Legs	Yes	Yes	164 [illegible]10	In labour	Normal	G.	G.	Pre-eclampsia. D.A.A.
1034	28	2	38	Nil	+	Clear	Legs	Yes	Yes	124 76	In labour	Normal	G.	G.	
1067	32	1	38	Nil		Clear	Legs	Slight	Nil	112 98	In labour	Forceps	G.	G.	Transverse lie, Manual Rotation and Forceps, D.A.A.
1147	25	1	36	Yes	++	--	Legs	Nil	Nil	180 120	On labour	Normal	D.	G.	Pre-eclampsia Chronic Nephritis.
1238	30	5	39	Nil	Trace	Clear	Legs	Nil	Nil	178 110	In labour	Normal	G.	G.	
1324	30	4	37	Nil	++	Clear	Legs	Nil	Nil	172 92	In labour	Normal	G.	G.	A.R.M.
1419	22	1	40	Nil	+	Clear	Legs	Nil	Nil	152 84	In labour	Forceps	G.	G.	Pre-eclampsia. D.A.A.
1431	23	1	40	Nil	+	Clear	Legs	Yes	Yes	130 80	In labour	Normal	G.	G.	
1506	27	1	40	Nil	+	Clear	Legs	Yes	Yes	160 100	In labour	Normal	G.	G.	Pre-eclampsia.
1524	24	1	37	Nil	+	Clear	Legs	Nil	Nil	106 76	In labour	Normal	G.	G.	Cystitis. D.A.A.
1565	23	1	40	Nil	++	Clear	Legs	Yes	Yes	146 100	In labour	Normal	G.	G.	Pre-eclampsia.
1614	20	1	40	Nil	+	Clear	Legs	Nil	Nil	160 90	In labour	Forceps	G.	D.	P.O.P., Manual Rotation, Pre-eclampsia.
1653	30	1	36	Nil	+	Clear	Legs	Nil	Nil	148 100	In labour	Normal	G.	G.	D.A.A.
1683	26	1	35	Nil	+	Clear	Legs	Nil	Nil	154 80	In labour	Normal	G.	G.	Pre-eclampsia.
1684	22	1	36	Nil	++	Clear	Legs	Nil	Nil	158 124	In labour	Forceps	G.	G.	Twins Pre-eclampsia.
1619	20	1	40	Nil	++	--	Legs	Yes	Nil	178 120	In labour	Forceps	D.	D.	Twins Pre-eclampsia.
1723	35	6	40	Nil	Trace	Clear	Legs	Nil	Nil	156 100	In labour	Normal	G.	G.	Anaemia.
1905	25	3	40	Nil	Trace	Clear	Legs	Slight	Slight	146 102	In labour	Normal	G.	G.	
1909	28	5	39	Nil	Trace	Clear	Legs	Nil	Nil	120 90	In labour	Normal	G.	G.	D.A.A.
1934	27	1	41	Nil	Trace	Clear	Legs	Slight	Slight	150 110	In labour	Normal	G.	G.	
1961	19	1	39	Nil	+	Clear	Legs	Nil	Nil	154 108	In labour	Normal	G.	G.	D.A.A.
2055	22	1	40	Nil	++	Clear	Legs	Nil	Nil	142 110	In labour	Normal	G.	D.	
2062	35	7	40	Nil	Trace	Clear	Legs	Nil	Nil	162 110	In labour	Normal	G.	G.	D.A.A.
1986	23	1	39	Nil	+	Clear	Legs	Yes	Yes	148 106	In labour	Normal	G.	D.	
2090	28	1	36	Nil	++	Clear	Legs	Yes	Yes	168 86	In labour	Normal	G.	G.	A.R.M. Pre-eclampsia, D.A.A.
2176	37	6	38	Nil	++	Clear	Legs	Yes	Yes	232 160	In labour	Normal	G.	S.B.	Pre-eclampsia.
2849	21	2	32	Nil	+++	Clear	Legs	Nil	Yes	160 100	In labour	Normal	G.	G.	Pre-eclampsia.
2206	24	1	40	Nil	+++	Clear	Legs	Yes	Yes	178 120	In labour	Normal	G.	S.B.	Pre-eclampsia.

ECLAMPSIA.

17 cases.

No mother died.

One baby was stillborn and one died, a mortality of 11.8%.

Reg. No.	Age	Gravida	Maturity	Condition on admission (including if in labour)	No. before admission	FITS Total	FITS Onset	URINE Albumen on admission	URINE Albumen on discharge	URINE Quantity in first 24 hrs.	Oedema	Highest Blood Pressure		Head ache	Eye Signs	No. of days in Hospital before delivery	Type of labour	Result M.	Result C.	REMARKS
G.C.H. (1937)																				
EMERGENCY																				
295	24	1	35	Oedema	Nil	5	Post-partum	+	Clear	21 ozs.	Generalised ...	198	140	Yes	Yes	1	Normal	G.	G.	
T.Y.H. (1937)																				
EMERGENCY																				
495	21	1	12	Oedema of legs and Abd. wall	Nil	1	Intra-partum	+	Clear	21 ozs.	Legs, Abd. wall	172	128	Nil	Nil	1	Forceps	G.	G.	
1523	19	1	40	Good	Nil	3	Post-partum	+	Clear	40 ozs.	Legs	146	104	Nil	Yes	1	Normal	G.	G.	
1734	28	1	39	Good	Nil	1	Post-partum	Clear	Clear	62 ozs.	General ...	164	112	Nil	Nil	-	Normal	G.	G.	Avitaminosis B_1.
1799	18	1	40	Had 2 fits at home. Good ...	2	8	Ante-partum	++	Clear	12 ozs.	Legs	138	96	Nil	Yes	--	Forceps	G.	G.	
1891	17	1	40	Good	1	1	Post-partum	Trace	Clear	10 ozs.	Legs	151	91	Nil	Nil	—	Normal	G.	G.	
T.Y.H. (1938)																				
BOOKED																				
2044/37	27	1	40	Good	2	2	Ante-partum	Clear	Clear	46 ozs.	Legs	160	108	Nil	Nil	7	Normal	G.	G.	
EMERGENCY																				
2020/37	19	1	40	Marked Oedema	1	6	Intra-partum	++	Clear	20 ozs.	Face, Limbs	171	121	Yes	Yes	—	Forceps	G.	D.	A.R.M.
192	23	1	35	Semi-comatose	3	5	Ante-partum	++	Clear	25 ozs.	Legs	150	110	Yes	Yes	—	Forceps	G.	G.	
228	36	5	39	Marked Oedema of legs ...		2	Post-partum	+	Clear	35 ozs.	Legs	182	116	Nil	Yes	—	Normal	G.	G.	Syphilis. D.A.A.
391	20	1	40	Oedema	-	1	Post-partum	++	Clear	62 ozs.	Legs	158	102	Yes	Nil	—	Normal	G.	G.	Twins.
531	25	2	40	Oedema		2	Post-partum	+	+	12 ozs.	Legs	180	120	Nil	Yes	--	Forceps	G.	G.	P.O.P. Manual Rotation. Ascariasis.
665	21	1	40	Oedema		1	Post-partum	Clear	Clear	16 ozs.	Legs	160	116	Yes	Yes	1	Normal	G.	G.	D.A.A. D.A.A
1023	33	1	35	Marked Oedema		1	Ante-partum	+++	Clear	39 ozs.	Legs	198	102	Nil	Nil	6	Forceps	G.	G.	Induction of labour.
1298	26	1	39	Oedema	-	1	Ante-partum	Trace	Clear	17 ozs.	Legs	156	96	Nil	Nil	1	Forceps	G.	S.B.	P.O.P. Manual Rotation.. D.A.A.
1527	19	1	40	Oedema	-	1	Ante-partum	+	Clear	18 ozs.	Legs	150	106	Nil	Nil	1	Forceps	G.	G.	
1699	30	4	37	Oedema	—	6	Post-partum	Trace	Clear	15 ozs.	Legs	166	106	Yes	Yes	—	Normal	G.	G.	Twins.

CARDIAC DISEASE.

10 cases.

3 mothers died, a mortality of 30%.

One baby was stillborn and one died, a mortality of 25%.

Reg. No.	*Age*	*Gravida*	*Maturity*	*Lesion*	*Degree of failure of Compensation*	*Days in Hsp. before Delivery*	*Method of Delivery*	*Result M.*	*Result C.*	*REMARKS*
G.C.H. (1937)										
EMERGENCY										
214	35	5	37	Mitral Stenosis ...	Marked	In labour	Normal	D.	G.	Avitaminosis B.
T.Y.H. (1937)										
BOOKED										
723	40	12	31	Mitral Stenosis ...	Moderate	7	Normal	P.	D.	D.A.A.
EMERGENCY										
634	30	4	30	Mitral Stenosis ...	Marked	11	Not in labour	P.	—	D.A.A.
923	29	2	36	Mitral Stenosis ...	Moderate	1	Normal	G.	G.	
960	27	2	38	Mitral Stenosis ...	Moderate	1	Normal	G.	G.	D.A.A.
1584	31	6	24	Mitral Stenosis ...	Moderate	8	Normal	G.	G.	With Regurgitation.
1925	30	5	37	Mitral Stenosis ...	Moderate	1	Normal	G.	G.	
T.Y.H. (1938)										
EMERGENCY										
1385	28	3	35	Unknown	Marked	3	Normal	D.	S.B.	
1455	38	8	36	Aortic Incompetence	Marked	2	—	D.	—	Died undelivered.
1853	36	13	40	Beri-beri	Slight	—	Normal	G.	G.	Anaemia.

HYDRAMNIOS.

24 cases.

No mother died.

7 babies were stillborn and 5 died, a mortality of 50%.

Reg. No.	*Age*	*Gravida*	*Maturity*	*Girth of Abdo.*	*Treatment*	*Result M.*	*Result C.*	*Remarks*
G.C.H. (1937)								
BOOKED								
26	32	5	44	40 inches	Nil	G.	G.	
T.Y.H. (1937)								
EMERGENCY								
1622/36	20	1	40	37 inches	Nil	G.	G.	
1629/36	28	5	38	47½ inches	Nil	G.	S.B.	D.A.A.
112	20	1	40	41½ inches	Nil	G.	G.	D.A.A.
198	22	1	29	42 inches	Nil	G.	D. D.	Twins.
282	12	9	40	39 inches	Binder applied	G.	G.	
298	41	8	43	43 inches	Binder applied	G.	G.	Pendulous abdomen.
120	40	10	40	42 inches	Binder applied	G.	G. G.	Twins.
1134	23	1	28	41 inches	Nil	G.	D.	Placental tumour.
1145	19	1	37	36 inches	Nil	G.	S.B.	P.O.P. D.A.A.
1423	36	11	38	36 inches	Nil	G.	G.	Pendulous abdomen.
1511	31	8	40	39 inches	Nil	G.	G.	Prolapse of anus.
1885	34	6	32	36 inches	Nil	G.	S.B.	
1906	35	3	40	35½ inches	Nil	G.	G.	
T.Y.H. (1938)								
BOOKED								
42	23	1	40	37 inches	Nil	G.	S.B.	
355	25	2	40	40½ inches	Nil	G.	G.	
622	24	3	38	38 inches	Nil	G.	G.	
EMERGENCY								
18	25	1	37	38 inches	Nil	G.	S.B.	
326	28	4	40	39 inches	Nil	G.	G.	
422	36	1	41	37 inches	Nil	G.	S.B.	Monster.
527	26	1	37	38½ inches	Nil	G.	G.	
1206	31	1	27	43 inches	Nil	G.	S.B. D.	Twins.
1461	35	5	40	40 inches	Nil	G.	G.	
2847	30	5	40	39½ inches	Nil	G.	D.	

TRIAL LABOUR FOR SUSPECTED DISPROPORTION.

8 cases.

No mother died.

2 babies were stillborn and 1 died, a mortality of 37.5%.

Reg. No.	*Age*	*Gravida*	*Maturity*	*Onset of Labour* Spontaneous	Induced	*Method of Delivery*	*Int. Spac.*	*Conj. Int.*	*Ect. Crist.*	*D.C.*	*1st St.*	*2nd St.*	*Weight* lb. oz.	*Length*	*Circum. of Head*	*M. C.*	*Remarks*
T.Y.H. (1937)																	
BOOKED																	
350	21	1	42	Yes	—	Normal	9″	10″	7″	—	32½ hrs.	25 mins.	7.2	22½″	—	G. G.	
1263	21	1	41	Yes	No	Perforation	8	10	6½	—	24½ hrs.	11 12 hrs.	8.1	21	—	G. S.B.	P.O.P
1362	24	2	41	No	No	Caesarean Section	8½	9½	6½	3½		20½ hours	6.11	19½	13½	G. G.	Flat pelvis.
1414	28	2	39	Yes	No	Normal	9½	10½	6½	—	15½ hrs.	4½ hrs.	7.1	20	13½	G. G.	
1753	23	2	40	Yes	No	Perforation	9	9½	7	—	19½ hrs.	7½ hrs.	6.1	20	—	G. S.B.	Contracted pelvis.
EMERGENCY																	
736	34	3	40	Yes	No	Normal	8	8½	6½	—	2½ hrs.	30 mins.	7.6	20	—	G. G.	Minor flat pelvis.
T.Y.H. (1938)																	
EMERGENCY																	
1305	19	2	40	Yes	—	Forceps	8½	9½	6½	—	8½ hrs.	2 hrs.	5.4	18	13½	G. D.	
2044	22	1	40	Yes	—	Spontaneous	8 4/5	9 3/5	7	—	53½ hrs.	3 hrs.	7.8	21	13	G. G.	Bad flexion.

INDUCTION OF LABOUR (Spontaneous delivery).

13 cases.

One mother died, a mortality of 7.9%.

4 babies were stillborn and 2 died, a mortality of 46.2%.

