of this investigation are reported, with particular reference to influenza and adenoidal-pharyngeal-conjunctival virus infections.

Specimens of blood were taken from 1,001 volunteers in December, 1954, and again from 711 of these in March, 1955. Evidence of influenza B infection was found in 134 (19%) of those from whom two specimens were taken, the proportion in individual stations varying from 4 to 43%. A few cases of influenza A infection were detected, but only two volunteers showed evidence of A.P.C. virus infection.

Only 15 (11%) of the 134 volunteers whose serum showed a rising titre to influenza virus B were admitted to sick-quarters suffering from a respiratory illness. In two units those who had a rising titre were interviewed at the end of the investigation: approximately a third had had typical influenza during the period, a third had had minor respiratory symptoms, and the remaining third could remember no illness.

In volunteers from five stations where well-defined outbreaks of influenza B occurred, evidence of a significant association was found between pre-epidemic levels of antibody to soluble antigen and subsequent susceptibility to infection.

In March, 1955, cases of influenza-like illness associated with A.P.C. virus infection were detected in one station. The clinical and laboratory findings are described.

We thank the following medical officers of the Royal Air Force for their help in this investigation: Squadron Leaders W. L. Birrell, E. M. Coates, D. D. Forbes, D. A. Hanley, R. N. Ibbotson, J. G. Kingan, H. E. Lewis, A. L. Röss, and F. H. Walker; Flight Lieutenants D. Campbell, A. F. Champion, J. Cottam, J. D. Heaton, J. W. Myles, J. D. Ralph, and B. G. Spilsbury. We are also indebted to Mrs. R. Mackneson and Mr. G. Thompson for technical assistance. We wish to thank the Director-General of Medical Services, Royal Air Force, for permission to publish this report.

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To help physically handicapped people to join in social activities, the London County Council has bought a special coach, equipped with lifting apparatus, able to accommodate up to 12 disabled persons and two or three wheel-chairs. Full-depth double doors at the rear of the vehicle conceal a hydraulic "tail-lift" by means of which the wheel-chairs and their occupants can be elevated to floor-level.

IDIOPATHIC HYPERTROPHY OF THE HEART IN AFRICAN CHILDREN

A REPORT OF FOUR CASES

BY

H. ALTMAN, M.B., B.Sc., M.R.C.P.
M.R.C.P.Ed., D.C.H.

AND

H. STEIN, M.B., M.R.C.P.Ed., D.C.H.

From the Baragwanath Hospital and the University of the Witwatersrand

Idiopathic cardiac hypertrophy is characterized by congestive cardiac failure with general enlargement of the heart, without a distinct congenital anomaly or known cardiovascular disease as a cause. The condition is rare. Kugel and Stoloff (1933) described seven cases in children. On reviewing the literature, Kugel (1939) found 52 cases reported as idiopathic cardiac hypertrophy. However, after further analysis, only 17 of the 52 reported cases showed pure hypertrophy of the heart without myocardial degeneration or inflammatory exudate or fibrosis. In the established cases death occurred within the first six years of life. At necropsy the heart was dilated and the muscle hypertrophied. The valves were normal. Ante-mortem bland thrombi were occasionally found attached to the endocardium.

The object of this paper is to present four cases of idiopathic cardiac hypertrophy in African children, to discuss the differential diagnosis, and to assess the possible relationship of our cases to the obscure cases of heart disease in African adults described by Bedford and Konstam (1946), Davies (1948), Gillanders (1951), Ball *et al.* (1954), O'Brien (1954), and the *British Medical Journal* (1954).

Case 1

An African girl aged 6 was admitted to hospital on June 26, 1951, with a five-day history of cough. There had been no previous illnesses of note, and the history of gestation, birth, and development had been quite normal. Her dietary history was satisfactory, and her six siblings were all healthy.

