the parts of the colon developed from the midgut and hindgut loops—that is, about the distal fourth of the transverse colon.

The most cogent objection to the view that these two cases represent parasympathetic overaction due to sympathetic deprivation is that in each case the contractions were painful. Pain fibres from the small and large intestines reach the cord, so far as we know, by way of the splanchnic nerves. Bentley and Smithwick (1940) showed that the appreciation of pain in the small intestine is abolished after bilateral splanchnicectomy, and Ray and Neill (1947) showed the same in the large intestine. The operation of splanchnicectomy has not, so far as I know, ever been followed by muscular overaction in the bowel.

We thus have two objections to the explanation of sympathetic deprivation put forward to account for the clinical syndrome here discussed—that surgical interruption of the sympathetic nerves at the same site does not produce this effect, and that it does abolish pain, which in these cases was not abolished.

Three suggestions may be made to account for this discrepancy. First, that the interruption of nerve fibres by malignant infiltration may be selective, picking out the motor fibres before the sensory ones. We know that in pressure lesions (tourniquet paralysis and obstetric paralysis) and traction lesions (brachial plexus injuries) involving nerve trunks motor power may be lost with little or no sensory disturbance. In this particular instance the motor fibres (at any rate, the post-ganglionic ones in the plexus) are non-medullated, while the sensory ones are medullated; and this chemical and histological difference may be accompanied by a differential resistance to infiltration.

Secondly, that the sensory and motor fibres may not run together in all individuals as they do in the majority. One fact that is appearing as the result of the great increase in operations on the sympathetic system is the astonishing and unpredictable course of the sympathetic pathways, particularly at the thoraco-abdominal junction. Boyd (1948) has shown that it is impossible, by the most complete ablation of all known sympathetic nerves and ganglia, to produce functional denervation of the sweat glands in the D/2 and L1 areas.

Thirdly, that painful sensations, blocked in their normal pathways, may find others. The motor cortex orders movements, and when the muscles or nerves that normally subserve those movements are destroyed will often accomplish the same movements by some other mechanism. The sensory cortex awaits messages, and if the orthodox routes are out of action will often receive them by underground channels. The pain of angina pectoris ceases after bilateral resection of the first four thoracic ganglia, but it comes back eventually. The pain of hip disease is cured for a time by antero-lateral cord section, but the relief is only temporary.

I should like to express my gratitude to Professor F. H. Bentley, Professor Michael Boyd, Sir Stanford Cade, Dr. Geoffrey Evans, Professor F. A. Knott, Professor G. A. G. Mitchell, Professor A. H. T. Robb-Smith, Sir Charles Symonds, and Mr. A. G. M. Weddell (the order is alphabetical), with whom I have discussed these cases.

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SOME CAUSES OF SLIGHT CARDIAC ENLARGEMENT

BY

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In routine radiological examination cardiac enlargement without physical signs of valvular disease or hypertension is sometimes seen. Among 2,500 recruits examined this problem arose in 25 men, and an attempt has been made to find the cause of the enlargement in 16 of these who were followed up and re-examined three to six years later. Two further cases seen after the original series (Parkinson and Hartley, 1946) are added.

Method

Opinions differ as to the best method of determining whether true enlargement is present. Hodges and Eyster (1926), Ungerleider and Gubner (1942), and Sherman and Ducey (1944) prefer exact cardiac mensuration. On the other hand, Bedford and Treadgold (1931) say that screen examination is a more reliable means of recognizing heart disease than is actual measurement by orthodiagram. Comeau and White (1942) find prediction tables the best approach to the problem, but both agree that the important factor in estimating heart size is the impression gained by a reliable and experienced radiologist. Parkinson (1936) thought that cardiometry was too complicated for routine clinical use, and that as sound an opinion could be given from screen examination without measurements.

In the type of case discussed measurements are of little use, as they usually fall between the minimum and maximum limits of normal. Careful radioscopy in the three routine positions by more than one experienced observer is more accurate in arriving at a conclusion, and this was the method here adopted. In every case the second examination was made without reference to the previous report. Table I shows the possible causes of radiological enlargement without physical signs of valvular disease or hypertension.