Reg. No.	Age	Gravida	Maturity	Indication	Int. Spin.	Int. Crist.	Ext. Conj.	D.C.	Duration of Labour 1st St.	Duration of Labour 2nd St.	Child Weight lb. oz.	Child Length	Child Circum. of Head	Result M.	Result C.	I.D.I.	Method Drug	Method Instrumental	REMARKS
G.C.H. (1937)																			
EMERGENCY																			
295	24	1	35	Pre-eclampsia	9″	10½″	8″	—	—	—	4 8	19″	—	G.	G.	10½ hrs.	Yes	A.R.M.	Post-partum Eclampsia.
311	27	7	38	Pre-eclampsia	9½	10½	7½	—	--	—	6 2	19	—	G.	G.	1½ hrs.	Yes	A.R.M.	
T.Y.H. (1937)																			
BOOKED																			
723	40	12	31	Mitral Stenosis ...	9	10	7	—	—	—	3 6	13	—	G.	D.	70½ hrs.	Yes	A.R.M.	D.A.A.
EMERGENCY																			
339	41	8	32	Toxaemia	9½	10½	8	—	6 hours	—	6 2	19½	—	D.	G.	10½ hrs.	Yes	Yes	Cardiac failure, Pulmonary Tb.
613	28	1	42	Pre-eclampsia	9½	10½	7½	—	--	—	5 11	19½	—	G.	S.B.	15 hrs.	Yes	A.R.M.	
678	32	7	38	Pre-eclampsia	9½	10½	7½	—	--	—	1 12	19	—	G.	G.	2½ hrs.	Yes	A.R.M.	D.A.A.
753	25	1	30	Toxaemia	9½	10½	7	—	—	—	3 1	18	—	G.	D.	15½ hrs.	Yes	A.R.M.	
717	37	6	36	Avitaminosis B ...	9½	10½	6½	—	--	--	5 7	19	—	G.	G.	41½ hrs.	Yes	A.R.M.	
904	22	1	34	Pre-eclampsia ...	9	10	7	—	7 days	½ hr.	2 0	—	—	G.	S.B.	7 days	Yes	A.R.M.	
951	40	6	36	Pre-eclampsia	9½	10½	7½	—	3 hrs. & 10 mins.		8 9	22	--	G.	S.B.	55½ hrs.	Yes	A.R.M.	
1274	27	3	36	Pre-eclampsia	9	10	7½	—	12 hrs.	3 hrs.	6 2	20		G.	G.	14½ hrs.	—	A.R.M.	Chronic Nephritis.
1354	24	2	40	Pre-eclampsia	10	10½	8	—	17 hrs. & 20 mins.		6 0	18	--	G.	G.	17½ hrs.	Yes	A.R.M.	D.A.A.
T.Y.H. (1938)																			
EMERGENCY																			
726	36	11	32	Chronic Nephritis ...		—	—	--	5 hrs.	3 hrs.	5 1	18	--	G.	S.B.	8 hrs.	Nil	A.R.M.	Breech.

INDUCTION OF LABOUR (Forceps delivery).

2 cases.

No maternal or foetal mortality.

Reg. No.	Age	Gravida	Maturity	Indication For Induction	Indication For Forceps	Int. Spin.	Crist. Ext.	Int. Conj.	D.C.	Duration of Labour 1st St.	Duration of Labour 2nd St.	Child Weight lb. oz.	Child Length	Child Circum. of Head	Result M.	Result C.	I.D.I	Method Drug	Method Instrumental
T.Y.H. (1937)																			
EMERGENCY																			
495	21	1	42	Pre-eclampsia ...	Eclampsia	8½″	9½″	7½″	-	51½ hours	--	6 6	20	13″	G.	G.	11 hrs.	--	A.R.M.
T.Y.H. (1938)																			
EMERGENCY																			
1023	33	1	35	Increasing Toxaemia	Eclampsia	9½	10½	7½	—	9½ hrs.	2½ hrs.	5 8	18½	—	G.	G.	22½ hrs.	Yes	A.R.M.

FORCEPS (Labour not induced).

86 cases.

7 mothers died, a mortality of 8.1%.

21 babies were stillborn and 7 died, a mortality of 32.6%.

Reg. No.	Age	Gra. cida	Matu. rity	Indication	Int. Spin.	Int. Crist.	Ext. Cong.	D.C.	Duration of Labour 1st St.	2nd St.	Child Weight lb. oz.	Length of Head	Circum.	Result M.	C.	REMARKS
G.C.H. (1937)																
EMERGENCY																
725/36	21	1	40	Delayed 2nd Stage...	8½″	10″	7½″	—	24 hrs.	2 hrs.	6	0	19″	-	G.	G.	
127	20	1	40	Uterine Inertia ...	9½	10	8	—	55½ hours	..	7	1	20		G.	G.	Foetal and Maternal distress.
T.Y.H. (1937)																
BOOKED																
301	37	3	40	Uterine Inertia ...	9	10	7	..	106 hrs.	1½ hrs.	7	0	21		G.	G.	Occipito-posterior. Spontaneous Rotation. P.P.H.
1532	11	9	37	Foetal distress ...	9	10	7	—	36½ hrs.	2½ hrs.	8	0	21		D.	S.B.	
1742	12	11	40	Maternal distress ...	9	10½	7½	—	17½ hrs.	3 hrs.	7	12	20	13″	G.	S.B.	V3 Manual Rotation.
1753	25	2	40	Contracted pelvis ...	9	9½	7	..	19½ hrs.	7½ hrs.	6	1	20		G.	S.B.	P.O.P. Failed Forceps. Perforation and Extraction.
1853	28	1	35	Maternal distress ...	9½	10½	7		5 hours		5	1	19		G.	G.	Toxaemia. D.A.A.
EMERGENCY																
75	33	1	40	Maternal distress ...	9½	10½	7½		15 hrs.	2 hrs.	5	8	19″		G.	G.	Subinvolution.
94	25	1	41	Uterine inertia ...	9	10	7		148½ hrs.	2 hrs.	6	12	19		G.	G.	Maternal distress. D.A.A.
112	26	1	40	Maternal distress ...	9½	10½	7½		28½ hrs.	1½ hrs.	6	10	21		G.	G.	D.A.A.
124	41	8	40	Foetal distress ...	9	10	7½	...	20 hrs.	5 hrs.	7	12	21		G.	G.	
128	17	1	41	Maternal distress ...	8½	9½	6½	-	15″ hrs.	6½ hrs.	7	4	21	13″	G.	G.	P.O.P. Delayed 2nd stage.
180	20	1	40	Foetal distress ...	9½	10½	7½	-	39½ hrs.	1 hr.	7	12	21	14	G.	G.	P.O.P. Manual Rotation. D.A.A.
191	35	1	38	Foetal distress ...	8½	9½	7	—	23 hrs.	5½ hrs.	6	0	19	12	G.	G.	Foetal shock.
514	40	5	35	P.O.P.	9	10	7½		36 hours		7	2	20½	13½	G.	G.	Manual Rotation.
553	27	1	35	Occipito-posterior ...	9	10	7½	-	12 hours		6	8	20	13	G.	G.	
656	42	9	40	Maternal distress ...	9	10	7		26½ hours		7	12	21	12½	G.	G.	P.O.P. Secondary Anaemia.
684	26	1	40	Uterine inertia ...	9	10	7½		62 hours		6	6	19½	13½	G.	D.	P.O.P. Maternal distress.
706	23	1	40	Tranverse lie	8½	10	7½	-	17 hrs.	1 hrs.	7	8	22	14	G.	G.	Manual Rotation. Foetal distress.
742	32	6	36	Tranverse lie	8½	9½	7	-	36½ hrs.	10 hrs.	7	8	21	13″	G.	S.B.	Manual Rotation. D.A.A.
1320	34	3	40	M. and F. distress ...	8½	9½	7		11½ hrs.	1 hrs.	7	0	21	14½	D.	S.B.	
1358	26	1	40	Maternal distress ...	9	10½	7½	..	6½ hours		7	10	21	—	G.	G.	P.O.P. Manual Rotation.
1709	19	1	40	Foetal distress ...	9	10	7	-	10 hrs.	1½ hrs.	6	12	21	13″	G.	S.B.	V1 Spontaneous Rotation.
1799	18	1	40	Eclampsia		..	-	—	3½ hrs.	15 mins.	6	8	19		G.	G.	
1911	29	3	38	P.O.P.	9	10	7		1½ hrs.	40 mins.	4	10	20		G.	G.	Prolapse of cord.
1943	25	1	42	Delayed labour ...	8½	9½	6½	-	1 hrs.	50 mins.	5	8	19		G.	G.	D.A.A.
1964	38	7	40	Foetal distress ...	9	10	7	..	6 hrs.	45 mins.	7	6	20	..	G.	G.	P.O.P. Manual Rotation. D.A.A.
1975	20	1	40	Foetal distress ...	9½	10½	7	..	12½ hrs.	1 hr.	7	6	21	-	G.	G.	P.O.P. Manual Rotation.
T.Y.H. (1938)																
BOOKED																
2001/37	29	1	34	Maternal distress ...	8½	9½	7½	-	35 hrs.	25 mins.	6	0	19½	-	G.	G.	P.O.P.
669	20	1	40	Prolonged 2nd stage..	—	—	—	—	3½ hrs.	3½ hrs.	8	1	20	..	G.	G.	Oedema of Vulva.
927	19	1	40	Uterine inertia ...	9½	10½	8½	—	53 hrs.	1 hr.	6	14	19½	14″	G.	S.B.	Transverse lie. Maternal and Foetal distress.
2093	25	1	38	M. and F. distress ...	9	10	7	..	17 hrs.	2 hrs.	6	10	20	13	G.	G.	P.O.P. Manual Rotation.
2161	31	1	37	Maternal distress ...	8½	9½	6½	—	11½ hrs.	3½ hrs.	6	8	20	13½	G.	S.B.	Transverse lie. Manual Rotation. D.A.A.
EMERGENCY																
2020/37	19	1	40	Eclampsia	—	-	-	—	35 hrs.	15 mins.	4	12	18	-	G.	D.	A.F.M.
68	32	2	37	M. and F. distress ...	9½	10½	7½	-	13½ hrs.	43 hrs.	7	2	20	—	G.	S.B.	
											5	4	18	-		S.B.	Twins.
80	29	1	38	Delayed labour ...	9½	10½	7½	-	46 hours		3	1	16	-	G.	S.B.	
153	37	3	37	Prolapse of cord ...	..		-	—	15½ hours		7	2	21		G.	S.B.	D.A.A.

FORCEPS (Labour not induced).—*(Continued).*

Reg. No.	Age	Gravida	Maturity	Indication	Int. Spin.	Int. Crist.	Ext. Cong.	D.C.	Duration of Labour 1st St.	Duration of Labour 2nd St.	Child Weight lb. oz.	Child Length	Child Circum. of Head	Result M.	Result C.	REMARKS
T.Y.H. (1938)																
EMERGENCY																
176	19	1	38	Prolapse of cord ...	8½	9½	7	—	1 hour		6	2	19	—	G.	S.B. D.A.A.
192	23	1	35	Eclampsia	—	—	—	—	4½ hours		6	1	18	—	G.	G. A.F.M.
288	22	1	40	Uterine inertia ...	8½	9½	7½	—	51½ hrs.	2½ hrs.	6	0	20	12	G.	S.B. P.O.P. Manual Rotation.
313	28	1	40	M. and F. distress ...	9½	10½	7½	—	26 hrs.	3½ hrs.	8	0	19		G.	G. P.O.P. Manual Rotation.
357	20	1	40	Uterine inertia ...	10	11	7	—	50 hours		6	10	19	-	G.	G. Foetal distress.
360	20	1	38	M.R. of Placenta ...	—	—	—	—	21 hrs.	45 mins.	6	14	21	—	D.	S.B.
441	26	2	40	P.O.P.	9½	10½	7½	—	19½ hours		7	6	21	—	G.	G. Manual Rotation.
474	26	1	40	Uterine contraction ...	10	10½	7	—	71 hrs.	2 hrs.	7	1	21		D.	S.B. P.O.P., Manual Rotation. Avitaminosis B . M. & F. distress
531	25	2	40	Maternal distress ...	—	—	—	-	2½ hrs.	5½ hrs.	7	6	20	-	G.	G. P.O.P. M. Rotation. Post-partum Eclampsia.
585	28	1	37	Foetal distress ...	10½	11	7		6½ hrs.	7½ hrs.	7	1	20	—	G.	G. P.O.P. Manual Rotation. D.A.A.
590	28	2	40	Foetal distress ...	10	11½	7½	—	4 hrs.	7½ hrs.	6	4	21½	—	G.	G. Obliquity of uterus. Rupture of urethra. P.O.P.
612	40	8	39	Maternal distress ...	9½	10½	8½	—	2½ hrs.	2 5/6 hrs.	6	12	19	—	G.	G. P.O.P. Manual Rotation.
613	25	1	39	P.O.P.	9½	10½	7½	—	18¾ hrs.	4½ hrs.	6	0	17½	-	G.	G.
747	25	3	41	Minor pelvic contract	9½	10½	7	—	34 hrs.	36 hrs.	8	0	19		G.	G. P.O.P. Manual Rotation. Foetal distress.
702	29	1	34	M. and F. distress ...	9½	10½	7½	—	21½ hrs.	3½ hrs.	6	0	21		G.	G P.O.P. Manual Rotation.
765	30	6	39	Face presentation ...	—	—	—	—	2 hrs.	3 5/6 hrs.	7	12	21		G.	S.B.
805	25	1	40	Maternal distress ...	8	9½	6½	—	12½ hrs.	4½ hrs.	6	0	19		G.	G.
1067	32	1	38	Tranverse lie	9	10	6½	—	17 hrs.	3½ hrs.	5	12	20		G.	G. Manual Rotation. D.A.A.
1135	21	1	40	M. and F. distress ...	9½	10½	7½	—	11½ hrs.	3 hrs.	6	4	18	12½	G.	G. D.A.A.
1176	34	1	37	Foetal distress ...	8½	10	8	—	11 hrs.	2 5/6 hrs.	6	0	20	13½	G.	G.
1200	27	2	41	Maternal distress ...	—	-		-	6½ hrs.	2½ hrs.	7	0	20	15½	G.	S.B.
1228	21	1	43	M. and F. distress ...	9	10½	7	—	23½ hrs.	25 mins.	6	6	20	13½	G.	G. P.O.P.
1289	22	1	40	Foetal distress ...	9	10	7½	—	26 hrs.	2½ hrs.	6	8	19	13½	G.	G. P.O.P.
1298	26	1	39	Eclampsia	9½	11	7½	—	21½ hrs.	55 mins.	7	1	21½	13½	G.	S.B. P.O.P. Manual Rotation. D.A.A.
1395	19	2	40	Pelvic contraction ...	8½	9½	6½	—	8½ hrs.	2 hrs.	5	4	18	13½	G.	D. Minor degree. D.A.A.
1419	22	1	40	Pre-eclampsia	9½	10½	7½	—	39 hrs.	2½ hrs.	7	1	20	14½	G.	G. D.A.A.
1447	25	1	40	Foetal distress ...	9	10½	7	-	15¾ hrs.	2 hrs.	6	8	19	13	G.	G. P.O.P. Manual Rotation.
1480	28	1	38	Prolonged 2nd stage..	8½	9	6½	—	18½ hrs.	3½ hrs.	6	0	19½	13½	G.	G. D.A.A.
1527	19	1	40	Eclampsia	9	10	7½	—	16½ hrs.	2 hrs.	7	0	19	13½	G.	G.
1589	21	1	39	Foetal distress ...	9½	10	7½	—	27½ hrs.	2½ hrs.	7	0	19	13	G.	G. P.O.P. Manual Rotation.
1597	21	1	39	Foetal distress ...	9½	10	7½	-	27½ hrs.	2½ hrs.	6	12	19	13	G.	G.
1611	29	1	41	Maternal distress ...	9	10	7	—	13 5/6 hrs.	2 5/6 hrs.	6	12	20	13½	G.	G.
1614	29	1	40	Foetal distress ...	9½	10½	7½	—	15 hrs.	30 mins.	6	0	19	13½	G.	D. P.O.P. Manual Rotation.
1621	37	2	38	M. and F. distress ...	8½	9½	6½	-	5½ hrs.	2½ hrs.	5	12	18	13½	G.	G. P.O.P. Manual Rotation.
1632	28	1	38	Prolonged 2nd stage..	9	10½	7	—	40½ hrs.	3½ hrs.	6	0	19½	12½	G.	G.
1655	20	1	38	M. and F. distress ...	9	10	7		10 5/6 hrs.	20 mins.	6	0	19	13	G.	G. Impacted head in Transverse lie.
											5	8	18	—	G.	
1664	22	1	36	M. and F. distress ...	10	11	7½		16 hrs.	2 5/6 hrs.	5	0	18	12½	G.	G. Twins. P.O.P. Manual Rotation.
1696	22	1	40	Transverse lie	9½	10½	7½		4 hrs.	35 mins.	7	0	20	13½	G.	G. Manual Rotation.
											5	0	17½	13	D.	
1619	20	1	40	Maternal distress ...	9	10	7	—	20 hrs.	2½ hrs.	3	12	17	—	D.	D. Twins. Pre-eclampsia.
1740	28	2	40	M. and F. distress ...	8	8½	6½	—	7 hrs.	25 mins.	6	12	19½	13½	G.	G. Transverse lie. Minor pelvic contraction. D.A.A.
1744	22	1	40	Posthumous	9	10	7	—	21 hours		6	10	19	—	D.	G. Post humous forceps delivery.
1785	26	1	38	M. and F. distress ...	9	10	7	—	22 hrs.	50 mins.	6	1	19	13½	G.	D. P.O.P. Manual Rotation. D.A.A.
1824	28	1	40	Foetal distress ...	9½	10½	7½	—	19½ hrs.	2½ hrs.	5	0	18½	13½	G.	G.
2020	28	5	41	Pelvic contraction ...	9½	10½	7½	—	16 hrs.	1 hr.	6	8	20	14½	G.	G. Foetal distress.
2100	29	1	40	Uterine inertia ...	9	10½	7½	—	66¾ hrs.	5⅝ hrs.	7	0	21	—	D.	S.B. P.O.P. Maternal and Foetal distress.
2050	26	1	40	Foetal distress ...	9	10	7	-	5 5/6 hrs.	37 mins.	4	8	17	12½	G.	S.B. P.O.P. Manual Rotation. Prolapse of cord.
2267	25	1	40	Foetal distress ...	11½	13	9½	—	12½ hrs.	3⅜ hrs.	6	2	19½	13	G.	G. P.O.P. Manual Rotation.
2278	31	1	40	Prolonged 2nd stage..	9	10	7	—	17½ hrs.	3½ hrs.	6	6	19	13	G.	G. Maternal and Foetal distress.
2307	21	1	40	Prolonged 2nd stage..	9½	10½	7½	—	1 hrs.	6¼ hrs.	7	1	20	14	G.	G. Maternal distress. P.O.P. Manual Rotation.

