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UNIVENTRICULAR ASYSTOLE AFTER MYOCARDIAL INFARCTION

BY

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Two patients with fatal cardiac arrest after myocardial infarction in which there was asystole of one ventricle are presented.

Case Histories

Case 1.—J. L., aged 77, was admitted to the Cardiff Royal Infirmary on December 26, 1959, because of cardiac failure. He gave a history of shortness of breath on exertion for two months and of pain behind the middle of the sternum on exercise for one month. This was relieved by rest. On two occasions during this time he awoke at night short of breath. For several weeks he had had giddy attacks in which he thought he would faint, but he never actually did so. He had suffered from a cough with expectoration of mucoid sputum for one week.

On admission the jugular venous pressure was slightly raised and there was oedema of the sacrum and ankles but no enlargement of the liver. The pulse was irregular and a systolic murmur was heard at the apex. The blood pressure was 140/70 mm. Hg. A few wheezes were present throughout both lungs. The electrocardiogram (Fig. 1) taken on December 28, 1959, showed a vertical heart pattern with marked clockwise rotation, neutral axis deviation, and sinus rhythm with normal P waves. There was no evidence of hypertrophy of either ventricle or of bundle branch block. The T waves were upright in all the praecordial leads, but the RS–T segments were depressed slightly in V7 and aVF, suggesting myocardial ischaemia. Postero-anterior and lateral chest films (Fig. 2) showed a small effusion into the right pleural cavity and a moderate-sized one on the left. The heart was considerably enlarged, the lung fields showed the appearances of pulmonary venous hypertension, and the aorta was calcified in its arched and upper descending portions. The urine was turbid and alkaline but contained no albumin, and the deposit showed amorphous debris only. The blood urea on December 29, 1959, was 104 mg. per 100 ml. The haemoglobin was 11.4 g. (77%) and the red blood cells were slightly hypochromic and showed anisocytosis and poikilocytosis: the total white and differential count was normal.

The patient was treated for heart failure with digoxin and chlorothiazide, and was given glyceryl trinitrite for the anginal pain. While on treatment he developed acute retention of urine: because an enlarged prostate was found, it was decided to perform cystoscopy and prostatectomy.

On January 1, 1960, at 9.45 a.m. the patient was given 0.6 mg. of atropine, and at 10.30 a.m. anaesthesia was commenced. Oxygen, nitrous oxide, and halothane were administered and a cuffed endotracheal tube was inserted: induction of anaesthesia was smooth. At 10.40 a.m., while the patient was being wheeled from the anaesthetic room to the operating theatre, he became cyanosed and pulseless and spontaneous respiration ceased. Cardiac arrest was diagnosed, the chest and pericardium were opened immediately, and cardiac massage was begun within three minutes. Five minutes later the writer arrived in the theatre and took over manual cardiac compression. The right ventricle was contracting strongly, but the left was atonic and asystolic. Despite manual compression and several intracardiac injections of 1:10,000 adrenaline and 1% calcium chloride the two ventricles remained in this condition until the terminal few minutes when fine fibrillation developed in the left ventricle. The latter was corrected temporarily by electrical defibrillation at the second attempt, but thereafter both ventricles became atonic and asystolic and manual cardiac compression was abandoned at 11.45 a.m. At this time the pupils were fixed and dilated.

Necropsy showed a grossly enlarged heart which weighed 560 g. The left ventricle was hypertrophied and a fibrous scar occupied its lateral wall. There was severe atheroma of the coronary arteries with recent occlusion of the residual lumen of the left anterior descending branch by antemortem thrombus. The aorta was also the site of severe atheroma. The heart valves were normal. Both lungs were deeply congested and oedematous. There was benign enlargement of the prostate causing obstruction of the urethra, and the kidneys showed granularity of the surface and irregular narrowing of the cortex. The bladder was distended to the level of the umbilicus with clear urine. No other relevant changes were found. Histological examination was not performed.

