A STUDY IN CARDIAC PROGNOSIS

BY

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Cardiac prognosis depends upon contemporary statistical knowledge and general experience; also upon the personal experience of the physician himself.

General experience is often a safe guide; for example, subacute bacterial endocarditis invariably used to be fatal, usually within a year, before the discovery of penicillin; and, as another example, a minimal blowing mitral murmur without cardiac enlargement was, and is, regarded as entailing no risk to life or health. But time produces continuous changes in the prognostic scene, and to-day 75% of patients with subacute bacterial endocarditis may look forward to a good recovery and a usefully active life. This transmuting effect of time has already influenced the outlook in mitral stenosis, persistent ductus arteriosus, syphilis of the cardiovascular system, and coronary disease. Further changes may soon be looked for in, for example, hypertensive heart disease and atrial septal defect. It is for this reason that orthodox prognosis, founded upon statistics, is apt to be unreliable when applied to individual cases.

It was therefore thought that a study of patients who had survived for long periods in spite of the presence of diseases usually considered serious might stress the value in prognosis of the individual clinical approach. Each case is dealt with separately, and an attempt is made to analyse from general principles the reason for individual survival or absence of deterioration. Such general principles may help the formation of a more reliable individual prognosis than will obsolete or obsolescent statistical figures.

The cases and their survival years are as follows: mitral stenosis, 14 years; aortic stenosis, 45 years; rheumatic aortic incompetence 49 years; very severe rheumatic pancarditis, 20 years; syphilitic aortitis with aortic regurgitation and angina of effort, 15 years; subacute bacterial endocarditis (aortic), 7½ years; right bundle-branch block, 18 years; angina of effort, due to coronary disease, 26 years; posterior myocardial infarction, 23 years; hypertension 25 years (male), and 11 years (female).

General Principles

Cardiac prognosis will always depend upon the heart being able to maintain a normal pumping efficiency. The factors concerned in this are the physical state of the heart muscle; potential change in the state of the heart muscle; maintenance of an easy balance between the systemic and pulmonary circulations; the presence or absence of mechanical handicaps to the functional activity of the left or right ventricle.

The normal heart muscle can maintain an adequate output at rest, and can increase this sufficiently to enable the individual to maintain his or her daily activities without symptoms. A similar result can be attained by a previously diseased but now compensated heart. Two distinct questions have thus to be determined in every case. Is the function of the heart normal? Is the structure of the heart normal? It is true, with few exceptions, that the absence of symptoms suggests absence of failure, and that if there is no enlargement of the heart there is no organic disease. Symptoms, however, may be latent, in the presence of serious disease, if both ventricles are equally impaired in functional efficiency.

Circulatory Balance

The importance of this factor of balance between the two pumping systems, systemic and pulmonary, is often overlooked, but its significance cannot be overemphasized. Most cases of cardiac failure are due either to rheumatism or to coronary disease. In both of these examples the left heart is predominantly affected—in rheumatism by mitral or aortic valvulitis, in coronary disease by left ventricular ischaemia. Both types of lesion cause primary failure of the left auricle or the left ventricle, and resultant but secondary increase in the work of the right ventricle. If either type of lesion develops rapidly pulmonary complications in the shape of congestion, paroxysmal dyspnoea or acute pulmonary oedema will follow.

If, however, an exact mechanical balance persists between the two ventricles and their circulations a diseased heart muscle may retain its functional efficiency to a quite remarkable degree. Examples of exactly balanced left and right ventricular myocardial disease, without valvular or other mechanical complications, are rare, however. Diphtheria and severe anaemia can produce such a result. In the past, before the days of diphtheria immunization and adequate treatment of pernicious anaemia, ambulatory patients collapsed and died suddenly from either disease, their heart muscle being riddled with fatty degeneration, as shown microscopically.

