patient had an acute bacterial endocarditis which was apparently cured by penicillin therapy, and that she died from acute heart failure.

We wish to thank Mr. James Wyatt, consultant to the puerperal sepsis unit, for his interest and help; Sir Bernard Spilsbury for the post-mortem reports on Cases II and VI; Mr. B. C. Maybury and Prof. W. G. Barnard for access to clinical and necropsy reports on Case III; Sisters Corcoran and Walshe and the nursing staff of the puerperal sepsis unit for their active co-operation in the nursing of the patients; Mr. E. W. Gregory for technical assistance; and the M.R.C. Penicillin Clinical Trials Committee for supplies of penicillin.

REFERENCES

- Christie, R. V. (1945). *British Medical Journal*, **1**, 232.
 Colebrook, L., and Purdie, A. W. (1937). *Lancet*, **2**, 1237.
 Cunliffe, A. C., Gillam, G. G., and Williams, R. (1943). *Ibid.*, **2**, 355.
 Dawson, M. H., and Hunter, T. H. (1945). *J. Amer. med. Ass.*, **127**, 129.
 Fleming, A. (1944). *Lancet*, **2**, 620.
 Florey, M. E., and Florey, H. W. (1943). *Ibid.*, **1**, 387.
 Fry, R. M. (1938). *Ibid.*, **1**, 199.
 Hill, A. M., and Butler, H. M. (1940). *Med. J. Austral.*, **1**, 273.
 Katz, L. N., and Elek, S. R. (1944). *J. Amer. med. Ass.*, **124**, 149.
 Keefer, C. S., Blake, F. G., Marshall, E. K., jun., Lockwood, J. S., and Wood, W. B. (1943). *Ibid.*, **122**, 1219.
 Kehr, H. L., and Adelman, M. (1942). *Amer. J. Dis. Child.*, **64**, 487.
 Keith, H. M., and Heilman, F. R. (1943). *Ibid.*, **65**, 77.
 Koletsky, S. (1941). *Ohio State med. J.*, **37**, 866.
 Loewe, L., Rosenblatt, P., Greene, H. J., and Russell, M. (1944). *J. Amer. med. Ass.*, **124**, 144.
 MacDonald, I. (1939). *Med. J. Austral.*, **2**, 471.
 McLellan, N. W., and Goldbloom, A. (1942). *Canad. med. Ass. J.*, **46**, 136.
 Ramsay, A. M., and Gillespie, M. (1941). *J. Obstet. Gynaec. Brit. Emp.*, **48**, 569.
 Rosenthal, A. H., and Stone, F. M. (1940). *J. Amer. med. Ass.*, **114**, 840.
 Thayer, W. S. (1931). *Gibson Lectures, Edinb. med. J.*, **38**, 237.
 Willis, F. A. (1943). *Proc. Mayo Clin.*, **17**, 216.

STAPHYLOCOCCUS PYOGENES SEPTICAEMIA TREATED WITH PENICILLIN

REPORT OF TWO CASES

BY

LEONARD HOWELLS, M.D., B.Sc., M.R.C.P.

Lieut.-Col., R.A.M.C.; Officer in Charge, Medical Division of a General Hospital

R. R. HUGHES, M.D., M.R.C.P.

Major, R.A.M.C.; Medical Specialist

AND

R. RANKIN, M.B., B.Sc., Ch.B.

Capt., R.A.M.C.; Graded Pathologist

The following is an account of two cases of *Staphylococcus pyogenes* septicaemia treated with penicillin.

Case I

The patient was a marine aged 22. On Sept. 28, 1944, he developed headache, sweating, slight cough, and pain in the back; he remained on duty all night and was sent to the sick-bay the next day. On the 30th he was admitted to hospital still complaining of the above symptoms. On examination he was rather apathetic, temperature 101.6°, pulse rate 100, and respiratory rate 22 a minute; his throat was slightly injected, but no other abnormality was found.

