The influence of collateral vascularisation on haemodynamic performance during abdominal aortic surgery

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The extent of periaortic collateral vascularisation has been proposed as a possible mechanism of an altered haemodynamic response to infra-renal aortic cross-clamp in patients undergoing by-pass grafting for aorto-iliac occlusive disease (AOD) compared with patients undergoing abdominal aortic aneurysm (AAA) resection. The haemodynamic responses following clamping, during the clamp time and following clamp release were studied in 18 patients undergoing AAA resection and 12 patients undergoing bypass grafting for AOD. The role of preoperative aortography in predicting cardiovascular performance during aortic vascular surgery was assessed. During the cross-clamp period LVSWI and CI decreased while SVR increased in the AAA group while the AOD group showed an improved CI, stable LVSWI and reduced SVR, which correlated with the extent of periaortic vascularisation on preoperative aortography. Chronic collateral circulation associated with AOD may permit continuous lower extremity perfusion during aortic cross-clamp. The extent of periaortic collateralisation may influence the choice of monitoring techniques and anaesthetic management.

Key Words:
ANAESTHESIA: cardiovascular; SURGERY: abdominal aortic; cross-clamp; MONITORING: stroke volume, after-load.

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Significant changes in cardiac index and systemic vascular resistance have been reported following infrarenal aortic cross-clamping and release.[1–4] Studies comparing the haemodynamic responses to aortic cross-clamping and release in patients undergoing abdominal aortic aneurysm (AAA) resection and bypass grafting for aorto-occlusive disease (AOD) have noted either no difference between the two patient populations[5–6] or less marked haemodynamic changes in the AOD group.[7] The extent of collateral circulatory changes associated with aorto-iliac occlusive disease has been proposed as the mechanism for a different haemodynamic response to aortic cross-clamp compared with patients undergoing abdominal aortic aneurysm resection. Johnson *et al.*[8] postulate that the extent of collateral vascularisation seen on preoperative aortography may selectively predict the magnitude of changes in stroke volume and systemic vascular resistance that will follow cross-clamp application.

The objectives of this study were to compare the haemodynamic changes following cross-clamp application, during the cross-clamping period and after cross-clamp release in patients with AOD and AAA; to correlate the degree of preoperative collateral vascularisation, as seen on aortography, with the haemodynamic changes following aortic cross-clamp and to assess the role of preoperative aortography as a predictor of cardiovascular performance during aortic vascular surgery.

Methods

Following ethics committee approval and informed consent, 30 patients, ASA physical status II–III (23 male, 7 female), were studied prospectively. Eighteen patients were scheduled for AAA resection while 12 patients underwent bypass grafting for AOD. Preoperative cardiac status was assessed in each patient by standard physical examination, electrocardiogram, chest roentgenogram, Goldman-Caldera Cardiac Risk Index[9] (CRI) and resting left ventricular ejection fraction determination with radionuclide ventriculography and 2D echocardiography. In

TABLE I Periaortic Vascular collateralisation score (vessels visible on preoperative angiography above infrarenal cross-clamp site)

0	None
1	Lumbar
2	Lumbar and superior mesenteric/coeliac
3	Lumbar, superior mesenteric/coeliac, lower intercostal
4	Lumbar, superior mesenteric/coeliac, lower intercostal and internal mammary

the AAA group the aortic aneurysm dimensions were determined by abdominal ultrasound. AAA patients with suprarenal or iliac involvement underwent aortography and were excluded from this study. Patients with AOD underwent preoperative aortography. The extent of periaortic collateral vascularisation was assessed using a scoring system, outlined in Table I, by a radiologist who was unaware of the study methods.

A standardised anaesthetic technique was employed. Premedication with diazepam 0.15 $mg \cdot kg^{-1}$ PO was given minutes before surgery. Radial and pulmonary artery catheters were inserted under local anaesthesia. Fentanyl 3–6 $\mu g \cdot kg^{-1}$ was administered IV prior to induction of anaesthesia with thiopentone 2–3 $mg \cdot kg^{-1}$. Pancuronium 0.1 $mg \cdot kg^{-1}$ IV was given to facilitate tracheal intubation and controlled normocapnic ventilation was commenced with 67 per cent N_2O/33 per cent O_2 and 0.5–1 per cent isoflurane. Standard CM5 ECG monitoring to detect intraoperative dysrhythmias and myocardial ischaemia was employed. Pulmonary capillary wedge pressure (PCWP) was maintained between 10 and 15 mmHg using crystalloid infusions and blood replacement was commenced when loss exceeded 10 per cent of the estimated blood volume. Systolic blood pressure elevations in excess of 20 per cent of base line were controlled with a nitroglycerin infusion.