Another dramatic example of this situation was that of a case of cardiac haemochromatosis (Bourne and Cureton, 1953). This man, aged 51, died with evidence of severe myocardial disease. All the usual phenomena of haemochromatosis of the heart were present: a very low voltage electrocardiogram; a low blood pressure (115/85 to 98/80); episodes of auricular fibrillation and flutter; and the rapid development, in a few days, of congestive heart failure, with oedema, ascites, and pleural effusion. Moreover, histological examination of the myocardium later showed widespread and severe cardiac haemochromatosis.

In spite of a myocarditis of this degree, from which he died on August 4, he had played 36 holes of golf on July 26. But the majority of patients with cardiac disease lack this benefit of an equally balanced diminution in the efficiency of both left and right ventricles.

Mitral Stenosis for 14 Years

When first seen Mrs. A. was aged 25. At the age of 40 she still had a loud first sound at the apex, a typical pre-systolic murmur, and a rumbling diastolic murmur filling most of diastole and localized to the apex. Her symptoms fourteen years ago were of the functional "nervous" type, including palpitation, exhaustion, giddiness, and a left chest ache not associated with exercise. She also had a little shortness of breath. She was overweight. The blood pressure was 116/80. The rhythm was regular. Hyperaesthesia was present in the left submammary region. Radiologically the heart was normal in size, measuring 11.0 cm. There was no prominence of the pulmonary conus. The barium swallow showed no evidence of auricular enlargement at any time. The electrocardiogram remained normal from the first to last. Twelve years ago she became pregnant and her condition remained unchanged both during and after the pregnancy. Fourteen years from the date of the first examination the size and shape of the heart remained unchanged. The latest transverse diameter measurement by orthodiagraph was 10.5 cm. She remained free from significant shortness of breath in spite of having been persistently overweight. A little palpitation and a feeling of tiredness, especially if she was worried, were constant throughout.

It is clear that the murmurs of mitral stenosis in her case do not indicate a lesion which has affected the function or the structure of her heart. There has been no sufficient mitral narrowing to interfere with the proper emptying of the pulmonary veins. The signs of pulmonary hypertension, shortness of breath on exertion, orthopnoea, and cough on

exertion have remained absent, as have the physical signs of pulmonary hypertension: accentuation of the pulmonary second sound with perhaps splitting of it; the presence of an opening snap; pulmonary conus pulsation; radiological and electrocardiographic evidence of auricular enlargement and of right ventricular hypertrophy. It is obvious that the prognosis in such cases depends on these latter direct evidences of the valvular narrowing and not on the presence of the typical mitral stenotic murmurs. These only indicate the position of the lesion, and give no guide to its extent.

To apply the general principles formulated above, the heart muscle in her case was normal, no recrudescence of rheumatic infection had occurred in the heart muscle. The normal balance between the systemic and pulmonary circulations remained unaffected by the minimal mitral deformity. This minimal mitral deformity was insufficient to cause any handicap to the functional activity of the right ventricle in the shape of pulmonary hypertension or of the left ventricle in the shape of diminishing output per beat. She may well continue in good health for an indefinite period.

Aortic Stenosis for 45 Years

Dr. B. died at the age of 64. When he was 18 Sir Thomas Lewis diagnosed aortic stenosis. The condition had therefore probably been present for fifty years at the time of his death. His youthful activities were normal. In spite of the murmur he joined the R.A.M.C. in 1916. He practised in Bangkok for twenty-five years, and retired to England, where he did general practice without symptoms until the age of 55. He then began to notice slight shortness of breath when climbing hills and stairs, but ascribed this to a mild degree of chronic bronchitis and emphysema, which indeed he had. At the age of 57 the electrocardiogram showed left ventricular strain. A year later he began to notice very slight angina of effort when hurrying up hills and stairs. This was never sufficient to need glyceryl trinitrate. At the age of 60 he was conscious of feeling older. At this time the typical systolic thrill and murmur of aortic stenosis were well developed. The heart rate was 86, the rhythm was regular, and the blood pressure was 152/102. The electrocardiogram still showed the typical picture of left ventricular strain and left ventricular hypertrophy. Radiologically the transverse diameter of the heart by orthodiagraph was 15 cm. Calcification of the aortic valve was visible radiologically.

