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Rise in Pulmonary Arterial Pressure following Release of Aortic Crossclamp in Abdominal Aortic Aneurysmectomy

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Many reports[1–6] have described the hemodynamic effects of aortic cross-clamping (XC) and declamping (XD) during surgery for abdominal aortic aneurysms (AAA). Although systemic arterial hypotension following XD is well established, reports of changes in pulmonary arterial pressure are not well documented. During routine monitoring of patients during AAA surgery, we noted a transient rise in pulmonary arterial pressure (PAP) following XD. We, therefore, prospectively evaluated changes in pulmonary arterial pressure after XD in patients undergoing AAA surgery.

MATERIALS AND METHODS

Eighteen consecutive patients (NYHA Class I or II) undergoing elective surgical treatment of infrarenal AAA were selected for study after institutional approval and informed consent were obtained. Five patients were excluded from the results (*vide infra*). The

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average age of the remaining 13 patients was 60.7 yr (range 59–70 yr), height 163 cm (range 157–172 cm), and weight 55 kg (range 49–68 kg); 11 patients were men. The patients had no history of angina pectoris, myocardial infarction, valvular heart disease, or arterial hypertension (diastolic > 105 mmHg). All had regular sinus rhythm and no significant abnormalities in preoperative ECG, except for eight patients who showed non-specific ST-T changes. Preoperative pulmonary function tests were within the normal range, except for three patients whose 1-s forced expired volume was less than 70%. Chest roentgenograms showed no significant abnormalities.

The patients were premedicated with intramuscular atropine 0.5 mg and hydroxyzine 50 mg 30 min prior to anesthesia. Anesthesia was induced with thiamylal 5 mg/kg iv and tracheal intubation was facilitated by administration of succinylcholine 1 mg/kg iv. An arterial cannula was inserted into the radial artery to measure systemic arterial blood pressure. A 7F thermodilution flow-directed pulmonary artery catheter (American Edwards Laboratory, Santa Ana, CA) was introduced *via* the right internal jugular vein for measurements of central venous blood pressure (CVP), PAP, pulmonary arterial wedge pressure (PAWP), and cardiac output in nine patients. In four patients, mixed venous oxygen saturation was continuously measured with an oximeter incorporated into a thermodilution flow-directed pulmonary artery catheter (American Edwards Laboratory, Santa Ana, CA). Expired carbon dioxide concentration at the proximal end of the endotracheal tube was monitored with an infrared carbon dioxide analyzer (Datex CD-300, Helsinki, Finland) in the same four patients. Nitrous oxide with 40–50% oxygen and enflurane (1–2%) were used for maintenance of anesthesia, supplemented with pancuronium bromide for muscle relaxation. Intraoperative maintenance fluid consisted of lactated Ringer's solution given at a rate of 300–350 ml/h. Trimethaphan, 15–30 $\mu g \cdot kg^{-1} \cdot min^{-1}$, was administered just prior to XC to alleviate an abrupt increase in afterload. This dose was tailored to accommodate systemic arterial pressure fluctuations, and the duration of infusion was approximately 2 min, although it varied among cases. Blood and plasma transfusions were titrated to replace blood loss during reconstruction of the abdominal aorta with a bifurcation graft. Just prior to aortic declamping, 150 ml of lactated Ringer's solution was infused in addition to the maintenance fluid volume. Aortic declamping was performed in a stepwise manner over a duration of 30 s, starting from the right iliac artery to the left iliac artery. The anesthetic concentrations were kept constant in each case during the study. Five cases were excluded from the study because they needed more than 200 ml of blood and/or plasma transfusion within 10 min immediately after XD to replace blood loss. The mean aortic cross-clamp time was 42.1 min (range 32–79 min). The mean blood loss during surgery was 536 ml (range 230–880 ml).