By Oct. 1 his condition had deteriorated; his temperature was swinging to 102° F., and the pulse and respiratory rates were rising. He was confused, restless, and irritable, his heart apex beat was in the mid-clavicular line in the fifth left intercostal space, there was some neck rigidity, and Kernig's sign was slightly positive. In view of these findings a clinical diagnosis of meningococcal meningitis was made. Lumbar puncture was done under pentothal anaesthesia; the cerebrospinal fluid pressure was 200 mm., and the fluid was slightly murky in appearance; on laboratory examination it was found to contain 40 red blood cells and 9 white blood cells per c.mm. and 40 mg. of protein (no excess of globulin), 70 mg. of glucose, and 700 mg. of chlorides per 100 c.cm. The urine was loaded with albumin (0.75 g. Esbach), and on microscopical examination scanty epithelial cells, leucocytes, and granular casts were found. Blood examination showed a haemoglobin of 89%, red blood cells 4,750,000 per c.mm., and white blood cells 14,000 per c.mm., with 85% polymorphonuclears.

On Oct. 2 the patient was still confused and irritable, with a temperature swinging to 102° F. and raised pulse and respiratory rates. On examination the neck rigidity and Kernig's sign were more pronounced, the fundi were normal, all tendon reflexes were absent, and plantar responses showed voluntary withdrawal of both feet. The heart was enlarged, with the apex beat $4\frac{1}{2}$ in. from the midline in the fifth left intercostal space, a systolic murmur was heard at the apex and base of the heart, and the blood pressure was 140/60. The white cell count had now risen to 23,600 per c.mm., with 84% polymorphs, the blood urea was 55 mg. per 100 c.cm.,

the urine was still loaded with albumin, and leucocytes and granular casts were still present.

During the day the patient's pulse rate had been rising steadily, and at 22.00 hours had reached 142 a minute. At this time he suddenly developed a left hemiparesis involving the limbs and face, and on examination his heart was found to be more dilated and the systolic murmur still present. In view of these developments it was now felt that a clinical diagnosis of infective endocarditis could be made; although this had not yet been confirmed by blood culture the dangerously ill condition of the patient made immediate specific treatment essential. Penicillin therapy was therefore started; he was given 100,000 units at 23.30 hours, followed by 40,000 units intramuscularly 3-hourly day and night.

By 09.00 hours on Oct. 3 he had been given 220,000 units of penicillin. His general condition had improved somewhat and his pulse rate had fallen to 108 a minute, but he was still in a state of low muttering delirium, his neck rigidity was more marked, and his hemiplegia was unchanged. The urine was loaded with albumin, the blood urea was 80 mg. per 100 c.cm., and the cerebrospinal fluid now contained 376 red blood cells per c.mm. Blood and cerebrospinal fluid cultures were made on this date and later; both were reported to contain *Staph. pyogenes*. The bacteriological report was as follows: "A smooth-growing well-pigmented strain of *Staph. pyogenes* was isolated after 36 hours. The organism was coagulase-positive, moderately haemolytic, and showed a sensitivity to sodium penicillin equalling that of the standard 'Oxford' strain." When re-examined at 19.00 hours his general condition was unchanged. He was still delirious, the neck rigidity and hemiparesis were still pronounced although he could now use his left arm a little, his heart apex beat was 1 in. outside the left mid-clavicular line in the fifth intercostal space, and a soft short apical diastolic murmur could be heard. The spleen was not palpable.

On Oct. 4 there was an improvement in the patient's general condition, his temperature had settled to the region of 99° F., and his pulse rate was steadily falling. He was, however, still delirious and the neck rigidity and hemiparesis were severe; the heart apex beat was well outside the left mid-clavicular line, and the soft apical diastolic murmur could still be heard. The blood urea was 60 mg. per 100 c.cm. On the 5th the patient's temperature was normal and his pulse and respiratory rates were falling rapidly. He was still very ill, but his condition had improved in the past 24 hours and he was having short lucid intervals. A crop of purpuric spots had appeared on the toes of both feet. The cerebrospinal fluid pressure was 250 mm.; the fluid contained 167 red blood cells per c.mm., and on culture was sterile. By 20.30 hours he had been given 1,020,000 units of penicillin; his condition was rapidly improving; he was fully conscious, with only occasional short periods of confusion, and was able to use his left hand to hold a feeding-cup.

By Oct. 6 the patient's condition had very much improved and he had been afebrile for the past 24 hours. He was still having occasional slight delirium and his neck rigidity was more pronounced; his hemiparesis was improving and he was now moving his left arm and leg a good deal. There was a left extensor plantar response. The heart apex beat was still well outside the mid-clavicular line in the fifth left intercostal space; there was a soft localized aortic systolic and a harsh apical diastolic murmur. The liver and spleen were not palpable. The blood urea was 55 mg. per 100 c.cm., the urine still contained albumin, and there were some leucocytes and an occasional granular cast in the deposit.