During the next three years his shortness of breath increased, and two months before his death signs of congestive failure began to show themselves in the form of peripheral oedema and orthopnoea. Auricular fibrillation was now present. Radiologically the heart had enlarged to a transverse diameter of 17.5 cm. The post-mortem examination showed what was apparently a calcified aortic stenosis of the acquired type.

His attack of early rheumatism appeared to have been restricted to the aortic valve. It caused slow fusion of the cusps with the development of aortic stenosis rather than distortion and destruction, producing aortic regurgitation. The remarkably slow development of the lesion was characteristic, and the heart muscle, which had never been seriously injured by acute rheumatism, remained not only in a state of normal health but able to hypertrophy, at its leisure, to meet the slowly increasing mechanical handicap of the narrowing aortic ring. No deterioration in the heart muscle occurred until evidence of coronary ischaemia began to appear in the form of angina of effort. It is probable that in this case, as in the following one, the cause of the ultimate failure was the fact that the myocardium of a man of 64 was unable to maintain the left ventricular output through an aortic valve whose narrowing had reached a critical diminution. A noticeable point indicating the myocardial failure was the increase in the heart size by orthodiagraph from 15 cm. to 17.5 cm. over the last three years of his life.

Aortic Regurgitation (Rheumatic) for 49 Years

Mr. C. had had aortic regurgitation for more than 49 years. The murmur was heard by the late Sir Maurice Cassidy when the patient was 20 years old. He was aged 66 at the time of my first examination of him. The lesion was almost certainly rheumatic in origin. He had remained symptom-free for 46 years after the diagnosis of his valvular lesion. He played Rugby football up to the age of 41 in spite of it. It was not until the age of 65, after an attack of pneumonia, that he became short of breath on exertion. This rather rapidly increased in severity, and he began to notice some orthopnoea. The physical signs found were as follows: increased arterial pulsation, both visible and palpable; considerable displacement of the apex beat to the left; a rapid regular heart rate of 110; pulsus alternans, with systolic figures of 180 and 190 and diastolic of 90 mm.; the typical murmur of aortic regurgitation, heard at the aortic base and down the left sternal border to the apex beat. Radiologically the heart was very large, the transverse diameter being 20.5 cm., and some pulmonary congestion was visible. The electrocardiogram showed the tall broadened R waves of left ventricular hypertrophy and the inverted T wave of the left ventricular strain in the left ventricular leads. During the next two years he fractured his femur, which was pinned, and later underwent a prostaticectomy, from which he made a good recovery.

The noticeable points relative to prognosis were as follows: the state of the heart muscle was obviously remarkably good throughout most of his active life; nor was there any deterioration in the normal state of the muscle, probably because he escaped further attacks of rheumatic carditis. The valvular handicap, although considerable, was not too great for the compensatory power of the healthy and hypertrophied left ventricular muscle, which was able to eject the full systemic requirement, and in addition the amount of the aortic reflux, without suffering any serious mechanical disability. The handicap which was probably responsible for the occurrence of symptoms after his 48 years of normal life was the inevitable one of increasing age.

In many cases of organic heart disease which have remained adequately compensated for decades the passage of years begins to produce deterioration in the arterial system. These changes may affect either the peripheral arterial bed in the heart, as elsewhere in the body, or the elasticity, the health, and the permeability of the larger coronary arteries.