Hemodynamic measurements were performed at the following intervals: prior to XC, 30 s post-XC, 5 min post-XC, prior to XD, 30 s post-XD, 2 min post-XD, 5 min post-XD, and 10 min post-XD. Measurements prior to XC were performed before the start of trimethaphan infusion, and those prior to XD were performed before the additional infusion of 150 ml of lactated Ringer's solution. In all patients, heart rate (HR); systolic, diastolic, and mean arterial blood pressures (MAP); systolic, diastolic, and mean pulmonary arterial blood pressures (MPAP); and CVP were continuously monitored using a Hewlett-Packard® system 78342A or 78171A (McMinnville, OR). Thermodilution cardiac outputs were measured in duplicate with a cardiac output computer (9520A, American Edwards Laboratory, Santa Ana, CA). Hemodynamic indices were calculated from measured variables using standard formulae.[7]

Data were expressed as mean ± SEM. Statistical analysis included analysis of variance for repeated measurements on each parameter. Obtained data were compared with the control (pre-cross-clamping) value using Dunnet's method. P values less than 0.05 were considered significant.

Results

MAP decreased significantly to 80 ± 4 mmHg at 30 s post-XD, compared with the pre-XC values of 97 ± 5 mmHg, while no significant increase was observed after XC. MPAP increased significantly to 16.4 ± 1.2 and 17.5 ± 0.6 mmHg at 2 and 5 min post-XD, respectively, compared with pre-XC values of 12.4 ± 1.0 mmHg. PAWP, CVP, and HR did not show a significant change (fig. 1). Cardiac index (CI) decreased significantly from 2.58 ± 0.08 to 1.77 ± 0.12 l/min/m at 30 s post-XC, and SVRI increased significantly from 2835 ± 190 to 5114 ± 1687 dynes · sec · m^2/cm^5 at 30 s post-XC and to 4206 ± 370 dynes · sec · m^2/cm^5 at 2 min post-XC. However, PVRI did not change significantly, although it tended to increase at 2 and 5 min post-XD (fig. 2). LVSWI did not show a significant change, while RVSWI increased significantly to 4.8 ± 0.7 and 5.2 ± 0.8 gm/m^2 at 2 and 5 min post-XD, respectively, compared with pre-XC values of 2.7 ± 0.4 gm/m (fig. 3).

Mixed venous oxygen saturation measured continuously in four cases decreased rapidly to 53–71% immediately after XD. In these cases, increases in carbon dioxide concentration (6.6–7.5%) in end-expiratory gas

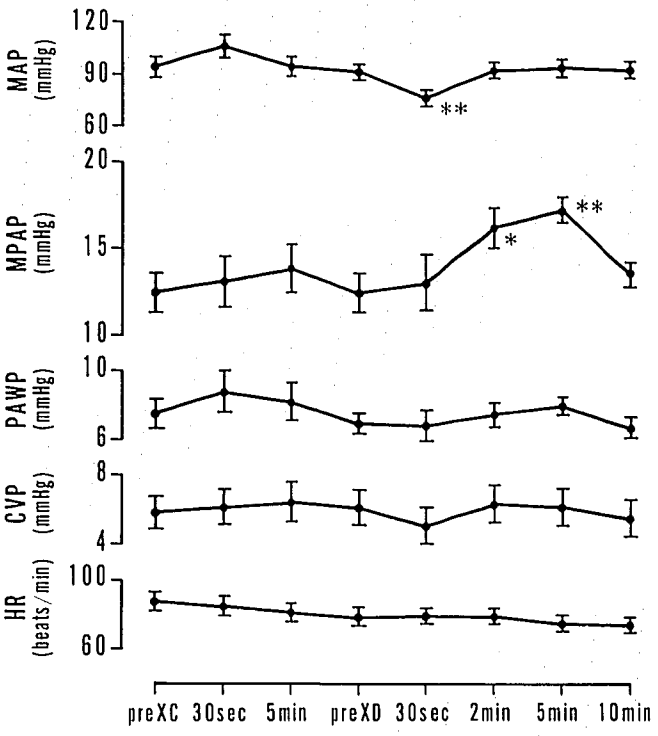

FIG. 1. Hemodynamic measurements in 13 patients. MAP = mean arterial pressure; MPAP = mean pulmonary arterial pressure; PAWP = pulmonary arterial wedge pressure; CVP = central venous pressure; HR = heart rate; preXC = pre-cross-clamping time; preXD = pre-declamping time. Values are means ± SEM, n = 13. Statistical analysis by analysis of variance and Dunnet's multiple comparison test. * $P < 0.05$, ** $0.01 < P < 0.05$, compared with pre-XC values.