On admission the child was in congestive cardiac failure. The heart was clinically enlarged, but there were no significant murmurs. The blood pressure was 90/65 mm. Hg. The patient's state of nutrition was good and she weighed 49 lb. (22.2 kg.). She was afebrile. The urine contained a moderate amount of albumin but no cells or casts. The electrocardiogram revealed no abnormality. X-ray examination of the chest showed marked generalized enlargement of the heart, which pulsated poorly; the lungs, apart from congestion, were clear. The Mantoux test (1 in 1,000) was positive. The haemoglobin was 16 g. per 100 ml.; leucocyte count, 4,200 per c.mm.; erythrocyte sedimentation rate, 1 mm. in the first hour. The streptococcal antihæmolytic titre was 100 units per ml. of serum. The blood urea was 29 mg. per 100 ml., and the total serum proteins 6.1 g. per 100 ml. (2.8 g. albumin, 3.3 g. globulin). The thymol turbidity, thymol flocculation, and Takata-Ara liver-function tests were normal.

Because of the gross enlargement and poor pulsation of the heart, pericardial effusion was considered as a possible diagnosis, and two attempts at paracentesis yielded negative results. Treatment consisted of sedation, digoxin, and mersalyl, but the child's condition steadily deteriorated, and she died 11 weeks after admission. A few days before death there was an episode of pain in the chest, hæmoptysis, and hæmaturia.

The necropsy was performed by Dr. J. Higginson. The pericardium was normal. Both ventricles were dilated, and the muscle was hypertrophied. The right auricle was dilated, and ante-mortem thrombus was found in the right auricular appendix. The left ventricle contained ante-mortem apical thrombus. The endocardial and coronary arteries were normal. There was an infarct in the apex of the middle lobe of the right lung. The hilar lymph nodes showed caseous tuberculosis. There was a small cortical infarct in the right kidney. The liver showed congestion with mild fatty change. Histological section of the myocardium showed some interstitial oedema and hypertrophy of the muscle fibres. Cellular infiltration was not present. The endocardium was slightly thickened in some areas and covered in parts by firmly attached thrombi. Neither the heart nor skeletal muscles had excess glycogen deposition.

Case 2

An albino African girl aged 2 was admitted to hospital on November 6, 1953, with a two-weeks history of cough, shortness of breath, and swelling of the face and legs. The patient's twin brother, also an albino, was quite well. Two older siblings had died of gastroenteritis. The mother and father were well. The history of pregnancy, birth, and development revealed nothing unusual. There had been no previous illnesses. The feeding history was good.

On admission the child was in severe congestive cardiac failure. The heart was enlarged but there were no cardiac murmurs. The blood pressure was 75/45 mm. Hg. She was afebrile. Her state of nutrition was moderately good, and she weighed 25 lb. (11.3 kg.). The urine contained a small quantity of albumin but no cells or casts. The electrocardiogram showed right bundle-branch block, with inversion and flattening of T waves in all precordial leads. X-ray examination of the heart and lungs revealed gross cardiomegaly and consolidation of the lower lobe of the right lung. On fluoroscopy the pulsation of the heart was poor and there was enlargement of both ventricles.

The Mantoux tests (1 in 1,000 and 1 in 100 dilutions) were negative. The haemoglobin was 15 g. per 100 ml.; leucocyte count, 6,600 per c.mm.; fasting blood sugar, 70 mg. per 100 ml.; blood urea, 33 mg. per 100 ml. The Takata-Ara liver-function test was negative, the thymol turbidity was 4.5 units, and the colloidal red test was one-plus positive.

Treatment consisted of digoxin and mersalyl for the cardiac failure, and penicillin for the lung lesion. The patient's condition improved at first but then retrogressed. She suddenly collapsed five weeks after admission with a severe haemoptysis and died.

Necropsy (Dr. J. Higginson) showed a grossly enlarged heart with dilatation and hypertrophy of all the chambers, particularly the ventricles. The valves were normal. There was slight endocardial thickening at the apex of the left ventricle, and ante-mortem thrombus was found in the right auricle. The coronary arteries and the pericardium were normal. There were recent infarcts in the upper lobes of both lungs, and old pneumonic consolidation of the right lower lobe. All the other organs were very congested.

