Should Initial Clamping for Abdominal Aortic Aneurysm Repair be Proximal or Distal to Minimise Embolisation?

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Objectives: to determine whether clamping proximally or distally on the infrarenal aorta during abdominal aortic aneurysm (AAA) repair increases the overall embolic potential.

Materials and methods: a sheath was placed in the mid-infrarenal aorta of 16 dogs. In eight animals a cross-clamp was placed at the aortic trifurcation, and in another eight animals it was placed in the immediate subrenal position. Under fluoroscopy blood flow within the infrarenal aorta was evaluated by contrast and particle injections. Greyscale analysis was used to calculate contrast density. Particle distribution was followed fluoroscopically and confirmed pathologically.

Results: fifty-seven ± 24% of injected contrast remained within the aorta with distal clamping while 97 ± 7% did so with proximal clamping (p<0.01). With distal aortic clamping 6.2 ± 1.3 out of 10 injected particles remained within the aorta after 15 seconds and only 0.8 ± 0.8 remained after 5 min. With proximal aortic clamping, all 10 of the particles remained within the aortic lumen for the full 5 minutes (p<0.001).

Conclusions: initial distal clamping minimises distal embolisation, but may result in renal and/or visceral embolisation. Initial proximal clamping prevents proximal embolisation and does not promote distal embolisation. We recommend initial proximal clamping in aortic aneurysm surgery to minimise the overall risk of embolisation.

Key Words: Abdominal aortic aneurysm; Aortic surgery; Repair; Clamping; Embolisation.

Introduction

Complications of abdominal aortic aneurysm (AAA) repair include distal embolisation, which has been reported to occur in anywhere from less than 1% to 25% of cases.1 The presence of distal emboli can be detected by physical examination during the intra- and postoperative periods, and these emboli treated using standard methods. Traditional wisdom holds that during infrarenal AAA repair distal cross-clamp(s) should be applied prior to proximal occlusion to minimise the risk of embolisation to the extremities. This recommendation has been perpetuated throughout the era of modern vascular surgery and has found its place among the major vascular surgery textbooks despite a lack of documented intraoperative observations or controlled studies confirming its veracity.1–6 However, this recommendation does not take into account proximal embolisation to the renal and/or visceral branches which is a less well-defined and less frequently reported complication of AAA repair. These emboli are difficult to diagnose and treat and are associated with a high morbidity.

Using a canine model and digital fluoroscopy we evaluated whether clamping the infrarenal aorta proximally or distally can increase the overall embolic potential of material within its lumen.

Materials and Methods

Preparation

Sixteen mongrel dogs weighing 30–40 kilograms were kept in accordance with “Principles of laboratory animal care” and “Guidelines for the care and use of laboratory animals”.7 All animals were pretreated with atropine HCI (0.04mg/kg) (Abbott Labs, Chicago, IL, U.S.A.) and acepromazine (0.05mg/kg) (Abbott Labs, Chicago, IL, U.S.A.) administered intramuscularly. Intravenous access was established and the dogs were anesthetised with intravenous Nembutal (30mg/kg...
body weight) (Abbott Labs, Chicago, IL, U.S.A.) and maintained on Harvard pump ventilators. Animals were positioned on a fluoro-ready table and through a midline coeliotomy the aorta was dissected from the aortic bifurcation to the renal arteries. The inferior mesenteric artery (IMA) and all lumbar arteries were doubly ligated with 3–0 silk ties and divided. Systemic heparin was administered at 100U/kg body weight. An 8-French introducer sheath was placed using direct aortic puncture midway between the iliac bifurcation and the inferiormost renal artery. The sheath was positioned no more than 5mm into the aorta and was non-occlusive. Proper positioning was confirmed angiographically.

Data acquisition

The study was performed in two parts. Each portion utilised a different method for evaluating blood flow patterns within the infrarenal aorta with either proximal or distal cross-clamps in place. In the first part radio-opaque contrast was injected through the sheath to allow observation of flow patterns. In the second part radio-opaque particles were introduced through the sheath to simulate embolic particles. Proximal clamps were placed in the immediate subrenal position and distal clamps were placed across the iliac trifurcation.

Contrast injections. After applying either a proximal or distal aortic clamp 3ml of 350mg/ml contrast, Iohexol (Nycomed, Inc., Princeton, NJ, U.S.A.) was injected through the sheath over 3 s. The distribution of contrast was followed with continuous digital cine-fluoroscopy for 15 s. After this observation period a 10ml sample of blood was withdrawn from the sheath and set aside to be analysed later for residual contrast density. The clamp was released and free circulation allowed for 5 min. The procedure was then repeated with the clamp in the opposite (distal or proximal) position.