Case 2.—K. W., a man of 34 years, was sitting in his parked car when at 7.45 p.m. on July 23, 1960, he was suddenly seized with severe pain in the region of the lower sternum, an ache in the jaws, and nausea. He had never had a similar attack before, but he

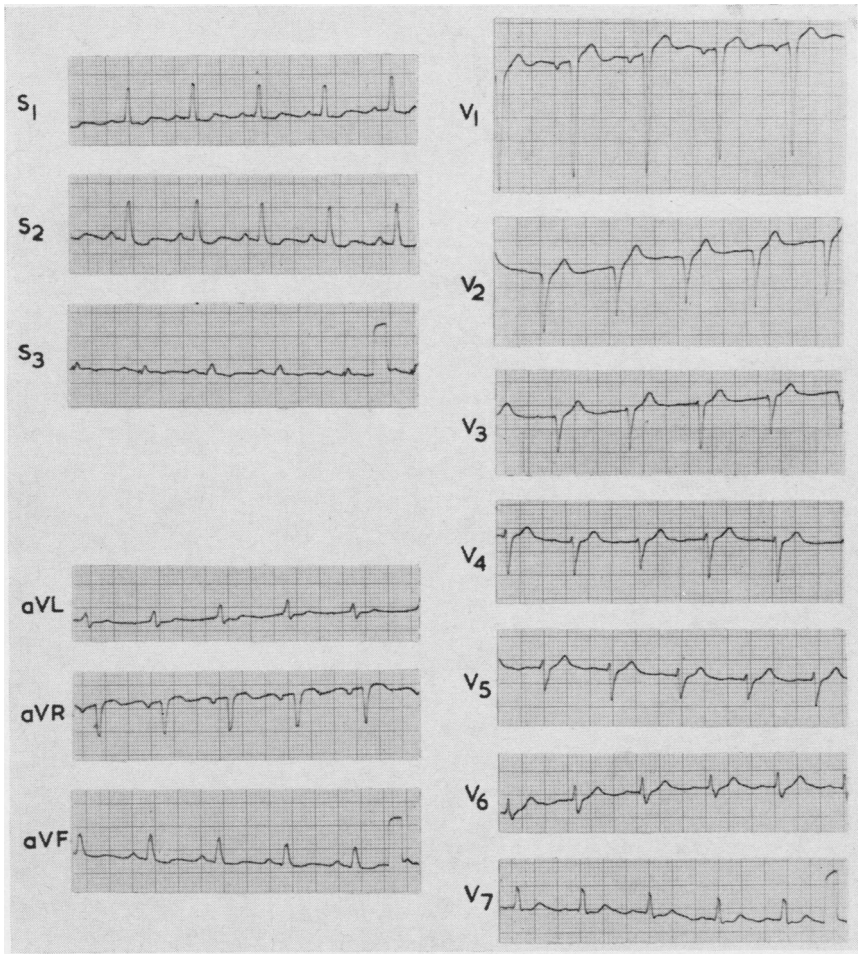

FIG. 1. *Case* 1. *Electrocardiogram showing slight depression of the RS-T segments in leads V7 and aVF suggestive of myocardial ischaemia.*

had had some indigestion, and a radiological examination in 1949 had shown a duodenal ulcer.

He drove to Cardiff Royal Infirmary, walked into the casualty department, undressed, and lay down on a couch unaided. On examination at 8 p.m. he did not look ill and was not collapsed, but he was flushed and a purplish tinge was observed in his neck. The pulse was regular, its rate being 72, and the blood-pressure was 120/70 mm. Hg. No abnormal physical signs were found in the cardiovascular system, chest, or abdomen, and the pupils reacted normally. A diagnosis of myocardial infarction was made and the patient was kept lying down. He continued to complain of an ache in the jaws, he sighed frequently, and he eructated considerable quantities of wind.

At 8.40 p.m., whilst being interviewed by the house physician, the patient suddenly became cyanosed, the skin of the trunk being mottled ; he stopped breathing, the pulses disappeared, and he became unconscious. Cardiac arrest was diagnosed.

