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Pathomechanism of Spontaneous Cardiac Rupture Following Infarction

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Summary. Eighty-seven cardiac ruptures were encountered among 870 cases of coronary occlusion. In about 40% the rupture resulted from small infarcts not exceeding 3 to 4 cm. These usually occurred if one of the small coronary artery branches which supply the left ventricle was occluded. Rupture of the posterior wall was due to occlusion of the circumflex branch at a point distant to is origin. In cases of small infarcts, the myocardium remains capable of contraction and the blood pressure is insignificantly reduced so that conditions for a cardiac rupture are favorable.

Key-Words: Heart, rupture of—Infarct of heart—Pathomechanism of heart rupture—Heart pathology.

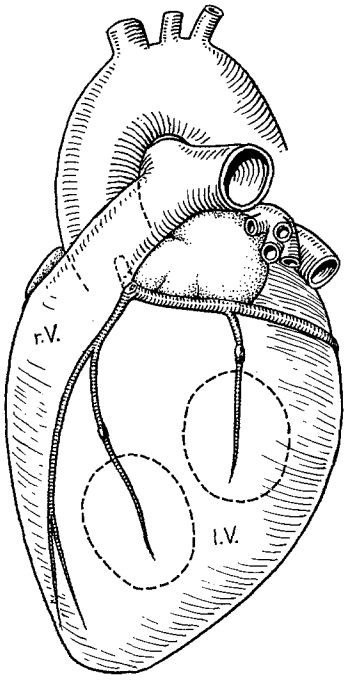
Zusammenfassung. Im Sektionsmaterial, aus 870 Kreuzgefäßthrombosen bestehend, wurden 87 Herzrupturfälle gefunden. In 40% war der Infarkt recht klein, der Durchmesser der Ruptur betrug nicht mehr als 3—4 cm. Ein derartiger Befund wurde dann erwogen, wenn ein kleiner Ast einer Coronararterie verstopft war oder ein Diagonalast, der auf die linke vordere Herzkammerwand hinüberlief. Eine Hinterwandruptur kann im allgemeinen nur dann erfolgen, wenn der linke umlaufende Kranzgefäßzweig an einer Stelle verlegt wurde, die von der Abgrenzstelle weit entfernt lag. Wenn der Infarkt klein ist, behält das Myokard noch seine Kraft, der Blutdruck senkt sich nur minimal; unter diesen Umständen sind die Verhältnisse zur Entstehung einer Herzruptur günstig.

Neither clinical and pathological observations nor even experimental investigations have succeeded in fully elucidating the mechanism of cardiac ruptures. Mönckeberg (1924) claims that ruptures of the heart cannot take place unless the left ventricle retains its muscular strength so that it is able to exert sufficient force when the heart contracts. It has further been observed that infarction combined with hypertension predisposes to cardiac rupture. Also other mechanisms have been implicated in the pathognesis of ruptures, such as physical exertion, psychic tension, digitalis therapy, anticoagulant treatment and the softening effect of leucocytes (Griffith *et al.*, 1961; Lodge-Patch, 1951; Maher *et al.*, 1956; Nesvadba, 1955; Sigler, 1910; Zeman and Rodstein, 1960). Again, other workers do not attribute any pathogenic role to these factors (Crawford and Morris, 1960; London and London, 1965; Mitchell and Parish, 1960). It is affirmed by Lunseth and Ruwald (1956), that cardiac rupture resembles dissecting aneurysma of the aorta and that both are brought about by the same mechanisms. With a view to elucidating the role of mechanical factors, Kohn (1959) carried out a model experiment. He likens the tear of the ventricular wall to the fatigue of metals. The infarcted muscle takes no part in contraction; moving passively and being repeatedly bent, the myocardial fibre is fatigued and undergoes rupture.

Hudson (1965) states in his work on pathology that the mechanism of cardiac ruptures has, so far, not been unequivocally cleared up. It is known that a considerable percentage of cardiac ruptures occurs at the incipient stage of infarctions, one or two days after the appearance of the initial symptoms.

Observations

A large material of sudden and unexplained fatalities is referred to the Institute of Forensic Medicine for the purposes of official autopsy. We have examined 870 cases of coronary occlusion in the course of the last two years and found cardiac rupture in 10 per cent,



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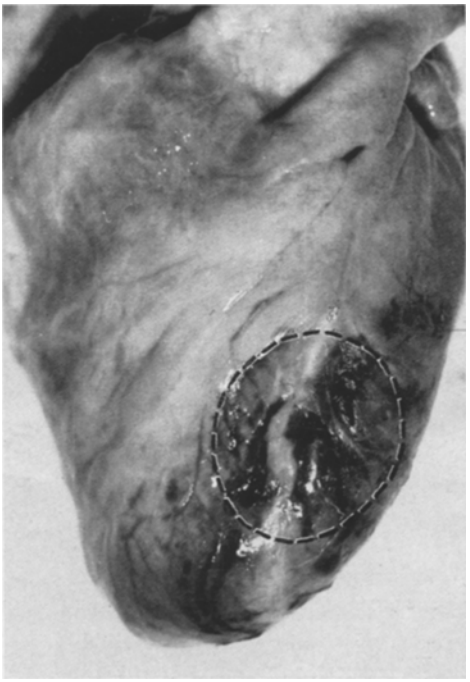

