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The Outlook for Children with Congenital Aortic Stenosis.

I. B. BRAVERMAN and S. GIBSON. *Amer. Heart J.*, **53**, 487, 1957.

Since few detailed accounts have appeared of the course and prognosis of congenital aortic stenosis in children, the authors have reviewed 85 such cases in patients under 16 years (67 boys and 18 girls) seen during the past 8 years at the Children's Memorial Hospital, Chicago. None had a history of rheumatism, all had the classic physical signs of aortic stenosis, 2 had associated patent ductus arteriosus, 3 had aortic coarctation, and one had fibrocystic disease.

An examination has been made or a report obtained on 73 of these patients since January, 1956, their ages ranging from 3 months to 19 years. Of these, 41 have no symptoms, 26 have symptoms, and 6 (8%) are dead. Of the children who died, 4 did so very suddenly and 3 had at least one syncopal attack before the fatal one; in 4 the heart was enlarged and in 3 there were electrocardiographic signs of left heart strain.

Of the 67 survivors, 44 can compete with their contemporaries, 14 cannot do so fully, and 9 cannot compete at all. Of the 26 patients with symptoms, 9 are easily fatigued, 6 have headaches, 4 became dizzy, 4 are dyspnoeic on exertion, 4 have epistaxis, and 3 have leg pains; in 5 of these children the heart is enlarged and in 10 there are cardiographic indications of left heart strain. Many of the 41 symptomless survivors are very active and several are athletes; 11 have large hearts and 14 have evidence of left heart strain.

The authors conclude that congenital aortic stenosis is not an innocent disease, and suggest that patients who are severely handicapped or have cardiac enlargement or electrocardiographic changes should be operated upon as soon as the procedure is proved to be relatively safe.

D. Emslie-Smith

Percutaneous Left Ventricular Puncture in the Assessment of Aortic Stenosis. P. FLEMING and R. GIBSON. *Thorax*, **12**, 37, 1957.

In order to determine the value of percutaneous left ventricular puncture in the preoperative assessment of aortic stenosis, 28 pressure records, have been analysed. All the patients concerned were considered to have aortic stenosis, and in 17 there was also clinical evidence of aortic regurgitation. The left ventricular pressure pulse in aortic stenosis is described. An "a" wave due to left atrial systole is usually present in the end-diastolic portion. The systolic pressure is high (ranging from 140 to 325 mm. Hg in the present series) and the pulse wave is more sharply peaked than normal, both isometric contraction time and ventricular systolic upstroke time being abbreviated. Owing to lengthening of the phase of reduced ejection, however, the total ventricular ejection period is prolonged.

Aortic valvotomy was subsequently performed on 18 patients, and a surgical estimate of the degree of stenosis was available for 16 of these. Neither the peak systolic pressure gradient across the valve nor the prolongation of the ventricular ejection period (corrected for differences

in cycle length) proved to correlate closely with severity of stenosis, presumably because these parameters are appreciably influenced by variations in stroke volume and hence by the presence and degree of regurgitation. The calculated aortic valve area is not expected to agree precisely with the true area since in the presence of regurgitation measurement of output by the Fick principle underestimates the actual forward flow, while if stenosis is dominant the formula tends to overestimate valve size. Nevertheless, there was sufficient correlation to suggest that the calculated value may be of help in assessing doubtful cases. It is tentatively suggested that if it exceeds 1 sq. cm. and the degree of regurgitation is uncertain aortic stenosis is unlikely to be severe enough to warrant valvotomy.

Analysis of the brachial arterial pressure records confirmed the unreliability of the pulse pressure as an index of the degree of stenosis. The brachial systolic upstroke time, however, appears to be a more helpful measurement; this was invariably prolonged, and when related to the normal value for the pulse rate, correlated fairly well with the severity of the stenosis. *S. G. Owen*

Retrograde Perfusion of the Coronary Sinus for Direct Vision Aortic Surgery. V. L. GOTT, J. L. GONZALEZ, M. N. ZUHDI, R. L. VARCO, and C. W. LILLEHEI. *Surg. Gynec. Obstet.*, **104**, 319, 1957.

