The onset of the disease was found generally to occur in the age group 20-45 years, and when disseminated sclerosis is suspected in patients outside these limits other aetiological factors should be carefully considered.

For the development of disseminated sclerosis personal susceptibility is of importance, and in our experience the incidence of the disease is greatest in those with a sympatheticotonic diathesis.

Reference is made to those clinical signs which are of importance in distinguishing disseminated sclerosis from the manifestations of hysteria. In this connexion significance should be attached to the association of overactive knee-jerks and sluggish or readily exhausted abdominal skin reflexes. Other signs of diagnostic value are the mental state of the patient, the presence of neurovascular phenomena, and the triad of ocular imbalance, mydriasis, and hippus.

Stress is laid on the need for recognition of the disease before permanent damage has been inflicted on the central nervous system. Whatever advances in treatment the future may hold, the degree of recovery will depend on the stage at which a diagnosis is first established.

REFERENCES

- Adams, D. K. (1921). *Lancet*, **1**, 420.
 — (1923). *Glasg. med. J.*, **100**, 290.
 — (1927). *British Medical Journal*, **2**, 13.
 — Blacklock, J. W. S., Dunlop, E. M., and Scott, W. H. (1924). *Quart. J. Med.*, **17**, 129.
 — and Sutherland, J. M. (1948). *Surgo (Glasg. Univ. med. J.)*, **14**, 111.
 Adie, W. J. (1929). *Proc. R. Soc. Med.*, **22**, 1257.
 — (1932). *British Medical Journal*, **2**, 997.
 Bailey, G. H., and Gardner, R. E. (1940). *J. exp. Med.*, **72**, 499.
 Böhmig, W. (1922). *Dtsch. Z. Nervenheilk.*, **75**, 24.
 Brain, W. R. (1930). *Quart. J. Med.*, **23**, 343.
 — (1947). *Diseases of the Nervous System*, 3rd ed., p. 500. Oxford Univ. Press, London.
 Bramwell, B. (1917). *Edinb. med. J.*, **18**, 96.
 Browning, C. H., and Mackenzie, I. (1924). *Recent Methods in the Diagnosis and Treatment of Syphilis*, 2nd ed. Constable, London.
 Buzzard, T. (1897). *Lancet*, **1**, 1.
 Campbell, A. M. G. (1947). *Quart. J. Med.*, **16**, 312.
 — Daniel, P., Porter, R. J., Russell, W. R., Smith, H. V., and Innes, J. R. M. (1947). *Brain*, **70**, 50.
 Collier, J., and Adie, W. J. (1933). In *A Textbook of the Practice of Medicine* edited by F. W. Price, 4th ed., p. 1603. Oxford Med. Pub., London.
 Cottrell, S. S., and Wilson, S. A. K. (1926). *J. Neurol. Psychiat.*, **7**, 1.
 Curtius, F. (1933). *Multiple Sklerose und Erbanlage*. Thieme, Leipzig.
 Dreyfus, H. (1921). *Z. ges. Neurol. Psychiat.*, **73**, 479.
 Ferraro, A. (1944). *Arch. Neurol. Psychiat., Chicago*, **52**, 443.
 Gowers, W. R. (1893). *Manual of Diseases of the Nervous System*, 2nd ed., 2, 544. Churchill, London.
 Gunn, R. M. (1904). *Lancet*, **2**, 412.
 Head, H. (1920). *British Medical Journal*, **2**, 691.
 Klauder, J. V. (1947). *J. Amer. med. Ass.*, **134**, 245.
 Lagrange, H., and Marquiez, R. (1924). *Rev. Neurol.*, **1**, 712.
 Langworthy, O. R. (1948). *Arch. Neurol. Psychiat., Chicago*, **59**, 13.
 MacAlpine, D. (1946). *Brain*, **69**, 233.
 Marburg, O. (1920). *Z. Augenheilk.*, **44**, 126.
 Margulis, M. S., Soloviev, V. D., and Shubladze, A. K. (1946). *J. Neurol. Neurosurg. Psychiat.*, **9**, 63.
 Marie, P. (1895). *Lectures on Diseases of the Spinal Cord*. New Sydenham Soc., London.
 Mattauschek, E., and Pilcz, A. (1913). *Z. ges. Neurol. Psychiat.*, **15**, 608.
 Morawitz, P. (1904). *Dtsch. Arch. klin. Med.*, **82**, 151.
 National Multiple Sclerosis Society (1947). *Multiple Sclerosis, Diagnosis and Treatment*. New York.
 Purves-Stewart, Sir J. (1945). *The Diagnosis of Nervous Diseases*, 9th ed. Arnold, London.
 Russell, J. S. R. (1910). In Allbutt and Rolleston's *System of Medicine*, **7**, 809. Macmillan, London.
 Steiner, G. (1918). *Neurol. Zib. Referat.*, **37**, 535.
 — (1941). *Detroit med. News*, **32**, 7.
 von Hoesslin, R. (1934). *Über multiple Sklerose*. Lehmann, Munich.
 Walshe, F. M. R. (1947). *Diseases of the Nervous System*, 5th ed., p. 180. Livingstone, Edinburgh.
 Wartenberg, R. (1944). *Arch. Neurol. Psychiat., Chicago*, **52**, 341.
 Weill, G. (1923). *Ann. Oculist., Paris*, **160**, 793.
 Wilson, I. G. H. (1927). *British Medical Journal*, **2**, 1220.
 Wilson, S. A. K. (1940). *Neurology*, **1**, 148. Arnold, London.

