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.

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CONGENITAL HEART DISEASE SIMULATING FOETAL DISTRESS

A REPORT OF TWO CASES

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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 (mal'adie 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.

A skiagram of the chest was taken, and the radiologist reported as follows: "The heart shadow is normal for a patient of this age. Lung fields clear."

The mother had not had any illnesses during the first three months of her pregnancy.

Case 2

A primigravida at term, aged 38, was seen at a routine visit in the antenatal clinic on Aug. 1, 1947. The fundus uteri was at term, the position left occipito-lateral, and the head engaged. The foetal heart could be heard beating strongly at a rate of 44, and it was regular. The maternal pulse rate was 96, and was also regular.

The patient was admitted to hospital, and artificial rupture of the membranes was performed with a Drew-Smythe catheter shortly after admission. Vaginal examination revealed that the cervix was not taken up; the external os was closed, but it was easily stretched to admit one finger; there was no cord palpable.

A 15-minute chart of the foetal heart rate was kept. Labour began at 7 p.m. on Aug. 1, and the cervix was fully dilated at 11.45 p.m. A normal delivery of a live female infant, weighing 8 lb. 5 oz. (3.8 kg.), took place at 1 a.m. on the 2nd. The infant was a good colour at birth, and cried lustily after the air passages had been cleared of mucus. The foetal heart rate varied between 44 and 52 during labour; the rhythm was regular throughout both the first and the second stages of labour, and it was unaffected either by the pains or the descent of the foetal head on to the pelvic floor.

At birth the infant's heart rate was 40, and the rhythm was regular; no murmurs were heard. No meconium was passed during labour and the placenta seemed healthy; there was no cord complication. The infant was breast-fed four-hourly, and appeared to be perfectly healthy. On Aug. 4 a loud harsh systolic murmur was heard all over the praecordium. The senior physician, Dr. Barnes, examined the child on Aug. 7, and reported as follows: "Healthy looking infant. No cyanosis. The heart appears enlarged to the left. Apex beat in the fourth left intercostal space in the anterior axillary line. Heart rate slow at 41, but regular: there is a harsh systolic murmur audible all over the praecordium, with its maximum intensity beneath the centre of the sternum."

An electrocardiogram showed complete heart-block; the ventricular rate was 41, and the auricular rate 130; both had regular rhythm. A skiagram of the chest was taken on Aug. 13, and the radiologist reported as follows: "Heart contours normal in this projection. Lung fields clear."

A diagnosis of interventricular septal defect with congenital complete heart-block was made. The infant was seen by the paediatrician in the out-patient department one month later. The heart rate was 42 with regular rhythm; the systolic murmur was still present; there was no change in its quality, intensity, or propagation. The child appeared healthy and was being breast-fed four-hourly; she had had no cyanotic attacks.

I saw the child on Sept. 16. She was being breast-fed three-hourly, each feed being complemented with "ostermilk." She had had no cyanotic attacks and her weight was 9 lb. 12 oz. (4.4 kg.). The heart rate was 40 with regular rhythm. A second skiagram of the chest was taken, and the radiologist reported as follows: "Cardiac outline within normal limits for the age of the child."

The mother's electrocardiogram revealed no abnormality. She had had no illnesses during the first three months of her pregnancy.

Commentary

In Case 1 the irregularity of the foetal heart, together with a rate below 100, strongly suggested foetal distress, and a cord complication was considered the most likely cause. A caesarean section was seriously contemplated as the mode of treatment, but, in view of the patient's age and the fact that the duration of the foetal distress was unknown, it was decided that an immediate surgical induction should be done, and a 15-minute chart of the foetal heart rate was kept. I am quite unable to account for the behaviour of this foetal heart before delivery, for

the presence of an interventricular septal defect without conduction disturbance should not lead to cardiac arrhythmia.

Case 2 is even more instructive, because congenital heart disease was suspected when the foetal bradycardia was discovered in the antenatal clinic (Case 1 having been seen exactly one month previously). In this case the foetal heart was regular and easily audible—quite unlike foetal distress, where irregularity and muffled heart sounds are common characteristics.

Over 100 cases of congenital heart-block have been recorded. The diagnosis, however, is seldom made antenatally, and the case now reported brings the total number of cases diagnosed antenatally to 10. A summary of the main features of these cases, in all of which the diagnosis was confirmed by electrocardiograms, is given in the accompanying table.

Author	Sex	Slowest Foetal Heart Rate	Slowest Rate after Birth	Time of Antenatal Diagnosis	Duration of Life	Remarks
Yater, 1929 ..	M	47	47	2 weeks	18 days	Marked cyanosis. Necropsy revealed multiple congenital anomalies
Witt, 1934 ..	M	44	44	2 months	75 days	Infant died in a cyanotic attack. Necropsy revealed coarctation of aorta
Heubner, 1938	No record	80	60	Not given	Alive at 11 months and healthy	Block disappeared at this age
Ottow, 1939 ..	F	32	52	24 hours	Alive at 5 months and healthy	Block still present at 5 months
Geiger and Hines, 1940	F	58	60	17 "	Alive at 5 months and healthy	Block still present at 5 months
Thomson, 1943	M	60	52	7 "	1 year	Died of Stokes-Adams seizure. Necropsy revealed no interventricular defect
" "	F	45	68	4½ weeks	Alive at 4 years and healthy	Block still present at 4 years
Hammond, Stone, and Hyams, 1944	F	40	40	1 month	Alive at 7 months and healthy	Infant delivered by a lower segment caesarean section shortly after onset of labour because of slow foetal heart
Plant and Steven, 1945	M	50	50	5 weeks	6 days	Died in a cyanotic attack. Necropsy revealed no gross structural defect, but histological examination failed to show subendocardial fibres which could be identified as Purkinje fibres
Sankey, 1947	F	44	40	9 hours	Alive at 7 weeks and healthy	Block still present at 7 weeks

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Postscript.—Since writing this article it has come to my knowledge that the infant with congenital complete heart-block suddenly collapsed at home at the age of 4 months and died within 24 hours. The cause of death appeared to be bronchopneumonia, but a post-mortem examination was not allowed.