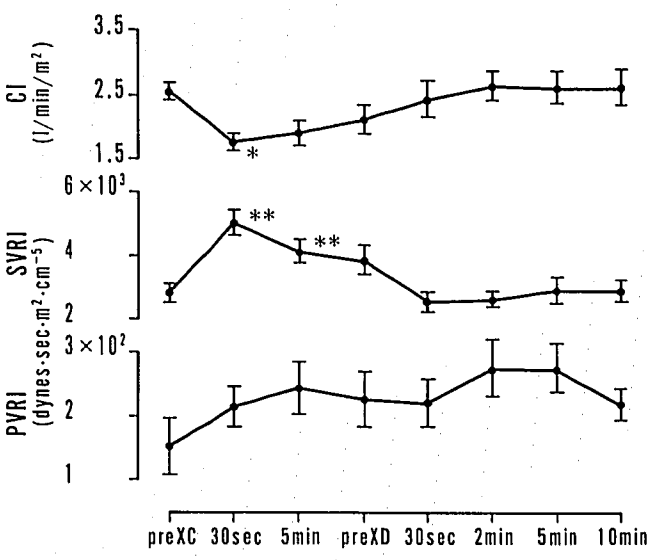

FIG. 2. Hemodynamic measurements in 13 patients. CI = cardiac index; SVRI and PVRI = systemic and pulmonary vascular resistance indices; preXC = pre-cross-clamping time; preXD = pre-declamping time. Values are means ± SEM, n = 13. Statistical analysis by analysis of variance and Dunnet's multiple comparison test. * $P < 0.05$, ** $0.01 < P < 0.05$, compared with pre-XC values.

samples were also observed following XD. Recordings of PAP elevation, decreased mixed venous oxygen saturation, and increased end-expiratory carbon dioxide concentration after XD are shown in figure 4.

DISCUSSION

An interesting aspect of the present study was significant increases in MPAP at 2 and 5 min post-XD in patients with AAA. Although there has been mention of systemic hemodynamic changes following XC and XD,[1-6] reports of detailed measurements of pulmonary hemodynamic changes on XD have been rare. Silverstein *et al.*[2] measured MPAP after XD. They reported that it decreased by 20% of the pre-XD value within 1–3 min after XD, which, however, was not significant compared to that of the pre-XD value. There are several possibilities as to why their data is different from ours. Firstly, Silverstein *et al.* had not described the number of patients who had AAA and atherosclerotic occlusive disease, respectively. Periaortic collateral vessels are reportedly different between the two diseases.[8] If the metabolic effects distal to XC are one of the causes of the increase in MPAP, responses of pulmonary vasculature to XD in patients with AAA would be more exaggerated compared to that of atherosclerotic occlusive disease. Secondly, the period just after XD is important, since PAP had increased significantly during this period in our cases. Thus, their measurement time of 1–3 min after XD is vague compared with our results. Thirdly, their control values were different from ours. We measured pre-XD values prior to an additional infusion of lactated Ringer's solution in order to minimize the influence of volume loading on our re-

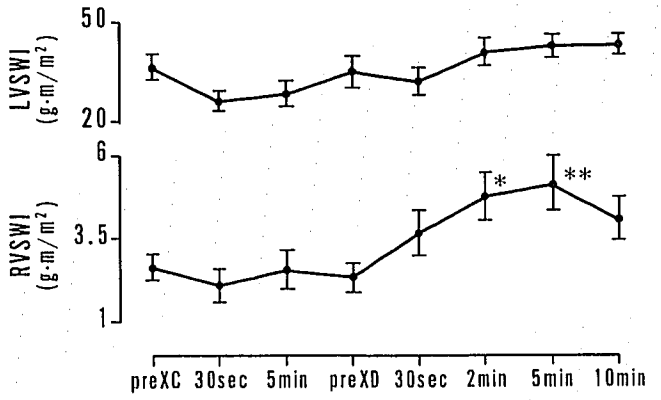

FIG. 3. Hemodynamic measurements in 13 patients. LVSWI = left ventricular stroke work index; RVSWI = right ventricular stroke work index; preXC = pre-cross-clamping time; preXD = pre-declamping time. Values are means ± SEM, n = 13. Statistical analysis by analysis of variance and Dunnet's multiple comparison test. * $P < 0.05$, ** $0.01 < P < 0.05$, compared with pre-XC values.