AFTER-HISTORY OF SUCCESSFULLY TREATED CASES OF SUBACUTE BACTERIAL ENDOCARDITIS

BY

HENRY MATTHEW, M.B., Ch.B., F.R.C.P.Ed.

Senior Registrar, Royal Infirmary, Edinburgh

The outstanding achievement of penicillin in overcoming infection in subacute bacterial endocarditis and in procuring a 65% cure (Christie, 1948) has presented new problems in prognosis. The immediate prognosis has received much attention, and an evaluation of the facts on which it may be based has often been presented (Jones *et al.*, 1947; Christie, 1946, 1948, 1949). The long-term influence of the infective process on the heart has naturally not received equal attention owing to the relatively short time since successful treatment of the infection was initiated. As penicillin has now been available for the treatment of subacute bacterial endocarditis since 1944 in America and since 1945 in this country, sufficient cases have been followed over a period of years for a tentative expression of remote prognosis to be offered.

Prognosis in rheumatic heart disease itself is always difficult owing to the tendency to additional damage due to relapse or reinfection, *ouvert* or subclinical. When it is appreciated, as Matthew and Gilchrist (1949) and MacIlwaine (1947) have shown, that the bacterial infection is frequently engrafted on a heart the seat of concomitant active rheumatic carditis, the difficulties of the assessment of the prognosis in subacute bacterial endocarditis will be seen to be further increased. Therefore prognostication of the after-effects of a bacterial infection engrafted on such a background cannot be easy, nor is it likely to be accurate. Deterioration will be difficult to ascribe either to the natural trend of the original rheumatic infection (DeGraff and Lingg, 1935) or the occurrence of rheumatic relapse, or to the after-effects of successful penicillin therapy for the superimposed bacterial infection. Despite such difficulties, it is important to know the fate of survivors from a disease which up to six years ago carried a 96% mortality (Lichtman, 1943).

Of a total of over 40 patients treated for subacute bacterial endocarditis, 20 have been cured for 27 months or longer. These 20 unselected patients provide the information on which assessment of remote prognosis is based. These patients have been closely followed up for periods varying from 2½ to 5½ years. Since discharge from hospital no survivor has relapsed or become reinfected, although at least one has had a further attack of rheumatic fever. The absence of relapse or reinfection is noteworthy, as Christie (1949) has indicated that the incidence of delayed relapse is of the order of 2% per annum.

A severe infection of the endocardium and myocardium, often of six months' duration before being arrested, is likely to lead to further cardiac damage. Apportioning of the degree of damage by the bacterial infection, in a heart already the seat of rheumatic or congenital pathology, is difficult for the reasons already advanced. Deterioration, if noted, cannot invariably be ascribed to the healing of the bacterial infection, as the relentless progress of the underlying rheumatic lesion will, especially in the elderly patient, contribute to a greater or less extent.

The cardiac efficiency of successfully treated cases can best be assessed by a study of three factors: (1) a comparison of the patient's capacity for effort before infection and after recovery; (2) changes in the heart size as noted

radiologically; (3) the detection of physical signs indicative of new valvular lesions or an increase in the severity of established lesions.