Retrograde perfusion, via the coronary sinus, of oxygenated blood, in combination with a pump-oxygenator, permits work on the open ascending aorta or coronary arteries for periods of up to 20 minutes in the dog. Flow studies of the coronary sinus venous drainage demonstrate in most instances a higher coronary flow in the bypassed heart even though the perfusion pressure in the aorta remains lower than for the non-by-passed heart. Twenty dogs were perfused seeking a survival experiment in each. Their hearts tolerated well 15 to 20 minutes of retrograde perfusion if the head of pressure was adjusted properly. The method of back perfusion via the coronary sinus has been tested in 7 clinical cases to date. In all instances the method appeared to protect the myocardium well against anoxia and coronary air embolism, permitting direct-vision reparative surgery for aortic stenosis and regurgitation, ruptured sinus of Valsalva, aortic-pulmonary septal defect, and complete transposition of the great vessels. The human heart responded well to this altered environment for periods up to 15 minutes.—[Authors' summary.]

Plasma Lipids and Proteins and Their Relationship to Coronary Disease among Navajo Indians. I. H. PAGE, L. A. LEWIS, and J. GILBERT. *Circulation (N. Y.)*, **13**, 675-679, 1956.

A review of the hospital records showed that myocardial infarction was diagnosed in only 5 full-blooded Navajo Indians among 10,276 admissions over 4 years to the Navajo Hospital, Arizona, and even in these 5 cases it was not confirmed by electrocardiography. Again, not one case of coronary arterial disease was observed among 60,405 out-patient attendances. In contrast, out of

20,289 white patients admitted to St. Joseph's Hospital, Albuquerque (about 150 miles distant), 146 had myocardial infarction; the age distribution was similar in the two groups. The Navajos are well nourished, and usually eat a typically American diet, often containing foods of high cholesterol and high fat content; they tend to consume less fruit and vegetables but more fried food.

The mean total plasma protein level among the Navajos was only slightly lower than that among a group of persons from Cleveland, Ohio; the albumin level was lower, while the globulin level tended to be slightly higher, especially the α - and γ -globulin fractions. The lipoprotein components were also very similar in the two populations. The mean total cholesterol value was about 50 mg. per 100 ml. lower in the Navajo group and did not alter with age. It is concluded that the lower serum cholesterol levels and the extremely low incidence of coronary arterial disease among the Navajo Indians cannot be attributed to a lower intake of fat, and that hereditary factors may be responsible.

Robert de Mowbray

Observations on the Morbid Anatomy of Coronary Artery Disease. A. W. BRANWOOD and G. L. MONTGOMERY. *Scot. med. J.*, 1, 367, 1956.

An investigation of the pathological changes in the heart in 144 cases of sudden death and of clinically diagnosed coronary thrombosis is reported from the University of Edinburgh.

In all of the 26 cases of sudden death outside hospital a previous history of angina was obtained, and in none was an extracardiac cause of death found. Only 8 of these cases, however, showed coronary occlusion by recent thrombus or atheromatous softening; in 6 there was a non-occlusive thrombus and the rest showed no occluding material. There were 6 cases of recent infarction, only 3 of which had thrombi. In all cases there was gross atheromatous stenosis of all the coronary arteries and also evidence of myocardial fibrosis. In all 13 cases of sudden death in hospital (mainly post-operative) there was severe coronary stenosis and myocardial fibrosis; in this group were 3 recent infarcts without thrombosis and 3 cases of recent thrombosis.

Of the largest group examined, namely, 101 cases of clinical coronary thrombosis confirmed by electrocardiography, a recent infarct was found at necropsy in only 61, myocardial fibrosis alone in 25, minor muscle changes of dubious significance in 10, and no evidence of infarct or fibrosis in 5. Of the 61 cases of infarction, coronary occlusion was found in 24; in the rest non-occlusive thrombus was present in 23, no recent thrombus was detected in 13, and coronary embolism in one case. In most cases severe stenosis affected all three coronary arteries. Further, since only about 40 per cent of cases of infarction had a coronary occlusion the authors conclude that infarction is related to over-all coronary insufficiency rather than to occlusion of a particular artery.

