

FATALITY FOLLOWING CARDIAC CATHETERIZATION INJURY

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The widespread use of cardiac catheterization has revealed certain dangers inherent in the procedure when the catheter is advanced beyond the tricuspid valve. Ventricular and other ectopic rhythms are a well recognized risk, but less is known about the effect of damage to the endocardium produced by the catheter in man (Zimdahl, 1951). This paper presents in detail a case that terminated fatally, and the possible mechanism of such trauma is discussed in the light of this and other similar cases that have been reported.

CASE REPORT

The patient was a male clerk, aged 44 years, who was suffering from calcareous mitral stenosis with pulmonary hypertension, and calcareous aortic stenosis. In view of his increasing dyspnoea on exertion and paroxysmal dyspnoea at rest, it was felt that mitral valvotomy was probably urgently indicated. Since the contribution of the aortic valvular lesion to his symptoms could not be fully assessed without hæmodynamic study, cardiac catheterization was advised.

After sedation with sodium amytal and local anaesthesia with procaine, a No. 9 American cardiac catheter was introduced into a left median antecubital vein and passed into the right atrium under fluoroscopic control, whence it was advanced into the main pulmonary trunk without difficulty. The catheter could not be further inserted into the right pulmonary artery and was therefore withdrawn a short distance for re-positioning. During withdrawal the tip slipped back through the pulmonary valve, and passed laterally to the left cardiac border (Fig. 1A). To determine its exact site, the patient was turned into the right oblique position and the catheter was seen to be pointing posteriorly (Fig. 1B). It was further withdrawn and then advanced gently and without difficulty through the pulmonary valve into the right pulmonary artery. The operator was experienced, and there was no undue resistance to the passage of the catheter. These manoeuvres occupied approximately five minutes. Apart from occasional ectopic beats, the pulse was regular throughout. The patient said that he "felt fine," apart from a slight substernal ache. The pulse was of good volume, regular, and the rate had not changed. A few moments later he became pale, sweated, vomited, and complained of faintness; the pulse became slow (regular and at 50 a minute) and impalpable at the wrist. The systolic blood pressure rapidly fell to 40 mm. Hg. Severe hypotension and cardiac pain persisted for 12 hours, the pulse rate increasing to 120 a minute, and a pericardial friction rub being heard on the following day. Consciousness was maintained throughout, and the heart could be felt to be beating strongly.

He remained severely ill for the next 24 hours, but improved somewhat next day, the blood pressure rising to 130/80. A cardiogram immediately after the onset of the pain showed sinus tachycardia and increase in voltage of the T wave in V3, when compared with the previous tracing (Fig. 2A). A further tracing next day (Fig. 2B) revealed further increase in voltage of the T wave and S-T elevation consistent with pericarditis. The serum potassium rose to 8 m-eq. per litre, but returned to normal following glucose and insulin therapy. Distension of the abdomen, reduction in bowel sounds, and a copious vomit suggested acute dilatation of the stomach or paralytic ileus. In spite of treatment for acute circulatory failure, electrolyte imbalance, and ileus, he died suddenly, approximately 40 hours after catheterization. The jugular venous pressure remained normal and the lungs clear during the illness.

Necropsy. (Dr. C. V. Harrison.) The pericardium was flaccid and contained 150 ml. of fluid blood. Both surfaces were covered by a fine film of fibrin.

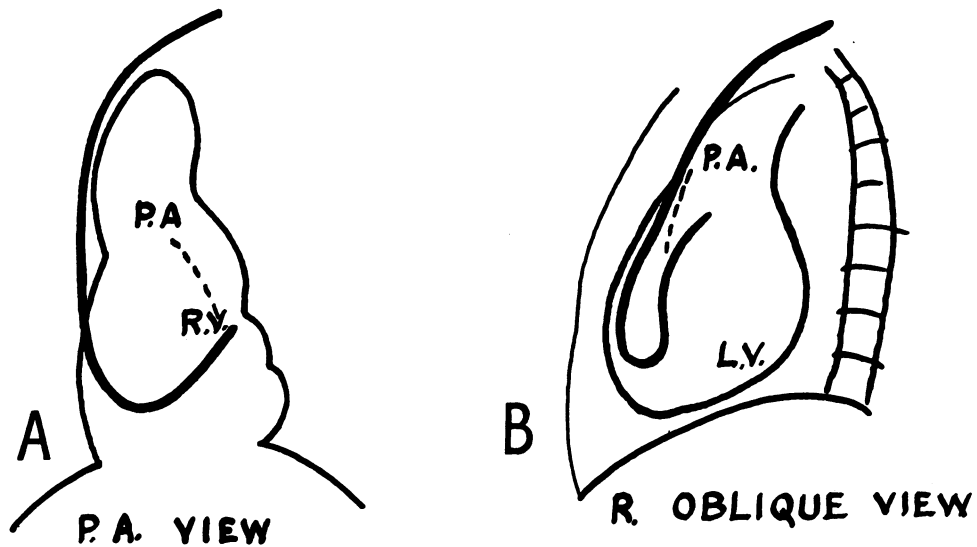


FIG. 1.—Position of catheter in right ventricle after slipping back through pulmonary valve.