Fulminating Rheumatic Carditis, with Perfect Recovery 20 Years Later

Mr. D. was first seen in 1936, when he was aged 34. He was suffering from a very acute attack of typical rheumatic fever with painful synovitis of wrists, a knee, and an ankle. During the acute stage of the disease he developed pericarditis with a loud friction rub, heart block with failure of auriculo-ventricular conduction at every third or fourth beat, and finally symptoms of acute rheumatic meningitis associated with lymphocytosis and other evidences of inflammation in the cerebrospinal fluid (Bourne, 1936). He was ill for three months, but subsequently made an uninterrupted recovery. After convalescence his heart was clinically normal. The P-R interval was normal. Radiologically the heart was not enlarged. The T wave was upright in lead I and diminished in size in lead II.

He was seen at intervals during the next 19 years, usually for symptoms due to alcoholism. During that time the only cardiological abnormalities were a slight systolic murmur at the apex and a progressive flattening of the T waves in the three standard leads. He suffered from no shortness of breath or chest pain.

In 1953 he began to complain of symptoms suggestive of cirrhosis of the liver. When he was examined in 1954 he was found to have ascites. After paracentesis the typical

hobnail liver was discovered; there was no venous distension in the neck, and no peripheral oedema or other evidence of constrictive pericarditis. He developed active pulmonary tuberculosis during this year and died early in 1956. To the end his heart remained clinically and radiologically normal.

Although the likelihood of permanent cardiac complications in acute rheumatism is 90% or more in children below the age of 10, in adults over the age of 20 the cardiac risk becomes progressively less with advancing years. No more acute example of severe active cardiac rheumatism could have been found, yet for twenty years neither his cardiac function nor his cardiac physical signs deviated from the normal.

Syphilitic Aortic Regurgitation, and Angina of Effort. Symptomless 15 Years Later

Mrs. E. and her husband were caretakers of a city building. She was first seen in October, 1941, when she was 42 years of age. Since the previous December, following a bad air raid, she had begun to notice shortness of breath on exertion and discomfort across her upper chest, which came on if she hurried or climbed stairs or walked against a wind. She was found to have a well-marked aortic regurgitation. The blood W.R. was strongly positive. The blood pressure during her first treatment in hospital was 170/50, subsiding with rest to 140/44. Treatment was by bismuth injections and neoarsphenamine. The treatment was repeated.

Six years later her physical condition was unchanged, the blood pressure being 180/62. Her shortness of breath and angina of effort had disappeared, and in April, 1948, she was doing her own shopping and household washing, and in fact was leading a full normal life. The aortic diastolic murmur persisted. The heart was only slightly enlarged radiologically, but the left ventricle was somewhat rounded. Three years later the aortic leak remained considerable, the blood pressure being 190/45. When last seen on April 19, 1956, she was still free from shortness of breath or anginal pain. Peripheral arterial pulsation was increased. The pulse pressure remained high (210/60). Radiologically the left ventricle was again seen to be rounded, but measurement of the heart by orthodiagraph still showed no great enlargement. Electrocardiograms during the period of her attendance over the last ten years have shown the gradual development of the left ventricular strain pattern.

She thus remains well fifteen years after the first diagnosis of syphilitic aortic valve incompetence, and of angina of effort from coronary ischaemia due to syphilitic aortitis.

The prognosis in such a case depends entirely upon the state of the heart muscle. The chief cardiovascular targets of syphilitic infection are the aorta and the aortic valve cusps. The aortitis may interfere with coronary flow either during the early stage of active progressive inflammation when obstruction results from the inflammatory swelling or after the active inflammation has been followed by fibrosis and progressive scarring, again producing coronary interference. The inflammatory swelling is amenable to treatment, the fibrous scarring is not.

In this case the left ventricular muscle remained healthy, for syphilis does not directly attack heart muscle. The angina of effort was due to temporary narrowing of the coronary mouths from active inflammation. Successful treatment of this restored the normal passage-way and removed the pain. Healthy heart muscle can always cope with an aortic leak by hypertrophy and compensatory dilatation. The prognosis in syphilitic cases will therefore depend entirely upon the presence or absence of coronary obstruction by the aortitis and upon the stage of aortitis reached by the disease. If the ischaemic lesion is sufficiently early prognosis may be excellent. The aortic valvular distortion due to syphilis is by comparison of relatively little importance.