On Oct. 7 he was using his left arm and leg a good deal although weakness persisted; his condition was otherwise unchanged, and he was delirious at times. The next day an aortic diastolic murmur was heard for the first time. The white blood count was 14,150 per c.mm. with 81% polymorphs. A course of sulphapyridine was started with an initial dose of 2 g., followed by 2 g. four hours later, then 1 g. 4-hourly by mouth.

On Oct. 9 his general condition was very good, his appetite was satisfactory, and he was sleeping well with only very few short periods of delirium. He was still having an occasional rise of temperature to 99 or 100° F. On examination the heart apex beat was in the mid-clavicular line in the fifth left intercostal space; there was no palpable thrill. Auscultation revealed a rough aortic systolic murmur which was conducted along the subclavian and carotid arteries, and also a soft short aortic diastolic murmur conducted down the left border of the sternum. The pulse was collapsing, the blood pressure 140/40, and a pistol-shot murmur was audible over the femoral arteries. The spleen was not palpable. He was now using the left arm normally, but there were a slight weakness of the left lower face, a spastic left leg, and a left extensor plantar response. The abdominal reflexes were present but diminished on the left side. The blood urea had now fallen to 45 mg. per 100 c.cm. Radiographs showed definite enlargement of the heart to the left, with the general configuration of an early aortic lesion. There was also some hyperaemia at the base of the right lung.

The patient's condition was still improving on Oct. 10 and his neurological signs had almost completely cleared. On this date a

number of large haemorrhagic blisters appeared on the hands and feet. Red blood cells were found in the urine for the first time (specimens had been examined daily for these since admission). By the 12th he had had a total of 3,020,000 units of penicillin, and although there was an occasional rise of temperature to 99 or 100° the drug was discontinued. By Oct. 18 the patient's condition was excellent and he had been afebrile for the past 7 days. There was still some slight weakness of the left face, the to-and-fro aortic bruit was unchanged, and the heart was enlarged; the heart sounds, however, were much stronger. The blood count showed a haemoglobin of 79%; the red blood cells numbered 3,700,000 and white cells 10,600 per c.mm., with 68% polymorphs. The blood sedimentation rate was 4 mm. in 1 hour, and the urine still contained a few leucocytes, red cells, and granular casts. The sulphapyridine was now discontinued after a total dose of 50 g.

The patient was still very well on Oct. 23. He had been afebrile since the 11th, but the pulse rate was rising, and varied between 84 and 108 a minute. On examination there was slight weakness of the left lower face and of the left arm and leg, the abdominal reflexes were somewhat diminished on the left side, the left knee- and ankle-jerks were both slightly increased, and both plantar responses were flexor. The heart apex beat was $3\frac{1}{2}$ in. from the midline in the fifth left intercostal space, the to-and-fro aortic murmur was still present but now very faint, and there was still a pistol-shot sound over the femoral and brachial vessels. The systolic blood pressure was 148 mm., the diastolic uncertain but probably about 48 mm. Hg.

On Nov. 11 he was feeling very well, and his appetite and sleep were good. He continued to be afebrile, but his pulse rate remained at 90 to 100 a minute. On examination there was a slight weakness of the left lower face; the left arm and leg were both normal and the plantar responses flexor. The heart apex beat was still diffuse and heaving, 4 in. from the midline in the fifth left intercostal space. On auscultation a rough systolic bruit could be heard over the whole of the praecordium, but was loudest in the aortic area and was conducted slightly along the carotid and subclavian arteries; a soft diastolic bruit was also audible over the whole praecordium, but loudest in the pulmonary area. The lungs and abdomen were normal; the spleen was not palpable. The blood sedimentation rate was 0.5 mm. in 1 hour; the Kahn test was negative and the blood count showed a haemoglobin of 85%, red cells 4,010,000, and white cells 9,300 per c.mm., with 69% polymorphs. The pulse was collapsing in type, the blood pressure 150/30, and a pistol-shot murmur was audible over both brachial and femoral vessels. On Nov. 11 he was evacuated to the United Kingdom.