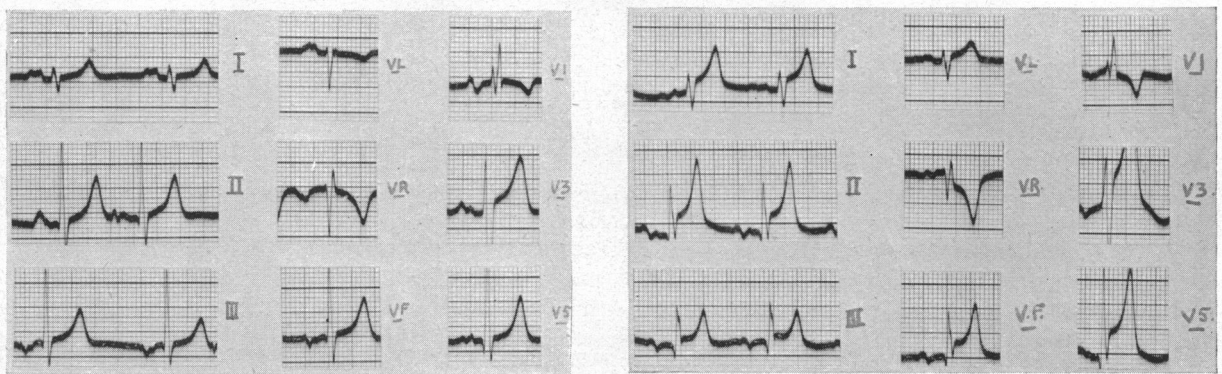


FIG. 2.—Cardiograms. (A) Before cardiac catheterization. Incomplete right bundle branch block and right ventricular hypertrophy. (B) 24 hours later. Increase in voltage of T wave in leads II, V3, and V5. Elevation of S-T segment 3–4 mm. in all leads, except VL, VR, and V1, indicating pericarditis.

Heart. 258 g. The right atrium was normal and the coronary sinus was healthy and free from signs of trauma or thrombus, as were the tricuspid and pulmonary valves. The right ventricle was not dilated but was hypertrophied to a thickness of 6 mm. in the conus region. There was a focus of hæmorrhagic discoloration in the epicardial fat over the right ventricle. This was about 15 mm. diameter and lay 5 mm. to the right of the descending left coronary artery, 35 mm. below the base of the aortic cusps. There was no sign of perforation of the muscle but the area of discoloration lay over a point where the ventricular cavity passed behind one of the larger columnæ carnæ (Fig. 3).

The left atrium was somewhat dilated and was moderately hypertrophied (2–3 mm. thick). The mitral valve showed gross stenosis, the posterior cusp and the two ends of the anterior cusps being calcified, and the chordæ tendinæ being fused.

The left ventricle was not dilated nor hypertrophied (15 mm. thick). The aortic valve was tightly stenosed and calcified.

Other organs. The stomach was dilated and contained 920 ml. of black fluid. There was some similar dilatation of the first and second parts of the duodenum but none in the rest of the intestine. The liver,