Exercise Tolerance

Table I shows the comparison of cardiac grade (New York Heart Association, 1939) before and 2½ to 5½ years after treatment. The preponderance of cases with good

TABLE I.—The Cardiac Grade Before and After Treatment

Cardiac Grade	Before Infection	After Infection	Died
1	11*	11†	—
2a	7	7*	1
2b	2†	0	—
3	0	1	—

* One deterioration. † One improvement (congenital heart).

exercise tolerance before acquiring infection is in accordance with the findings of Libman and Friedberg (1948) that subacute bacterial endocarditis rarely occurs in the presence of advanced rheumatic heart disease. The information in Table I is revealing. Of 11 cases in Grade 1 (symptom-free before treatment) only one has shown deterioration in capacity for exertion, and that only to a slight extent. In seven patients classified as Grade 2a before the bacterial infection was acquired no deterioration was observed with the passage of the years.

One patient with congenital pulmonary stenosis was convinced that his exercise tolerance had improved considerably subsequent to the infection. In one patient, aged 34, classified as Grade 2b, further deterioration has become manifest by advancement into Grade 3 (congestive heart failure).

There has thus been a remarkable maintenance of capacity for effort in the survivors. All but two patients have resumed their former occupations or are now undertaking employment as strenuous as that prior to the onset of the infection. Of the two patients not working, one developed a right-sided hemiplegia during treatment and, although employable, has not yet been suitably placed; the other, in Grade 3, is naturally unfit for work. The occupations of the survivors range from busy housewife to general labourer, farm-worker, electrician and shop-assistant.

Four female patients have married since their illness, two have borne children without difficulty, and one is healthy at the sixth month of pregnancy at the time of review.

The degree to which subjective evidence of increased myocardial damage is absent is remarkable. Investigation of the second factor of assessment—x-ray examination for alteration in heart size—tends to substantiate the subjective evidence of lack of permanent damage from the infective process.

Heart Size

X-ray records were available of the size of the heart in two instances shortly before the onset of infection. In the remaining cases the heart size was assessed in the postero-anterior and both oblique views, if possible during the first week in hospital. This latter time of examination naturally varied with the duration of infection before admission. The variation, however, does not appear to confuse the deductions that can be made.

All patients had radiographs taken at the time of their follow-up, and the heart size was compared with that before or early in the infection (Table II).

The grades of heart size employed are: normal; slight enlargement, for a heart having one or more chambers

TABLE II.—Change in Size of Heart Found at Follow-up. In Only Four Instances has the Heart Enlarged

Heart Size	Before or Early in Infection	At Follow-up			
		Normal	Slight	+	++
Normal	5	5			
Slight enlargement ..	10		8	1	
+	5			3	2

just outside the limits of normal; one "plus" for a heart definitely enlarged in one or more chambers; and two "plusses" for a grossly enlarged heart. In only four instances did the heart enlarge during or after subsidence of the infection. No heart which was normal in size before treatment subsequently enlarged; increase, if it did occur, took place in a heart already enlarged to some degree.

Investigation of the four cases with evidence of change in heart size shows that the enlargement has not been slowly progressive over the years since cure. It occurred to its greatest extent during the weeks of penicillin treatment, and thereafter the size has remained almost stationary. It may be noted in passing that in no instances did the heart enlarge during treatment, only to regress during convalescence—a finding contrary to that of Jones *et al.* (1947), who report three cases showing enlargement which subsequently lessened, although only to a very slight extent. It is therefore assumed that, should the heart enlarge during treatment, it is likely to remain enlarged. That cardiac enlargement during the process of eradication of infection is the result of the bacterial infection cannot be certain. A concomitant rheumatic infection might as readily produce such a change in size.

Further investigation of the four cases with an increase in size shows no constant feature such as age, rhythm, valvular lesions, length of time prior to treatment, past or present rheumatic history, or electrocardiographic evidence of myocardial damage sustained during the bacterial infection, any of which might be responsible for the increase in size. Nor does the increase in heart size correlate with any deterioration in exercise tolerance except in one of the four cases—the patient formerly in Grade 2b and now in Grade 3 has considerable enlargement of the left ventricle and auricle. Jones *et al.* (1947) found that cardiac enlargement occurring at the time of, or subsequent to, treatment was usually associated with decreasing capacity for effort. There is therefore no doubt that heart size on termination of treatment is an important observation in assessment of remote prognosis. Although deterioration may not be evident, it is to be expected that the larger heart will fail earlier.