Subacute Bacterial Endocarditis of the Aortic Valve. Normal Function after 7½ Years

Mr. F. had suffered from rheumatic fever at the age of 6. At the age of 45 a dental extraction was performed under penicillin cover lasting for 24 hours. Three months later he noticed irritability and lassitude, and a month before he was examined, five months from his dental extraction, he began to suffer from severe night sweats and transient pain in the shoulder joints. On examination he looked a little pale. The heart rate was 88, the rhythm was regular, and the blood pressure was 130/54. The murmur of aortic regurgitation was present. No mitral diastolic or systolic murmurs were audible. The spleen was not palpable. Radiologically the heart was slightly enlarged, the transverse diameter being 14.3 cm. by orthodiagraph. This enlargement chiefly involved the left ventricle. Watch was kept on his temperature, and fever was found to be present. *Streptococcus viridans* was isolated from his blood culture. He was treated for six weeks with full doses of penicillin, and made an excellent recovery.

Seven and a half years later he was in good health. He was free from shortness of breath, chest pain, or other symptoms, and was living a normal active life. Over this period of time the blood pressure gradually rose to 176/76.

About 75% of patients with subacute bacterial endocarditis make a perfect recovery except for the inflammatory distortion of the affected valve. The remaining 25% die of gradually increasing renal failure, the result of multiple embolism, of progressive heart failure due to myocardial failure, probably due to embolism similar in type to that causing the renal lesion, the remainder from cerebral or pulmonary thrombo-embolic complications. This patient, who was left with distortion of the aortic valve only, was very little inconvenienced by this, for the disease has no long-term direct effect upon the heart muscle. Fortunately, also, the valvular fibrosis nearly always produces regurgitation and not stenosis, probably because, for some unknown reason, subacute bacterial endocarditis is far more likely to occur in mitral and aortic valves in which regurgitation rather than stenosis was originally present. In this case the healthy left ventricular muscle was able to accommodate, per beat, both the normal diastolic filling from the left auricle and the extra diastolic flow from the aorta due to the aortic valve deformity. Furthermore, the left ventricular muscle was able to eject its double load without distress.

Right Bundle-branch Lesion Present for 18 Years Without Change

Mr. G. had had rheumatic fever in 1930 at the age of 41. He was first seen in August, 1935, when he was 46. He at that time complained of heart consciousness and palpitation, clearly due to premature beats. He was, however, playing tennis and golf and living a normal life. A year later he was re-examined. No physical abnormality was discovered. The blood pressure was 130/72. The three-lead electrocardiogram was normal. There was no cardiac enlargement radiologically. In 1938, at the age of 49, the clinical condition was unchanged, but the electrocardiogram showed a complete right bundle-branch lesion. This has persisted without change for eighteen years.

He remained free from significant symptoms until January, 1954, when he was 65. He then rather suddenly noticed shortness of breath while walking at an ordinary speed, and pain in the left arm, produced by walking and synchronous with the dyspnoea. This disappeared when he rested, and returned when he resumed his walk. Two months later he was seized with severe chest pain radiating to the left arm. Collapse and sweating were both present. On examination he was found to have auricular fibrillation. Although the electrocardiogram was inconclusive there was no doubt clinically that he had had a myocardial infarction. He was treated in the usual way by rest followed by gradual resumption of his normal life, but at a considerably lower level of activity.

The electrocardiogram is highly regarded, rightly, as giving invaluable information in cardiac conditions. By the lay public and by some practitioners it is so highly revered as the final court of appeal that deviations from the normal in it are considered to indicate grave trouble. There is no electrocardiographic picture which shows a greater disturbance of the normal pattern graph than a bundle-branch lesion, left or right. It is important, therefore, to know that such an apparently gross abnormality can be present for years (in this case a period of eighteen years) as the only indication that there is anything wrong. Undue emphasis on an isolated electrocardiographic finding could in such a case induce a state of premature cardiac invalidism long before any real reason for this was present.