Artificial ventilation with oxygen was commenced forthwith. A face-piece was used at first, but this was replaced by an intratracheal tube after the chest had been opened. Thoracotomy was started within two minutes and manual cardiac compression was begun through the intact pericardium within four minutes of collapse. The heart was dilated and atonic and no pulsation could be seen or felt through the pericardium. Three millilitres of 1:10,000 adrenaline was injected into the heart after 10 to 15 minutes of cardiac compression, and the dose was repeated after five minutes. Ten minutes later 5 ml. of 10% calcium chloride was injected into the heart. None of these injections produced any response, the heart remaining flaccid and devoid of visible or palpable pulsation. Fifty minutes after starting intermittent cardiac com-

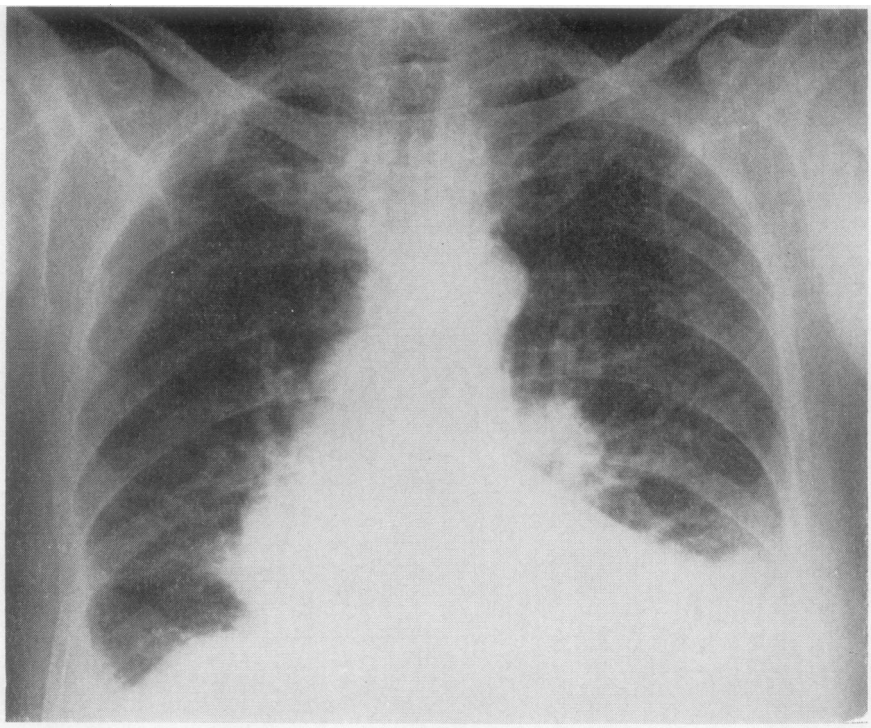

FIG. 2a

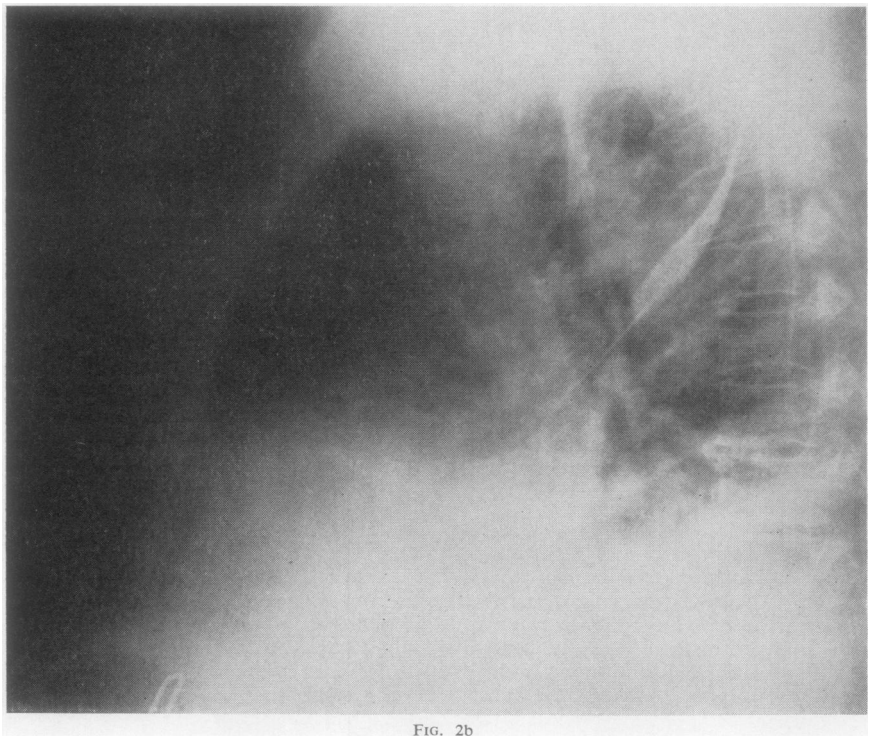

FIG. 2b

FIG. 2. *Case* 1. (a) *Postero-anterior and* (b) *left lateral radiographs showing a large heart, pulmonary venous hypertension, bilateral pleural effusions, that on the left side being the larger, and calcification of the aorta.*