TABLE I.—*Showing Possible Causes of Radiological Cardiac Enlargement in the Absence of Physical Signs of Valvular Disease or Hypertension*

1. Physiological.—Childhood, athleticism, bradycardia, and pregnancy.
2. Apparent.—Scoliosis, depressed sternum, and raised diaphragm.
3. Familial cardiomegaly and congenital idiopathic hypertrophy. Friedreich's ataxia.
4. Organic heart disease.—V.D.H. before the development of murmurs, congenital heart disease, repeated coronary thrombosis, cardiac aneurysm, and antecedent hypertension.
5. Abnormal rhythm.—Complete heart-block, paroxysmal tachycardia with failure, auricular fibrillation, and bundle-branch block.
6. Arteriovenous aneurysms and Paget's disease of bone.
7. Metabolic, endocrine, and blood disease.—Anaemia, thyrotoxicosis, myxoedema, and beriberi.

Other rarer causes are anomalous coronary arteries, Fiedler's myocarditis, amyloid disease, Gaucher's disease, Von Gierke's disease, and acromegaly.

Among the 18 cases were examples falling into the first four groups only. Table II shows the final conclusion reached.

The "Directory of Psychiatric Out-patient Facilities for Adults and Children in England and Wales" may be obtained for 1s. (11s. a dozen) from the National Association for Mental Health, Maurice Craig House, 39, Queen Anne Street, London, W.1.

TABLE II.—*Showing the Final Diagnosis made in 18 Cases of Cardiac Enlargement in the Absence of Physical Signs of Valvular Disease or Hypertension*

Case No.	Final Diagnosis
1	Bradycardia and depressed sternum
2	" and scoliosis
3	" and depressed sternum
4	" and scoliosis
5	Scoliosis
6	
7	Full-sized heart. Aorta more prominent than normal
8	" " Right-axis deviation "
9	" " " "
10	" " " "
11	Familial cardiomegaly " "
12	Probable early mitral stenosis
13	Aortic incompetence (rheumatic)
14	" " { " }
15	" " { " }
16	" " { " }
17	" " { " }
18	Probable aortic incompetence (rheumatic)

Apparent Cardiac Enlargement

It is well known that scoliosis of minor degree, depressed sternum, or a raised diaphragm may cause apparent radiological enlargement. Although scoliosis or depressed sternum was recognized as a contributory cause in four men at the first examination, the heart in all was still considered to be larger than normal. Reconsideration at the second examination showed that all four had, in addition, a bradycardia.

Case 1.—Aged 40. *First examination*, October, 1941:—B.P. 130/85. The mitral first sound was loud and there was a moderate apical systolic murmur. Radiologically the left ventricle was suspected of being too large. *Second examination*, May, 1947:—Pulse rate 60. There was a minor degree of depressed sternum. The apical first sound was loud and a moderate apical systolic murmur was present. *Diagnosis*: the heart was now considered to be within normal limits in view of the bradycardia and minor depression of the sternum.

Case 2.—Aged 39. *First examination*, July, 1941:—B.P. 150/85. Apical and pulmonary short but loud systolic murmurs. Electrocardiogram: R, small and slurred, T₂ diphasic. Full-sized heart on radiology. *Second examination*, May, 1947:—Slight depression of the sternum. Short soft apical and pulmonary systolic murmurs. Pulse 60. Electrocardiogram unchanged. Radiology: full-sized heart. Slight displacement by scoliosis. *Diagnosis*: bradycardia and scoliosis.

(Note.—A diphasic T₂ was found among healthy young aircrew in the 1939–45 war (Stewart and Manning, 1944; Wood *et al.*, 1941).)

Case 3.—Aged 18. *First examination*, February, 1943:—Positive rheumatic history. B.P. 120/80. The pulmonary second sound was loud. Radiology showed slight enlargement, and early aortic incompetence was suspected. *Second examination*, April, 1947:—Short soft apical systolic murmur. Clear third heart sound. Electrocardiogram normal. Depressed sternum, minor degree. Radiology: full-sized heart; normal size in both oblique positions. *Diagnosis*: bradycardia and depressed sternum cause the appearance of full size.

Case 4.—Aged 20. *First examination*, May, 1943.—B.P. 140/70. Apex beat external to the nipple line and too forcible. Radiology: probably slight enlargement, but heart lies to the left. Possibly early aortic incompetence. *Second examination*, March, 1947:—Apex beat beyond the mid-clavicular line. Short soft apical systolic murmur. Pulse rate 60. Radiology: full-sized heart. Deep breathing shows it to be of normal size. *Diagnosis*: bradycardia and scoliosis cause the appearance of enlargement.