ANTE-NATAL VERSION (*i.e.* before labour).

3 cases.

Reg. No.	*Age*	*Gravida*	*Maturity*	*Anæsthetic*	*Position at Delivery*	*Weight of Child* lb.	oz.	*Result* M.	C.	*REMARKS*
T.Y.H. (1937)										
BOOKED										
1714	31	4	38	Ether	Vertex I	7	2	G.	G.	External Version. D.A.A.
T.Y.H. (1938)										
BOOKED										
287	28	6	40	Yes	Vertex I	6	6	G.	G.	External. D.A.A.
1776	25	3	43	Ether	Vertex	8	0	G.	G.	External.

VERSION (In Labour).

23 cases.

No mother died.

19 babies were stillborn and 1 died, a mortality of 86.9%.

Reg. No.	*Age*	*Gravida*	*Maturity*	*Indication*	*Bipolar, External or Podalic*	*Weight of Child* lb.	oz.	*Result* M.	C.	*REMARKS*
G.C.H. (1937)										
EMERGENCY										
218	33	7	39	Brow presentation	Bipolar	7	1	G.	S.B.	Marginal Placenta Praevia.
T.Y.H. (1937)										
BOOKED										
1016	27	2	38	Shoulder presentation...	Internal	2	4	G.	S.B.	Marginal Placenta Praevia. Old Tb. Hip. D.A.A.
EMERGENCY										
367	39	4	34	Transverse presentation	Internal	5	2	G.	G.	Prolapsed arm and shoulder.
454	31	6	40	Shoulder presentation...	Internal	7	10	G.	S.B.	Prolapsed arm. Extraction.
530	23	3	32	Transverse presentation	External	3	0	G.	S.B.	
661	40	9	33	Shoulder presentation...	Internal	4	4	G.	S.B.	Prolapsed left hand.
985	28	3	34	Transverse presentation	Bipolar	2	1	G.	S.B.	Central Placenta Praevia. Foetal Monstrosity. D.A.A.
1295	29	2	42	Shoulder presentation...	Internal	5	0	G.	S.B.	2nd of twins.
1453	31	5	34	Transverse presentation	Internal	6	0	G.	S.B.	Lateral Placenta Praevia.
1568	21	2	40	Transverse presentation	Internal	8	4	G.	S.B.	Prolapsed hand and arm.
1770	36	8	29	Shoulder presentation...	Internal	2	8	G.	D.	2nd of twins. Extraction.
1949	35	6	40	Transverse presentation	Internal	7	2	G.	S.B.	Prolapsed cord and left arm.
T.Y.H. (1938)										
EMERGENCY										
2023/37	22	3	40	Transverse to Shoulder	Internal	5	12	G.	S.B.	Lateral Placenta Praevia.
136	28	4	39	Transverse to Breech...	Internal	6	0	G.	S.B.	Lateral Placenta Praevia. D.A.A.
198	23	1	40	Breech	External	6	6	G.	G.	
416	25	3	36	Transverse left shoulder presenting	Internal	4	0	G.	S.B.	Prolapsed left hand. D.A.A.
740	25	2	37	Transverse presentation	External	4	0	G.	S.B.	A.E.M. Willett's Forceps.
857	25	3	34	Transverse presentation	Internal	4	12	G.	S.B.	Impacted shoulder with prolapsed hand.
1026	28	4	25	Transverse presentation	Bipolar	1	8	G.	S.B.	Placenta Praevia.
1315	32	2	20	Marginal Placenta Praevia	Bipolar	3	8	G.	S.B.	Shoulder presentation.
1834	35	6	29	Transverse presentation	Internal	4	8	G.	S.B.	Prolapsed cord, hand, leg. Extraction.
1886	32	2	37	Transverse L. Placenta Praevia ...	Bipolar	1	13	G.	S.B.	Miscarriage.
2818	31	5	40	Shoulder presentation.	Internal	7	8	G.	G.	Prolapsed of cord and hand.

CAESAREAN SECTION.

8 cases.

No mother died.

One baby died, a mortality of 12.5%.

Reg. No.	Age	Gravida	Maturity	Indication	Int. Spin.	Int. Crist.	Ext. Conj.	D.C	Duration of 1st St.	Duration of 2nd St.	Child Weight lb. oz.	Child Length	Child Circum. of Head	Result M.	Result C.	Admitted for Trial Labour	Type of Operation	Remarks
G.C.H. (1937)																		
EMERGENCY																		
22	23	2	38	Obstructed labour ...	8½″	10″	7″	—	7½ hrs.	4 hrs.	8.0	20″	14″	G.	G.	—	Lower segment	Cicatricial Contraction of vagina.
T.Y.H. (1937)																		
BOOKED																		
887	26	2	41	Flat Pelvis	8	10	6½	4½	—	—	7.10	21	—	G.	G.	No.	Lower segment	Occipital posterior.
1962	24	2	41	Flat Pelvis	8½	9¹	6½	3½	7½ hours		6.11	19½	13½	G.	G.	Yes.	Lower segment	
EMERGENCY																		
688	28	5	37	C. Placenta Praevia...	—	—	—	—	—	—	6.8	20	—	G.	D.	No.	Classical	Fallopian tubes cut and tied.
T.Y.H. (1938)																		
BOOKED																		
73	28	1	40	Disproportion	8¾	10½	7½	—	...	—	7.12	20½	—	G.	G.	No.	Classical	
483	29	4	39	Flat Pelvis	9½	10½	8	—	—	—	5.10	19	12½	G.	G.	No.	Classical	Spondylolisthesis.
EMERGENCY																		
518	20	1	40	Contraction of uterus	8½	9½	6¼	—	48 hours		6.0	20½	—	G.	G.	No	Lower segment	M. and F. distress.
1295	23	3	40	Stricture of vagina...	8	10	6½	—	8 hrs.	15/6 hrs.	4.12	18½	—	G.	G.	No.	Classical	Urethro-vaginal fistula.

EMBRYOTOMY AND CRANIOTOMY.

4 cases.

No mother died.

Reg. No.	Age	Gravida	Maturity	Indication	Previous Treatment	Int. Spin.	Int. Crist.	Ext. Conj.	D.C.	Duration of labour 1st Stage	Duration of labour 2nd Stage	Weight of Child lbs.	Result to Mother	Type of Operation	Remarks
G.C.H. (1937)															
EMERGENCY															
74	24	3	40	Pelvic deformity...	...Nil ...	9½″	10½″	8″	—	5 hrs. 20 min.	2 hrs.	6.2	G.	Perforation	Old Tb hip.
T.Y.H. (1937)															
BOOKED															
1263	21	1	41	Maternal distress	P.O.P.	Forceps failed...	8	10	6½	—	21½ hrs.	1 1/12 hrs.	8.4	G.	Perforation	
1753	23	2	40	Contracted Pelvis	...Forceps failed...	9	9½	7	—	19½ hrs.	7¾ hrs.	6.1	G.	Perforation	
T.Y.H. (1938)															
EMERGENCY															
1105	40	8	40	Brow presentation	...Forceps failed...	8½	9½	6½	—	2 hrs.	5 hrs.	6.4	G.	Craniotomy	Dead foetus.

PERINEAL LACERATION AND EPISIOTOMY.

388 Lacerations.

65 Episiotomies.

(Incidence of Laceration and Episiotomy = 9.95% of Total Deliveries.)

A. LACERATION OF PERINEUM.

Type of Labour.		*1st or 2nd Degree.*	*3rd Degree.*
Natural Forces:	Vertex	327	12
	Breech	8	—
Forceps Delivery:	Vertex	35	5
Perforation:	Vertex	1	—
		371	17

B. EPISIOTOMY.

Type of Labour.		*Central Episiotomy.*
Natural Forces:	Vertex	41
	Breech	21
Forceps Delivery:	Vertex	3
		65

MANUAL REMOVAL OF PLACENTA.

10 cases.

3 mothers died, a mortality of 30%.

4 babies were stillborn, a mortality of 40%.

Reg. No.	*Age*	*Gravida*	*Maturity*	*Method of Delivery*	*Length of 3rd Stage*	*Indication*	*Morbidity*	*Result M. C.*	*Amt. of Bleeding*	*REMARKS*
G.C.H. (1937)										
EMERGENCY										
312	19	1	30	Normal	5¼ hrs.	Retained placenta	Yes	G. S.B.	?	B.B.A.
T.Y.H. (1937)										
EMERGENCY										
265	28	3	40	Normal	2¼ hrs.	Partial adherent placenta...	Nil	G. G.	40 ozs.	
277	21	1	40	Breech...	1½ hrs.	P.P.H.	Yes	G. G.	20 ozs.	D.A.A.
399	41	8	32	Induction	1½ hrs.	P.P.H.	No	D. G.	25 ozs.	
1586	37	3	40	Right Cleidotomy ...	5½ hrs.	Retained placenta	Yes	G. S.B.	Normal	Contraction ring of uterus.
1789	20	2	39	Normal	1¼ hrs.	Retained placenta	Nil	G. G.	Normal	
T.Y.H. (1938)										
EMERGENCY										
2014/37	27	2	41	Normal	1¼ hrs.	Retained placenta	Nil	G. G.	30 ozs.	
360	20	1	38	Forceps	1 hour	Adherent placenta	Yes	D. S.B.	20 ozs.	
2100	20	1	40	Forceps	1¼ hrs.	Retained placenta	Nil	D. S.B.	30 ozs.	
2127	34	7	40	Normal	8.50-60 hrs.	Retained placenta	Nil	G. G.	30 ozs.	D.A.A.

POST-PARTUM HAEMORRHAGE.

80 cases.

4 mothers died, a mortality of 5%.
8 babies were stillborn and 4 died, a mortality of 15%.

