Pain, blood loss, and death from leaking abdominal aortic aneurysms

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Abstract
The amount of blood lost from the circulation due to leaking abdominal aortic aneurysms was estimated in absolute figures by the difference in weight of the retroperitoneal tissues in 25 cases of sudden death and 25 controls matched for age, sex, height and weight. The proportion of total blood volume lost was calculated using established formulae. Eight subjects lost less than 500 ml or 10% of blood volume and only six lost more than 1500 ml or 25%. These figures suggest that most deaths are not due simply to haemorrhage and that death can occur after only a small loss of blood. There was no difference between subjects and controls in terms of heart weight, degree of coronary artery stenosis, and previous myocardial infarcts, nor was there any correlation of these with amount of blood lost. Endogenous cerebral opiates are known to switch off homeostatic cardiovascular responses to haemorrhage. This provides a possible explanation for circulatory collapse in patients with leaking abdominal aortic aneurysms who characteristically experience severe backache or abdominal pain.

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The volume of blood loss is generally considered to be a primary factor causing death in patients with ruptured abdominal aortic aneurysms. Indeed, for those who undergo surgery, there is a well documented association between volume of blood transfused and mortality.1 Most patients, however, die before, or soon after, arrival at hospital and do not reach the operating table.2 Their mode of death is sudden cardiovascular collapse, but the assumption that this is due to catastrophic blood loss may not be correct. We have been impressed by the variable and often small amount of blood in the retroperitoneal tissues at necropsy and set out to devise a method for its measurement.

Methods
Fifty cases of sudden death due to haemorrhage from a ruptured abdominal aortic aneurysm were encountered over three years. Haemorrhage was confined to the retroperitoneal space in 47 and was both retro- and intraperitoneal in three. All patients died at home or on admission to hospital and none had undergone surgery. The history obtained from relatives was that of severe backache or abdominal pain, which was followed by sudden collapse and death.

The study group comprised 25 unselected cases of retroperitoneal haemorrhage, 20 men and five women, aged 64-91 years (mean 77 years). The range of height was 158-193 cm (mean 176 cm) and of weight 45-91 kg (mean 75 kg). In each case the loose connective tissue and fat in the retroperitoneal space between the diaphragm and the pelvic brim was dissected free of the abdominal wall and separated from the aneurysm, great vessels, and viscera. This haemorrhagic tissue was weighed to the nearest 5 g.

Retroperitoneal tissue was similarly dissected and weighed from 25 control subjects who had died from a variety of other causes. They were matched for age (±5 years), sex, height (±2·5 cm), and weight (±5 kg).

The differences in weight of retroperitoneal tissue between the study and control groups were used to provide an estimate of blood loss in absolute figures. Total blood volume was calculated according to the following formulae:

Blood volume (male) =
0·0236 × H0·725 × W0·425 − 1·229
Blood volume (female) =
0·0248 × H0·755 × W0·425 − 1·054

where H is height in centimetres and W is body weight in kilograms. The percentage of total blood volume lost was then calculated for each subject in the study group.

In addition, the cardiac health of each subject in both study and control groups was assessed by weighing the heart, noting maximum degree of coronary artery stenosis by reference to a chart showing loss of lumen in steps of 10%,4 and by recording previous myocardial infarcts.

Results
The range of estimated absolute blood loss was 70-2680 ml (mean 917 ml) and the percentage of total blood volume lost was 2-45% (mean 19%). The distribution is shown in the figure. Eight subjects lost less than 500 ml or about 10% of total blood volume. "Catastrophic" blood loss estimated as over 1500 ml or 25% of total blood volume occurred in only six.

With regard to cardiac health, the range of heart weights in the study group was 295-620 g (mean 431 g); 15 subjects had severe...
coronary artery stenosis (more than 80%), six had moderate loss of lumen (50–80%) and four mild stenosis (less than 50%). Old myocardial infarcts were present in three of those with severe coronary artery stenosis. No difference could be shown in heart weight or severity of coronary artery stenosis between subjects in the study and control groups, nor could these be related to magnitude of blood loss. The number of observations, however, may have been too small for a valid comparison.

Discussion
It has been established that “rupture” of an abdominal aortic aneurysm is more of a leak and that it remains confined to the retroperitoneal space in most cases. Death usually occurs quite rapidly after the onset of symptoms and only 54% of patients survive more than six hours after the onset of symptoms, and of these, 64% are dead on arrival at hospital. Clearly, transfusion is vital for patients undergoing surgery, but even then the volumes of blood and colloid administered relate more to postoperative cardiac and renal complications than to death on the operating table. As for those who die before reaching hospital, the amount of extravasated blood may not be the only, or even the main, factor causing death in most, as indicated by estimates in this study, and alternative explanations need to be considered.

A limited comparison of heart weight, degree of coronary artery stenosis, and previous myocardial infarct between subjects in the study and control groups showed no difference. In any event, a recent report of 227 “ruptured” and 311 “non-ruptured” abdominal aortic aneurysms after surgical repair showed a comparable 5 year survival in both sets of patients and this was also the same as that of an age, and sex matched “normal” population. Abdominal aortic aneurysms are manifestations of generalised atherosclerosis, but their fate is unrelated to the degree of cardiac or cerebrovascular involvement by the same disease.

If death in this situation is not necessarily due to massive blood loss or a poor cardiac health, then its mechanism may lie in a failure of normal physiological responses. The events that lead up to sudden cardiovascular collapse are dominated by severe pain, experienced in the back or abdomen, and often “tearing” in character. Little work seems to have been done on the nervous pathways that mediate pain in retroperitoneal events. In cats, at least, the receptive fields of afferent fibres are mainly found on large vessels, the peritoneum, and in relation to the lumbar vertebral column. Their function is unclear but they may be involved in “nociception”. Recent studies have shown that endogenous cerebral opiates of δ subtype switch off homeostatic cardiovascular responses when blood loss exceeds 25% of total blood volume, and it has been suggested that selective receptor antagonists such as naltindrole may restore this mechanism. This inhibitory reflex may be activated by severe pain and it may contribute to circulatory collapse at lower levels of blood loss. This being the case, the fate of patients with leaking abdominal aneurysms might be improved by early administration of analogesics or selective opiate antagonists to delay, or help prevent, the onset of irretrievable cardiovascular collapse.