Comment on Case I

This was a typical case of infective endocarditis due to *Staph. pyogenes* treated with penicillin and sulphapyridine. No primary focus was found, there being no evidence of staphylococcal infection and no history of any such condition. When the case was first seen the diagnosis was obscured by a subarachnoid haemorrhage and a hemiparesis, presumably the result of the rupture of a mycotic aneurysm; by the fifth day of the disease, however, the cardiac changes were definite enough to venture a clinical diagnosis, this being confirmed later by isolation of the organism from the blood and cerebrospinal fluid. Penicillin treatment was begun on the fifth day of the disease, and within 52 hours the temperature had fallen to normal: 3,020,000 units were given over a period of nine days. However, as he still showed occasional rises of temperature, this was followed by a course of 30 g. of sulphapyridine.

During the early stages of the illness the patient had the usual features of a septicaemia: temperature swinging to 102° F., rapid pulse and respiratory rates, a white blood cell count up to 23,000 per c.mm., and a haemoglobin which fell from 89% on the fourth day to 79% on the nineteenth day of illness. After treatment of the disease the temperature had been normal for 26 days, the white cell count was 9,300 per c.mm., the haemoglobin had risen to 85%, and the blood sedimentation rate was 0.5 mm. in 1 hour. One unsatisfactory feature was a pulse rate remaining persistently at the 90-100 level despite complete bed-rest.

The embolic phenomena were of particular interest. On the day after admission to hospital the patient was noted to have signs of slight meningeal irritation, and on lumbar puncture his cerebrospinal fluid was found to contain 40 red blood cells per c.mm. The meningeal signs gradually increased in severity, and 24 hours later he suddenly developed intense neck rigidity and a left hemiparesis, and the cerebrospinal fluid contained 376 red cells per c.mm. The neck rigidity gradually subsided during the next week. The hemiparesis was more persistent,

but it improved steadily, and on discharge all that remained was slight weakness of the left lower face and minimal changes in the reflexes on the left side. It seems likely that the patient had an infected cerebral embolus which produced a mycotic aneurysm very early in the course of the disease, leaking slightly at first, and later more severely to produce a focal cerebral lesion. Other embolic manifestations were a crop of purpuric spots and, later, numerous large haemorrhagic vesicles on the toes and feet. Osler's nodes were not seen; there were no retinal emboli and no splenic enlargement at any time.

The renal changes provided some interesting and confusing features. At the time of the intracranial haemorrhage (the fourth day of the disease) there was heavy albuminuria, with granular casts in the urine and a blood urea estimated two days later at 80 mg. per 100 c.cm.; such changes are not uncommon with acute intracranial conditions like haemorrhage and meningitis, and are apparently not associated with renal damage. Together with the mental disturbance they raised the possibility of uraemia at that stage of the illness. The urine was examined for red blood cells each day, but these were not found until the 13th day of the disease; by this time the blood urea had fallen to 45 mg. per 100 c.cm., and there remained only a trace of albumin together with granular casts in the urine. These later changes were probably due to an embolic focal nephritis, and persisted for some time.

When questioned about previous heart trouble, after the acute phase of the illness was past, the patient admitted to an attack of "rheumatism" in childhood. This seems to have left no gross heart lesion, as he was able to exercise without undue discomfort and no heart lesion was discovered at several medical examinations during his naval service. On admission to hospital his heart was clinically normal; during the acute stages of the disease it rapidly enlarged, various changing murmurs were produced, and on the fifth day of the illness the blood pressure was 140/60. It was not until the 11th day that an aortic diastolic murmur was heard, and the following day his case had the classical features of an aortic regurgitation. On consideration of these findings it seems possible that there may have been some slight rheumatic damage to the aortic valve which determined the localization of the septicaemia in this situation, and that this damage was much increased by the present illness. As the septicaemic condition subsided the heart diminished in size, and we are under the impression that the murmurs became less intense, but at the time of discharge from hospital there could be no doubt that he had a severe aortic leak.

While, in view of the short period of observation, it is realized that the result in this case cannot be regarded as an undoubted cure, we feel that it is most encouraging and that penicillin is worthy of trial in other cases of this condition.

Case II

The patient, a sapper aged 24, was admitted to hospital on Aug. 27, 1944. His illness had begun on Aug. 21 with a sore throat, fever, and general malaise. On admission he looked ill; his temperature was 103° F., pulse rate 100, and respiratory rate 25 a minute. The tonsils were considerably enlarged, oedematous, and covered by a yellowish follicular exudate. There were no abnormal signs in the heart, lungs, abdomen, or central nervous system. Examination of a throat swab was reported upon as follows: "Direct film showed Vincent's organisms and a few Gram-positive cocci, and on culture non-haemolytic streptococci and a scanty growth of staphylococci were obtained; examination for K.L.B. was negative." He was given sulphamylamide, 2 g. initial dose, followed by 1 g. every four hours. On Aug. 28 he had two rigors, the temperature reaching 103.8° F.