Reg. No.	*Age*	*Gravida*	*Maturity*	*Relevant Features*	*Predisposing Cause*	*Treatment*	*Result M.*	*Result C.*	*Amt. of Bleeding*	*REMARKS*
G.C.H. (1937)										
BOOKED										
283	29	3	41	Oedema	Atony	General	G.	G.	20 ozs.	
EMERGENCY										
68	27	1	40	Cervical laceration	Atony, Trauma	Plug and douche	G.	G.	18 ozs.	
141	30	1	38	Cervical laceration	Trauma	Suture	G.	G.	15 ozs.	
143	21	2	41	Nil	Adherent placenta	Manual Removal	G.	G.	16 ozs.	
T.Y.H. (1937)										
BOOKED										
301	37	3	40	Uterine inertia	Atony	Bimanual comp. of uterus	G.	G.	10 ozs.	Twilight sleep.
538	20	1	38	Puerperal Pyrexia	Atony	General	G.	D.	50 ozs.	
1019	27	3	40	Traumatic	Cervical laceration	Suture	G.	G.	70 ozs.	
1867	27	6	40	Haemolytic Streptococci	Retained membranes	General	G.	G.	20 ozs.	D.A.A.
EMERGENCY										
1614/36	20	2	35	Nil	Atony	General	G.	G.	30 ozs.	
1620/36	22	1	35	Nil	Traumatic Atony	General	G.	G.	10 ozs.	D.A.A.
14	20	1	37	Twins	Atony	General	G.	G.	40 ozs.	
32	23	1	39	Full bladder	Atony	General	G.	G.	30 ozs.	
136	39	9	40	Oedema	Traumatic Atony	General	G.	G.	30 ozs.	D.A.A.
216	21	2	40	Nil	Atony	General	G.	G.	30 ozs.	
237	21	1	40	Marked oedema	Atony	General	G.	G.	30 ozs.	D.A.A.
264	45	13	24	M. Placenta Praevia	Atony	Willett's Forceps	G.	D.	25 ozs.	
265	28	3	40	Partial adherent placenta	Atony	Manual Removal	G.	G.	40 ozs.	
277	21	1	40	Nil	Atony	M.R. of Placenta	G.	G.	20 ozs.	D.A.A.
339	41	8	32	Induction of labour	Atony	M.R. of Placenta	D.	G.	25 ozs.	
369	25	3	40	Nil	Atony	General	G.	G.	30 ozs.	D.A.A.
431	43	12	32	Severe A.P.H.	Atony	Manual comp. of uterus	D.	S.B.	40 ozs.	
474	37	13	38	Toxaemic A.P.H.	Atony	General	G.	G.	10 ozs.	
696	25	5	40	Slight oedema	Atony	General	G.	G.	10 ozs.	
828	26	4	40	Nil	Cervical laceration	Suture	G.	G.	20 ozs.	
941	25	2	40	Nil	Cervical laceration	Suture	G.	G.	25 ozs.	
901	23	3	40	Acute Avitaminosis B	Atony	General	G.	S.B.	20 ozs.	D.A.A.
1232	20	1	40	Nil	Perineal laceration	Suture	G.	G.	15 ozs.	D.A.A.
1310	19	1	40	Nil	Atony	General	G.	G.	30 ozs.	
1393	22	1	40	Nil	Atony	General	G.	G.	15 ozs.	
1561	27	1	40	Oedema	Atony	General	G.	G.	30 ozs.	
1581	30	5	40	Avitaminosis B	Vulval haematoma	Incision-Plugging	G.	G.	20 ozs.	
1597	32	6	40	Nil	Atony	General	G.	G.	15 ozs.	
1614	25	1	36	Nil	Bilateral cervical tear	Suture	G.	G.	15 ozs.	
1654	27	1	40	Nil	Atony	General	G.	G.	20 ozs.	
1788	20	2	36	Nil	Atony	General	G.	G.	25 ozs.	
1792	20	2	36	Nil	Atony	General	G.	G.	20 ozs.	
1894	24	2	40	Nil	Atony	General	G.	G.	40 ozs.	
1903	25	2	40	Retained products	Atony	General	G.	G.	50 ozs.	D.A.A.
1975	20	1	40	P.O.P.	Atony	General	G.	G.	20 ozs.	Manual Rotation and Forceps.

POST-PARTUM HAEMORRHAGE.—(*Continued*).

Reg. No.	*Age*	*Gravida*	*Maturity*	*Relevant Features*	*Predisposing Cause*	*Treatment*	*Result M.*	*Result C.*	*Amt. of Bleeding*	*REMARKS*
T.Y.H. (1938)										
BOOKED										
2021/37	21	1	39	Nil	Atony	General	G.	G.	10 ozs.	
128	21	1	43	Cervical Tear	Atony	Suture	G.	G.	20 ozs.	
1423	19	1	33	Nil	Atony	General	G.	G.	20 ozs.	Twins.
EMERGENCY										
2014/37	27	2	41	Nil	Retained Placenta	Manual Removal	G.	G.	30 ozs.	
2015/37	20	1	37	Nil	Atony	General	G.	G.	15 ozs.	
80	29	1	38	Toxaemia	Atony	General		S.B.	30 ozs.	Twins.
94	21	2	40	Nil	Atony	General	G.	S.B.		
133	19	2	39	Nil	Atony	General	G.	G.	30 ozs.	
136	28	4	39	Placenta Praevia	Atony	General	G.	G.	25 ozs.	
154	29	6	36	Placenta Praevia	Atony	General	G.	S.B.	20 ozs.	D.A.A.
161	21	1	40	Nil	Atony	General	G.	S.B.	30 ozs.	D.A.A.
262	26	2	40	Laceration of cervix	Atony	Suture	G.	G.	10 ozs.	
273	27	2	37	Nil	Atony	General	G.	G.	20 ozs.	
328	19	1	39	Nil	Retained Placenta	Injection Expression	G.	G.	20 ozs.	
352	18	1	40	Nil	Atony	General	G.	G.	30 ozs.	D.A.A.
439	27	5	40	Nil	Retained Placenta	Insertion of cord	G.	G.	15 ozs.	
460	24	4	40	Nil	Atony	General	G.	G.	25 ozs.	
538	22	2	40	Nil	Retained Membs. Atony	General	G.	G.	10 ozs.	
601	23	1	40	Oedema	Atony	General	G.	G.	20 ozs.	
670	17	1	40	Nil	Atony	General	G.	G.	20 ozs.	
1044	38	9	40	Nil	Atony	General	G.	G.	20 ozs.	D.A.A.
1082	27	2	36	Nil	Atony	General	G.	G.	30 ozs.	
1166	22	1	38	Oedema	Atony	General	G.	G.	20 ozs.	
1185	28	6	40	Pyrexia	Cervical Tear	Suture	G.	G.	15 ozs.	
1191	30	3	40	Nil	Amniotic Atony	General	G.	G.	20 ozs.	
1200	27	2	40	Nil	Cervical Tear	Suture	G.	G.	15 ozs.	
1340	33	6	39	Nil	Atony	General	G.	S.B.	22 ozs.	
1400	29	5	40	Nil	Atony	General	G.	G.	15 ozs.	
1544	28	3	38	Nil	Atony	General	G.	G.	20 ozs.	
1593	35	5	40	Nil	Atony	General	G.	G.	20 ozs.	
1603	21	2	39	Nil	Atony	General	G.	G.	30 ozs.	
1635	25	1	39	Multiple Lacerations	Atony	General	G.	G.	28 ozs.	
1682	24	1	40	Nil	Atony	Suture, General	G.	G.	50 ozs.	
1866	26	1	40	Nil	Atony	General	G.	G.	15 ozs.	
1928	26	2	28	L. Placenta Praevia	Atony	General		D.	20 ozs.	Twins
2100	29	1	40	Retained Placenta	Atony	Manual Removal	D.	D.		
2127	31	7	40	Retained Placenta	Atony	General	G.	S.B.	30 ozs.	
2154	23	2	40	Nil	Atony	General	G.	G.	36 ozs.	D.A.A.
2217	39	3	38	Nil	Atony	General	G.	G.	20 ozs.	
2270	30	1	40	Multiple fibromyomata	Atony	General	G.	G.	30 ozs.	
2301	20	2	40	Nil	Atony	General	D.	G.	60 ozs.	Severe haemorrhage.

MATERNAL MORBIDITY.

246 Cases.

All cases with pyrexia and all maternal deaths are included as morbid.

The definition of puerperal pyrexia, as adopted at this clinic, is: "A temperature of 100.4 F. or over, occurring during the puerperium while the patient is under observation, not including the first twenty-four hours."

	1937	*1938*	*Total*
Booked Cases.			
Number of cases delivered	128	126	254
Cases of Pyrexia	9	6	15
Maternal Deaths	—	—	—
Morbidity Rate	7.03%	4.8 %	5.9 %
Emergency Cases.			
Number of cases delivered	2114	2141	4255
Cases of Pyrexia	113	103	216
Maternal Deaths without pyrexia	7	8	15
Morbidity Rate	5.68%	5.2 %	5.44%
Morbidity Rate for whole clinic	5.75%	5.11%	5.46%

Details of Morbid Cases.	*1937*	*1938*
Booked Cases. (Total Number 15.)		
Puerperal Infection	5 (4 H.S.)	1 (H.S.)
Mastitis	—	4
Urinary Tract Infection	3	—
Mucous Colitis	—	1
Cellulitis of Arm	1	—
Emergency Cases. (Total Number 231.)		
Puerperal Infection		
Uterine	37 (16 H.S.)	32 (13 H.S.)
Perineal Sepsis	4 (1 H.S.)	1
Mastitis	26	29 (2 H.S.)
Urinary Tract Infection	20 (1 H.S.)	5
Chronic Nephritis	1	—
Haematuria	1	—
Parotitis	—	1
Tonsillitis	2	2 (1 H.S.)
Bronchitis	5	7
Pneumonia	—	4
Bacillary Dysentery	2	7
Typhoid Fever	1	1
Enteritis	1	2
Influenza	1	—
Malaria	—	1
Smallpox	—	2
Syphilis	4	3
Cold Abscess of Rib	—	1
Fever of unknown origin	8	5
Maternal Deaths without Pyrexia	7	8

Monthly distribution of the Pyrexia cases, and of the incidence of Haemolytic Streptococcus infection, for the two years:—

	No.	*H.S.*		*No.*	*H.S.*
January	14	2	July	18	0
February	14	4	August	21	4
March	20	6	September	13	2
April	12	2	October	34	2
May	17	0	November	25	4
June	23	6	December	20	7

The parity of the cases was as follows:—

No. of Cases:

Para	1	2	3	4	5	6	7	8	9	10 (or over)
BOOKED	8	3	0	0	1	2	0	0	1	0
EMERGENCY	113	37	18	20	9	9	6	1	1	2

MATERNAL MORTALITY.

22 Deaths (1 Booked, 21 Emergency).

Mortality Rate 0.48%.

T.Y.H. (1937)

BOOKED

No. 1532. *Pyelitis, Bacillary Dysentery.*

Para 9, age 41, maturity 37 weeks. Patient was admitted from ante-natal clinic for hospital treatment for oedema of legs one month previously. She was in hospital for one week and left in good condition. Readmitted three weeks later with history of fever and pain in the back: temperature 101.6 F., pulse 100, marked albuminuria, B.P. 86/46. Pyelitis diagnosed. Came into labour five days later and was delivered by forceps after labour lasting 38 hours. Temperature rose to 104 with rigors on day after labour. On the second day temperature rose to 105 and 12 loose stools were passed. Bacillary Dysentery was diagnosed and the patient transferred to the Queen Mary Hospital, where she subsequently died.

EMERGENCY

No. 339. *Pregnancy Toxaemia, Post-partum Haemorrhage.*

Para 8, age 41, maturity 31 weeks. Patient admitted with generalised oedema, B.P. 168/104, marked albuminuria. Twenty-four hours later the blood-pressure had risen to 184/120. Membranes ruptured and a living child of 6lb. 2oz. was delivered after labour lasting 10 hours. Patient collapsed after delivery. The placenta was adherent and was removed manually on account of haemorrhage. Patient died 2 hours after delivery.

No. 431. *Ante-partum and Post-partum Haemorrhage.*

Para 12, age 43, maturity 32 weeks. Patient was admitted in a collapsed state complaining of abdominal pain and severe vaginal haemorrhage. Pulse 92, temperature 98. The cervix was 2 fingers dilated, membranes intact, no placental tissue could be felt, Vertex I position. The membranes were ruptured and the vagina was packed. Submammary saline was given and repeated later. After removal of the plug Willett's forceps were applied, followed by two doses of Pituitrin ½c.c. at half-hourly intervals. 500 c.c. of Saline was given intravenously and patient was delivered of a stillborn child weighing 6lb. 2oz. five hours after admission. This was followed by a post-partum haemorrhage, and inspite of all treatment she died in half an hour. After death it was learned from the relatives that the patient had been taking drugs to try and interrupt the pregnancy.

T.Y.H. (1937)
EMERGENCY

No. 988. *Avitaminosis B1, Cardiac Failure.*

Primipara, age 30, maturity 36 weeks. On admission there was evidence of Avitaminosis B1; oedema, absence of knee and ankle jerks, impaired sensation of finger tips, pulse rate 110, B.P. 128/94. The oedema was of moderate degree in the legs, feet and hands, and slight in the anterior abdominal wall and forearms. Heart and lungs showed no gross physical signs on admission. Urine showed no pathological changes. With the onset of strong labour pains patient began to show signs of cardiac failure: dyspnoea, rales at both bases, cyanosis and restlessness. There was vomiting. Later the pulse became irregular and the patient died undelivered.

No. 1320. *Contracted pelvis, Myocardial Degeneration (?Avitaminosis B1).*

Para 3, age 34, maturity 40 weeks. Interspinous 8½ ins., Intercristal 9¾ ins., External Conjugate 7 ins. History of oedema of the legs for one month. Two previous still-births. On admission patient had slight albuminuria and B.P. 144/94. After prolonged 2nd stage pulse rate and temperature rose, forceps were applied under ether anaesthesia and a still-born child was extracted. Soon afterwards the patient became cyanosed and stopped breathing. Post-mortem examination showed right sided dilatation of the heart with myocardial degeneration.

No. 1732. *Subtertian Malaria.*

Para 6, age 36, maturity 36 weeks. On admission there was evidence of chronic malaria: spleen enlarged three fingers below costal margin, anaemia marked. Following labour there was a rigor and ring and crescent forms of malarial parasites were found in the blood smear. Treatment with quinine followed by atebrin was given. Death, with preceding signs of cardiac failure, occurred on the 6th day of the puerperium.

No. 1889. *Pregnancy Toxaemia. Burns.*

Para 4, age 41, maturity 31 weeks. History of generalised oedema for 12 days and a burn of the right foot (caused by a misguided endeavour to relieve the oedema). Albuminuria marked, B.P. 126/102, absence of knee and ankle jerks. Delivered of still-born child on day after admission after labour lasting less than 2 hours. The condition of her foot became worse and on the fourth day after admission she showed signs of cardiac failure and died.

G.C.H. (1937)
EMERGENCY

No. 214. *Mitral Stenosis, Avitaminosis B1, Cardiac Failure.*

Para 5, age 35, maturity 34 weeks. The patient was admitted with dyspnoea and oedema of the legs feet and vulva for a fortnight. The B.P. was 142/80, the urine was clear. There were signs of mitral stenosis with congestive heart failure and Avitaminosis B1. There was spontaneous delivery of a child weighing 4lb. 14ozs. three hours after admission. The patient's heart grew steadily worse and death took place 11 hours after delivery. Post-mortem examination showed mitral stenosis and signs of avitaminosis B1.

T.Y.H. (1938)
EMERGENCY

No. 107. *Pregnancy Toxaemia, Avitaminosis B1, Cardiac Failure.*

Para 8, age 35, maturity 38 weeks. History of fever and rigors 2 weeks before admission. On admission marked oedema of feet, legs, thighs, vulva, abdominal wall. Pulse 132, B.P. 150/94. Marked albuminuria, knee jerks absent. Normal labour lasting 7 hours. After delivery the B.P. rose to 164/108, and on the 5th day of the puerperium cardiac symptoms developed. The lungs became waterlogged and she collapsed suddenly on the 7th day.