Assessment of the age of the thrombi and infarcts by histological criteria showed that while most infarcts were at least several days old, the associated coronary thrombi appeared to be only 24 to 36 hours old. Therefore, the authors conclude, unless the sequence of histo-

logical changes in thrombi is very much delayed in atheromatous coronary arteries, it would seem that coronary thrombosis is often not the cause, but the terminal event in the presence of an already established recent myocardial infarct.

M. C. Berenbaum

Clinical and Anatomic Features in Five Hundred Patients with Fatal Acute Myocardial Infarction. K. T. LEE, W. A. THOMAS, E. R. RABIN, and R. M. O'NEAL. *Circulation*, 15, 197, 1957.

The clinical and anatomical features of 500 cases of acute myocardial infarction coming to necropsy at the Washington University School of Medicine, St. Louis, Missouri, during the period 1910-54 are described. In 429 of these cases it was possible to date the time of infarction. There was a preceding history of angina pectoris in half this number, and in the other half fatal myocardial infarction occurred without previous warning. Almost half the patients had evidence of old myocardial infarction in addition to a recent infarct, although only 25 per cent gave a history of past infarction. Chest pain was present in 64 per cent of the 429 patients. In 19 per cent of the whole series with recent infarction a correct diagnosis was not made in life. *James W. Brown*

Rupture of Papillary Muscles: Occurrence of Rupture of the Posterior Muscle in Posterior Myocardial Infarction. R. J. SANDERS, K. T. NEUBUERGER, and A. RAVIN. *Dis. Chest*, 31, 316, 1957.

The authors present clinical and necropsy details of 5 cases in which death occurred, as the result of rupture of a papillary muscle complicating myocardial infarction.

Rupture of a papillary muscle usually occurs 3 to 17 days after the onset of symptoms of myocardial infarction shown electrocardiographically to be situated posteriorly. Clinically, there is a sudden onset of dyspnoea with signs of peripheral circulatory collapse and rapidly progressive pulmonary oedema. A harsh systolic apical murmur makes its appearance, or one previously present becomes accentuated, though sometimes it cannot be heard because of very loud respiratory noises. Death usually occurs from left ventricular failure with intractable pulmonary oedema. The prognosis is poor.

Only one case in the present series was diagnosed ante mortem, but the authors consider that it should be possible to differentiate rupture of a papillary muscle from acute cardiac dilatation and from rupture of the interventricular septum. In the latter event the systolic murmur is parasternal rather than apical, conduction defects are often present, and the electrocardiogram shows changes of anterior infarction. *Marcel Malden*

Limited Use of Anticoagulants in Acute Myocardial Infarction. Analysis of One Thousand "Good-risk" Cases. H. I. RUSSEK and B. L. ZOHMAN. *J. Amer. med. Ass.*, 163, 922, 1957.

The authors have studied the mortality and incidence of thrombo-embolic complications in 1000 "good-risk" cases of acute myocardial infarction, which were not treated with anticoagulants. The series consisted of two

groups: (1) 489 cases selected and studied in retrospect; and (2) a further 511 cases selected for study at the time of their first examination. All patients were treated conservatively with 3 to 4 weeks' bed rest and leg exercises from the onset.

In Group 1 the over-all mortality was 3.1 per cent, mortality after the first 48 hours 1.7 per cent; and incidence of thromboembolism 0.8 per cent. In Group 2 the figures were 3.5 per cent, 1.8 per cent, and 3.7 per cent respectively. Thrombo-embolic incidents in the latter group were all mild in degree, with no deaths, and no case of cerebral or peripheral arterial embolism occurred. In the combined series there were 33 fatal cases (3.3%), in only 9 of which anticoagulant treatment might have saved life (3 deaths from recurrent thrombosis, one from cerebral embolism, and 5 from unknown causes). Of the remainder, 16 deaths occurred unexpectedly during the first 48 hours, 4 were from non-cardiac causes, and 4 were from rupture of the left ventricle. Thus death from thrombo-embolic causes occurred in less than 1 per cent of good-risk cases.

On the basis of these findings, and in view of the risk of hemorrhagic complications and of the adverse effect of the disturbance to the patient inseparable from treatment with anticoagulants in hospital, the authors consider that the use of these drugs is contraindicated in good-risk cases of acute myocardial infarction, though this conclusion does not detract from their established value in properly selected cases.