On Aug. 29 there was a slight deterioration in his general condition, with no change in the appearance of the throat but a decrease in the fever during the day. A pleural friction rub was heard in the right axillary region. The urine contained a trace of albumin. Examination of a blood film for malaria parasites was negative. The total white cell count was 7,200 per c.mm., with 50% polymorphonuclears, 8% small lymphocytes, 30% large lymphocytes, and 12% monocytes. He had now received 12 g. of sulphamylamide with no apparent improvement, so it was decided to substitute sulphathiazole, 1 g. every four hours, instead.

During Aug. 30 and 31 the fever varied from 97 to 103° F., the pulse rate rose to 128 a minute, and he was now seriously ill with no change in the condition of the throat. On Sept. 1 the spleen was palpable clinically for the first time, the pleural friction rub

persisted, and he began to expectorate blood-stained sputum. The tonsils were still greatly enlarged and covered by a yellowish exudate. Sputum examination for tubercle bacilli was negative.

Blood culture on Sept. 2 gave a good growth of *Staph. pyogenes* (confirmed two days later, when another good growth was obtained). The organism had the following characteristics: a smooth moderately pigmented strain of *Staph. pyogenes*, non-haemolytic, coagulase-positive; penicillin sensitivity equal to that of the standard "Oxford" strain. It was now clear that the patient had a general septicaemia, and in spite of having received 12 g. of sulphanilamide and 25 g. of sulphathiazole he was getting steadily worse. Sulphathiazole treatment was stopped, and at 22.00 hours penicillin was given instead, in doses of 16,666 units, by intramuscular injection every three hours. The temperature at 22.00 hours was 102° F., and the pulse rate 110 a minute. No pathogens were obtained from culture of a throat swab. The total white cell count was still relatively low considering the nature of the infection—namely, 6,800 per c.mm., with 50% polymorphs, 27% small lymphocytes, 19% large lymphocytes, and 4% monocytes. On Sept. 3 and 4 a slight lowering of the temperature and some improvement in his general condition were apparent. On the 5th the temperature dropped to normal and was accompanied by a striking improvement in the patient's general condition and in the local appearance of the throat. The spleen was no longer palpable and the pleural friction rub could not be heard.

Subsequent Progress.—There was a rise of temperature to 100° F. on Sept. 6 and 7 and to 99° F. on Sept. 9 and 10, but subsequently the temperature and pulse rate remained at normal levels. Penicillin was discontinued on Sept. 11, as the patient had now received 1,133,288 units. By this time all clinical evidence of septicaemia had gone and the appearance of the throat was normal. He continued to make an uneventful recovery, with no residual signs of disease in any system, and on Sept. 20 was evacuated to the United Kingdom for a period of convalescence.

Comment on Case II

This was a case of *Staph. pyogenes* septicaemia associated with acute tonsillitis. The patient's resistance against the infection was poor, as was evidenced by a serious deterioration in his clinical condition and by the unsatisfactory white blood cell response. Treatment with 12 g. of sulphanilamide followed by 25 g. of sulphathiazole failed to produce any improvement in the local tonsillar infection or in the general condition of the patient. Penicillin was administered at a critical stage of the illness, and within 56 hours was followed by a dramatic improvement and, a few days later, by a complete and uncomplicated recovery.

Summary

The above two cases present encouraging features in the treatment of *Staph. pyogenes* septicaemia with penicillin. In the first case the favourable result was probably assisted by sulphapyridine therapy, but in the second case sulphanilamide and sulphathiazole each failed to produce any improvement in the condition. It is felt that these results may encourage others to employ and observe the effect of penicillin therapy in similar cases.

We wish to thank Col. F. G. Flood, M.C., officer commanding the military hospital, for permission to publish this report.