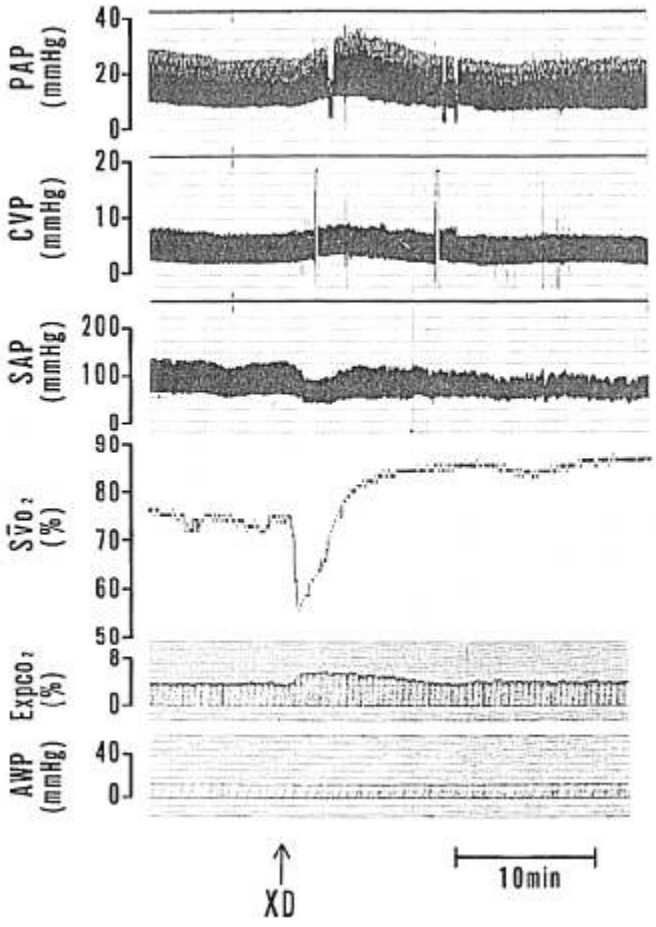

FIG. 4. Recordings showing alterations in pulmonary arterial pressure, central venous pressure, systemic arterial pressure, mixed venous oxygen saturation, and end-expiratory carbon dioxide concentration in pre- and post-aortic declamping period. SAP = systemic arterial blood pressure; PAP = pulmonary arterial blood pressure; CVP = central venous blood pressure; XD = aortic declamping. $S\bar{v}_{O_2}$ = mixed venous oxygen saturation; Exp_{CO_2} = expiratory carbon dioxide concentration; AWP = airway pressure.

sults. Assuming that they had obtained pre-XD values after an additional volume load, pre-XD PAP would be relatively high, and would be declining immediately after XD.

Decreased mixed venous oxygen saturation after XD could reflect a return of hypoxic venous blood from the lower extemities and/or an increase in oxygen extraction to compensate for the oxygen debt occurring during the XC period.[4-6] Since hypoxic pulmonary vasoconstriction is reportedly evoked by a synergistic effect of decreased alveolar oxygen tension and mixed venous oxygen tension,[9-11] hypoxic mixed venous blood might temporarily cause pulmonary vasoconstriction. Damask *et al.*[5] reported that increased Pa_{CO_2} coincides with higher carbon dioxide production after XD. In a recent report, it was noted that acute hypercapnea exerts a moderate degree of pulmonary vasoconstriction and enhances hypoxic pulmonary vasoconstriction, although this depends upon experimental circumstances and the species chosen for the study.[12-14] Therefore, results presented in figure 4 might explain our present observation of an increase in MPAP after XD. However, the absence of a significant increase in PVR after XD in our patients contradicts this speculation.

In conclusion, we report a significant increase in MPAP 2 and 5 min after XD in patients undergoing AAA surgery. The mechanism of this phenomenon remains to be investigated further.

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