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Fig. 1. Diagram illustrating origin and occurrence of small infarcts

Fig. 2. An autopsy case of cardiac rupture, comp. with Fig. 1.

i.e. in 87 cases, a figure in agreement with literary data. Conditions regarding age and sex were likewise in harmony with the findings of other authors. The site of the thrombi was the anterior descending branch in 33, the circumflex branch in 24 and the vessels which run up the ventricular wall, i.e. the so-called diagonal branches, in 30 cases. No cases of cardiac rupture due to occlusion of the right coronary artery were encountered. In the course of numerous routinely performed dissections it was possible to observe certain phenomens which seemed to constitute a characteristic and well circumscribed entity in the pathomechanism, and to throw light on the origin of many postinfarction ruptures.

Relying on our observations and on literary data, we have come to the conclusion that infarction is followed by rupture when only a small area of the myocardium is necrotized, i.e. when only a small coronary vessel with a limited area of supply is occluded. We were guided in our estimation concerning the extent of infarction by the necrosis that was visible to the naked eye and further by the size of the area supplied by the affected small vessel.

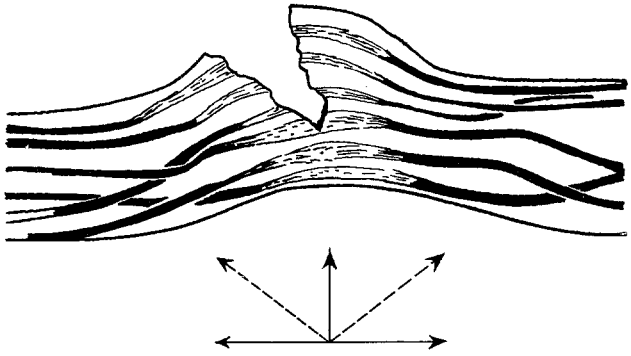

Fig. 3. The combined forces of passive stretching due to contraction of undamaged adjacent myocardium and to the blood pressure from the inside, may increase the vector and cause ruptur of the small infarct

Necroses with diameters not exceeding 3 to 4 cm were regarded as infarctions of small extent. Myocardial rupture takes, thus, place if one of the following branches is obstructed.

a) The anterior wall of the left ventricle is almost invariably ruptured if those minute branches are obstructed which arise from the left circumflex branch at right angles and run to the anterior wall of the left ventricle.

b) The ventricle may undergo rupture next to the septum if a minor branch becomes obstructed at the broom-like arborization of the left descending branch.

c) Rupture of the posterior wall was observed when the obstruction of the left circumflex coronary vessel occurred at a point relatively distant from its origin.

Discussion

Thrombosis of a major branch of the coronary artery damages a large myocardial area. The strength of the muscles diminishes, the blood pressure is suddenly reduced, ventricular contractions are incomplete and weak in such cases.

If, on the other hand, only a small portion of the myocardium is infarcted, the muscles of the left ventricle remain strong and a so-called blowout rupture occurs in the necrotized area. That rupture follows necroses of small extent in a few hours is proved, among others, by the observation that the nacrotized area has in such cases not even time enough to assume the characteristic colour of clay that can otherwise be perceived by the naked eye. Our observations explain also the pathogenic role of hypertension in postinfarction ruptures. Small infarctions do not affect the muscular strength of the ventricle, and no or hardly any post-infarction fall of blood pressure takes place. Therefore, rupture does not occur only because of hypertension, but blood pressure remains high because only a small portion of the myocardium is impaired. Gábor succeeded in preventing ruptures by having reduced blood pressure by means of medication. That the obstruction of a minor branch induces rupture can be explained also anatomically: the thinner the branch, i.e. the more it has the character of an end artery, the more remote will be the possibility of a development of anastomoses and collaterals.

The type of ruptures, discussed in the foregoing, is uniform and well amenable to clinical and pathological interpretation. Ruptures occur, according to our observations, through the described mechanism in about 40 per cent of the cases.

Myocardial blood supply is far from being uniform: it varies according to the division of the coronary arteries, the width of their lumen, and the location of arteriosclerotic strictures. This is the reason why literary data are contradictory, and it is indeed hardly possible to ascribe all kinds of ruptures to one and the same mechanism.

The above observations, made in the course of daily routine work in the dissecting room, might prove useful for clinical practice. The available literature contains no reports on attempts to draw, in respect of myocardial ruptures, prognostic conclusions from the clinical symptoms of infarction. Since small infarcts accompanied by hypertension are, according to our observations, a factor predisposing to cardiac tear, the question arises as to whether it would be advisable to try to elaborate methods for the prevention of cardiac rupture in small infarcts by medication.

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