Histological section of the myocardium showed hypertrophy of the muscle fibres, but no cellular infiltration. There was slight fibro-elastic thickening of the endocardium of the left ventricle. The liver sections showed congestion but not fibrotic change. There was no evidence of excess glycogen deposition in the skeletal or heart muscle.

Case 3

An African girl aged 15 months was admitted to hospital on May 28, 1953, with a two-months history of cough, followed four weeks later by swelling of the body. The pregnancy and delivery had been normal, and the feeding history throughout had been good. The family history was non-contributory.

On admission the child was in severe congestive cardiac failure. The heart was enlarged clinically but no cardiac murmurs were heard. The blood pressure was 85/65 mm. Hg. The urine contained a trace of albumin but no erythrocytes or casts. The child weighed 20 lb. (9 kg.) and there was no external evidence of malnutrition. X-ray examination of the chest showed gross cardiomegaly, the heart having a globular contour. The lung fields were clear. The electrocardiogram revealed a right bundle-branch block.

Treatment was started with sedation and mersalyl, and a low-salt diet was given. However, the child deteriorated rapidly. A triple rhythm became apparent, the systemic congestion became worse, and she died six days after admission.

At necropsy the pericardium contained 2 oz. (57 ml.) of straw-coloured fluid, but was otherwise healthy. The heart was grossly enlarged owing to dilatation of all the chambers, with particular dilatation and hypertrophy of the ventricles. The valves, coronary arteries, and endocardium appeared healthy. Ante-mortem thrombus was found at the base and apex of the left ventricle. The lungs were congested, with early bronchopneumonia. The liver was congested, with no obvious fatty infiltration. All the other organs appeared healthy. Histological sections of the heart showed hypertrophy of the muscle fibres of both ventricles with ante-mortem thrombus attached to the endocardium of the left ventricle. There was slight cellular infiltration of the myocardium with some fibroblastic repair. No excess glycogen was demonstrated.

Case 4

An African boy aged 4 was admitted to hospital on June 18, 1954, with a five-weeks history of cough, increasing breathlessness, and swelling of the face and legs. He had had no illnesses of note, and the feeding history appeared to be satisfactory. The family history revealed nothing significant.

Examination revealed a well-nourished child weighing 34 lb. (15.4 kg.) in severe congestive cardiac failure. The heart was enlarged and there was a diffuse apex beat. A soft systolic murmur and a triple rhythm were heard at the apex. The extremities were cold and the pulse volume was poor. The blood pressure was 70/40 mm. Hg. Mild albuminuria was present, but no cells or casts were found in the urine. X-ray examination revealed a marked cardiomegaly due mainly to the enlarged left ventricle, with a small effusion at the right lung base. On fluoroscopy poor pulsation of the heart was evident. Electrocardiography suggested left ventricular hypertrophy. The blood sedimentation rate was 1 mm. in the first hour; haemoglobin, 12.3 g. per 100 ml.; leucocytes, 3,800 per c.mm. The serum contained 50 units of streptococcal antihæmolytic O per ml.; total serum proteins, 6.2 g. per 100 ml. (3 g. albumin, 3.2 g. globulin). The fasting blood sugar was 62 mg. per 100 ml., and rose to 91 mg. after 5 min. (0.3 ml.) of adrenaline subcutaneously.

Treatment consisted of sedation, mersalyl, and digitalis. There was a gradual improvement during the following three weeks, but the child then relapsed with severe systemic congestion and pulmonary oedema, and died 26 days after admission.

At necropsy the pericardium contained a few millilitres of pale straw-coloured fluid. The heart showed hypertrophy of the myocardium with dilatation of all the chambers. The valves were healthy. There was minimal thickening of the endocardium of the left auricle and ventricle. The coronary arteries were healthy, and no intracardiac thrombi were present. The liver and lungs were congested. Microscopical examination of the heart showed hypertrophy of the ventricular muscle, particularly of the left ventricle, with minor endocardial thickening. The liver showed centrilobular congestion with slight fatty change but no periportal fibrosis. The other organs were congested. Pathologist's comment (Dr. J. Higginson): "This is a case of idiopathic cardiac hypertrophy, associated with a mild degree of fibroelastosis."