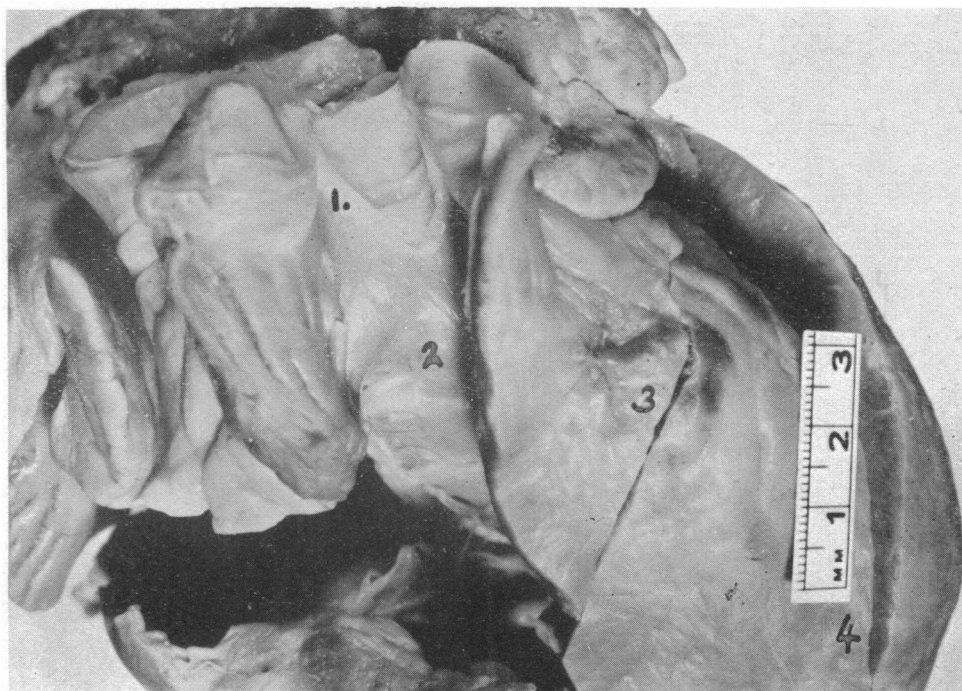


FIG. 3.—Photograph of the heart. (1) Pulmonary valve. (2) Right ventricular outflow tract. (3) Hæmorrhage into the epicardial fat overlying the endocardial lesion which lies behind the large columnæ carneæ. (4) Interventricular septum.

spleen and kidneys did not show any sign of passive congestion. The lungs were congested (right 760 g., left 620 g.).

Histology. Examination of the atrial muscle did not reveal any evidence of active rheumatism, nor was there any sign of ischæmic necrosis of the ventricular muscle supplied by the descending left coronary artery.

Sections taken from the area of suspected trauma all showed a little hæmorrhage into the subepicardial fat but only one showed any myocardial damage. Beneath one of the columnæ carneæ was a small endocardial thrombus (Fig. 4) old enough to have excited a slight infiltration of polymorphonuclears at its edge and of macrophages in the underlying endocardium. Adjacent to this was a small focus of recent muscle necrosis (measuring $350 \times 800 \mu$) just beneath the endocardium. This appeared to be of about the same age as the thrombus. There were also a few torn muscle fibres but these had not excited any reaction and it was concluded that they were torn when examining the heart post mortem.

DISCUSSION

It is possible tentatively to reconstruct the course of events. On withdrawal of the catheter through the pulmonary valve, the tip moved laterally and became trapped behind the thick columnæ carneæ. In this position it may have been held firm while the ventricular wall contracted down upon it with each heart beat. This was sufficient to cause a small area of necrosis and bruising, with oozing of blood into the epicardial fat and into the pericardium. The delay of one to two minutes in withdrawing the catheter from this position, and rotation of the patient into the right oblique position where his heart could have been impaled upon the catheter, may well have contributed further to the production of the lesion. At no time was the catheter advanced with force against resistance.

The tiny myocardial necrosis and the small hæmopericardium would not seem to be sufficient to cause the rapid onset of irreversible circulatory failure, or the severe and prolonged cardiac pain.