pression 3 ml. of 1:10,000 adrenaline and 5 ml. of 10% calcium chloride were given. Within one minute the heart regained tone and atrial contractions were felt. Fifty minutes later, one hour and 40 minutes after the onset of circulatory arrest, the left ventricle started to contract rhythmically.

At this stage the patient was taken into the casualty theatre, and at 10.45 p.m. the pericardium was opened and massage was continued. At 10.55 p.m. the writer arrived. At this time the left ventricle was contracting rhythmically and quite forcibly, but the right ventricle was atonic and asystolic. The pupils reacted briskly to light and were not dilated. Manual cardiac compression was continued until 12.15 a.m. on July 24, 1960, three further injections of adrenaline and one of calcium chloride having been given. Throughout this period, except for the last few minutes, the left ventricle continued to contract rhythmically, but the right ventricle remained in asystole and ultimately dilated markedly. Neither ventricle fibrillated. At midnight the pupils became fixed and dilated. At 12.10 a.m. pace-maker electrodes were implanted into the walls of the right and left ventricles, but no response could be obtained even with the full output of 9 volts.

Necropsy showed that the heart weighed 400 g. The wall of the left ventricle was 1.5 cm. thick and that of the right ventricle was 0.5 cm. thick. Over the surface of the heart there were subpericardial areas of bruising. Intramyocardial and subendocardial haemorrhage was present in the posterior wall of the left ventricle and the interventricular septum. The heart valves were normal. The coronary arteries were grossly stenosed by atheroma. The left coronary artery and its anterior descending branch were almost completely occluded, and the right coronary artery was completely occluded macroscopically by greyish firm tissue. Several plaques of atheroma were present in the thoracic and abdominal portions of the aorta, especially around the origins of the larger branches and inside the mouths of the innominate and carotid arteries (Fig. 3). These took the form of raised, discrete, flat lesions measuring up to 0.75 cm. in diameter. Well-developed fatty streaking was present in the thoracic and abdominal parts of the aorta. There was copious mucoid, slightly blood-stained fluid in the air passages. The right lung showed oedema and central congestion in the lower lobe, and the left lung displayed some collapse and oedema. The liver and brain were congested. The other organs were normal.

Histological examination showed that the right main coronary artery was almost totally occluded by large protrusions of organized atheroma (Fig. 4). The media and adventitia contained numerous capillaries with prominent groups of endothelial capillary nuclei. The left main coronary artery showed similar changes (Fig. 5) but with a little more patency of the lumen and calcification of the atheroma. The myocardium showed extravasation of red blood cells into the interstitial tissue. Some muscle fibres showed nuclear and striation changes compatible with early infarction. Intimal atheroma was present in the aorta. The lungs showed marked capillary congestion and some poor-staining, low-protein oedema. Groups of siderocytes were present in the right lower lobe. The liver was congested and was the site of slight fatty change. The renal glomeruli showed capillary thickening in some areas with hyaline material, and interstitial oedema was present.