Fresh Valvular Damage

The finding of fresh evidence of valvular lesions in a heart the seat of rheumatic pathology cannot be ascribed solely to the bacterial infection and the healing process. However, Rosenblatt and Loewe (1945) and Bloomfield *et al.* (1945) have demonstrated cases in which the mutilation of the valves immediately after penicillin therapy has been such as to constitute a grave mechanical handicap and to contribute materially to heart failure. In a series of fatal cases (Matthew and Gilchrist, 1949) no such destruction was evident at necropsy in 13 instances, except that in one case an aortic cusp had perforated as the result of the infection.

A comparison of the auscultatory findings at the outset of treatment and at the time of follow-up is presented in

TABLE III.—*Valvular Lesion Before and After Treatment. The Term "Mitral" is Employed to Cover Mitral Stenosis and Mitral Damage*

Valvular Lesion	Before Treatment	After Treatment
Mitral	9	8
Aortic incompetence and mitral	7	8
Aortic stenosis	1	Died
Congenital	3	3

Table III. In only one instance is there now evidence of additional valvular damage, aortic incompetence having appeared where previously mitral stenosis was the sole lesion. In three other patients the heart murmurs have permanently altered in character. This finding suggests that in these four instances it is probable that the healing of the infective process had some effect on the structure of the valve cusps.

Arrhythmias

Once again it must be emphasized that an alteration in rhythm cannot be attributed with any degree of certainty to the effects of the bacterial infection. However, three patients now suffer from occasional attacks of palpitation which have been shown by electrocardiogram to be due to paroxysmal auricular fibrillation. One additional patient has developed frequent ventricular extrasystoles. These arrhythmias do not disturb the patients unduly, but in one instance the cardiac grade has deteriorated to Grade 2a.

One Death

One patient died 29 months after treatment. He was 49 years old and was the only case of pure aortic stenosis in the series. He gave no rheumatic history, nor was there an aortic diastolic murmur or evidence of rheumatic heart disease, thus suggesting that he comes into the category of pure aortic stenosis (Kiloh, 1950). Death occurred from congestive heart failure of rapid onset and increasing in severity over the course of two months. There was no suspicion of reinfection or relapse of the bacterial infection, nor was there subjective evidence of active rheumatism. Details of his last illness when correlated with a history of effort pain for two years prior to acquiring the bacterial infection indicated death from congestive heart failure subsequent to ischaemic myocardial damage. As death occurred in the patient's home in the north of Scotland a post-mortem examination was not made.

Discussion

The significant prognostic features affecting the immediate outcome of a case of subacute bacterial endocarditis have been clearly enunciated (Jones *et al.*, 1947; Christie, 1948; Bramwell, 1948; Matthew and Gilchrist, 1949). Sufficient time has now elapsed since the availability of penicillin to suggest features of significance pertinent to the remote prognosis of this disease. The period since treatment does not permit of dogmatic statement, but points are now evident which, observed at the time of cure of the initial infection and subsequently, allow a tentative opinion to be expressed.

This disease, carrying in 1943 a 96% mortality rate (Lichtman, 1943), has had the immediate mortality rate reduced to around 30% (Wagner, 1948). Penicillin can unostentatiously be credited with this remarkable improvement. As 25% of cases sustain myocardial damage of some magnitude during the infection (Seabury, 1947), it might be assumed that antibiotic therapy, in overcoming the infection, would leave the patient a cardiac cripple. That this does not pertain is amply demonstrated by a study of this series of 20 cases followed for 27 to 63 months. Apart from one death, deterioration in capacity for effort has

been restricted to two instances; ten patients remain in cardiac Grade 1 before and after infection and six maintain a grading of 2a despite the infection.

Other investigators have had less encouraging results in the subsequent progress of their cases and have endeavoured to explain on various grounds the deterioration and, on occasion, death of their patients long after eradication of the infection. Kaplan *et al.* (1949), in a study of 18 cases followed up from 25 to 61 months after the infection was cured, found that in 12 instances the infection had behaved as an intercurrent process, the healing in no way aggravating the pre-existing heart disease. Of the remaining six cases, three had a greatly reduced exercise tolerance and three died of congestive heart failure. The severity of the underlying rheumatic infection might have brought about such developments; however, no information is given regarding the age, cardiac grade, or rhythm which might assist in determining this point. One factor common to these six cases and not noted in any survivor is that all had a high pulse pressure on termination of treatment or shortly afterwards. In five this was due to a severe degree of aortic incompetence and in the sixth to a patent ductus arteriosus.