Particle injections. Following completion of the contrast protocol a proximal aortic clamp was reapplied in 8 animals and a distal aortic clamp was reapplied in 8 animals. Ten small (2mm in diameter), radio-opaque, rounded metallic particles were gently released into the aorta via the sheath. The distribution of the particles was followed with digital cine-fluoroscopy continuously for 30s and intermittently for 5 min.

Animals were euthanised using an anaesthetic overdose of Nembutal 120mg/kg IV bolus followed by KCI (Abbott Labs, Chicago, IL, U.S.A.) 80mEq IV bolus.

Data analysis

Contrast injections. Fluoroscopic images of the experimental samples acquired at the time of the experimental manipulations were scanned into a computer and a greyscale analysis of contrast density was performed on these digitised images. A contrast normogram was then created by mixing blood and full-strength contrast in varying concentrations. Fluoroscopic images of these mixtures were taken and scanned into a computer (Fig. 1). A greyscale analysis for contrast density was again performed utilising these digitised images.

There were 256 shades of grey ranging from 0 (black) to 255 (white) defined in this analysis. By comparing the values of our experimental samples with those from the normogram, in which the actual amount of contrast was known, the quantity of residual contrast within the infrarenal aorta was determined.

Particle injections. At the end of the 5-min observation period the number of particles remaining in the infrarenal aorta was counted and the distribution of any embolic particles was noted. The location of the emboli was subsequently confirmed by pathologic examination.

Statistics

Results from both the contrast and particle injections were analysed using a paired, two-tailed, Student’s t-test.

Results

Contrast injections. Greyscale analysis revealed that with a proximal clamp in place 97±7% of the contrast remained within the infrarenal aorta after the 15-second observation period. With distal clamps in place only 57±24% of injected contrast remained within the infrarenal aorta after 15s (p<0.01). Comparative fluoroscopic images taken after 30s are shown in Fig. 2.

Particle injections. With proximal aortic clamping all 10 of the particles remained within the infrarenal aortic lumen for the entire 5-minute observation period in all animals. With distal aortic clamping 6.2±1.3 of the injected particles remained within the infrarenal aorta after the first 15s (p<.01). After five minutes of observation only 0.8±0.8 of the particles remained within the infrarenal aorta (p<0.001) (Table 1). In other words, with distal clamping more than 90% of the particles had been displaced from the infrarenal aorta after 5 min. The majority of these became lodged in either renal or visceral branches (Fig. 3). These findings were confirmed at postmortem examination.
Fig. 1. Mixtures of blood and contrast material in varying concentrations from full strength contrast (far left) to blood only (far right).

Discussion

Thromboembolism is a commonly recognised and important complication of aortic surgery, the prevention of which continues to be a major area of interest to vascular surgeons. Emboli associated with infrarenal AAA repair may present either distally to the extremities or proximally to the renal and/or visceral vessels. Emboli which lodge in the renal, superior mesenteric, or coeliac artery distributions may impact greatly on the overall morbidity of aortic surgery.

Distal embolism following aortic surgery, first reported by Blakemore and Vorhees in 1954, is far more common than proximal embolisation. The incidence of limb ischaemia due to distal embolism during repair of non-ruptured infrarenal aortic aneurysms has recently been estimated to be 3.3%. The actual incidence of clinically significant proximal embolisation is difficult to estimate because some of these emboli may be silent or unrecognised, and because reports on renal and visceral complications following AAA repair tend to include a variety of aetiologies. The incidence of small bowel ischaemia from all causes following aortic surgery has been estimated to be between 0.15% and 0.4%, of which embolic causes represent only a fraction. In a series of combined aortic aneurysm and occlusive cases, Iliopoulos et al. found an incidence of 1.9% for renal microembolisation syndrome, characterised by a non-oliguric increase in serum creatinine from 2 to 6 times greater than preoperative baseline without acid–base or electrolyte disturbances. Renal artery clamping prior to aortic clamping has been suggested as the best chance to prevent this syndrome; however, our results suggest that these potential emboli would then simply go to other vessels, such as the SMA and coeliac arteries.

Distal emboli are relatively easy to diagnose either intraoperatively or postoperatively by physical examination. These emboli can be treated using standard balloon catheter embolectomy methods, or in more complex cases with fluoroscopically assisted techniques. Proximal embolisation on the other hand, although rare, can be devastating. These emboli are more difficult to recognise than distal emboli for two reasons. First, several factors can cause or contribute to both intestinal ischaemia and/or renal dysfunction postoperatively. The pathogenesis of intestinal ischaemia after aortic surgery includes, but is not limited
Fig. 2. Angiograms taken 30 seconds after introduction of contrast. Proximal clamp in place with large amount of residual contrast remaining in infrarenal aorta (A). Distal clamp in place showing absence of residual contrast (B). S = sheath, C = clamp.

