Editorial: Dissecting the dissecting aneurysm

The histological definition of the normal aorta is an essential prerequisite for the understanding of the changes seen in dissecting aneurysms. Studies of the normal aorta are rare (Rottino, 1939; Carlson et al., 1970), but a very detailed description has recently been given by Schlatmann and Becker (1977a). The latter authors noted an increase in frequency and severity of medial fibrosis and medionecrosis and an increasing frequency of severer grades of elastic fragmentation with age. On the other hand, the frequency of severer grades of cystic medial necrosis decreased with increasing age. Carlson et al. (1970) reported increasing incidence of milder degrees of cystic medial necrosis and of acellular foci (medionecrosis) with increasing age. Neither group of investigators could find any difference in severity or incidence of these abnormalities between hypertensive and normotensive patients when the comparisons were corrected for age. The earliest author (Rottino, 1939) observed loss of muscle cells with a collapsing together of the elastic lamellae and disintegration of elastic fibres, and sometimes mucinous areas. These changes were more frequent and more severe with increasing age and in the hypertensive patients. However, Rottino made no comparison of his hypertensive patients with age-matched non-hypertensives.

What is the relation to dissecting aneurysm of these observations on the normal aorta? It is usually stated that cystic medial necrosis is the most frequent cause of aortic dissecting aneurysm. It was present in 62% of surgically resected thoracic aneurysms (Pomerance et al., 1977). Any lesion seen frequently in dissection will inevitably be regarded as producing the dissection. However, if the same lesion is also encountered frequently in the normal aorta then doubts arise whether these lesions do cause the dissection. In 1976 Hirst and Gore posed the question, 'Is cystic medionecrosis the cause of dissecting aortic aneurysm?' and produced a well-reasoned argument that it is not. Schlatmann and Becker (1977b) analysed the changes seen in aneurysms of the aorta and found no fundamental differences between these and the changes in normal aortas: only some differences of degree of severity of the changes. These authors suggested that the abnormalities they saw were facets of the cycle of stress damage and repair that is going on continuously in the aorta.

A relatively new but increasingly frequent stress on the ascending aorta is cannulation, incision, and clamping during open heart surgery. Fukuda et al. (1976) described three patients who developed dissecting aneurysms of the ascending aorta at, or within four months of, replacement of a stenotic aortic valve. All three showed cystic medial necrosis of the aorta.

Salama and Blesovsky (1970) reported one dissection in an atheromatous plaque and two false aneurysms in 420 cannulations of the ascending aorta. All five of the aortic dissections reported by Litchford et al. (1976) arose in atheromatous plaques and occurred among 5000 aortic cannulations or clamping. Lam et al. (1977) described further cases with normal media or with minimal medial necrosis and intimal atheroma. Muna et al. (1977) reported one atheromatous and one cystic medial necrotic dissection in 847 aortic valve replacements and found an overall incidence of dissection in 0.07% of published accounts of aortic valve replacements. This figure has to be regarded with caution on two counts. Firstly, in some cases (for instance, Derkac et al., 1974) the aorta was aneurysmal before valve replacement and therefore might well have spontaneously dissected had the operation not been performed. Secondly, all series report sudden deaths with no necropsy and a proportion of these could well be due to acute aortic dissection. It is unlikely that these two sources of error exactly counterbalance one another.

Hirst et al. (1958) in their review found dissecting aneurysm in 1.1% of coroners' non-traumatic necropsies and in one in 10,756 hospital admissions (0.0093%). More recently in Nottingham and Sheffield, Talbot (1974) found dissecting aneurysm given as the cause of death in 2.5% of coroners' non-traumatic necropsies. This compared with an incidence of 0.015 to 0.017% of admissions to the largest hospitals in these two cities.

On the figures available there may be an increased risk of developing a dissecting aneurysm after surgical intervention in the ascending aorta, but it can only be a small one.

If a sizable proportion of the population have abnormally structured aortas, and if there is
relatively little risk of dissection after planned mechanical injury during open heart surgery, why are not dissecting aneurysms more common?

The answer to this question may well lie with biochemical or ultrastructural studies to reveal a more consistent and fundamental defect in one or more of the constituents, elastic, collagen, and smooth muscle, of the aortic wall, rather than in more quantitative studies of lesions at the light microscopical level.

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References


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