No. 360. *Central Placenta Praevia, Uterine Sepsis, Delayed Post-partum Haemorrhage.*

Primipara, age 20, maturity 38 weeks. Mentally deficient patient, central placenta praevia, with concomitant ruptured and discharging Bartholin's abscess, with gonococcus, staphylococcus albus and streptococcus faecalis in culture. Treated by application of Willett's forceps and later low forceps. Portion of placenta had to be removed manually. 10 c.c. of Prontosil were injected at time of removal and 5 gm. given daily for four days. Fever on 3rd day with foul vaginal discharge. On 7th day there was a severe uterine haemorrhage, which was arrested, but the patient died soon afterwards.

No. 393. *Smallpox.*

Para 7, age 35, maturity 40 weeks. Normal labour. Third day after delivery patient developed smallpox and died of this disease after removal to the isolation hospital.

T.Y.H. (1938)
EMERGENCY

No. 431. *Smallpox.*

Para 3, age 24, muturity 30 weeks. Normal labour. On day of delivery patient developed high temperature and prodromal smallpox rash. Died after removal to isolation hospital.

No. 459. *Septic endometritis, Salpingo-oophoritis, Peritonitis.*

Primipara, age 24, maturity 40 weeks. Labour normal and unassisted, lasting 8½ hours. Placenta and membranes delivered intact. On the 2nd day there was a sharp rise in temperature. No H.S. found in vaginal swabs. Lochia became very offensive despite treatment with streptocide etc. Temperature ranged between 102 and 104, with Pulse of 110 to 120. On 10th day patient discharged herself against advice, but collapsed on leaving Hospital. Post-mortem examination showed peritonitis following ascending genital tract infection.

No. 474. *Primary Uterine Inertia, Avitaminosis B1, Cardiac Failure.*

Primipara, age 26, maturity 40 weeks. Evidence of avitaminosis B1 : absence of knee jerks, tenderness of calf muscles, oedema of legs, B.P. 104/86, trace of albuminuria. First stage lasted 92 hours, with head in P.O.P. position. When the membranes ruptured the cord prolapsed and was replaced manually. Manual rotation of the head failed. There was a difficult forceps extraction and a still-born child. Three hours later the patient died of acute cardiac failure.

No. 1147. *Toxaemia of Pregnancy, Cardiac Failure.*

Primipara, age 25, maturity 36 weeks. History of oedema of legs for 2 months. Cough and dyspnoea for 3 days. B.P. 170/122, albuminuria marked, granular and hyaline casts present. Knee jerks present. Oedema of legs, lower abdomen, base of lungs. Normal, easy delivery. Patient became very cyanosed and died with signs of heart failure on third day.

No. 1385. *Albuminuria of Pregnancy, Avitaminosis B1, Myocardial Degeneration.*

Para 3, age 28, maturity 36 weeks. History of dimness of vision, dyspnoea, numbness of lower extremities for 1½ months. Marked general oedema, tenderness of calf muscles, absence of knee and ankle jerks. Moderate albuminuria, B.P. 120/66, left-sided enlargement of heart. Labour normal, lasting 1½ hours.

T.Y.H. (1938)
EMERGENCY

After delivery oedema became worse, lungs became waterlogged and patient died from cardiac failure 25 hours after delivery.

No. 1455. *Avitaminosis B1. Cardiac Failure.*

Para 8, age 38, maturity 36 weeks. History of oedema and breathlessness with numbness of lower extremities for 6 weeks, dyspnoea for last 5 days, inability to walk for one day. On admission marked general oedema, reflexes absent, slight albuminuria, B.P. 144/90. Signs of acute heart failure, died in a few hours without labour having started.

No. 1619. *Toxaemia of Pregnancy, Acute Parotitis.*

Primipara, age 20, maturity 40 weeks. History of oedema of legs for 1 month, admitted with marked oedema of legs and lower abdomen, vomiting, some tenderness of calf muscles, knee jerks absent. Marked albuminuria, B.P. 164/118. Twins delivered by forceps on account of primary uterine inertia with rising blood pressure and maternal distress. Labour lasted 36 hours. On second day temperature rose to 102, pulse 110, and a swelling of the left parotid gland appeared. On the following day the right parotid also became involved and the patient was transferred to the Queen Mary Hospital, where she subsequently died.

No. 1744. *Avitaminosis B1, Cardiac Failure. Posthumous Forceps Delivery.*

Primipara, age 22, maturity 40 weeks. History of oedema of legs and general anasarca for 5 months, with cough, dyspnoea and palpitation for the last 3 days. Admitted with clinical picture of cardiac beri-beri and cardiac failure. Knee jerks absent, B.P. 160/70, urine blood-stained, not tested. Labour was in progress. Patient was given 1,000 units Vitamin B intravenously, with cardiac stimulants and sedatives. Three hours after admission she became cyanosed and pulse imperceptible. At the time of death cervix was just fully dilated, so forceps were applied posthumously and a living male child delivered.

No. 2100. *Primary uterine inertial, Obstetric Shock.*

Primipara, age 29, maturity 40 weeks. There was a history of oedema of the legs for 3 weeks. Admitted in labour. The occiput was posterior and failed to

T.Y.H. (1938)
EMERGENCY

rotate. Labour was prolonged, the first and second stages lasting 72 hours. Delivery was finally effected with forceps, face to pubes, after attempts at rotation had failed. The baby was still-born. The placenta was retained and Crede's method with saline injection of the cord failed to bring about separation. 4¾ hours later, after treating shock with coramine and intravenous glucose and saline, the placenta was removed manually. For a time the patient rallied, but in half an hour she collapsed and died.

No. 2108. *Lobar Pneumonia.*

Para 2, age 27, maturity 38 weeks. History of two attacks with fever and rigors before admission. On admission moderate oedema of legs, tenderness of calves with weakness and numbness. Knee jerks absent. No albuminuria, B.P. 128/88. Normal delivery, membranes and placenta complete. One day after delivery patient had a rigor, Temperature 103, Pulse 120, with cough and pain in the back. Death from lobar pneumonia, with cardiac failure, on 7th day.

No. 2270. *Multiple fibroids of uterus, Post-partum haemorrhage.*

Para 4, age 39, maturity 40 weeks. Normal delivery, placenta and membranes intact. Post-partum haemorrhage started ¾ hour after delivery and resisted all treatment. Ergometrin, .5 mg. intramuscular and .125 mg. intravenous, failed to stimulate uterine contractions. The uterus was plugged and intravenous saline administered. The patient died 3 hours after delivery. Post-mortem examination revealed multiple small uterine fibroids.

INFANTS REPORT.

Total No. of Babies born ...	4,553	Alive	4,216
		Still-born	178
		Neo-natal Deaths	159

Full Term Infants:	*G.C.H. (1937)*	*T.Y.H. (1937)*	*T.Y.H. (1938)*	*Total*
Babies discharged alive	286	1,730	2,011	4,027
Still-born Babies	9	45	46	100
Neo-natal Deaths	5	21	35	61
Total	300	1,796	2,092	4,188

Premature Infants (Birth weight 5 lb. and under):

Babies discharged alive	16	68	105	189
Still-born Babies	2	33	43	78
Neo-natal Deaths	5	45	48	98
Total	23	146	196	365

STILL-BIRTHS.

There were 178 Still-births (including 78 premature babies).
Still-birth rate = 3.9%.

Reg. No.	*Sex*	*Method of Delivery*	*Maternal Complication*	*Cause of Death (P.M. if done)*	*REMARKS*
G.C.H. (1937)					
BOOKED					
266	F.	Normal	Nil	Macerated foetus	
EMERGENCY					
36	F.	Normal	Ankylostomiasis Avitaminosis	Prematurity	
57	M.	Breech	Nil	Asphyxia Severe Shock	
74	M.	Perforation	Pelvic deformity	Perforation	Old Tb. hip. Obstructed labour.
170	F.	Normal	Generalised Oedema	Unknown	Toxaemia.
186	F.	Normal	Ante-partum Haemorrhage	Unknown	
203	M.	Normal	B. Paratyphosus B.	Unknown	
218	F.	Brow presentation	Marginal Placenta Praevia	Macerated foetus	
220	F.	Breech	Nil	Prematurity	Macerated foetus.
247	F.	Normal	Syphilis	Macerated foetus	
512	M.	Normal	Retained Placenta	Prematurity	
T.Y.H. (1937)					
BOOKED					
28	F.	Normal	Nil	Prematurity	Macerated foetus.
1016	F.	Internal Version	Marginal Placenta Praevia	Prematurity	
1263	F.	Occipito-posterior	Maternal distress	Perforation	
1552	M.	Forceps	Nil	Foetal distress	
1742	M.	M. R. and Forceps	Pendulous Abdomen	Macerated foetus	
EMERGENCY					
1629/36	M.	Normal	Hydramnios	Generalised oedema	Macerated foetus.
127	M.	Normal	Syphilis	Prematurity	
223	F.	Breech	Nil	Prematurity	
238	F.	Breech	Syphilis	Prematurity	Ascites.
247	M.	Breech	Syphilis	Prematurity	
248	F.	Normal	Nil	Unknown	
285	M.	Normal	Syphilis	Syphilis	
327	M.	Normal	Nil	White Asphyxia	
354	M.	Normal	Nil	Unknown	
380	M.	Normal	Nil	Unknown	
395	M.	Normal	Nil	Prematurity	Macerated foetus. B.B.A.
451	F.	Normal	Atonic P.P.H.	Prematurity	
454	F.	Shoulder presentation	Internal Version. Extraction	Macerated foetus	
516	F.	Normal	Nil	Prematurity	Macerated foetus.
550	F.	Transverse	External Version	Prematurity	
582	F.	Breech	Nil	Prematurity	Macerated foetus.
596	M.	Normal	Syphilis	Prematurity	Macerated foetus.
655	F.	Normal	Marginal Placenta Praevia	Prematurity	
657	M.	Normal	Lateral Placenta Praevia	Prematurity	
661	M.	Shoulder to Breech	Internal Version. Extraction	Prematurity	
613	F.	Normal	Pre-eclampsia	Toxaemia	

STILL-BIRTHS.—(*Continued*).

Reg. No.	*Sex*	*Method of Delivery*	*Maternal Complication*	*Cause of Death (P.M. if done)*	*REMARKS*
T.Y.H. (1937)					
EMERGENCY					
731	M.	Breech	Nil	Prematurity	
753	F.	Breech	Nil	Prematurity	
742	M.	Forceps	Nil	Prematurity	Ascites.
804	M.	Breech	Syphilis	Macerated foetus	
821	F.	Breech	Nil	Deformity	
871	M.	Normal	Nil	Prematurity	Macerated foetus.
900	F.	Normal	Pre-eclampsia	Prematurity	
901	M.	Normal	Acute Avitaminosis B1	Syphilis	
980	M.	Assisted Breech	Nil	Foetal Shock	
985	M.	Transverse presentation	Central Placenta Praevia	Prematurity	Foetal Monstrosity.
1007	M.	Spontaneous	Lateral Placenta Praevia	Prematurity	
1039	F.	Face presentation	Nil	Unknown	Spontaneous delivery.
904	M.	Shoulder presentation	Pre-eclampsia	Prematurity Extraction	Macerated foetus.
1055	F.	Normal	Syphilis	Prematurity	Macerated foetus.
1097	F.	Assisted Breech	Pre-eclampsia	Prematurity	Foetal Shock.
1138	F.	Normal	Toxaemia	Asphyxia	Foetal Shock.
1145	M.	P.O.P. Spontaneous	Hydramnios	Foetal Oedema	
1190	M.	Breech	Nil	Foetal Shock	
1251	M.	Normal	Cerebro-spinal Meningitis	White Asphyxia	
1252	F.	Normal	Nil	Prematurity	Twins.
,,	F.	Breech	Nil	,,	
1276	F.	Normal	Oedema	Asphyxia	Prolapse of cord.
1295	M.	Assisted	Toxaemia	Asphyxia	2nd of Twins.
1328	F.	Normal	Syphilis	Prematurity	
1320	F.	Forceps	Maternal distress	Foetal distress	
1358	F.	Normal	Syphilis	Macerated foetus	
1347	M.	Normal	Nil	Toxaemia	
1373	M.	Normal	Nil	Macerated foetus	
1398	F.	Normal	Lateral Placenta Praevia	Asphyxia	
1414	M.	Breech	Syphilis	Prematurity	Macerated foetus.
1453	F.	Shoulder presentation	Lateral Placenta Praevia	Asphyxia	
1501	M.	Normal	Nil	Macerated foetus	
1539	F.	Normal	Syphilis	Macerated foetus	
1548	M.	Normal	Tuberculosis, Ano-perineal fistula	Foetal ascites	
1568	M.	Transverse presentation	Nil	Asphyxia	Internal Version.
1620	M.	Normal	Nil	Asphyxia	
1586	M.	Right Cleidotomy	Pre-eclampsia	Macerated foetus	
1675	M.	Normal	Marginal Placenta Praevia	Asphyxia Prematurity	
1684	F.	Normal	Elderly Primipara	Foetal Shock	Precipitate labour.
1709	F.	Forceps	Congestion of breasts	Asphyxia. Foetal Shock	
1710	F.	Normal	Syphilis	Prematurity	
T.Y.H. (1937)					
EMERGENCY					
1718	F.	Breech	Nil	Prematurity	Macerated foetus.
1753	M.	Perforation	Contracted Pelvis	Perforation	Failed forceps.
1780	M.	Normal	Oedema-Vulvitis	Prematurity	
1791	M.	Normal	Accidental Haemorrhage	Prematurity	