Kaplan postulates that the healing of the infected valve under the influence of penicillin may induce a severer degree of aortic incompetence than would occur in the natural process of the rheumatic endocarditis. Rosenblatt and Loewe (1945) were early aware of this possibility. Kaplan emphasizes that the degree of aortic leak must be sufficient to cause a "dynamic" burden on the heart mechanism. In the series under consideration the one patient who died suffered from aortic stenosis and had no aortic regurgitation. He did, however, suffer from myocardial ischaemia.

Seven cases in this series had aortic regurgitation (Table III). In three the diastolic pressure shortly after the completion of penicillin therapy was below 60 mm. Hg and has since remained below that level. This could be considered as imposing a "dynamic" burden on the myocardium. None of these seven cases shows any deterioration in exercise tolerance. One patient, who had only an organic mitral systolic murmur at the end of treatment, now also has aortic regurgitation with a blood pressure of 134/60. Her cardiac grade has deteriorated from Grade 1 to Grade 2a. She also suffers from paroxysmal auricular fibrillation. Rapoport and Ellis (1948) report 11 cases studied two years after successful penicillin treatment. No deterioration occurred in the four cases of their series showing aortic valve involvement. The degree of valvular damage is not, however, stated. In fact, all 11 of their cases were "at least as well and as active, and some could do more," as compared with their condition before the bacterial infection.

Bramwell (1948) is inclined to vest the healing process under the influence of penicillin, at least in the instance of the mitral valve, with diagrammatically opposite powers to those of erosion and destruction. On the evidence of one patient who originally had aortic incompetence at the time of infection and who two years later developed mitral stenosis, and on the fact that in two other patients the electrocardiograph showed an increased tendency to right axis deviation, it was postulated that, in the process of healing, a stenotic lesion may develop or increase in severity at the mitral valve. The three cases mentioned were the only instances in which deterioration in exercise tolerance occurred in the 18 patients reported on two years after eradication of infection.

In my series no patient developed a stenotic valvular lesion, nor did stenosis once present tend to increase,

although the patient dying of aortic stenosis may have been in this category. The alteration in the pitch of the mitral systolic murmur observed in three patients is not necessarily indicative of incipient stenosis. The development of right axis deviation can be attributed to the natural progress of rheumatic heart disease without evoking the healing process of the bacterial infection even as a contributory factor. Based on the slender evidence of one case in this series and another in which the infection has not been eradicated long enough, the effect of the healing process, at least on the pulmonary valve, seems to be the opposite of a stenosing tendency.

A boy aged 14 suffering from congenital pulmonary stenosis with superimposed subacute bacterial endocarditis was unable to trot the length of a football pitch before infection. Now, four years after infection, he can partake energetically in a game of football without undue breathlessness. This improvement cannot be the result of the natural evolution of pure pulmonary stenosis, the tendency in such cases being relentless deterioration (Allanby and Campbell, 1949). It would appear that the infection had freed the stenosis after the nature of a Brock (1948) operation. The other patient with infection eradicated from a stenosed pulmonary valve or infundibulum possesses a capacity for effort greater than she has enjoyed for years. Neither patient has a pulmonary diastolic murmur, only a systolic thrill and murmur at the base of the heart with conclusive x-ray and electrocardiographic evidence of the diagnosis. This evidence that healing of the infected process possesses destructive rather than fibrotic powers in the instance of the pulmonary valve has been noted by Pollack *et al.* (1948). As has already been shown, at least in this series, a similar development has fortunately not occurred in healing aortic valves.

Conclusions

In the vast majority of cases of subacute bacterial endocarditis it would appear that, provided the infection is overcome and congestive heart failure avoided at the time of treatment and shortly afterwards, the disease can be regarded as an incident which leaves little or no evidence of additional permanent damage in a heart the seat of congenital or rheumatic disease. In the few cases in which deterioration occurs, it may be anticipated if attention be paid to the following facts: the cardiac grade of the patient when the infection is acquired; the detection of a severe degree of aortic regurgitation after elimination of infection; and the heart size, radiologically, during and immediately after eradication of infection.

Summary

Twenty cases of healed subacute bacterial endocarditis have been followed for periods of between 27 and 63 months. No case relapsed or became reinfected after successful treatment of the bacterial infection. One death occurred.

The effect of the bacterial infection in two cases of congenital pulmonary stenosis is described.