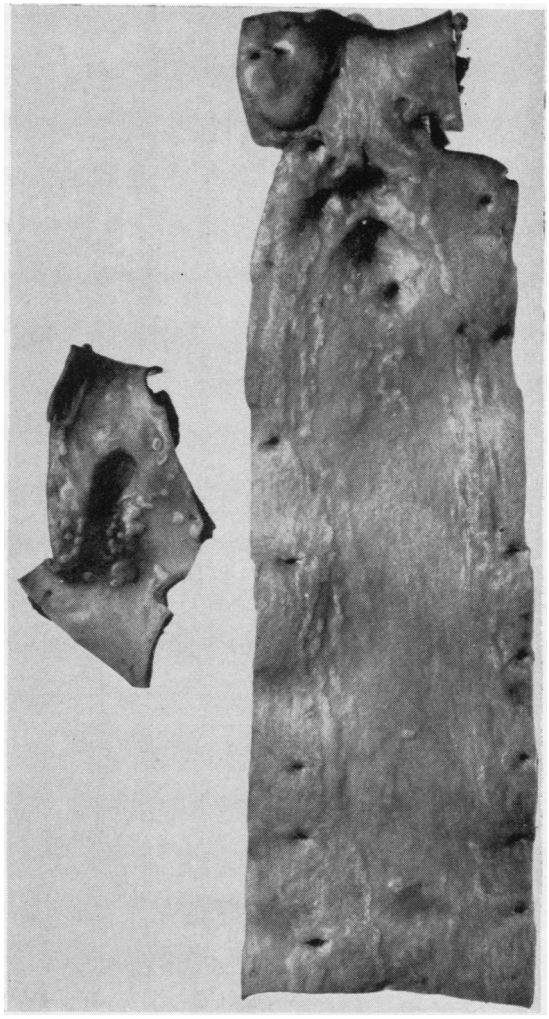

FIG. 3. *Case* 2. *Descending thoracic and upper abdominal aorta and orifice of innominate artery showing fatty streaking of the aorta and unusual distribution of lesions within the orifices of the major branches.*

The cause of death was considered to be due to a myocardial infarct of the posterior wall of the left ventricle and the interventricular septum.

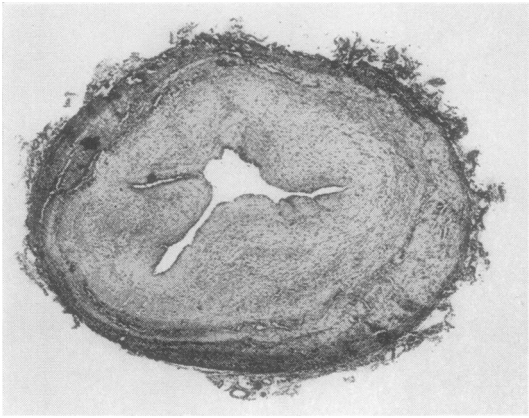

FIG. 4

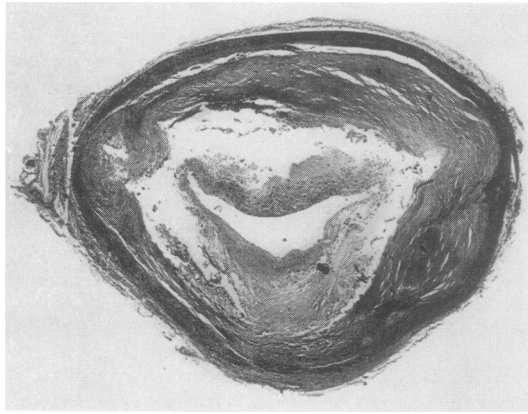

FIG. 5

FIG. 4. *Case 2. Right coronary artery showing gross stenosis of the lumen by active atheroma with advanced organization.*

FIG. 5. *Left coronary artery showing severe stenosis by atheroma with calcification.*

DISCUSSION

These two cases are of great interest from several points of view. Firstly, no other account of asystole of a single ventricle has been found in the literature, though colleagues have told me that they have seen it. In both of the cases reported here one ventricle was completely asystolic for the whole of a considerable part of the period of observation whilst its fellow contracted rhythmically and strongly. In the first patient this state of affairs was present when the heart was exposed by opening the pericardium within three minutes of the onset of cardiac arrest, and it persisted unchanged until fine fibrillation supervened terminally almost an hour later. In the second patient no initial pulsation could be felt through the intact pericardium from the time when the latter was exposed within four minutes of the onset of cardiac arrest until 51 minutes later when rhythmical atrial contractions were felt. Left ventricular contractions were first appreciated one hour and 44 minutes after the onset of cardiac arrest. It is possible that during this period fine ventricular fibrillation was overlooked because the pericardium was intact. When the pericardium was opened two hours and five minutes after the heart stopped, however, the findings stated were confirmed, for the left ventricle was beating rhythmically and quite strongly whereas the right ventricle was asystolic. This state of affairs continued until a few minutes before rhythmic cardiac compression was abandoned three and a half hours after the onset of cardiac arrest. In both cases, therefore, one ventricle was asystolic while its fellow beat rhythmically and powerfully for a considerable period of time, namely one hour and one hour and 50 minutes respectively.