The 150th annual report by the Managers of Glasgow Royal Infirmary, for the year 1944, shows that a very large volume of work was done for the sick and injured from all parts of Britain, including a great many Service cases treated in the wards. For a long time wards have been set aside for the treatment of burns, and more of these injuries have been dealt with at the Glasgow Royal Infirmary than at any other hospital in the British Isles. In view of the need for further research on various aspects of the treatment of burns, the Medical Research Council, in agreement with the managers of the Infirmary, decided to appoint a junior surgeon to give whole-time assistance to Mr. A. M. Clark, who has been in charge of the burns wards for several years, and to establish a laboratory team to survey the problem along with other members of the staff. During last year 318 patients suffering from burns and scalds were admitted. The outstanding features were the greatly reduced death rate—5.6%—and the elimination to a very large extent of streptococcal infection, the main source of trouble in burns. All out-patients with burn injuries are now treated in the Unit; they number about a thousand a year. During 1944 the Infirmary received a very considerable supply of penicillin for the treatment of military and civilian cases. Large supplies were distributed to other hospitals in Glasgow, Lanarkshire, Stirlingshire, and Renfrewshire, and the Infirmary is not only a distributive centre but an advisory centre for the use of penicillin.

LOCAL PENICILLIN THERAPY IN OPHTHALMIA NEONATORUM*

BY

ARNOLD SORSBY, M.D., F.R.C.S.

(White Oak (L.C.C.) Hospital, Swanley)

In an earlier communication (Sorsby and Hoffa, 1945) an account was given of the use of penicillin for local therapy in 47 cases of ophthalmia neonatorum. It was shown that consistent results could be obtained when drops in a concentration of not less than 2,500 units per c.cm. were used; of 22 cases so treated 21 recovered, against only 3 out of 8, 4 out of 7, and 6 out of 10 treated with penicillin in concentrations of 500, 1,000, and 1,500 units respectively. The gratifying results obtained with penicillin in adequate concentration had been achieved by instilling one drop of the solution at half-hourly intervals for 3 hours, then hourly for 24 hours, and 2-hourly subsequently. None of these 21 successfully treated cases took longer than 100 hours to show a clinical cure, and in 6 cases clinical cure was obtained within 3 to 24 hours. It was indicated that all the common causal organisms of ophthalmia neonatorum, including the virus of inclusion blennorrhoea, appeared to respond to penicillin, though perhaps to varying degrees, diphtheroid bacilli probably being the most resistant.

A Further Series of 38 Cases

Like the last 22 cases in the previous series these 38 new cases have all been treated with penicillin in a concentration of 2,500 units per c.cm., but the frequency of instillation has been different: at longer intervals in 13 and at shorter intervals in 25 cases.

(a) *Thirteen cases treated with drops of penicillin at intervals of 1 hour for 6 hours, then 2-hourly for 24 hours, and subsequently at 3-hourly intervals.*—All these responded well, but one case relapsed after 24 hours and another after 5 days (both of these being finally cleared by a course of sulphamezathine for 5 days and 2 days respectively). The clinical details on these 13 cases are summarized in Tables I and II.

TABLE I.—Time taken for Clinical Cure: in Relation to Severity and Distribution of the Affection

	No. of Cases	Clinical Cure in
Mild:		
Unilateral	3	4, 48, and 56 hours respectively
Bilateral	0	
Moderate:		
Unilateral	2	30* and 38 hours respectively
Bilateral	3	58, 60,† and 60 hours respectively
Severe:		
Unilateral	1	20 hours
Bilateral	4	4, 18, 26, and 34 hours respectively

* Mild relapse after 24 hours. † Relapse after 5 days.

TABLE II.—Time taken for Clinical Cure: in Relation to Causal Organism and Severity of the Affection

	No. of Cases	Clinical Cure in
Gonococcus:		
Mild	1	4 hours
Moderate	0	
Severe	3	20, 26, and 34 hours respectively
Gram-positive cocci		
Severe	1	18 hours
Staph. aureus:		
Severe	1	4 "
Diphtheroids:		
Mild	1	56 "
Inclusion bodies present (with Gram-positive cocci and Gram-positive bacilli respectively):		
Moderate	2	30* and 38 hours respectively
No organisms or inclusion bodies present:		
Mild	1	48 hours
Moderate	3	58, 60,† and 60 hours respectively

* Mild relapse after 24 hours. Inclusion bodies were also present in scrapings from the mother's cervix.

† Relapse after 5 days. The culture had shown the presence of *Ps. pyocyanea*, probably a contaminant.

These results do not appear to be substantially different from those obtained in the 22 cases treated at more frequent intervals.

* A report to the Penicillin Clinical Trials Committee of the Medical Research Council.