The exercise tolerance, radiological heart-size, and auscultatory features at the time of treatment and after the lapse of years are compared and the significant points identified.

The patients providing the information for this paper were all under the care of Dr. A. Rae Gilchrist, to whom I am grateful for help and criticism.

REFERENCES

- Allanby, K. D., and Campbell, M. (1949). *Guy's Hosp. Rep.*, **98**, 18.
Bloomfield, A. L., Armstrong, C. D., and Kirby, W. M. M. (1945). *J. clin. Invest.*, **24**, 251.
Bramwell, C. (1948). *Lancet*, **2**, 481.
Brock, R. C. (1948). *British Medical Journal*, **1**, 1121.

- Christie, R. V. (1946). *British Medical Journal*, **1**, 381.
— (1948). *Ibid.*, **1**, 1.
— (1949). *Ibid.*, **2**, 950.
DeGraff, A. C., and Lingg, C. (1935). *Amer. Heart J.*, **10**, 459.
Jones, A. M., Herring, R., Langley, F. A., and Oleesky, S. (1947). *Brit. Heart J.*, **9**, 38.
Kaplan, S. R., Rosenman, R. H., Katz, L. N., and Brams, W. A. (1949). *J. Amer. med. Ass.*, **141**, 114.
Kiloh, G. A. (1950). *Brit. Heart J.*, **12**, 33.
Libman, E., and Friedberg, C. K. (1948). *Subacute Bacterial Endocarditis*, 2nd ed. Oxford Univ. Press.
Lichtman, S. S. (1943). *Ann. intern. Med.*, **19**, 787.
MacIlwaine, Y. (1947). *J. Path. Bact.*, **59**, 557.
Matthew, H., and Gilchrist, A. R. (1949). *Edinb. med. J.*, **56**, Trans. Med.-Chir. Soc., **128**, 25.
New York Heart Association (1939). *Nomenclature and Criteria for Diagnosis of Diseases of the Heart*, 4th ed. N.Y. Tuberculosis and Health Association.
Pollack, A. A., Taylor, B. E., Odel, H. M., and Burchell, H. B. (1948). *Proc. Mayo Clin.*, **23**, 516.
Rapoport, B., and Ellis, L. B. (1948). *New Engl. J. Med.*, **239**, 951.
Rosenblatt, P., and Loewe, L. (1945). *Arch. intern. Med.*, **76**, 1.
Seabury, J. H. (1947). *Ibid.*, **79**, 1.
Wagner, B. M. (1948). *Amer. J. med. Sci.*, **215**, 84.

CONGENITAL MALARIA

REPORT OF THREE CASES

BY

B. S. JONES, M.D., F.R.C.S.Ed.

Surgical Specialist, Colonial Medical Service

A considerable divergence of opinion persists regarding the frequency with which congenital malaria may occur. That it is occasionally encountered there can remain no doubt. Jones and Brown (1924) reported two authentic cases in London, while cases have also been recorded by Beekel (1910), who describes 12 instances of undoubted placental transmission, by Tanner and Hewlett (1935), and by Van Nitsen (1932), who demonstrated malarial schizonts in the spleens of six infants, either born dead or dying soon after birth. Chen *et al.* (1944) have summarized 39 cases of congenital malaria from the world literature, while Wickramasuriya (1935) believes that transplacental foetal infection occurs more often than is normally conceded and records six proved cases of *Plasmodium falciparum* infection.

Most British and American authors, however, while recognizing the possibility of congenitally transmitted malaria, regard its occurrence as a rarity. Thus English (1946), who was in charge of a large maternity hospital in Singapore for 20 years, encountered only one case of congenital malaria amongst the 5,551 deliveries during 1938—a European infant aged 6 days.

During and since the recent war many European women have elected to produce their babies in Nigerian hospitals, and the possibility of congenital malaria has become a significant consideration. Since congenital transmission is still generally regarded as a remote contingency, a pyrexial illness in the first few days of life is more likely to raise suspicions of neonatal infection, while a convulsive seizure will more probably suggest an intracranial birth injury. Even less likely is the possibility of congenital malaria to be entertained in the cases of such women who return to the United Kingdom to have their babies. Therefore three cases personally encountered are recorded.

Most non-African labours in Lagos are conducted at the Creek Hospital, the patients being mainly Europeans, Syrians, or Lebanese of varying duration of residence in the country, though almost all may be assumed to have been subject to subtertian malaria at one time or another.