STILL-BIRTHS.—*(Continued 2).*

Reg. No.	*Sex*	*Method of Delivery*	*Maternal Complication*	*Cause of Death (P.M. if done)*	*REMARKS*
T.Y.H. (1937)					
EMERGENCY					
1885	F.	Normal	Hydramnios	Prematurity	
1889	M.	Breech	Pre-eclampsia	Prematurity	
1910	M.	Normal	Nil	Prematurity	
1949	F.	Transverse to Breech	Internal Version	Foetal Shock	
1952	F.	Normal	Nil	Prematurity	
1974	F.	Breech	Placenta Praevia	Asphyxia Prematurity	Prolapse of cord.
2016	M.	Normal	Ante-partum Haemorrhage	Haemorrhage from Placenta	
T.Y.H. (1938)					
BOOKED					
42	F.	Breech	Hydramnios	Macerated foetus	
130	F.	Breech	Nil	Prematurity	
927	M.	Forceps	Uterine inertia	Cerebral haemorrhage	Transverse lie of head.
1071	M.	Normal	Nil	Anoxaemia	
1273	F.	Normal	Nil	Cerebral haemorrhage	
1706	M.	Normal	Malaria (S.T.)	Prematurity	
2161	M.	Forceps	Nil	Inter-cranial haemorrhage	Transverse lie. Manual Rotation.
EMERGENCY					
2011/37	F.	Breech	Syphilis	Asphyxia	
2023/37	F.	Shoulder	Lateral Placenta Praevia	Macerated foetus	Internal Version.
2049/37	M.	Assisted	Contraction Ring	Foetal Ascites	
13	M.	Normal	Syphilis	Blue Asphyxia	
32	M.	Breech	Syphilis	Macerated foetus	
33	F.	Breech	Oedema	Cerebral haemorrhage	
68	M.	Forceps	Nil	Cerebral haemorrhage	Shock.
80	F.	Forceps	Toxaemia-Dysentery-Cystitis	Macerated foetus	Twins.
..	F.	..	..	..	..
136	F.	Transverse to Breech	Lateral Placenta Praevia	Asphyxia	
153	F.	Forceps	Marginal Placenta Praevia	Prolapse of cord	
154	F.	Normal	Lateral Placenta Praevia	Syphilis	
176	M.	Forceps	Puerperal Pyrexia	Prolapse of cord	
180	M.	Normal	Small-pox	Prematurity	
190	M.	Normal	Pre-eclampsia	Prematurity	
235	M.	Normal	Nil	Strangulation in utero	
280	F.	Normal	Nil	Asphyxia	
288	F.	Forceps	Uterine inertia	Foetal distress. Shock	
302	F.	Breech	Toxaemia	Prematurity	2nd of Twins.
325	F.	Normal	Toxaemia Avitaminosis B1	Toxaemia of pregnancy	
360	M.	Forceps	Central Placenta Praevia	Maternal haemorrhage	
416	F.	Transverse to Breech	Internal Version	Prematurity	Prolapse of left hand.
422	F.	P.O.P.	Toxaemia	Macerated Monster	
434	F.	Normal	Syphilis	Prematurity	
471	F.	P.O.P.	Oedema	Prolapse of cord	Shock.
590	F.	Breech	Toxaemia	White asphyxia	
632	M.	Breech	Nil	Prematurity	
726	M.	Breech	Chronic Nephritis	Macerated foetus	
740	M.	Transverse to Vertex	External Version	Macerated foetus	

STILL-BIRTHS.—*(Continued3)*.

Reg. No.	*Sex*	*Method of Delivery*	*Maternal Complication*	*Cause of Death (P.M. if done)*	*REMARKS*
T.Y.H. (1938)					
EMERGENCY					
765	F.	Forceps	Syphilis	Asphyxia	Face presentation.
777	F.	Normal	Lateral Placenta Praevia	Prematurity	
785	F.	Normal	Ante-partum haemorrhage	Prematurity	
820	M.	Breech	Nil	Prematurity	
825	F.	Breech	Nil	Asphyxia	
857	M.	Transverse to Breech	Syphilis	Macerated foetus	
1020	M.	Normal	Placenta Praevia	Prematurity	Anoxaemia.
1026	F.	Transverse to Breech	Placenta Praevia	Prematurity	Anoxaemia.
1061	M.	Normal	Primary Uterine inertia	Blue Asphyxia	
1078	F.	Breech	Syphilis	Macerated foetus	
1117	M.	Normal	Lateral Placenta Praevia	Anoxaemia	
1146	F.	Normal	Syphilis	Prematurity	
1187	M.	Assisted	Syphilis	Macerated foetus	
1200	M.	Forceps	P.P.H.	Asphyxia	
1201	F.	Normal	Syphilis	Prematurity	
1206	M.	Normal	Nil	Prematurity	1st of Twins.
1227	F.	P.O.P.	Nil	Prematurity	Macerated foetus.
1287	M.	Normal	Accidental Haemorrhage	Asphyxia Anoxaemia	
1290	M.	Breech	Nil	Prematurity	
1309	F.	Normal	Nil	Prematurity	
1315	M.	Shoulder presentation	Marginal Placenta Praevia	Prematurity	
1320	F.	Normal	Accidental Haemorrhage	Asphyxia Anoxaemia	
1385	M.	Normal	Cardiac disease-Oedema	Prematurity-Toxaemia	
1394	F.	Transverse presentation	Nil	Ascites	Macerated foetus.
1298	M.	Forceps P.O.P.	Eclampsia	Atelectasis	
1396	F.	Normal	Syphilis	Macerated foetus	
1412	F.	Normal	Nil	Prematurity	
1519	M.	Breech	Nil	Prematurity	Macerated foetus.
1518	M.	Normal	Syphilis	Cerebral haemorrhage	
1405	M.	Craniotomy	Nil	Craniotomy	Brow presentation.
1543	M.	Normal	Nil	Prematurity-Ascites	
1598	M.	Normal	Nil	Prematurity	
1625	F.	Normal	Nil	Syphilis	
1730	M.	Breech	Missed abortion	Macerated foetus	
1749	M.	Normal	Nil	Atelectasis Asphyxia	
1800	M.	Normal	Ante-partum haemorrhage	Macerated foetus	
1845	M.	Normal	Nil	Anoxaemia	
1886	M.	Bipolar version	Pulmonary Tuberculosis	Prematurity Non-viability	Transverse lie. Miscarriage.
1967	M.	Normal	Oedema	Atelectasis	
1970	M.	Normal	Pyrexia	Hyperpyrexia	Macerated foetus.
1988	F.	Normal	Syphilis	Macerated foetus	
2051	F.	Normal	Malaria-Dysentery	Macerated foetus	
2090	M.	Normal	Nil	Prematurity	Prolapse of cord and hand.
2100	F.	Forceps	P.P.H.	Inter-cranial haemorrhage	
2050	F.	Forceps	Nil	Anoxaemia	P.O.P. Manual Rotation.
2097	M.	Normal	Nil	Atelectasis	
2127	F.	Normal	Syphilis	Prematurity	Macerated foetus.
2176	F.	Normal	Toxaemia	Atelectasis. Shock	
2202	M.	Normal	Oedema	Foetal Ascites. Hepatomegaly	
2258	M.	Normal	Accidental Haemorrhage	Premature separation of Placenta	
2319	F.	Normal	Nil	Prematurity	Macerated foetus.
2354	M.	Normal	Nil	Foetal Ascites	
2206	M.	Normal	Pre-eclampsia	Toxaemia	

NEO-NATAL DEATHS.

There were 159 Neo-natal deaths (including 98 premature babies).
Neo-natal death rate = 3.5%.

Reg. No.	Sex	Birth Weight lbs.	Birth Weight ozs.	Method of delivery	Maternal Complication	Cause of Death (P.M. if done)	Age	Method of Feeding	REMARKS
G.C.H. (1937)									
EMERGENCY									
25	M.	5	4	Normal	Syphilis	Unknown	2½ days	Breast	
120	M.	3	8	Normal	Nil	Prematurity	5½ days	Glucose water	
159	M.	3	1	Normal	Syphilis	Prematurity	14½ hrs.	—	
233	F.	2	8	Normal	Nil	Prematurity	6 hrs.	—	
241	M.	7	4	Normal	M. Placenta Praevia	Foetal Shock	5 hrs.	—	
246	M.	8	0	Breech	Nil	Foetal Shock	15 mins.	—	
249	M.	6	0	Normal	Marked oedema	Atelectasis	1 day	—	Blue Asphyxia.
279	M.	4	4	Normal	Nil	Foetal Shock	7½ hrs.	—	White Asphyxia.
303	M.	6	0	Normal	Nil	Blue Asphyxia	39 hrs.	—	
310	M.	3	11	Normal	Syphilis	Prematurity	7 hrs.	—	
T.Y.H. (1938)									
BOOKED									
538	F.	7	2	Normal	P.P.H.	Enlarged spleen and liver	6 days	Breast	Distended abdomen.
723	M.	3	0	Normal	Mitral Stenosis	Prematurity	20 hrs.	—	
EMERGENCY									
1605/36	F.	6	6	Breech	Nil	Shock	1 hr.	—	
1608/36	M.	2	6	Normal	Nil	Prematurity	26 hrs.	—	
1610/36	M.	4	0	Normal	Nil	Prematurity	2 days	Breast	
1616/36	M.	2	12	Breech	Nil	Prematurity	20 mins.	—	
20	F.	6	8	Normal	Nil	Blue Asphyxia	15 mins.	—	
37	F.	2	6	Normal	Nil	Prematurity	11 hrs.	—	
185	F.	5	0	Normal	Generalised oedema	Ascites	25 mins.	—	
198	M.	3	0	Normal	Nil	Prematurity	55 mins.	—	1st Twins.
,,	M.	2	12	Breech	Nil	Prematurity	9 hrs.	Breast	2nd Twins.
199	M.	3	4	Normal	Nil	Asphyxia	10 hrs.	Artificial	
253	M.	5	0	Breech	Nil	Prematurity	10 mins.	—	
264	F.	1	8	Normal	M. Placenta Praevia	Prematurity	15 mins.	—	
297	F.	6	2	Normal	Syphilis	Syphilis	26 hrs.	Breast	
316	F.	6	1	Normal	Nil	Cerebral haemorrhage	3 days	Breast	
317	M.	5	2	Normal	Nil	Swollen abdomen	53½ hrs.	Breast	
508	M.	3	4	Normal	Nil	Prematurity	6 hrs.	—	
559	M.	5	10	Normal	Syphilis	Syphilis	8 hrs.	—	
569	F.	1	0	Normal	Nil	Prematurity	8 hrs.		
590	F.	2	11	Normal	Nil	Prematurity	11 hrs.	Breast	Twins.
	F.	3	4	,,	,,	Prematurity	4½ days	Breast	

NEO-NATAL DEATHS.—*(Continued 1).*

Reg. No.	*Sex*	*Birth Weight lbs.*	*oz.*	*Method of delivery*	*Maternal Complication*	*Cause of Death (P.M. if done)*	*Age*	*Method of Feeding*	*REMARKS*
T.Y.H. (1938)									
EMERGENCY									
608	M.	4	8	Normal	Oedema	Prematurity	1½ days	Breast	
625	F.	2	6	Normal	Nil	Prematurity	5½ hrs.	—	
663	F.	2	8	Breech	Nil	Prematurity	2½ hrs.	—	
684	F.	6	6	Forceps	Uterine Inertia	Asphyxia	11 hrs.	—	
688	M.	6	8	Caesarean Section	C. Placenta Praevia	Asphyxia	4 hrs.	—	
788	M.	6	14	Normal	Toxaemia	Pre-eclampsia	24 hrs.	Breast	
823	F.	4	4	P.O.P.	Nil	Cerebral haemorrhage	51½ hrs.	Breast	2nd of Twins.
892	F.	5	10	Normal	Diarrhoea	Prematurity	10 days	Breast	
911	F.	4	0	Normal	Nil	Prematurity	7 hrs.	Breast	
955	M.	6	8	Normal	Nil	Asphyxia	1 hr.	—	
1040	F.	3	4	P.O.P. Face to Pubis	Nil	Prematurity	10 hrs.	Breast	
1068	M.	6	0	Normal	Nil	Cerebral haemorrhage	11½ hrs.	-	
1117	M.	6	8	Normal	Nil	Anencephalus	4 hrs.	-	
1127	M.	1	8	Normal	Oedema	Foetal Shock	2½ days	- -	
1134	F.	3	14	Normal	Hydramnios	Prematurity	20 hrs.	-	
1180	M.	4	6	Normal	Nil	Prematurity	31 hrs.	-	
1221	F.	3	6	Normal	Nil	Prematurity	2½ hrs.	-	
1297	M.	3	4	Normal	Nil	Prematurity	50 hrs.	Breast	
1310	M.	4	12	Normal	Avitaminosis B1	Prematurity	91½ hrs.	Breast	Twins.
..	F.	4	4	Breech	..	..	20½ hrs.	..	
1321	M.	5	12	Normal	Nil	Congenital Syphilis	12 hrs.	Breast	
1324	M.	6	8	Breech	Nil	Cerebral haemorrhage	3½ hrs.	—	
1381	F.	5	2	Normal	Oedema	Prematurity	58 hrs.	Breast	
1391	M.	4	4	Normal	Syphilis	Prematurity	9½ hrs.	—	
1507	M.	4	0	Normal	Toxaemia	Prematurity	87 hrs.	—	Foetal Shock.
1535	F.	2	0	Normal	Placenta Praevia	Prematurity	4 hrs.	- -	
1536	M.	3	4	Normal	Syphilis	Prematurity	21 hrs.	- -	Twins.
..	M.	3	0	Breech	..	..	5½ hrs.	-	
1508	F.	4	8	Normal	Oedema	Prematurity	3¾ hrs.	—	
1581	F.	6	4	Normal	Toxaemia	Icterus Gravis Neonatorum	4½ days	Breast	
1516	F.	7	14	Normal	Avitaminosis B1	Diarrhoea	17 days	Breast	
1600	M.	2	0	Breech	Nil	Prematurity	24 hrs.	..	2nd of Twins.
1634	F.	4	8	Normal	Nil	Prematurity	55 mins.	—	
1681	F.	4	0	Normal	Syphilis	Prematurity	14 hrs.	—	
1713	M.	4	0	Normal	Syphilis	Prematurity	5 5 6 hrs.	..	
1720	F.	3	8	Normal	Toxaemia	Prematurity	5½ hrs.	—	2nd of Twins.
1722	M.	3	8	Normal	Avitaminosis B1	Prematurity	14 hrs.	—	
1732	F.	3	0	Normal	Malaria	Prematurity	18 hrs.	- -	
1736	M.	7	12	Breech	Nil	Pneumonia	21 hrs.	—	
1775	M.	6	2	Normal	Nil	Icterus Gravis Neonatorum	4 days	Breast	
1779	M.	3	10	Normal	Nil	Prematurity	13½ hrs.	—	Twins.
..	F.	2	8	Shoulder presentation	Nil	..	63 hrs.	—	
1832	F.	3	12	Normal	Syphilis	Prematurity	11½ hrs.	—	
1985	M.	4	8	Normal	Nil	Foetal Ascites	1 hr.	—	