In Cases 5 and 6 a second examination showed scoliosis alone as the cause of the apparent enlargement. These show that a second examination will sometimes correct the previous view that more enlargement is present than can be accounted for by displacement.

Case 5.—Aged 17. *First examination*, February, 1944:—B.P. 130/75. Apex beat forcible. Short minimal systolic murmur at apex and on left of sternum. Electrocardiogram

normal. Radiology: probably slight enlargement. *Second examination*, April, 1947:—Short soft apical systolic murmur. Pulmonary second sound duplicated. Electrocardiogram normal. Full-sized heart, but right border is made up of spine. When the scoliosis is corrected by a 5-degree turn to the right the heart is normal in shape and size. *Diagnosis*: scoliosis.

Case 6.—Aged 19. *First examination*, September, 1941:—Positive history of rheumatic fever. B.P. 130/80. Apex beat in nipple line. Pulmonary second sound loud. Electrocardiogram normal. Radiology showed probable slight enlargement of the left ventricle, in addition to displacement by scoliosis. Possibly early aortic incompetence. *Second examination*, May, 1947:—Apex beat in mid-clavicular line, not forcible. Pulmonary second sound loud. Electrocardiogram normal. *Diagnosis*: radiology—scoliosis causes the appearance of enlargement.

Prominent Aorta

The interest in Cases 7 and 8 lies in the fact that even at the second examination cardiac enlargement was thought to be present. The radioscopic reports in each case are almost identical at both examinations. Yet if heart disease were present some progress over five years might have been expected. At the second examination, knowing the previous findings, it is justifiable to call both hearts normal, but if such a man is seen for the first time serious doubt would still arise. In both cases the ascending aorta was considered at each examination to be too prominent for the age of the patient. It is possible that the vascular pedicle in youth may be wider on occasion than the generally accepted normal.

Case 7.—Aged 19. *First examination*, October, 1941:—B.P. 155/90. Apex beat forcible and displaced. Electrocardiogram normal. Radiology: the heart lies slightly to the left. Possibly left ventricular enlargement. *Second examination*, May, 1947:—B.P. 140/70. Apex beat beyond the mid-clavicular line and too forcible. Short pulmonary systolic murmur. Electrocardiogram normal. Radiology showed a full-sized heart, with a vascular pedicle which is wide for his age. Ascending aorta prominent. Left ventricle possibly slightly enlarged. Oblique positions within normal limits. *Diagnosis*: probably no heart disease present.

Case 8.—Aged 21. *First examination*, December, 1942:—B.P. 130/80. Apex beat slightly forcible. Long piping apical systolic murmur, heard also in the aortic area. Electrocardiogram: left axis deviation. Radiology: heart full-sized, possibly slight enlargement. Possibly early aortic stenosis. *Second examination*, May, 1947:—Apex beat forcible. Short but noticeable apical piping systolic murmur. Electrocardiogram: left axis deviation. Radiology: heart full-sized only. Aorta too prominent for his age. *Diagnosis*: this may be a normal heart, but it is not certain even now.

Right Axis Deviation

Cases 9 and 10 are in many ways similar to Cases 7 and 8, but in these there is right axis deviation in the electrocardiogram. That it is unusual to find this at the age of 20 is true, but among 299 normal students studied by Wood *et al.* (1941) it was present in four. Slight widening of the QRS complex often occurs in normal youths, as has been shown by Graybiel *et al.* (1944) and by Stewart and Manning (1944). Graybiel *et al.* found QRS complexes as long as 0.13 second in normal aviators. Stewart and Manning found prolongation only in leads II and III. Five years after the original examination there was no change in Cases 9 and 10, and the presumption is that they have normal hearts.

Case 9.—Aged 19. *First examination*, November, 1942:—B.P. 130/75. Apical and pulmonary soft systolic murmurs. Electrocardiogram: right axis deviation. Radiology: slight but definite enlargement of heart. Possibly congenital heart disease. *Second examination*, May, 1947:—Apex beat beyond the mid-clavicular line. Not forcible. Soft apical systolic

murmur. Electrocardiogram: right axis deviation. Radioscopy: full-sized heart only. *Diagnosis*: no heart disease present.