Table 1. Number of particles remaining within the infrarenal aorta with either a proximal or a distal clamp in place after 15s and 5 min.

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<tr>
<th>Clamp position</th>
<th>Number of residual particles within infrarenal aorta</th>
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<tr>
<td>Proximal</td>
<td>15 seconds: 10 ± 0, 5 minutes: 10 ± 0</td>
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<tr>
<td>Distal</td>
<td>6.2 ± 1.3, 0.8 ± 0.8</td>
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patients have a decreased functional reserve preoperatively and will not tolerate even minimal embolic insults well. In addition to being more difficult to diagnose, these emboli can be difficult, if not impossible to treat.

It should also be noted that embolisation can occur to any outflow vessel of the aorta. This includes the lumbar arteries or the IMA if patent, although we did not evaluate this possibility in our study. Embolisation to these vessels may contribute to intestinal or spinal cord hypoperfusion.

The probable mechanism of the phenomenon observed in this study is the pressure wave reflected off the occluded iliac artery orifices. This generates turbulent flow and a net movement of contrast or particles in a retrograde fashion. Once the embolic material has moved into the suprarenal aorta it can then embolise to either the renal or visceral vessels. This cycle can continue as long as only distal clamps are in place. This proposed mechanism is not a new concept in aortic aneurysm surgery. In 1957 an autopsy series by Thurlbeck and Castleman documented atheromatous emboli to the kidneys in 17 of 22 (77.3%) patients following aortic surgery for either aneurysmal
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(17 cases) or occlusive disease (5 cases). There were eight cases of severe embolisation (2–8 emboli per slide) consisting of multiple infarcts within the kidneys and occasional visceral emboli. These authors were able to show that the infarcts were related to the emboli by the use of step and serial sections. They postulated that manipulation and clamping of the aorta fragmented atheromatous plaques, which were then churned up in the turbulent flow just proximal to the clamp and embolised to the nearest, i.e. to the renal vessels. They recommend careful handling and gentle, but decisive clamping to minimise this complication.

The authors of the present study do not suggest that initial proximal clamping can prevent all episodes of proximal or distal embolisation associated with aneurysm repair. Dislodging of mural thrombus or plaque with consequent potential embolisation can occur during any step of aneurysm surgery including the initial dissection, application of the clamps, graft placement, unclamping, or closure. Certainly, any embolic material which becomes freed while clamps are not applied will embolise distally, provided that the origin of these emboli is distal to the renal arteries. However, this study focuses on episodes of embolisation which occur following application of the first clamp when further manipulation of, or turbulent flow within, the aneurysm can produce free fragments of mural thrombus in the lumen.

Our study has several implications for standard aneurysm repair. First, there may be some benefit to initial proximal clamping in that this manoeuvre can facilitate dissection around a non-distended aneurysm and around the iliac arteries while not promoting distal embolisation. Second, if it becomes necessary to reposition the proximal clamp prior to opening the aneurysm, our results suggest that one or both of the iliacs should also be unclamped before releasing the proximal clamp to prevent the possibility of renal or visceral embolisation, especially if there has been extensive manipulation of the aneurysm. In this circumstance, protection from proximal embolisation would occur at the expense of a potential distal embolus. Additionally, when removing the proximal clamp after completion of the proximal anastomosis, one of the limbs should be left open in order to flush...
any potential atheroembolic material from the system. This study also has implications for endovascular AAA repair. Indeed, it was observations made during endovascular procedures that led us to perform these experiments. During contrast injection with the iliac arteries occluded, contrast entered the aneurysm, was rapidly redistributed in a retrograde fashion up into the suprarenal aorta, and subsequently was moved out into the renal and visceral arterial branches. While performing endovascular AAA repair mural thrombus or atheromatous material may be loosened by intraluminal manipulations with guidewires and catheters. Material thus freed may embolise during the course of the procedure. Embolisation is likely to occur distally to the extremities, but may occur proximally if free particulate matter is generated within the aneurysm while outflow through the iliac arteries is occluded. Temporary outflow occlusion is necessary to ensure precise placement of the proximal stent, avoiding downward displacement of the stent-graft during deployment. It is, therefore, especially important to ensure that disruption of plaque and thrombus does not occur during catheter-guidewire manipulations while both common iliac arteries are occluded.

The results of this study support the conclusion that initial clamping in aortic aneurysm surgery should be proximal rather than distal. While initial distal clamping certainly minimises distal embolisation, it greatly enhances the risk of proximal embolisation. Initial proximal clamping, however, minimises proximal embolisation and does not promote distal embolisation.

Acknowledgements

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References

7 “Principles of laboratory animal care” and “Guidelines for the care and use of laboratory animals”. NIH Publication No. 80-23, revised 1985.

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