NEO-NATAL DEATHS.—*(Continued 2).*

Reg. No.	*Sex*	*Birth Weight lbs.*	*oz.*	*Method of delivery*	*Maternal Complication*	*Cause of Death (P.M. if done)*	*Age*	*Method of Feeding*	*REMARKS*
T.Y.H. (1938)									
BOOKED									
622	F.	5	1	Normal	Hydramnios	Foetal Hepatomegaly	23¼ hrs.	—	
803	F.	6	4	Breech	Nil	Asphyxia	1 hr.	—	
1906	M.	6	2	Normal	Nil	Syphilis	33 hrs.	—	
EMERGENCY									
2020/37	M.	4	12	Forceps	Eclampsia	Prematurity	9½ hrs.	—	
31	M.	4	0	Normal	Syphilis	Prematurity. Lobar pneumonia	7 days	Breast	
38	M.	6	2	Normal	Nil	Unknown	3½ days	Breast	
61	M.	6	8	Normal	Nil	Unknown	3 days	Breast	
75	M.	3	0	Normal	Toxaemia	Prematurity	6 days	Breast	Twins.
..	F.	4	8	..	..	..	4½ days	..	Twins.
89	F.	3	8	Normal	Typhoid fever	Typhoid fever of mother	7 hrs.	—	
98	F.	3	6	Normal	Nil	Prematurity	2½ days	Breast	
107	M.	7	6	Normal	Pre-eclampsia	Unknown	3½ days	Breast	
112	F.	2	8	Breech	Nil	Prematurity	9 hrs.	—	
141	F.	3	0	Breech	Nil	Prematurity	3 5/6 hrs.	—	
143	M.	6	14	Normal	Syphilis	Pulmonary Embolism	2½ days	—	
171	M.	6	8	Normal	Nil	Unknown	2 days	—	
181	M.	1	12	Normal	Smallpox	Prematurity	6 hrs.	—	
187	F.	7	12	Normal	Nil	Asphyxia	6 hrs.	—	
231	F.	4	0	Normal	Nil	Prematurity	3 days	Breast	Twins.
,,	F.	4	6	..	..	,,	4 days	,,	Twins.
255	M.	5	12	Normal	Nil	Anencephalus	5 hrs.	—	
294	M.	6	1	Normal	Nil	Icterus Neonatorum	6 days	Breast	
302	F.	4	2	Normal	Toxaemia	Prematurity	26½ hrs.	—	1st of Twins.
398	F.	5	1	Normal	Nil	Prematurity	19 hrs.	—	
405	M.	2	14	Normal	Nil	Prematurity	2 hrs.	—	
573	M.	4	2	Normal	Nil	Prematurity	9½ hrs.	—	
624	M.	2	8	Normal	Syphilis	Prematurity	4 days	Breast	
662	F.	4	12	Normal	Placenta Praevia	Prematurity	44½ hrs.	—	
671	F.	5	12	Normal	Syphilis	Cerebral haemorrhage	34½ hrs.	—	
686	F.	1	10	Normal	Oedema	Prematurity	52 hrs.	—	1st of Twins.
698	M.	7	2	Normal	Nil	Anencephalus	21 hrs.	—	Monster.
716	M.	5	2	Normal	Placenta Praevia	Prematurity	1½ hrs.	—	
618	F.	6	8	Normal	Post-partum insanity	Cerebral haemorrhage	7 days	Breast	
732	F.	3	0	Normal	Pyrexia	Prematurity	3½ hrs.	—	
793	M.	3	1	Breech	Nil	Prematurity	5 hrs.	—	
804	F.	5	0	Normal	Syphilis	Prematurity	¾ hr.	—	
807	M.	5	0	Breech	Nil	Prematurity	10½ hrs.	—	
831	M.	6	8	Normal	Basal pneumonia	Broncho-pneumonia	38 hrs.	—	
881	F.	3	8	Normal	Nil	Prematurity Asphyxia	1 hr.	—	
926	M.	1	8	Normal	Nil	Prematurity	4 days	Breast	
976	F.	7	0	Normal	Cephal haematoma	Extra-cranial haemorrhage	7 days	Breast	
993	M.	3	4	P.O.P.	Syphilis	Prematurity	4 hrs.	—	

NEO-NATAL DEATHS.—(*Continued 3*).

Reg. No.	*Sex*	*Birth Weight* lbs.	ozs.	*Method of delivery*	*Maternal Complication*	*Cause of Death* (P.M. if done)	*Age*	*Method of Feeding*	*REMARKS*
EMERGENCY									
1011	M.	2	12	Normal	Nil	Prematurity	12½ hrs.	—	
1038	M.	3	0	Normal	Syphilis	Prematurity	2½ hrs.	—	
1052	M.	4	4	Normal	Nil	Broncho-pneumonia	2½ days	Breast	
1080	F.	7	12	Normal	Syphilis	Prematurity	23 hrs.	—	
1110	M.	6	0	Normal	Ankylostomiasis ...	Extradural haemorrhage ...	2½ days	—	
1164	F.	3	0	Normal	Placenta Praevia ...	Prematurity	3½ hrs.	—	
1206	M.	1	0	Breech	Nil	Prematurity	½ hr.	—	
1272	F.	3	1	Normal	Nil	Prematurity	3 hrs.	—	
1276	M.	4	4	Breech	Placenta Praevia ...	Prematurity	19 hrs.	—	
1330	F.	3	4	Breech	Nil	Prematurity	3½ hrs.	—	
1331	M.	6	4	Normal	Nil	Broncho-pneumonia	16½ hrs.	—	
1335	F.	4	8	Normal	Nil	Prematurity	29½ hrs.	—	
1381	M.	7	8	Normal	Oedema	Broncho-pneumonia	4 days	—	
1395	F.	5	4	Forceps	Pelvic contraction ...	Cerebral haemorrhage... ...	14½ hrs.	—	
1550	M.	5	0	Normal	Syphilis	Prematurity	1 hr.	—	
1614	M.	6	0	Forceps	Pre-eclampsia	Cerebral haemorrhage... ...	23½ hrs.	—	
1619	M.	5	0	Forceps	Pre-eclampsia	Shock following burns ...	23½ hrs.	—	Twins.
,,	M.	3	12	,, ...	,, ...	Prematurity	2 days	—	
1670	M.	6	12	Normal	Nil	Gastro-enteritis	8½ days	Breast	
1703	F.	4	11	Normal	2nd degree Prolapse	Prematurity	10½ hrs.	—	
1752	M.	3	4	Normal	Abscess of vulva ...	Prematurity	11 hrs.	—	
1781	M.	3	12	Normal	Nil	Prematurity	8¼ hrs.	—	
1801	M.	4	4	Normal	Oedema	Prematurity	6 days	Breast	
1803	M.	6	12	Normal	Nil	Convulsions	2 days	—	
1822	F.	2	8	Normal	Nil	Prematurity	2 months	Condensed milk	
1785	F.	6	4	Forceps	Nil	Blood disease of infant ...	6½ days	Breast	P.O.P.
1850	M.	5	4	Normal	Nil	Atelectasis...	17 hrs.	—	
1903	M.	4	8	Normal	Pyrexia	Congenital heart disease ...	33 hrs.	—	
1928	M.	3	12	Normal	Placenta Praevia ...	Prematurity	11 days	Condensed milk	Twins.
,,	M.	3	0	Breech	P.P.H.	,, ...	22 hrs.	—	
1948	F.	6	1	Normal	Nil	Partial Atelectasis	1 day	—	
2008	F.	2	8	P.O.P.	Malaria	Prematurity	8 hrs.	—	
2016	F.	3	12	Normal	Nil	Prematurity	2 days	—	
2055	F.	2	14	Normal	Toxaemia	Prematurity	12 hrs.	—	
2096	F.	2	6	Normal	Nil	Prematurity	4 hrs.	—	
1986	M.	3	8	Normal	Toxaemia	Prematurity	3 days	—	
2120	F.	5	14	Normal	Nil	Partial Atelectasis	6 days	Breast	
2227	F.	7	6	Normal	Syphilis	Gastric ulceration	2 days	—	Haemorrhage.
2347	M.	5	12	Normal	Hydramnios	Anencephalus Atelectasis ...	15 mins.	—	
2352	M.	5	12	Normal	Nil	Atelectasis...	17½ hrs.	—	
2358	F.	4	6	Normal	Nil	Prematurity	45 mins.	—	

FOETAL ABNORMALITIES.

Reg. No.	
G.C.H. (1937)	
EMERGENCY	
725/36	Right facial paralysis ... ...
819	Congenital Colonic Atresia ... ...
T.Y.H. (1937)	
EMERGENCY	
185	Foetal ascites. Generalised oedema ... .
196	Congenital Talipes Calcaneo-Valgus ... .
440	Complete Harelip and Cleft Palate ... ...
610	Harelip ... ...
630	Congenital Haemangioma of right leg ... ...
742	Foetal ascites and Hydro-thorax ... ...
985	Anencephalus. Absence of left arm. Talipes equino-varus, Double harelip. Extrusion of viscera ...
1117	Anencephalus ... ...
1415	Congenial dislocation of left knee joint... ... ...
T.Y.H. (1938)	
BOOKED	
965	Lipoma of 1st of Twins ... ...
1906	Cleft palate, Talipes Valgus (Right) ... ...
EMERGENCY	
55	Talipes Calcaneo Valgus ... ...
211	Hypo-padias ... ...
220	Phimosis ... ...
254	Complete Unilateral Harelip... ... ...
255	Anencephalus ... ...
332	Supernumerary digit ... .
422	Macerated Monster ... ...
441	Club foot (left) ... ...
642	Absence of rt. external auditory Meatus. Bilateral harelip, cleft patate, Dextro position of viscera ...
698	Anencephalus ... ...
934	Complete harelip and cleft palate ... ...
1016	Left sided harelip and cleft palate ... ...
1282	Right sided Talipes Equino-varus... ... ...
1910	Encephalocele ... ...
1934	Cleft palate... ... ...
2347	Anencephalus ... ...
2355	Bilateral harelip ... ...

OPHTHALMIA.

Reg. No.	*Number of days Treated*	*Remarks*
G.C.H. (1937)		
EMERGENCY		
129/36	5 days	
4	4 days	
221	1 day	Gonococcal.
T.Y.H. (1937)		
BOOKED		
870	7 days	Gonococcal.
EMERGENCY		
586	5 days	
669	3 days	D.A.A.
708	4 days	Gonococcal D.A.A.
1638	7 days	Gonococcal.
1664	3 days	
1704	4 days	Gonococcal.
T.Y.H. (1938)		
BOOKED		
2200	31 days	
EMERGENCY		
7	7 days	Gonococcal.
98	3 days	Ophthalmia Neonatorum.
504	11 days	Gonococcal. Neonatorum.
1140	11 days	G.C. in mother.
1353	10 days	Gonococcal Ophthalmia.
1526	7 days	Gonococcal Ophthalmia.
951	12 days	Gonorrhoea Syphilis.

REPORT OF GYNAECOLOGICAL DEPARTMENT.

From January 1937 until the middle of June 1937 Gyaecological cases were treated at the Government Civil Hospital, or at the Tsan Yuk Hospital, where a combined total of 23 beds was available. With the opening of the Queen Mary Hospital in June 1937, a 21 bed ward was set aside for Gynaecological cases, and all further patients were admitted to this Hospital.

The total number of admissions and operations for the two years is shown in the following table :—

	1937 (*G.C.H., T.Y.H., Q.M.H.*)	*1938* (*Q.M.H.*)
Number of admissions	458	428
Number of operations	305	300

Further details of the work done during the two years will be found in the following tables, each year being considered separately.

GYNAECOLOGICAL REPORT, 1937.

Table No. I

	T.Y.H.	*G.C.H. & Q.M.H.*
Number of admissions	130	328
Number of operations	73	232

NATURE & NUMBER OF OPERATIONS (1937).

Table No. II

	T.Y.H.	*G.C.H. & Q.M.H.*
Vulva:—		
Bartholin cyst, removal of	—	1
Post-partum atresia of vagina and vulva, partial divisions	1	—
Perineum:—		
Perinaeorrhaphy	—	1
Urethra:—		
Urethral caruncle, excision of	1	2
Urethro-vaginal fistula, repair of	—	1
Vagina:—		
Traumatic stenosis, posterior colpotomy ...	—	1
Vesico-vaginal fistula, repair of	—	2
Removal of vaginal septum	—	1
Colpo-perinaeorrhaphy for prolapse	—	21
Uterus:—		
Curettage (simple)	6	3
Curettage for incomplete abortion	8	10
Hysterectomy (Subtotal)	—	5
Hysterectomy (Total)	—	11
Wertheim's total hysterectomy	—	1

	T.Y.H.	G.C.H. & Q.M.H.
Abdominal hysterotomy	—	2
Removal of fibroid polyp	2	1
Removal of myxo-angio-fibroma	—	1
Ventral suspension	—	1
Cervix:—		
Amputation of	—	1
Polypus, removal of	2	8
Cauterization (Electro) for erosion	20	53
Dilatation and tubal insufflation	29	54
Fibroid, removal of	—	2
Tubes and Ovaries:—		
Ovariotomy	—	22
Salpingectomy	—	4
Salpingo-oopherectomy	—	12
Extra-uterine gestation	—	7
Miscellaneous:—		
Exploratory Laparotomy	—	1
Breast abscess	3	1
Abscess of pelvis	—	1
Nephrectomy	—	1
Dyspareunia, incision for	1	—
Total	73	232

Table No. III

NATURE AND NUMBER OF CASES TREATED WITHOUT OPERATION (1937).

	T.Y.H.	G.C.H. & Q.M.H.
Refused operation	2	1
Hyperemesis gravidarum	—	1
Pregnancy with syphilis, Gonorrhoea, Ascariasis	—	1
Pregnancy with fibroid	1	—
Pregnancy with Avitaminosis B_1	1	1
Pregnancy with Cystitis	—	1
Pregnancy with Condylomata, Syphilitic	—	1
Pregnancy with Malaria	—	1
Retroperitoneal Sarcoma	—	1
Fibroma of ovary (Inoperable)	—	1
Carcinoma of body of uterus (Inoperable)	1	1
Carcinoma of cervix (Radium treatment)	—	13
Carcinoma of cervix	2	4
Urethritis and Bartholin cyst	—	1
Ovarian cyst	2	2
Bartholin cyst and Haematuria	—	1
Fibromyoma	1	—
Incomplete abortion	1	4

	T.Y.H.	G.C.H. & Q.M.H.
Threatened abortion	7	2
Inevitable abortion	4	7
Cystocele and rectocele	—	1
Cystocele, malignant tumour of skull and pelvic bones, Primary cancer of Thyroid	1	—
Post abortive debility	1	—
Post partum beri-beri	—	1
Post partum vaginal stenosis	—	1
Extra-uterine gestation	2	—
Pelvic haemotocele	1	—
Endocervicitis	4	4
Cervical erosion and gonorrhoea	1	2
Ulceration of cervix and syphilis	3	1
Retroverted gravid uterus	—	2
Salpingo-oophoritis	6	13
Retroversion	3	2
Sterility	2	2
Pelvic cellulitis	—	2
Pelvic abscess	3	2
Leucorrhoea	1	—
Pyosalpinx	1	—
Chronic appendicitis	—	1
Accidental uterine haemorrhage following abdominal trauma	—	1
Vaginitis	3	1
Cystitis	1	1
Splenomegaly	—	2
Vesico-vaginal fistula, vaginal stenosis	1	—
Torn perineum	1	—
Chronic inflammatory granuloma	—	1
B. Coli Pyelitis, chancre	—	1
Parametritis	—	2
Alimentary stasis	—	1
Anaemia	—	1
Ovarian hormone disturbance	—	1
Avitaminosis A & B	—	1
Delayed menstruation	—	1
For observation	—	4
Total	57	96

Table No. IV

MORTALITY (1937).