Measured and derived indices of cardiac function were obtained three minutes before and following induction of anaesthesia, three minutes before and after aortic cross-clamping, and three minutes before and after aortic cross-clamp release. Measured haemodynamic data included heart rate (HR), systolic and diastolic arterial pressure, pulmonary artery systolic and diastolic pressures, pulmonary capillary wedge pressure (PCWP), central venous pressure (CVP) and mean cardiac output from triplicate 10 ml iced saline injections. Derived indices of cardiac function included cardiac index (CI), stroke volume index (SVI), mean arterial pressure (MAP), systemic vascular resistance (SVR), pulmonary vascular resistance (PVR) and left ventricular stroke work index (LVSWI). The data were expressed as mean ± standard error of the mean (SEM). Statistical analysis between and within the two groups included unpaired Student's t test, chi-square, Spearman rank correlation coefficient testing and repeated multivariate analysis of variance (MANOVA), where appropriate. $P < 0.05$ was considered significant.

TABLE II Patient characteristics (mean ± SEM)

	AAA	*AOD*
Age (years)	71 ± 1.65	60.3 ± 2.2 *p < 0.05
n	18	12
Body surface area m	1.73 ± 0.04	1.74 ± 0.06 NS
Sex (M/F)	12/6	11/1
Previous myocardial infarction	5	4 NS
Angina pectoris	3	4 NS
Hypertension	10	4 NS
Resting LVEF (%)	51 ± 2.3	53 ± 4.3 NS
Aortic cross-clamp time (min)	51 ± 4.1	68 ± 6.2 *p < 0.05
Volume infused (litre)	4.5 ± 1.2	3.0 ± 0.7 *p < 0.05

Results

Significant co-existing cardiovascular and peripheral vascular disease, as determined by history, physical examination and preoperative electrocardiogram, were present in both patient groups (Table II). No patient had a history of myocardial infarction in the six months before surgery and no signs of heart failure were apparent on preoperative evaluation. All patients were in Goldman's cardiac risk index (CRI) Group I and II on admission (less than 12 points) and no difference was found between the two groups studied. Patients presenting with AOD were significantly younger (60.3 ± 2.0 years) compared with patients presenting with AAA (71 ± 1.65 years). A male/female ratio of 11/1 was noted in the AOD group compared with a 12/6 ratio in the AAA group. While the incidence of pre-existing hypertension was higher in AAA group, this failed to achieve statistical significance. The resting LVEF, as determined by radionuclide ventriculography and 2D echocardiography, was similar in the two patient populations. The AOD group required less perioperative fluids and had a longer aortic cross-clamp time compared with the AAA group. Induction of anaesthesia resulted in significant reductions in CI, LVSWI and unchanged SVR in the two patient populations (Figures 1–3). Aortic cross-clamping was associated with similar reductions in CI and elevation of SVR in both groups studied. During the cross-clamp period, LVSWI and CI both decreased while SVR increased in the AAA group, while the AOD group demonstrated an improved CI, stable LVSWI and a reduced SVR. Following aortic cross-clamp release CI and LVSWI improved while SVR decreased in the AAA group. No significant changes in CI, LVSWI and SVR were noted in the AOD group.

The correlation between the extent of periaortic collat-

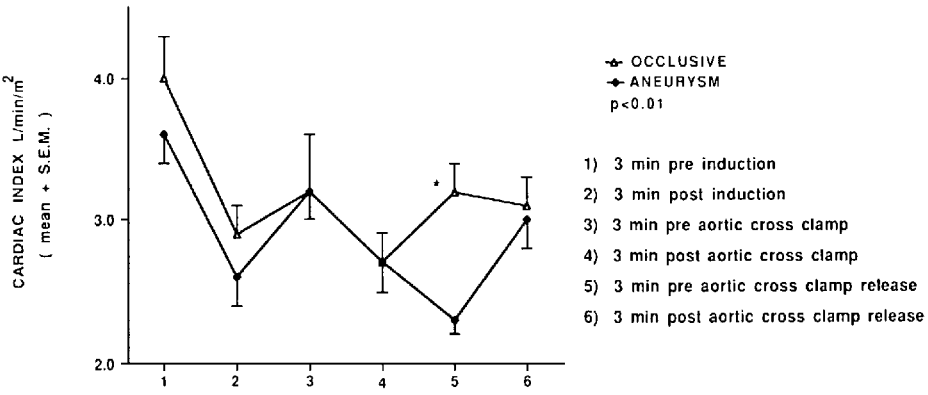

FIGURE 1 Cardiac index 3 minutes pre- and post-induction, 3 minutes pre- and post-aortic cross-clamp and 3 minutes pre- and post-aortic cross-clamp release.

