However, as R.S. virus is similar in every respect to the N.D.V. group, except for the ability to agglutinate red cells and elute from them, the question inevitably arises of the value of haemagglutination for taxonomic purposes.

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Medical Memoranda

Spontaneous Rupture of the Right Ventricle

Spontaneous rupture of the heart without localized disease at the rupture site is very rare. Rupture of the left ventricle has not been reported, so far as I am aware. Howell and Piggot (1950) mention three cases of rupture of the right ventricle in an article on the senile myocardium. Haemopericardium following breakdown of a myocardial infarct is very common, though this is almost invariably in the wall of the left ventricle. In these cases there is always coronary artery disease with necrosis of the muscle. It was the complete absence of such a lesion that drew attention to the two cases described below.

CASE 1

A man aged 66, suffering from chronic bronchitis since being "gassed" in the first world war, was operated on for carcinoma of the colon. A partial colectomy was completed without incident, but the man suddenly died two and a half hours after the operation, which lasted two and a half hours.

At post-mortem examination there was half a pint (284 ml.) of fresh blood and clot in the pericardium which had escaped from a small tear on the anterior surface of the right ventricle. The rupture was $\frac{1}{4}$ in. (3 mm.) long, and was situated immediately to the right of the interventricular septum, about midway along the course of the interventricular vessels. The heart was generally enlarged, weighing 450 g.; there was marked dilatation of the right ventricle, the volume of which was about three times normal, the wall being thinned and stretched, measuring 3 mm. in thickness, except at the pulmonary conus, where a thickness of 6 mm. indicated previous hypertrophy before the acute dilatation. The coronary arteries showed minimal atheroma, a few non-occlusive mural plaques being all that was visible. The myocardium was macroscopically free from infarction and fibrosis, and at the rupture site no localized degeneration could be found. There was very little epicardial fat on the heart and none at the actual site of rupture. The other

relevant post-mortem findings were severe chronic bronchitis and marked bullous emphysema.

There was nothing in the operative procedure that might have any direct bearing on the cause of death, and there were no metastases from the carcinoma of the colon.

Histological examination of the heart revealed no infarction or any other degeneration at the rupture site. There was some senile change throughout the myocardium, indicated by the presence of lipofuscin droplets in the cells, but this was no different in degree at the edges of the tear. Special staining did not demonstrate any mucoid degeneration.

CASE 2

A woman of 80, rather senile but with no known diseases, attempted to extinguish a fire in her television set by throwing a bucket of water over it. She then collapsed and died.

At necropsy there were no signs of electrocution, and, apart from generalized arteriosclerosis and some mild bronchitis and emphysema, there was no significant disease present. The pericardium contained about 100 ml. of clotted blood, again having escaped from a small tear, $\frac{1}{4}$ in. (6 mm.) long, on the anterior surface of the right ventricle, close to the interventricular groove and midway between the apex and the atrioventricular sulcus. The heart was of normal size, as were all chambers. There was excess pericardial fat, a layer a few millimetres thick overlying the site of the tear. The actual muscle layer here was 2 mm. thick. The muscle showed no infarction, and, though senile changes were advanced, the edges of the tear were no different from the rest of the heart. The coronary arteries were calcified and generally stenosed, but no thrombus or subintimal haemorrhage could be found on dissection.

Histology again revealed no infarction or mucoid degeneration. Senile pigmentation was evident, but the region of the rupture was in no way remarkable.

COMMENT

Two cases are reported in which the right ventricle had ruptured and given rise to a fatal haemopericardium in the absence of naked-eye or histological evidence of infarction. Both lesions occurred during periods of abnormal stress, the first following an anaesthetic and major operation in a man with a cor pulmonale and the second in a senile woman during a moment of sudden excitement and unusually intense physical activity.

In Case 1 the prolonged anaesthetic may have contributed by causing added respiratory embarrassment which led to acute strain on the already thinned right ventricle. The main factors in Case 2 seem to have been senile friability of the muscle and increase in pericardial fat at the expense of the myocardium. Rupture here was obviously related to sudden effort and presumably raised intraventricular pressure.

The possibility of these lesions being artifacts, due to damage caused when removing the sternum, was considered, but this was excluded as the pericardium was intact and the quantity of blood-clot present could not possibly have occurred after death.

In conclusion, it appears that, though infrequent, rupture of the right ventricle without associated infarction can occur, especially in old persons.

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