Case 10.—Aged 20. *First examination*, December, 1942:—B.P. 155/90. Apex beat forcible. Electrocardiogram: right axis deviation with slight widening of the QRS complex, T₁ large. Radioscopy showed slight enlargement of the heart. *Second examination*, April, 1947:—Apex beat beyond the mid-clavicular line and forcible. Electrocardiogram unchanged. Radioscopy: heart appears enlarged, but turning 5 degrees to the right partially corrects it, and a deep breath then makes the size almost normal. There is some scoliosis, but in spite of this there seems to be more enlargement than can be accounted for by this and the high diaphragm. The size of the left ventricle raises suspicion, but the electrocardiogram shows right axis deviation. *Diagnosis*: no heart disease present.

Familial Cardiomegaly

William Evans (1948) has recently described cardiomegaly occurring as a familial condition, and Case 11 in my series proved to be a member of the family described by him.

Case 11.—Aged 18. *First examination*, June, 1944:—B.P. 130/60. Frequent extrasystoles. Apex beat in the sixth space, displaced and too forcible. Radioscopy showed moderate to great enlargement, chiefly of the left ventricle. Unfortunately, an electrocardiogram was not taken, as at that date work in London was carried on under difficulties. *Second examination*, June, 1947:—Auricular fibrillation. Electrocardiogram showed a grossly abnormal QRST complex. Radioscopy revealed moderate to great enlargement. The patient's brother had recently died in the London Hospital from heart failure due to the same condition. *Diagnosis*: familial cardiomegaly.

Mitral Stenosis

The diagnosis of valvular heart disease is seldom made in the absence of the typical murmur. In mitral stenosis enlargement can appear before a presystolic murmur, and sometimes even before an apical systolic murmur is heard. The latter was seen in five men out of 264 with mitral valvular lesions (Parkinson and Hartley, 1946). Case 12 was thought to be an early example of mitral stenosis, though it is not proved.

Case 12.—Aged 19. *First examination*, November, 1942:—B.P. 155/80. Apex beat external to the nipple line. Short systolic murmur in all areas, loudest in the pulmonary area. Radioscopy showed slight general enlargement. Possibly congenital, possibly acquired heart disease. *Second examination*, May, 1947:—Apex beat forcible and in mid-clavicular line. Apical first sound loud and sudden. Pulmonary second sound duplicated. Soft systolic murmur in all areas. Electrocardiogram: sino-auricular block with ventricular escape. Radioscopy: very doubtful enlargement with convex left border, possibly due to a combination of scoliosis and low diaphragm. In the right oblique position, with barium in the oesophagus, the left auricle is probably enlarged and also the pulmonary artery. In the left oblique position the left auricular enlargement is not so convincing. *Diagnosis*: probably early mitral stenosis.

This case raises the question of the diagnosis of early mitral stenosis as discussed by Parkinson and Hartley (1946). If this is mitral stenosis it is of great interest that such little deterioration has occurred in a period of nearly five years. The loud first sound and the probable left auricular enlargement make the diagnosis most likely.

Aortic Incompetence (Rheumatic)

On the other hand, the suggestion is not usually made that the enlargement of aortic incompetence may appear before an aortic diastolic murmur is audible under normal conditions. In Cases 13, 14, 15, 16, and 17 of this series a tentative diagnosis of possible aortic incompetence (rheuma-

tic) was made at the first examination, based on the radioscopic findings only, although at that time examination included a special but futile search for an aortic diastolic murmur, in all positions and at different pulse rates. On second examination in each case a clear aortic diastolic murmur was heard.

Case 13.—Aged 41. *First examination*, November, 1942:—History of chorea. B.P. 130/80. Apical first sound split. Pulmonary second sound loud. Electrocardiogram normal. Radioscopy: slight to moderate enlargement of the left ventricle. Probably aortic incompetence. *Second examination*, May, 1947:—Pulse shows auricular fibrillation. An aortic diastolic murmur is heard. Radioscopy: general enlargement, with left ventricular enlargement and a prominent ascending aorta. Slight scoliosis with a low diaphragm tended to mask the radiological findings in this case. *Diagnosis*: aortic incompetence (rheumatic).