Discussion and Differential Diagnosis

Idiopathic cardiac hypertrophy presents as congestive heart failure in children, and can be diagnosed with certainty only at necropsy. There are certain well-defined pathological entities which should be considered in the differential diagnosis.

1. *Acute Isolated Myocarditis* (Fiedler's myocarditis; interstitial myocarditis).—This condition is very rare in childhood. Conlin and Mantz (1953), reviewing the literature, found only 22 reported cases in children under 2 years of age. The clinical picture of Fiedler's myocarditis (Scott and Saphir, 1929) is that of congestive heart failure with cardiomegaly, low blood pressure, and mild pyrexia, and with a sinus tachycardia out of proportion to the fever. Changes of rhythm such as paroxysmal tachycardia, auricular fibrillation, and heart block may occur. Systemic emboli are common (de la Chapelle and Graef, 1931). Electrocardiographic examination shows flattening of T waves in all leads. The P-R interval may be prolonged. The diagnosis can be made with certainty only at necropsy, which reveals varying degrees of cardiac hypertrophy and dilatation, with a pale, soft, and swollen myocardium. Microscopically, there is a diffuse infiltration of the myocardium with polymorphonuclear leucocytes, round cells, lymphocytes, plasma cells, and eosinophils. Intracardiac mural thrombi are often found.

2. *Anomalous Origin of Left Coronary Artery from Pulmonary Artery*.—Only 38 cases of this rare congenital anomaly of the arterial supply to the heart have been recorded (McKinley *et al.*, 1951; Johnson, 1954). The clinical diagnosis of the condition was first discussed by Bland *et al.* (1933). They reported a case of a 3-months-old infant who had recurrent attacks of discomfort precipitated by the exertion of feeding. During these attacks the child became pale and cold, and perspired freely. The electrocardiogram showed inversion of T waves in all leads. Cases reported more recently have also shown raised ST segments and deep Q waves similar to those seen in cases of coronary artery disease in adults (Kaunitz, 1947; Gasul and Loeffler, 1949). Fluoroscopy in these cases reveals gross cardiomegaly involving mainly the left ventricle. At necropsy the left ventricle is dilated and hypertrophied, and the myocardium is replaced to a large degree by fibrous tissue. The origin of the left coronary artery from the pulmonary artery is easily demonstrated.

3. *Glycogen Storage Disease of the Heart*.—This rare condition is due to an inborn error of metabolism and is transmitted as a Mendelian recessive gene. Di Sant' Agnese *et al.* (1950), who reviewed the literature, found only 14 authenticated cases. The clinical manifestations start in the first few months of life, with failure to thrive and attacks of dyspnoea and cyanosis. Examination reveals congestive heart failure and gross cardiomegaly with inconstant heart murmurs. The electrocardiogram shows inverted T waves and ST depression in all leads. A large globular heart is seen on x-ray examination. In all the described cases death occurred in the first year of life (Lambert *et al.*, 1953). At necropsy the heart is three to four times the normal size, with the left ventricle particularly grossly thickened. The myocardium is pale pink with the increased deposition of glycogen. The voluntary muscles are also infiltrated with glycogen—so much so that on occasion a macroglossia is present. Excess glycogen is also found in the liver. However, glycogen storage disease of the heart and that of the liver (von Gierke's disease) are separate entities. The low fasting blood sugar and poor adrenaline response classically seen in von Gierke's disease are not found in glycogen storage disease of the heart (Di Sant' Agnese *et al.*, 1950). The only certain way of making the diagnosis during life is by biopsy of skeletal muscle to demonstrate the increased glycogen deposition.