The reason for this patient's continued good cardiac health in spite of the presence of the bundle-branch lesion was no doubt the fact that the bundle and its branches occupy a small area of heart muscle tissue. If a pathological lesion happens to be so placed as to interfere with the bundle of His, or with either of its branches, such a lesion need only be very small in extent to produce complete interference either with the bundle or with its right or left subdivision. Such a lesion, although minimal, can distort the electrocardiogram grossly, but, being small, will not interfere with the general efficiency of the pumping power of the heart.

Angina of Effort from Coronary Disease (26 Years)

Mr. H. has had classical angina of effort for 26 years. At the age of 37 he first noticed pain in the chest only during exercise. This disappeared quickly when he rested. At that time the three-lead electrocardiogram showed no abnormality. This exercise pain was first present for a period of two years, after which he recovered so well as to be able to play cricket, tennis, and squash. At the age of 47 he noticed cramp in his left calf, which came on while playing tennis. His foot went white and cold. A year later his angina of effort reappeared. The pain was felt across the upper chest and it radiated to both arms. It was usually induced by walking for a distance of about 50 yards. It was accelerated by hurry and prevented by walking slowly. Stairs and emotion also precipitated it and glyceryl trinitrate relieved it. At this time the only abnormalities were a slightly raised diastolic blood pressure (152/100), and in the electrocardiogram a low T wave and a small Q wave in lead II, and an inverted T wave and larger Q wave in lead III.

He was seen again at the age of 50. The anginal pain was unchanged but he still suffered from intermittent claudication in the left leg after walking 50 yards or so. He was re-examined at the age of 52. His general health was remarkably improved and his anginal pain, though occasionally noticed, was much less severe. He had again suddenly suffered intermittent claudication, this time in the right lower leg. The dorsalis pedis and posterior tibial arteries could not be felt on the right side, but were present on the left.

Four years later he retired from his work as secretary to a hospital. His angina of effort was slight, but the intermittent claudication in the right leg still worried him. His condition remained unchanged until he was re-examined seven years later at the age of 63. His intermittent claudication was still somewhat of a handicap; his angina of effort was not evoked unless by considerable exertion. This time, however, he had recently suffered a thrombosis of the left central retinal vein. His heart was clinically normal, the rhythm regular, and the blood pressure 164/92. Radiologically the heart was bulky but within normal limits for a man of his physique. The electrocardiogram showed a flat T wave in lead I and inversion of the T wave from V4 to V7 inclusive. The T wave was also inverted in leads II, III, and VF, and the Q wave remained increased in size in lead III and was visible in leads II and VF.

He thus had had angina of effort for 26 years. The history from the start was classical, and the electrocardiogram throughout most of this time showed evidence of coronary

disease. In this case it is clear that the amount of coronary arteriosclerosis must have been small and that the coronary vessels remained free from further deterioration. It is also probable that some revascularization of the ischaemic area had developed. He had always been a highly strung sensitive person. It is also therefore likely that his visceral sensations were felt more easily from a less degree of local disease than would have been the case in a more phlegmatic patient. Not only was the coronary disease non-progressive, but the state of the heart muscle as a whole must have remained good, thanks to an adequate blood supply. No hypertensive factor was present to burden the heart.

Posterior Myocardial Infarction (23 Years)

Mr. I., aged 72, had suffered from a myocardial infarction at the age of 49 when he was under the care of Sir John Parkinson. He was in a nursing-home for six weeks. He made a good recovery and remained well for 23 years.