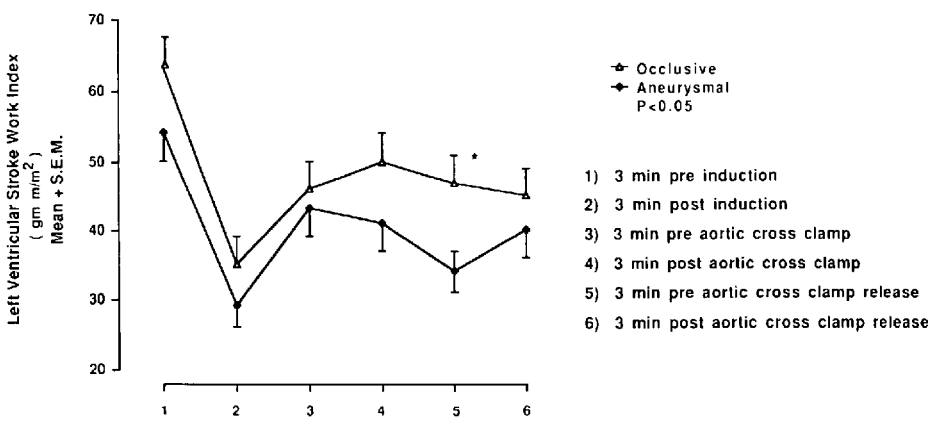

FIGURE 2 Left ventricular stroke work index 3 minutes pre- and post-induction, 3 minutes pre- and post-aortic cross-clamp and 3 minutes pre- and post-aortic clamp release.

eral vascularisation and changes in SVR during the aortic cross-clamp period is shown in Figure 4. Identical results were obtained when systemic vascular resistance changes were expressed as a percentage and as absolute values ($R_S = -0.86, p < 0.01$). The extent of periaortic vascular collateralisation related to the percentage change in SVR and CI during the aortic cross-clamp period. No correlation was noted between periaortic collateral vascularisation and changes in SVR and CI on aortic cross-clamp release. Nitroglycerin infusion, $0.25-1\ \mu g \cdot kg^{-1}$, was initiated to attenuate hypertensive responses or ECG evidence of myocardial ischaemia in four AAA patients. No such therapy was required in the AOD group. All 12 patients in the AOD group survived surgery. Two out of 18 patients in the AAA group died within the first 30 days following surgery; one death was associated with postoperative myocardial infarction and the other followed massive pulmonary embolism.

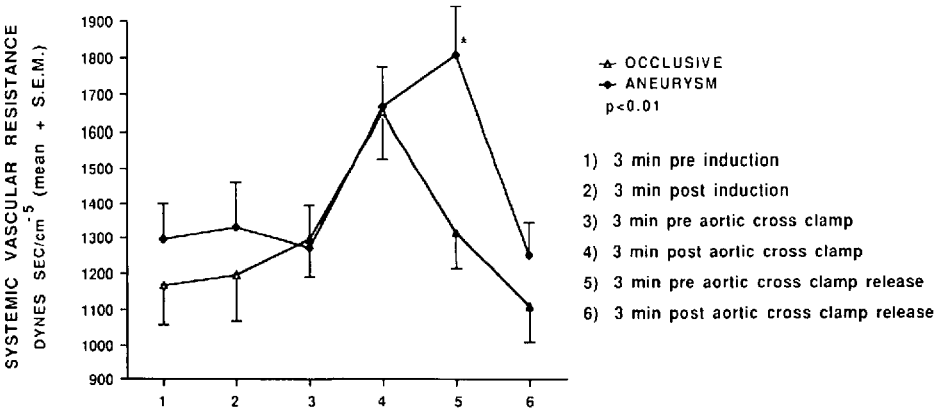

FIGURE 3 Systemic vascular resistance 3 minutes pre- and post-induction, 3 minutes pre- and post-aortic cross-clamp and 3 minutes pre- and post-aortic cross-clamp release.

Discussion

This study suggests that, while the changes in cardiac index and systemic vascular resistance following aortic cross-clamping may be similar in AAA and AOD patients, the haemodynamic changes during the cross-clamp period may differ. The extent of collateral circulatory changes associated with aorto-occlusive disease influenced the magnitude of SVR and CI changes during the cross-clamp period. The collateral circulatory changes associated with AOD may permit continuous lower extremity perfusion during the cross-clamp period. In contrast, the total or near total ischaemia in the lower limbs and pelvis following aortic cross-clamping in patients with no collateral circulation will produce increased anaerobic metabolism with reduction in total body oxygen consumption and carbon dioxide production.[10]

Other factors besides the extent of collateral circulatory changes may be responsible for the observed differences in cardiac performance during the aortic cross-clamp period. The AOD patients were younger, maintained stable left ventricular function, had a longer aortic cross-clamp time and required less intravenous fluid administration to maintain optimum left heart filling pressures compared with AAA patients.