There were 4 deaths.		
There were 4 deaths	1	3

Causes of death :—

1. Typhoid fever, incomplete abortion.
2. Tuberculous pyosalpinx and vaginal stenosis.
3. Ruptured extra-uterine pregnancy.
4. Hypernephroma.

Table No. V

CLASSIFICATION OF DISEASES (1937).

	T.Y.H.	*G.C.H. & Q.M.H.*
Vulva:—		
Post-partum atresia of vagina and vulva...	1	—
Bartholin cyst	—	3
Perineum:—		
Laceration of	1	1
Vagina:—		
Vaginal septum	—	1
Traumatic stenosis	—	3
Vesico-vaginal fistula	1	1
Vaginitis	3	1
Urethra:—		
Urethro-vaginal fistula	—	1
Urethral caruncle	1	2
Uterus:—		
Displacements—		
Retro-displacements (Retroversion)	4	3
Utero-vaginal prolapse	—	18
Retroverted gravid uterus	—	2
Cystocele and Rectocele	—	1
Cystocele and laceration of cervix and perineum	—	3
Uterine haemorrhage complicating secondary anaemia	1	—
Disorders of pregnancy—		
Incomplete abortion	7	14
Threatened abortion	7	2
Inevitable abortion	6	7
Hydatidiform mole	1	—
Disorders of menstruation—		
Dyspareunia	1	—
Metropathia Haemorrhagica	2	—
Endometritis	1	1
Parametritis	—	2
Cornual endometriosis, fimbrial cyst ...	—	1
Neoplasms—		
Submucous fibroid	—	1
Uterine fibroid	2	6
Fibroid polyp	2	—
Fibromyoma	—	6

	T.Y.H.	G.C.H. & Q.M.H.
Adenoma of body of uterus	1	1
Carcinoma of body of uterus	1	1
Retroperineal sarcoma	—	1
Myxo-angio-fibroma	—	1
Hypernephroma	—	1
Cervix:—		
Erosion of cervix	25	59
Ulceration of cervix	3	1
Hypertrophy of cervix	—	1
Polyp	2	6
Fibroid	—	2
Carcinoma	2	18
Tubes and Ovaries:—		
Inflammation—		
Acute salpingo-oophoritis	1	2
Chronic salpingo-oophoritis	5	12
Pyosalpinx	1	3
Pelvic abscess	3	4
Pyometra	—	1
Extra-uterine pregnancy	2	5
Pelvic haemotocele	1	2
Neoplasms—		
Carcinoma of left ovary	—	1
Ovarian pseudomucinous cystadenoma	—	23
Papilliferous cystadenoma	—	4
Ovarian cyst	2	6
Ovarian dermoid	—	1
Fibroma of ovary	—	1
Broad ligament cyst	—	1
Teratoma	—	1
Endometrioma of ovary	—	1
Sterility	31	58
Miscellaneous:—		
Pelvic cellulitis	—	2
Breast abscess	3	1
Syphilis and cystitis	1	1
Leucorrhoea	1	—
Pregnancy with cystitis	—	1
Pregnancy with Pulmonary tuberculosis	—	1
Pregnancy with Syphilis	—	1
Pregnancy with Avitaminosis B_1	1	1
Pregnancy with Condylomata of vulva	—	1

	T.Y.H.	*G.C.H.* & *Q.M.H.*
Pregnancy with Fibroid	1	1
Pregnancy with Cervical polyp	—	1
Pregnancy with Malaria	—	1
Hyperemesis gravidarum	—	1
Post abortive debility	1	—
Chronic appendicitis	—	1
Postpartum beri-beri	—	1
Splenomegaly	—	2
Anaemia	—	1
Ovarian hormone disturbance	—	1
Avitaminosis A & B	—	1
Delayed menstruation	—	1
Accidental uterine haemorrhage following abdominal trauma	—	1
Alimentary stasis	—	1
Chronic inflammatory granuloma	—	1
B. Coli Pyelitis, chancre	—	1
Chronic nephritis	—	1
Cystocele, malignant tumour of skull and pelvic bones, Primary cancer of Thyroid	1	—
For observation	—	4

Table No. I

GYNAECOLOGICAL REPORT, 1938.

Number of admissions	428
Number of operations	300

Table No. II

NATURE AND NUMBER OF OPERATIONS (1938).

Vulva:—	
Epithelioma, excision of, radium treatment	1
Fibroma of labium majus, removal of	2
Bartholin abscess, incision of	1
Abscess of vulva, incision of	1
Perineum:—	
Perinoeorrhaphy	2
Urethra:—	
Urethro-vaginal fistula, repair of	1
Urethral caruncle, removal of	5
Vagina:—	
Vaginal cyst, removal of	1
Recto-vaginal fistula, repair of	1
Ano-vaginal fistula, repair of	1
Fibroma of vaginal wall, removal of	1
Colpo-perinoeorrhaphy for prolapse	14
Vesico-vaginal fistula, repair of	1
Uterus:—	
Curettage (simple)	14
Curettage (for incomplete abortion)	4
Hysterectomy (Subtotal)	11
Hysterectomy (Total)	3
Wertheim's Total Hysterectomy	1
Myomectomy	2
Removal of fibroid polypi	4
Cervix:—	
Amputation of	2
Fibromyoma, removal of	2
Polypus, removal of	8
Cauterization (Electro) for erosion	100
Dilatation and tubal insufflation	31
Carcinoma, Radium treatment	15
Tubes and Ovaries:—	
Ovariotomy	23
Cauterization for salpingo-oophoritis	5
Salpingectomy	6
Salpingo-oophorectomy	6
Extra-uterine gestation	2
Caesarean section and sterilization	2
Sterilisation	1

Miscellaneous:—

Broad ligament cyst, removal of	4
Dermoid cyst, removal of	5
Fimbrial cyst, removal of	1
Endometriosis, removal of	1
Suppurating ovarian cyst, incision, drainage	1
Fibro-adenomatous polypi, Diathermy	1
Senile endometritis, Diathermy	3
Endometritis, ovarian cyst, removal, drainage	1
Exploratory Laparotomy	8
Mastitis	1
Total	300

Table No. III

NATURE AND NUMBER OF CASES TREATED WITHOUT OPERATION (1938).

Refused operation	2
Normal pregnancy	3
Provisional pregnancy	1
Pregnancy with pulmonary tuberculosis	1
Pregnancy with hypertrophy of cervix	1
Toxaemia of pregnancy	1
Normal uterus	2
Hyperemesis gravidarum	2
Hydatidiform mole	1
Incomplete abortion	5
Missed abortion	1
Inevitable abortion	4
Threatened abortion	9
Retained products of conception	2
External Haemorrhoids	1
Haemorrhage after biopsy	1
Dysmenorrhoea	2
Metropathia haemorrhagica	5
Sarcoma of uterus	1
Carcinoma of cervix	5
Carcinoma of body of uterus	1
Epithelioma of cervix	1
Fibro-adenomatous cervical polyp	1
Uterine fibroid	3
Ovarian cyst	7
Cystic ovary	1
Fimbrial cyst	1
Intraligamentous cyst	1
Abdominal tumour	1
Endocervicitis	8
Erosion and gonorrhoea	1
Gonococcal cystitis	1

Syphilitic cervicitis	1
Retroversion	2
Retroversion (Pessary inserted)	2
Acute anteflexion of uterus	1
Retroverted gravid uterus	1
Post-partum prolapse (Pessary inserted)	1
Prolapse	2
Urethritis	1
Urethritis and parametritis	1
Urethritis and cervicitis	1
Parametritis	3
Subinvolution	2
Sterility	4
Endometritis	2
Salpingo-oophoritis	10
Peritonitis	3
Pelvic inflammation	1
Gonococcal vulvo-vaginitis	1
Congenital absence of uterus and vagina	1
Post eclamptic debility	1
Post operative debility	1
Post partum beri-beri	1
Pleural effusion beri-beri	1
Tuberculous mesenteric lymph gland	1
Ischio rectal abscess	1
Laceration of perineum	1
Fractured spine first lumbar vertebra	1
Floating kidney	1
Diabetes	1
Hyperthyroidism	1
Total	128

Table No. IV

MORTALITY (1938).

There were 4 deaths.

Causes:—
1. Carinoma of cervix, post operative shock.
2. Toxaemia of pregnancy.
3. Inoperable ovarian cyst.
4. Parametritis, bilateral pyelo-nephritis, general debility, cardiac failure.

Table No. V

CLASSIFICATION OF DISEASES (1938).

Vulva:—

Fibroma of labium majus	2
Bartholin abscess	1
Abscess of vulva	1
Epithelioma	1

Perineum:—

Laceration	3

Vagina:—

Fibroma of vaginal wall	1
Vaginal cyst	1
Ano-vaginal fistula	1
Recto-vaginal fistula	1
Vesico-vaginal fistula	1
Gonococcal vulvo-vaginitis	1

Urethra:—

Urethro-vaginal fistula	1
Urethral caruncle	5
Urethritis and cervicitis	1
Urethritis and parametritis	1
Urethritis	1

Uterus:—

Congenital-Absence of uterus and vagina	1

Displacements—

Retro-displacement (Retroversion)	3
Utero-vaginal prolapse	17
Retroverted gravid uterus	1
Post-abortum retroversion	1
Normal uterus	2
Post partum retroversion	1
Acute anteflexion of uterus	1

Disorders of pregnancy—

Incomplete abortion	9
Inevitable abortion	4
Missed abortion	1
Tubal mole	1
Hydatidiform mole	3
Retained products of conception	6
Threatened abortion	9

Disorders of Menstruation—

Metrorrhagia	5
Dysmenorrhoea	4
Subinvolution	3
Metropathia haemorrhagica	1
Endometritis	9

Neoplasms—

Fibroid polyp	2
Uterine fibroid	11
Malignant adenoma	1

Sarcoma	1
Carcinoma of body of uterus	1
Submucous fibroid	5
Adenomyoma, Endometriosis	2
Carcinoma	1

Cervix:—

Erosion of cervix	109
Hypertrophied	1
Elongation and Rectocele	1
Syphilitic cervix	1
Polyp	9
Carcinoma	22
Fibromyoma	2
Epithelioma	1

Tubes and Ovaries:—

Inflammation—

Acute salpingo-oophoritis	3
Chronic salpingo-oophoritis	16
Pyosalpinx	2
Tubo-ovarian cyst	6
Extra-uterine pregnancy	4
Pelvic haemotocele	2

Neoplasms—

Adeno-carcinoma of left tube	1
Ovarian pseudomucinous cystadenoma	20
Papilliferous cyst	6
Dermoid cyst	6
Fibroma	1
Suppurating ovarian cyst	1
Tuberculosis of ovary	1
Broad ligament cyst	4
Carcinoma of ovary	1
Fimbrial cyst	3
Blood cyst of right ovary	2
Sterility	35

Miscellaneous:—

Tuberculous peritonitis	4
Pelvic peritonitis	2
Pelvic inflammation	5
Mastitis	1
Pregnancy with Pulmonary tuberculosis	1
Provisional pregnancy	1
Toxaemia of pregnancy	1
Normal pregnancy	3

Pregnancy with hypertrophy of cervix	1
Hyperemesis gravidarum	2
Ischio rectal abscess	1
Post eclamptic debility	1
Post operative debility	1
Tuberculous mesenteric lymph gland	1
Pleural effusion beri beri	1
Post partum beri beri	1
Hyperthyroidism	1
Gonococcal cystitis	1
Fractured spine, first lumbar vertebra	1
Interligamentous cyst	1
Chronic nephritis	1
Uterus subseptus with pregnancy	1
Floating kidney	1
Contracted pelvis	2
Abdominal tumour	1
Haemorrhage after biopsy	1
External haemorrhoids	1

Review of Books

"*A Manual of Practical Anatomy.*" By Thomas Walmsley. Second Edition. Part II. Thorax and Abdomen. Longmans, Green & Co., 12/6 net.

This volume opens with an Introduction of some 19 pages in which the author presents to the student notes of a general character on the structure of the trunk, its walls, its cavities and its contents. These notes include definitions of the various regions, thorax, abdomen and pelvis. Containing accounts of the general arrangement of the digestive tract, the genito-urinary system, the arterial and venous systems this Introduction contains much valuable information.

It is a novelty to meet a book which is not divided into chapters or sections named as such. At intervals one meets headings in heavy type such as "The Perineum," "The Lumbar Region." "The Abdomen." Even "The Thorax" comes as a surprise in the middle of a page. "The Thoracic Wall," the "Thoracic Cavity," the "Pleurae and Lungs" are headings in a medium type.

The plan of the author to avoid chapters and sections seems to suggest the most laudable view of the essential continuity of human structure.

The abdomen is not separable from the thorax nor the pelvis from the abdomen whether one's viewpoint is that of structure or of function.

The book conjures up a picture of a demonstration of the structure of the human frame presented by a master teacher making a running commentary with explanations as he proceeds with his class round. Very good and very interesting are these comments and explanations; for they cover questions of function, of embryology, of practical significance and include even areas of cutaneous sensibility in relation to abdominal and thoracic organs. It would not be difficult to visualise a scene such as is represented in the well known title page of Vesatins' "Fabrica," brought suitably up to date. The details of dissecting room technique which occupy so much space in many dissecting room manuals occur in the text undistinguished by special type and apparently are not held by the author to be of much consequence.

It might be widely agreed that details of dissecting technique might be dispensed with for students who can use their heads (and their hands) in independence of exact instructions, but not every teacher would recommend the method for all and sundry of his students.

Obviously a book such as this, loaded with information, must be bulky unless the type is small or the paper thin. The type may well prove too small for the comfort of many. While on this topic why will not an author represent to his publisher the need for a water proof cover to a dissecting room book?

The book contains 131 diagrams in its 323 pages of text. Many are new and vigorous but others are traditional or adaptations.

The X-rays, of which there are seven, are well reproduced and are informative.

It is good to see errors eliminated and new knowledge included. Particularly welcome are the elimination of the traditional description of the intercostal muscles and nerves; equally so are the inclusion of the "fascia lunata" of Elliot-Smith in the perineum, and the elimination of Alcock's canal.

Study of this book should be part of the training of a junior demonstrator and many senior students and young graduates would profit by reading it.

Acknowledgments.

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