4. *Endocardial Fibroelastosis*.—In recent reviews of the literature (Hill and Reilly, 1951; Blumberg and Lyon, 1952; Gowing, 1953) 93 cases of endocardial fibroelastosis have

been reported. The onset of the illness is in the first few months of life. The infants have progressive heart failure with gross cardiac enlargement. Heart murmurs may be heard when the valves are involved. The electrocardiograph shows left ventricular strain. Systemic emboli from intracardiac mural thrombi may occur. Pathologically the condition is characterized by cardiac hypertrophy together with a non-inflammatory fibrosis affecting the mural and occasionally the valvular endocardium—the left ventricle and left auricle being most commonly affected. Associated anomalies of the heart are found in 20% of the cases (Gowing, 1953). These include bicuspid aortic valve, coarctation of the aorta, persistent ductus arteriosus, and patent foramen ovale. Minor degrees of endocardial fibroelastosis may occur in hypertrophied hearts due to isolated myocarditis, glycogen storage disease, and anomalous origin of the left coronary artery.

5. *Rhabdomyoma*.—Sixty-five cases of this condition have been reported in the literature (Johnson, 1954). In half the cases described, death occurred in the first year of life. The clinical picture is that of progressive heart failure with cardiomegaly. 50% were associated with tuberous sclerosis. The tumours seen at necropsy may be single or multiple.

6. *"Endocardial Fibrosis" and "Nutritional Heart Disease" in Africans*.—Davies (1948) reported 36 cases of congestive heart failure of unknown aetiology which at necropsy showed cardiac hypertrophy with endocardial fibrosis. He stated that the condition was the cause of nearly 10% of cases of heart failure occurring in Africans living in East Africa. The majority of cases occurred in early adult life, and he reported one case in a child under 10 years of age. The clinical picture was that of progressive heart failure with little response to treatment. At necropsy the heart was dilated and hypertrophied, with fibrosis of the endocardium affecting the apex of the left ventricle and extending to a variable degree along the ventricular wall and infiltrating into the subendocardium. Thrombi were often found overlying the fibrous plaques.

Bedford and Konstam (1946) described similar cases occurring in West African soldiers. Gillanders (1951), at this hospital, reported a series of 30 cases of congestive cardiac failure of unknown aetiology occurring in African adults. Fluoroscopy revealed generalized cardiac enlargement with poor pulsation of the heart. These cases did not respond to digitalis, but early in the illness some improvement was evident on a balanced ward diet. At necropsy all chambers of the heart were dilated and hypertrophied, and mural thrombi were found in the left ventricle in five cases. Microscopical examination showed hypertrophy of the myocardium and a fine interstitial fibrosis of the ventricular endocardium. Cellular infiltration was occasionally present. Biopsy and necropsy of the liver in these cases revealed a varying degree of periportal fibrosis and haemosiderosis. Gillanders thought these findings were due to malnutrition, and labelled the condition "nutritional heart disease." He stated that similar cases occurred in childhood, but he did not describe any such case.

A similar group of 40 cases was described by Becker *et al.* (1953). The clinical presentation of these cases was identical with that described by Gillanders. However, with specific histological staining at necropsy, they demonstrated degeneration of the collagen and its associated ground and cement substances. The term "cardiac collagenosis" was suggested for the condition.

Summary and Conclusions

Four cases have been described that presented with congestive heart failure, with cardiac hypertrophy and dilatation, for which no obvious cause could be found. Clinically there was no evidence of rheumatic fever, chronic pulmonary disease, nephritis, or anaemia, while post-mortem examination excluded congenital heart disease, myocarditis, glycogen storage disease, endocardial fibroelastosis, and rhabdomyoma.

Clinically, our cases closely parallel Gillanders's description of nutritional heart disease, with the hypokinetic circulation, the poor movement of the heart as seen fluoroscopically, and the poor response to treatment. However, there was no evidence that malnutrition was a causal factor. Malignant malnutrition or kwashiorkor is a common condition amongst the African children admitted to this hospital, but in our cases the state of nutrition was much above the average, with no evidence of dermatosis, severe liver disturbance, oedema, or other stigmata of malnutrition. Thus until the aetiology of these cases is determined the term idiopathic cardiac hypertrophy seems appropriate.