Case 14.—Aged 21. *First examination*, July, 1944:—No history of rheumatic fever or chorea. B.P. 140/85. Depressed sternum and variable apical systolic murmur. Radioscopy: enlargement as well as displacement by the depressed sternum. *Second examination*, May, 1947:—Cup-like depression of the sternum. Apex beat well beyond the nipple line. Aortic diastolic murmur on the left of the sternum. Electrocardiogram shows right axis deviation. Radioscopy: considerable general enlargement. In spite of the depressed sternum the heart was thought to be too large. The left ventricle was slightly enlarged and the pulmonary artery was also slightly large. In view of the right axis deviation the question was raised whether the murmur could indicate pulmonary incompetence: the pulmonary artery was not large enough for this to be so. *Diagnosis*: aortic incompetence (rheumatic).

Case 15.—Aged 18. *First examination*, May, 1943:—B.P. 140/80. Apex beat forcible, a short apical systolic murmur. Radioscopy: left ventricle slightly enlarged. *Second examination*, April, 1947:—Slight depression of the sternum. Apex beat forcible and beyond the nipple line. Aortic second sound loud and an aortic diastolic murmur is present. Electrocardiogram: left axis deviation. Radioscopy: moderate general enlargement, left ventricle enlarged, and ascending aorta prominent. *Diagnosis*: aortic incompetence (rheumatic). It is possible that the diastolic murmur was obscured by the loud aortic second sound at the original examination.

Case 16.—Aged 18. Seen December, 1942. No rheumatic history. B.P. 115/70. Short apical systolic murmur only. Radioscopy: probably slight general enlargement. Possibly aortic incompetence. Later, in 1943, a diastolic murmur was recorded. *Diagnosis*: aortic incompetence (rheumatic).

Case 17.—Aged 18. Seen February, 1943. No rheumatic history. B.P. 120/75. Apex beat in nipple line and too forcible. Radioscopy showed left ventricle enlarged. *Diagnosis*: Possibly aortic incompetence. A letter from Sir John Conybeare, dated July, 1943, when this patient was in the R.A.F., confirmed that there was no diastolic murmur present, although one was audible in November, 1943. *Diagnosis*: aortic incompetence (rheumatic).

Case 18.—Aged 19. *First examination*, January, 1943:—B.P. 140/75. Apical and basal short systolic murmur. Radioscopy: slight general enlargement, with undue pulsation of the aorta. *Second examination*, April, 1947:—Apex beat beyond the mid-clavicular line and too forcible. Third sound well heard. Short soft pulmonary systolic murmur. Electrocardiogram normal. Radioscopy: slight general enlargement, left ventricle large, and ascending aorta prominent. Although repeated auscultation failed to show an aortic diastolic murmur, it seems probable from the radiological examination that he has aortic incompetence.

Cases 13, 14, 15, 16, and 17 show that enlargement may precede a constant diastolic murmur in cases of aortic incompetence. It is probable that phonocardiography would have helped in earlier diagnosis. It is not suggested that aortic incompetence should ever be diagnosed in the absence of the typical murmur but rather that such appearances as have been described should lead to repeated careful

auscultation until the diagnosis is certain. These 18 men illustrate difficulties in the assessment and exact diagnosis of doubtful enlargement.

Discussion

Cardiac hypertrophy of unknown cause has been discussed by Levy and Von Glahn (1937), who give the credit for the first descriptions to Jossierand and Gallavardin in 1901. The former authors describe 10 cases in patients aged 29 to 66. Arrhythmias were common. All the electrocardiograms were abnormal. Norris and Pote (1946) discuss four men, aged 21 to 30, who died of unexplained hypertrophy and dilatation of the heart, and all had abnormal electrocardiograms. The clinical features of this condition appear to be rapid progress of symptoms to failure and death, often with embolic phenomena, in a patient with a grossly large heart and an abnormal electrocardiogram. It might be asked whether our first 10 cases should not be included in this idiopathic group, but their symptoms, signs, electrocardiograms, and course would seem to exclude this.