The only other observation I could find of dissociated action of the ventricles is that of Edwards (1960), who states " when cardiac arrest takes place during cardiac surgery, the usual state is ventricular fibrillation, unless the ventricle is hypertrophied and strained, in which case asystole may occur. In cases of pulmonary stenosis and aortic stenosis the curious paradox of fibrillation in the normal ventricle and asystole in the hypertrophied ventricle may be seen if arrest takes place."

The second feature of interest in these two cases is the relationship between the site of coronary arterial occlusion and that of the asystolic ventricle. In Case 1 the anterior descending branch of the left coronary artery was occluded by antemortem thrombus, the left ventricle was hypertrophied, and its lateral wall was scarred. In this case it was the left ventricle which was asystolic. In Case 2 both right and left coronary arteries were grossly narrowed by atheroma, the right being totally occluded macroscopically. An infarct was considered to be present in the septum and posterior wall of the left ventricle, but it was the right ventricle which was asystolic. The wall of this ventricle was 0.5 cm. thick. It is interesting here that although the lumen of the right coronary artery was almost occluded and the right ventricle was asystolic the infarct affected the left ventricle.

These two cases establish the fact that coronary occlusion can cause asystole of only one ventricle and, when considered in conjunction with those

of Edwards cited above, they indicate that dissociated derangement of function of the two ventricles is a possibility.

Experimental Univentricular Asystole

In order to investigate further the possibility of dissociated action of the two ventricles, animal experiments were carried out in association with Dr. R. M. E. Seal. Cineangiography was performed on two occasions in association with Dr. L. R. West.

Methods.—Thirteen adult rabbits were anaesthetized with 2 ml. of intravenous combutal and the following experiments were performed.

Experiment A.—The chest and pericardium of one rabbit were opened and the trachea was then clamped until the animal died of asphyxia.

Experiment B.—Fifteen ml. of air was injected into the marginal ear vein of two rabbits, after which the chest was rapidly opened and the heart removed and observed.

Experiment C.—The chest and pericardium of eight rabbits were opened to expose the heart. Tracheostomy was then performed in seven of these rabbits and ventilation was carried out via the tracheostomy opening by a mechanical pump having a stroke volume of 50 millilitres. From 10 to 20 ml. of air was then injected into the marginal vein while the heart was under observation.

Experiment D.—Electrocardiographic tracings were obtained from standard leads and also from electrodes inserted into the myocardium of the right and left ventricles of three of the rabbits used in experiment C. Direct readings were thus obtained from each ventricle.

Experiment E.—Two animals were submitted to injection of air and cineangiography. In the first of these 8 ml. of air was injected into the marginal vein. The result was unsatisfactory. In the second case 10 ml. of air was injected into the left superior vena cava via a cardiac catheter and was followed after three minutes by an injection of 1 ml. of hypaque. The result was excellent.

Experiment F.—A movie film was made from two animals in experiment C and from the second animal in experiment E.

Results.—The following results were obtained.

Experiment A.—Isolated asystole of the left ventricle developed.

Experiment B.—Both hearts developed asystole of the left ventricle, and in one of them the left atrium was also asystolic. The right atrium and ventricle of both remained in sinus rhythm.

Experiment C.—In six of the eight rabbits asystole of the right ventricle developed, and in four of these the right atrium was also noted to be asystolic. The left atrium and ventricle continued to beat normally. Aspiration of air from the asystolic right atrium or ventricle sometimes restored rhythmic contraction, probably by relieving distension.

In one case after removing the heart from the chest the right and left ventricles continued to beat in a co-ordinated manner but, for a time, the right atrium and ventricle contracted twice as rapidly as the left atrium and ventricle. In the eighth rabbit all chambers continued to beat normally.

Experiment D.—Before injecting air direct leads from the two ventricles gave traces typical of the right and left ventricles respectively. After inducing asystole of the right ventricle with air both ventricles gave a left ventricular type of trace.