The literature is briefly reviewed, with particular reference to the rarer forms of heart disease occurring in infancy and childhood.

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MENTAL ABILITY AND SCHOOL ACHIEVEMENT OF PREMATURE CHILDREN AT 8 YEARS OF AGE*

BY

J. W. B. DOUGLAS, B.M.

Senior Lecturer in Public Health and Social Medicine,
University of Edinburgh

This is the fourth in a series of papers describing the growth and development of a national sample of premature children who have been followed through from birth and compared with a closely matched group of children born at term. Previous papers have described their health (Douglas and Mogford, 1953a), their growth (Douglas and Mogford, 1953b), and their age at walking (Douglas, 1956). The present paper describes the results of reading, vocabulary, and picture intelligence tests given when they were 8 years old.

*This survey is being made by a joint committee of the Institute of Child Health (University of London), the Society of Medical Officers of Health, and the Population Investigation Committee. The chairman of the committee is Professor James Young, the vice-chairman Professor A. A. Moncrieff, and the secretary Professor D. V. Glass. The Nuffield Foundation has financed this inquiry during the pre-school years, and grants for continuing it in the primary school period have been made by the board of governors of the Hospital for Sick Children, Great Ormond Street, through the Institute of Child Health, and by the Ford Foundation. Fourteen regional hospital boards have also made grants to the inquiry from their free moneys.

During the last thirty years many studies have been made of the mental development of premature children, and excellent critical reviews have been given by Benton (1940) and Alm (1953). Most workers agree that premature children are restless, irritable, and lacking in concentration, and report a relatively high incidence of severe mental defect among them. These severe defects are usually associated with a history of birth injury, which is relatively common among premature children, or with developmental abnormalities, such as mongolism, which are themselves associated with low birth weight. But the important question of whether premature children are retarded in development after these abnormal groups have been excluded has not yet been satisfactorily answered. Some workers—for example, Hess *et al.* (1934)—find no evidence of mental handicap except when there is a history of cerebral damage, whereas others—for example, Lofthus (1930), Brander (1936), and Blegen (1953)—report handicaps of varying severity.

This lack of consistency is not surprising, since a large proportion of the premature children enrolled in these studies were not traced and those that were tested were by no means a random selection of those sought. A further source of bias lies in the fact that premature children are more common in poorer families, among firstborn infants, and among females; and this selection must be taken into account when assessing their mental development. If, as in so many of these studies, their scores are compared with those of a randomly chosen group of children born at term, there will always remain the possibility that any handicaps found are the result of environmental differences rather than of prematurity itself. More detailed criticisms of previous studies are given by Benton (1940), Alm (1953), and Douglas and Mogford (1953a, 1953b).

Nature of Present Inquiry

A unique opportunity to make a controlled study of a representative group of premature children was provided by the Maternity Survey of 1946 (R.C.O.G. Survey, 1948). All children born during the first week of March of that year were enrolled in the inquiry, and among them were 707 legitimate single-born babies weighing 5½ lb. (2.5 kg.) or less at birth, and therefore, by international definition, "premature." Information was lacking on the home conditions of 31 of these premature children, but we have been able to match each of the remaining 676 with a child (the control) weighing more than 5½ lb. at birth who was selected from the remaining legitimate singletons born during the survey week. The selection was made on the basis of sex, ordinal position in the family, mother's age, social group, and degree of crowding in the home; and wherever possible the premature child was matched with a control living in the area of the same local authority.

The majority of premature children were well matched for all these characteristics, and there is little doubt that in this way we have removed the major sources of bias in assessing their development. For further details of matching see Douglas and Mogford (1953a). The resulting sample is too small to allow us to make precise statements about severe mental defects but is well suited to a comparison of the general distribution of mental ability in premature children and their controls.

Available Information

In March, 1954, when the survey children were 8 years old, they were tested for their ability to read and understand a list of words of graded difficulty and to appreciate relationships shown in a series of picture strips. Three scores were given—one for reading, one for word comprehension (vocabulary),