The part played by antecedent hypertension in cases of cardiac enlargement of unknown aetiology has been considered by Kaplan *et al.* (1938). Evidence based on microscopical examination is given that previous hypertension played a part in 18 cases out of 45 with such enlargement. They suggest that coronary sclerosis, auricular fibrillation, chronic anaemia, and hyperthyroidism may play a part. Willis (1939) says that it is undesirable to assume the pre-existence of hypertension in any enlarged heart in the absence of collateral physical signs of hypertension. Palmer (1937) showed that coronary sclerosis may cause slight enlargement of the heart. Smith and Bartels (1931) considered coronary thrombosis to be the probable cause of cardiac hypertrophy in two men, aged 35 and 36, seen at necropsy.

Summary

An account is given of the follow-up examination of 18 men three to six years after they had shown cardiac enlargement without signs of valvular disease or hypertension. The method used was physical examination and radioscopy in the three routine positions.

The causes of cardiac enlargement without signs of valvular disease or hypertension are listed, and the relative value of mensuration and experienced radiological examination in assessing cardiac enlargement is considered.

In 10 cases the enlargement was found to be apparent only. In four of these displacement with bradycardia was present: two showed only scoliosis; while in the remaining four the heart was radiologically full-sized—two had an aorta more prominent than is usually considered normal in youth, and two had right axis deviation.

In eight cases the enlargement was confirmed—one proved to be a case of familial cardiomegaly, which is discussed in detail; one was considered to be probable early mitral stenosis; five (over 25% of the total cases) had developed an aortic diastolic murmur and were confirmed as cases of aortic incompetence in which cardiac enlargement had preceded the diastolic murmur; and one case with similar radiological appearances was thought most probably to have aortic incompetence, although no diastolic murmur was heard.

Ultimate diagnosis in the majority of such cases depends on re-examination at a later date.

My thanks are due to Sir John Parkinson for his practical aid and advice in the execution of this follow-up.

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CONGENITAL HEART DISEASE SIMULATING FOETAL DISTRESS

A REPORT OF TWO CASES

BY

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Congenital heart disease is comparatively rare: according to Price (1937) it is found in about 2% of all patients suffering from organic morbus cordis. Still rarer is it for congenital heart disease to reveal itself *in utero*. The following two cases are of interest in that marked slowness of the foetal heart accompanying congenital heart disease closely simulated foetal distress.

Case 1

A primigravida at term, aged 21, was seen at a routine visit in the antenatal clinic on July 4, 1947. The fundus uteri was at term, the position right occipito-anterior, and the head engaged. The foetal heart could be heard beating quite strongly, but it was markedly slow, the rate varying from 72 to 108, and its rhythm was irregular; the patient did not appear to be in labour. A vaginal examination was made, and the cervix was found to be taken up, but there was no dilatation and no cord was palpable. A diagnosis was made of a probable cord complication—either a loop of cord round the foetal neck or body or a true knot in the cord.

The patient was admitted to hospital from the clinic, and artificial rupture of the membranes with a Drew-Smythe catheter was performed shortly after admission. A 15-minute chart of the foetal heart rate was kept. Labour began at 8 p.m. on July 4, the cervix was fully dilated at 2.30 a.m. on July 5, and a normal delivery of a live female infant, weighing 6 lb. 7 oz. (2.9 kg.), took place at 3.15 a.m. The infant cried well at birth and was a good colour. During labour the foetal heart rate ranged between 60 and 110, but it did not vary with the pains, although the rhythm throughout was irregular.

At birth the infant's heart rate was 120, but three hours later it had slowed to 80; on each occasion the rhythm was regular. No meconium was passed during labour, and the placenta looked healthy. Cord complications were absent. On examination of the infant shortly after birth there was no cyanosis; the heart rate was 86, and no murmurs were heard.

On July 10 a loud harsh systolic murmur could be heard all over the praecordium, its maximum intensity being beneath the sternum. An electrocardiogram was physiological for an infant of this age, and there was no heart block or arrhythmia.

The infant was seen by the paediatrician in the out-patient department one month later. The heart rate was 136 with a regular rhythm. The systolic murmur was still present; there was no change in its quality, intensity, or propagation, which are characteristic of uncomplicated interventricular septal defect (maladie de Roger). The child appeared healthy and was being breast-fed four-hourly; she had had no cyanotic attacks.

I saw the child on Sept. 2. She was then very well, and her weight was 8 lb. 5 oz. (3.8 kg.). She had had no cyanotic attacks.