The recent history was that he had had slight shortness of breath for a few days, and considerable swelling of the legs and feet for the same length of time. He had noticed that he had bruised rather easily during the preceding fortnight. His physical state during these 23 years had remained good. His activities were normal and he had been free from shortness of breath and chest pain.

On examination marked pitting oedema of the legs and feet was present. The heart, however, was clinically perfect. The blood pressure was 136/80. Radiologically the transverse diameter by orthodiagraph was 13 cm., which was normal for his age and physique. The urine contained no albumin. The electrocardiogram showed large Q waves and inversion of the T waves in leads II, III, and VF. It was clear that the oedema was not cardiac, since he had had no shortness of breath, nor was it renal, since there was no albuminuria. A tourniquet test was done on his arm and multiple fresh petechiae appeared below the band.

He had thus made a good functional and physical recovery from his posterior myocardial infarction, and this recovery was still persisting when he was seen 23 years later. The reasons for this were that temporary injury to the heart muscle was compensated for by scar formation at the site of the infarct, also, in all probability, by successful collateral circulation from the near-by coronary branches, and by hypertrophy of the healthy heart muscle to such a degree that, on balance and after a period of readjustment, he was left with as good a functioning heart as he had had before. No handicaps in the shape of hypertension or of serious atheroma of the other coronary branches interfered with this perfect recovery.

Hypertension for 25 Years

Mr. J., at the age of 15, was found by a doctor to have a higher blood pressure than normal. Two years later, when he was playing vigorous Rugby football, this discovery was confirmed. Figures were first available from an examination when he was aged 22, the blood pressure in the arms being 240/160 and in the legs, by palpation, 210. This excluded the possibility of coarctation of the aorta, further evidence of which condition remained absent. At the age of 23 the figures were 185/130. When first examined by me he was symptomless, having no shortness of breath on exertion, no chest pain, headaches, or visual defects. The retinal vessels showed an early grade I hypertensive change. The blood pressure was 230/140. The first sound at the apex was reduplicated. The aortic second sound was accentuated. The electrocardiogram was normal. Radiologically the heart was slightly bulky for his build and age, the transverse diameter being 13.2 cm. by orthodiagraph. The left ventricle was a little prominent and the aorta also.

Nine years later, when he was 41, he still felt perfectly well. The blood pressure was 212/136. Radiologically the heart was slightly larger, the transverse diameter being 14.7 cm. The electrocardiogram remained normal as regards the standard leads, but the T wave was a little flat in V5.

At the age of 42 he was re-examined. The blood pressure was 208/126. The retinal vessels showed no change from their previous state. Radiologically the heart was still slightly larger, the transverse diameter being 15.5 cm.

Three months later he was seized with the symptoms of myocardial infarction, from which he died very suddenly.

He had clearly suffered from prolonged severe hypertension. This remained harmless to his health or his life for 25 years, the absence of complications during that time being due to the excellent state of his arteries. The adequate supply to his heart muscle enabled the left ventricle to remain healthy and to support the hypertensive burden without in any way failing. Atheroma, however, supervened towards the end, and a fatal coronary thrombosis caused his death. The increasing heart size over the last few years was ominous, although symptoms were absent until the end.

He was a sensible and helpful patient, and it was a tragedy that his disease terminated shortly before the introduction of effective hypotensive drugs. Sympathectomy was thought of, but its failure in so many similar cases led to its not being tried, especially in a symptomless patient.

Hypertension, Unchanged for 11 Years

Mrs. K. was first seen at the age of 65 for attacks of giddiness. She had incidentally a raised blood pressure. The detailed history of her symptoms suggested that the attacks of giddiness were vestibular in origin, the function of the middle ear being affected by local arteriosclerosis. On examination the heart rate was 90 and the blood pressure 230/126. The heart was normal clinically and radiologically. The orthodiagram gave a normal transverse diameter of 11.5 cm. The electrocardiogram showed upright T waves in the left ventricular leads and no evidence of left ventricular hypertrophy.