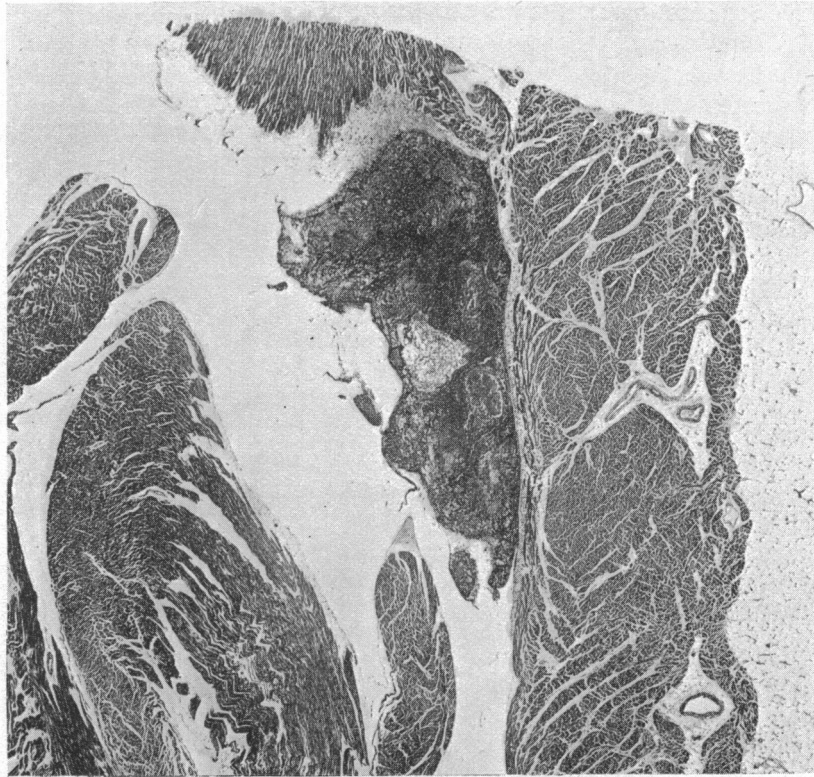


FIG. 4.—Photomicrograph of the area of trauma, showing small endocardial thrombosis, without perforation.

However, the descending branch of the left coronary artery was very close to the lesion and if the trauma had resulted in spasm of this vessel, the clinical picture could be explained by acute coronary insufficiency. Moreover, patients with aortic stenosis are, in any event, liable to sudden attacks of coronary insufficiency which are sometimes fatal. There was no evidence of an arrhythmia or of cardiac tamponade. It seems possible that the patient might have recovered had he not developed acute dilatation of the stomach, which may have been connected with hyperkalæmia, produced by the release of potassium from tissue rendered ischæmic by the prolonged hypotensive state.

Undue stiffness of the catheter may have been a factor in causing the damage, although the instrument employed had been used many times previously without mishap. It is of interest that Ellis *et al.* (1950) found no correlation between the stiffness of the catheter and the severity of endocardial lesions in 16 dogs subjected to cardiac catheterization.

Although endocardial thrombosis occurs not infrequently in dogs after cardiac catheterization (Goodale *et al.*, 1947; Ellis *et al.*, 1950), there are comparatively few reports of the same complication in man, and in all save one the lesions have been confined to the right atrium, superior vena cava, or coronary sinus. The exception was a case of Ebstein's disease described by Holling and Zak (1950) in which a thrombus formed on the septal wall of the left ventricle (which had been catheterized via an atrial-septal defect) and produced fatal embolization ten days after catheterization. Biörck and Krook (1951) reported cardiographic evidence of a subendocardial injury and reduction in right ventricular pressures during catheterization, which they ascribed to myocardial injury by the catheter.

McMichael and Mounsey (1951) reported five non-fatal cases in which a syndrome identical with that described here occurred either during or immediately after cardiac catheterization. In

one of these, the electrocardiogram showed changes suggestive of pericarditis, and in two of lateral ischæmia; while in two there was no change. One patient developed a pericardial rub. All recovered so that the exact mechanism of the condition was not discovered, but it was suggested that deep catheterization of the coronary sinus was responsible. Further evidence in favour of this hypothesis was the position of the catheter, which in each case appeared to have penetrated deep into the coronary sinus, with probable perforation into the pericardial sac in one case. Further support was obtained from the work of Goodale *et al.* (1948) on dogs in which thrombotic occlusion of the great cardiac vein induced by the catheter had resulted in cardiographic changes of S-T segment elevation and T wave inversion. Possible penetration of the pericardium via the coronary sinus by the cardiac catheter in man has also been reported by Stern *et al.* (1952).

In view of the lesion demonstrated here it seems possible that damage to the endocardium of the right ventricle, rather than deep catheterization of the coronary sinus could have been responsible in some of the other cases.

It is difficult to see how this hazard can be avoided with certainty, but it is suggested that the tip of the catheter when in the right ventricle should be kept as far away from the lateral cardiac border as possible and that should it approach close to the edge of the cardiac silhouette, it should be withdrawn instantly. Furthermore, with the catheter in this position, the patient should probably not be rotated into the right oblique position. Finally, it should again be stressed that cardiac catheterization beyond the tricuspid valve is a serious procedure, which involves a significant risk of morbidity and even of mortality. In view of the increasing awareness of the dangers involved, it is felt that such catheterizations are only justified when the information derived can be used for the therapeutic benefit of the individual patient.

SUMMARY

A case is described in which trauma to the endocardium of the outflow tract of the right ventricle, due to cardiac catheterization, resulted in death. Reference is made to similar clinical syndromes that have been separated, and suggestions are made for the prevention of such occurrences in the future.

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