V. Reade

Effect of Heparin on Lipemia-induced Angina Pectoris.

P. T. KUO and C. R. JOYNER. *J. Amer. med. Ass.*, 163, 727, 1957.

The effect of heparin on acute anginal pain induced by administration of a fatty meal in patients with coronary arterial disease and old myocardial infarction was studied at the Hospital of the University of Pennsylvania, Philadelphia. A total of 15 attacks of angina were induced in 7 patients after a latent period of 5 to 5½ hours. In 14 of the 15 attacks subjective relief was obtained 5 to 15 minutes after heparin administration, and this was confirmed objectively by improvement in the electrocardiogram, ballistocardiogram, and pneumogram. Similar but less permanent improvement was obtained with glyceryl trinitrate. Plasma turbidity and serum total fatty acid values rose sharply after the meal until heparin was given, and then fell suddenly. The serum lipoprotein pattern also showed considerable anodal migration after heparin. No such changes occurred when the pain was relieved by glyceryl trinitrate, and there were no changes in the serum cholesterol and phospholipid levels. No objective or subjective improvement followed intravenous injection of saline solution.

J. Warwick Buckler

Serum Transaminase Determinations as a Diagnostic Aid in Myocardial Infarction.

R. SHABETAL, A. IGLAUER, and D. M. ANDERSON. *Brit. med. J.*, 1, 555, 1957.

The level of activity of glutamic oxalacetic transaminase in the serum was estimated in 4 groups of patients at the

Jewish Hospital, Cincinnati. In 20 patients, none of whom was "suspected of a recent thrombo-embolic incident", the serum transaminase level was within the normal range of 10 to 40 units when estimated by Karmen's method.

Of 39 patients with suspected coronary arterial disease, 13 had clinical and electrocardiographic features typical of myocardial infarction. In 12 of these the serum transaminase level was raised. On the other hand 12 patients who on admission were suspected on clinical and electrocardiographic grounds of myocardial infarction, but in whom serial transaminase estimations gave normal results, were subsequently considered on other grounds not to have had infarctions. Raised serum transaminase levels were found in patients with congestive failure and a large liver, with cirrhosis, and with acute pancreatitis.

The serum transaminase level is normal for 6 to 10 hours after a myocardial infarction, reaching a peak 12 to 24 hours after the onset of pain and returning to normal within a week. Its estimation is useful in distinguishing the cardiac pain of ischaemia from that of infarction, but it is important to obtain blood samples within a few hours of the onset of pain.

D. Emslie-Smith

Serum Enzymes—I. Serum Lactic Dehydrogenase in Myocardial Infarction.

L. P. WHITE. *New Engl. J. Med.*, 255, 984, 1956.

The determination of serum levels of glutamic oxalacetic transaminase (G.O.T.), recently introduced as an aid in the differentiation of myocardial infarction and pulmonary infarction or embolism, suffers from three main defects: (1) abnormal levels of the enzyme persist only for 24 to 72 hours after the infarction occurs; (2) some patients with infarction of limited extent do not show abnormal levels of enzyme; and (3) the method is laborious. As the heart is rich in other enzymes, the practicability of utilizing the serum level of one of these has therefore been investigated. The enzymes thus studied were lactic dehydrogenase, aldolase, and hexose isomerase, the serum levels of which were determined and compared with that of G.O.T. in 50 patients in whom the diagnosis of myocardial infarction was entertained.

It was found that the serum level of lactic dehydrogenase was a good, if non-specific, index of the presence of myocardial infarction. Serum G.O.T. determination on the other hand appeared to be of little use in differentiating myocardial infarction, but was of help in the diagnosis of hepatitis. The superiority of lactic dehydrogenase determination depends on the simplicity of assay and on the greater degree and longer duration of the rise in level of this enzyme in the serum. Lactic dehydrogenase, in common with many other enzymes, is widely distributed throughout the body. Any cause of tissue destruction, such as infection, trauma, or infarction, may be associated with abnormal elevation of serum enzyme levels.

L. A. Elson