The haemodynamic consequences of aortic cross-clamping will be influenced by the patient's preoperative coronary circulation and myocardial function, the intravascular volume, the site of cross-clamp application, the anaesthetic agents and techniques employed and the surgical pathology.[11] The anticipated consequences of an abrupt aortic cross-clamping include an increased impedance to ventricular ejection (after-load), a decreased venous return (pre-load), with decreased velocity and shortening of myocardial muscle fibres.[12]

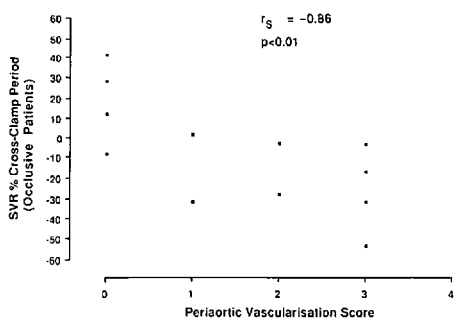

FIGURE 4 Vascularisation score – correlation with systemic vascular resistance changes during aortic cross-clamp period.

The haemodynamic consequences of aortic cross-clamping have been evaluated extensively in experimental and clinical studies. Clinical reports have demonstrated consistently a 15–35 per cent reduction in stroke volume and cardiac index, coupled with an increased arterial blood pressure and up to 40 per cent increase in systemic vascular resistance.[1–4] The effect of abdominal aortic cross-clamping on venous return is a composite of complimentary and opposing factors – diminished venous return due to exclusion of blood flow to the pelvis and

lower extremities, a possible redistribution of blood flow from the inferior vena cava to superior vena cava,[13] an increase in left ventricular end systolic and end diastolic volumes[14] and the underlying myocardial reserve or compliance.

The patient's preoperative cardiac status and myocardial reserve may exert a profound influence on the haemodynamic response to aortic cross-clamping. Attia *et al.*[15] noted different haemodynamic responses to aortic cross-clamping. Patients clinically free of underlying atherosclerotic heart disease demonstrated lower pulmonary artery, pulmonary capillary wedge and central venous pressure when the aorta was clamped while patients with clinical evidence of atherosclerotic heart disease responded to cross-clamping with increased right-and left-sided filling pressures. Three of ten patients in this series, with severe coronary artery disease, responded to cross-clamping with pulmonary capillary wedge pressure increases of 7 mmHg or greater and ECG evidence of myocardial ischaemia. Carroll[17] reported 2 of 14 patients undergoing aortic vascular surgery responded to cross-clamping with significant elevation of pulmonary artery occlusion pressure. In one of these cases ECG evidence of myocardial ischaemia appeared. Gooding *et al.*[17] confirmed that in addition to increasing pulmonary capillary wedge pressure on cross-clamping, patients with coronary artery disease sustained a greater reduction in cardiac index compared with patients judged free of coronary artery disease. The different responses to cross-clamping of patients with and without coronary artery disease suggests that patients with impaired myocardial contractility or increased left ventricular end-diastolic volumes may be unable to mobilize further the Frank Starling mechanism and may proceed to develop myocardial ischaemia and left ventricular failure following abrupt increases in after-load.

The increased systemic vascular resistance in the AAA patients in this study was associated with significant reductions in LVSWI and CI. In this group, the administration of low-dose nitroglycerin ($0.25\ \mu g \cdot kg^{-1}$ per minute) may be effective in maintaining myocardial contractility during the aortic cross-clamp period.[18] In contrast, AOD patients exhibiting a decreased systemic vascular resistance and increased cardiac index during the period of aortic cross-clamping may not require further afterload reduction.