She was seen eleven years later, when she was 76 years old. Her attacks had continued. Her description of the attacks was now not so clear, and it was difficult to disentangle the possible causes as between vestibular disturbance, vasovagal faintness, and functional or psychogenic influence. On examination she again had a nervous tachycardia of 90; the blood pressure was 220/120. The heart remained clinically normal. Radiologically there was some increase in size, the transverse diameter being 13.7 cm. The electrocardiogram was that of a normal horizontal heart. The unipolar limb leads and the unipolar precordial leads V1 to V6 were all used.

She clearly was a woman in whom there was a static, so-called benign, hypertension. Since the heart becomes more bulky in normal people in the 70's and 80's the increase in the transverse diameter is not so significant as it would have been in a younger individual. The hypertension as such was clearly harmless, and her case was a good example of the fact that the prognosis in elderly women with a raised blood pressure is so frequently extremely good.

Conclusions

It is not claimed that this record of long-term cases can provide any accurate statistical guide to the prognosis in the various conditions mentioned. The purpose is to offer proof that a prolonged period of good health may be expected in individual patients. From this it follows that the clinician must carefully analyse all his cases, as individuals, from the point of view of the state of the heart muscle at the time of examination, the changes of deterioration or improvement in the state of the heart muscle, the absence or the presence of mechanical handicaps which might interfere with cardiac efficiency, and the removability of these by medical or surgical means. He should also, so far as the facts will allow him, emphasize to his patients the possibilities of a good prognosis so that their co-operation in a positive constructive line of life may be obtained. It is only by

so doing that those individuals in whom a good prognosis is possible will be enabled to achieve it to its maximum extent.

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BLOOD TRANSFUSION IN OBSTETRIC HAEMORRHAGE WITH REFERENCE TO VALUE OF ESTIMATIONS OF BLOOD VOLUME

BY

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This paper is concerned with the position of blood transfusion and the value of blood-volume estimations in obstetric shock, mainly in cases of concealed accidental haemorrhage. Grant and Reeve (1951) and Crosby and Howard (1954) have shown that estimations of blood volume are of considerable value in the treatment of shock due to blood loss in battle casualties. This work is being followed up on civilian casualties in the Birmingham Accident Hospital by Clarke, Topley, and Flear (1955) and Clarke and Flear (1956). In view of this, one of us (A. D. T.) estimated the blood volume in a number of cases of obstetric haemorrhage as a guide to the amount of blood to administer. In the treatment of obstetric shock the problem of when and in what quantity to administer blood can be a difficult one. This is especially true of concealed accidental haemorrhage, and there is diversity of opinion regarding the best methods of local and general treatment. Clarke and Fisher (1956) state clearly the difficulties arising from the provision of pint bottles of blood. A bottle of blood contains 100 to 120 ml. of anticoagulant, and in fact we transfuse approximately 400 ml. of the whole blood in a bottle. This explains our stating the amounts of blood transfused in millilitres; thus a transfusion of five bottles approximates to 2 litres of whole blood.

By "severe accidental haemorrhage" we mean cases where the patient is admitted with concealed and/or revealed accidental haemorrhage and in whom the foetal heart cannot be heard. Crichton (1950), in an analysis of 120 cases in Capetown, gave a maternal mortality of 10.9%. Sheehan and Moore (1952) described 700 cases of severe accidental haemorrhage with 75 maternal deaths—a mortality of 10.7%—and their criterion of severity was a dead child *in utero*.

Our views on local treatment in accidental haemorrhage are based on those of the late Professor Marshall Allan (1940), of Melbourne, who taught that artificial rupture of the membranes was advisable. Professor Allan followed this treatment with pituitary extract, but we do not use intramuscular pituitary extract or oxytocin because of the possible association of afibrinogenaemia with excessive uterine action. We are coming more and more to the view that caesarean section has little to offer in this condition except in the mild case associated with