Experiment E.—Cineangiography showed gross distension of the right atrium and ventricle with a complete hold-up of dye by the air in the right atrium (asystole was not induced ; only 10 ml. of air was injected). With each cardiac cycle dye shuttled between the right atrium and the two venae cavae, and none reached the pulmonary artery or its branches. When air reached the lungs they became intensely radiolucent, but unfortunately the kilo-volt increased at this moment so that interpretation of this change was difficult.

Experiment F.—Distension and asystole of the right heart with continued co-ordinated contraction of the small left heart was well shown.

Interpretation of Results.—These experiments prove that air embolism or asphyxia can cause dissociated action of the ventricles and atria of the two sides. Asystole of either the right or left ventricle, with or without asystole of its corresponding atrium, and a two to one right-to-left heart rhythm were seen. Air appeared to produce asystole of the right atrium and ventricle by causing gross over-distension, and the condition could sometimes be relieved by aspirating the air.

Electrocardiographic recordings from the two ventricles showed that when one was rendered asystolic by air embolism the tracing obtained from it was the same as that from the contracting ventricle and merely reflected transmitted activity.

Air embolism was shown by cineangiography to produce complete obstruction to the circulation in the right heart, even when ventricular asystole

does not occur. The right heart becomes grossly overdistended.

The Mechanism of Univentricular Arrest

The mechanism of univentricular arrest is difficult to understand, for the two ventricles have always been considered to form a single functional unit. Heart muscle forms a syncytium, for the individual muscle fibres have no definite cell membranes and they are united to one another by branches. The muscle fibres of the two ventricles are in continuity. Despite this it does appear possible for one ventricle to contract while the other remains quiescent, or for one ventricle to fibrillate whilst its fellow is asystolic. These differences in behaviour of the two ventricles may be initiated by differences in their blood supply, resulting in local or variable metabolic errors or from paralytic overdistension of one or other ventricle. An alternative possibility is that a nervous reflex is initiated by, for instance, acute coronary occlusion, originating either in the coronary arteries themselves or in the ischaemic myocardium. Pressure receptors are known to exist in cardiac muscle (Sharpey-Schafer, Hayter, and Barlow, 1958; Sharpey-Schafer, 1956), and it has been suggested that they may be responsible for initiating vasovagal collapse when severe pressure transients develop in the left ventricle, if it contracts forcibly on an empty cavity. It may be that myocardial nerve endings are stimulated by acute ischaemia due to coronary occlusion and that they could initiate asystole of one ventricle, but there appears to be no experimental evidence to support this supposition.

Isolated arrest or impairment of function of one ventricle may well account for different clinical syndromes. For example, transient arrest or more prolonged impairment of function of the left ventricle would form a ready explanation for the development of acute pulmonary oedema. Such terms as right ventricular failure and left ventricular failure have long been in use without a clear picture of what is actually happening to the ventricles.

Several questions arise from these cases and require an answer: How often does isolated arrest or impaired contraction of one ventricle occur, and what are its causes? Is such a mechanism an important one in the production of clinical syndromes such as acute pulmonary oedema? Is asystole or fibrillation of one ventricle a common cause of fatal cardiac arrest? Can fibrillation of one ventricle occur whilst the other ventricle continues to contract rhythmically? The answers to these questions must await further experience and observations.

Summary

Two patients who died after myocardial infarction are presented in whom asystole of one ventricle was associated with rhythmical contraction of its fellow.

Experimental arrest of one ventricle was produced in a series of rabbits by injecting air into the marginal vein of the ear and is illustrated by a film made from two experiments.

The mechanism of univentricular asystole is discussed.

Several questions arise and require an answer: How often does isolated asystole or impaired contraction of one ventricle occur? How often is it a cause of fatal cardiac arrest? Is such a mechanism responsible for clinical syndromes such as acute pulmonary oedema?

My grateful thanks are due to my colleagues Professor H. Scarborough, Dr. L. Howells, and Mr. Gwyn Morris, who asked me to see their patients, to Dr. Lyn Rees and Dr. Dennis Wakeley for anaesthetic details, to Dr. P. Roche and Dr. R. T. Hughes, who performed the post-mortem examinations, to Dr. R. M. E. Seal and Dr. L. R. West, who collaborated in the experimental work, to Dr. R. T. Hughes for Figs. 3, 4, and 5, and to Mr. R. Marshall, who prepared the illustrations for Figs. 1 and 2.

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