During infrarenal cross-clamping, the lower extremities and pelvis undergo ischaemic vasodilatation and vasomotor paralysis. Lactic acid and products of anaerobic metabolism accumulate in ischaemic tissue during cross-clamping.[19] Declamping hypotension may result from hypovolaemia (pooling of blood in capacitance vessels), and the release of vasoactive metabolites and myocardial depressant factors.[20] Diastolic compliance (the relation between pulmonary capillary wedge pressure and end–diastolic index) may decrease after declamping, indicating myocardial dysfunction.[6]

In this study declamping hypotension was avoided and a stable cardiac output was achieved by volume loading to a high pulmonary capillary wedge pressure prior to aortic cross-clamp release. Cardiac output has been known to decrease following declamping in animal experiments and in patients undergoing aortic aneurysm resection.[21,22] In contrast, well-hydrated patients with AOD may increase their cardiac output following declamping.[23] Reactive hyperaemia developed gradually over several hours, and not immediately at the time of declamping, following revascularisation in patients with AOD.[24] In this study declamping was associated with a significant reduction in SVR and increase in cardiac index in AAA patients while no significant changes were noted in AOD patients.

Johnson *et al.*'s[8] recent evaluation of the influence of periaortic collateral vascularisation on haemodynamic changes immediately following cross-clamping and this study assessing the influence of the same factor on the haemodynamic changes during the cross-clamp period and declamping both raise important considerations for patient management. Well-hydrated patients with angiographic evidence of good periaortic vascularisation presenting with AOD will maintain a stable haemodynamic performance during the aortic cross-clamping period and following declamping. If no symptomatic heart disease is detected on preoperative investigation, right atrial or central venous monitoring may be substituted for pulmonary capillary wedge pressure monitoring in these patients because changes in CVP may accurately predict the direction and magnitude of changes in PCWP.[25] Patients with angiographic evidence of poor collateralisation, presenting with AOD, will develop significant haemodynamic changes during the cross-clamp period just like patients with AAA. In such cases, especially if symptomatic coronary artery disease or evidence of impaired left ventricular function is detected on preoperative investigation, pulmonary artery catheterisation should be undertaken to facilitate optimal intravenous fluid administration, to detect myocardial ischaemia promptly and to assess the haemodynamic changes associated with aortic cross-clamping and release. AAA and AOD patients with poor periaortic collateralisation both sustain significant increased SVR and decreased cardiac index and LVSWI during aortic cross-clamping. Prophylactic nitroglycerin infusion commencing prior to cross-clamp application should be administered to reduce SVR and sustain myocardial contractility.

The coronary arteriolar vasodilating properties of iso-

flurane are now well documented in experimental and clinical studies.[26,27] The clinical implications of isoflurane-induced coronary arteriolar dilatation in patients with coronary artery disease remain controversial.[28] The adverse coronary steal effects associated with one per cent end–tidal isoflurane administration have been highlighted by Reiz *et al.*[29] in patients with coronary artery disease presenting for aortic vascular surgery. Ten of 21 patients studied developed ECG and metabolic changes associated with myocardial ischaemia. In contrast, the same authors noted decreased cardiac index, systemic vascular resistance, coronary sinus blood flow, myocardial oxygen consumption and no myocardial lactate production in aortic vascular surgery patients with coronary artery disease and evidence of heart failure, using one per cent end–tidal halothane in 30 per cent oxygen.[30] Based on current information, nitrous oxide[31] and isoflurane should, if possible, be avoided in the aortic vascular surgery population because of the risk of insidious global or regional myocardial ischaemia.

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Résumé

L'importance de la vascularisation collatérale périaortique a été proposée comme étant un mécanisme pouvant altérer les réponses hémodynamiques au clampage aortique infra-rénal chez les patients devant subir une greffe aorto-iliaque comparativement aux patients devant subir une résection de l'anévrisme de l'aorte abdominale. Les réponses hémodynamiques après clampage, durant le clampage ainsi qu'après déclampage ont été étudiées chez 18 patients subissant une résection d'anévrisme de l'aorte abdominale et 12 patients subissant un pontage aorto-iliaque. Le rôle de l'aortographie préopératoire dans la prédiction de la performance cardiovasculaire durant la chirurgie fut évalué. Lors du clampage aortique, le travail d'éjection indexé du ventricule gauche ainsi que l'index cardiaque ont diminué alors que la résistance vasculaire systémique a augmenté chez les patients ayant subi une résection de l'anévrisme de l'aorte abdominale. Les patients ayant subi une greffe aorto-iliaque pour maladie athérosclérotique obstructive ont démontré une amélioration de l'index cardiaque, un travail d'éjection indexé du ventricule gauche stable et une diminution de la résistance vasculaire systémique qui était reliée à l'importance de la vascularisation péri-aortique telle que démontrée par l'aortographie préopératoire. L'établissement d'une circulation collatérale chronique en présence de maladie athérosclérotique obstructive de l'aorte peut permettre une perfusion continue des extrémités lors du clampage aortique. L'étendue de la collatéralisation péri-aortique peut influencer le choix des techniques de surveillance ainsi que la conduite anesthésique.