medicated. Our colleagues in radiology report that when patients are allowed to sit up soon after gas myelography, many will experience severe headaches despite evacuation of most of the air from the subarachnoid space.‡ It seems possible that cephalad movement of subarachnoid air is responsible for those headaches.

In neither of our patients was dural puncture evident at the time of injection. The first patient obtained a segmental block typical of low-volume epidural anesthesia. Apparently a small amount of air was forced through the dura upon entry of the needle into the epidural space. In the second patient, who had an obvious subarachnoid block despite negative aspiration, the appearance of subarachnoid air was not surprising.

We have not seen reports of early transient headaches after epidural anesthesia for surgical procedures or labor and delivery. Presumably such patients would be less likely to experience this problem because they are usually supine for several hours after the injection, and are often medicated with potent analgesics during the early postoperative or postdelivery periods.

Early, transient headaches are apparently the complication of a technique, *i.e.*, loss of resistance with air. This complication could probably be prevented by using saline solution rather than air. When it does occur after the subarachnoid injection of air, the duration of cephalgia can probably be reduced by the inhalation of 100 per cent oxygen, which would facilitate the reuptake of nitrogen from the air bubble.

‡ Williams, AL., Department of Radiology, Medical College of Wisconsin, 8700 W. Wisconsin Avenue, Milwaukee, Wisconsin 53226: Personal communication.

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Avoiding the Hemodynamic Consequences of Aortic Cross-clamping and Unclamping

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The management of patients undergoing abdominal aortic surgical procedures represents a major challenge for the anesthesiologist. The typical victim of aortic disorders is advanced in years and has generalized arteriosclerotic vascular disease, including coronary-artery and/or cerebral vascular insufficiency, as well as concurrent hypertension, diabetes mellitus, and renal dysfunction. The surgical procedure may last many hours, with significant blood loss, volume shifts, and electrolyte and temperature changes. Aortic cross-clamping and unclamping are particularly hazardous events during the course of abdominal aortic operations. Hypertension, myocardial ischemia, and arrhythmias have been reported to occur after application of the aortic cross-clamp, and recently some of the hemodynamic changes associated with these problems have been elucidated.[1] Removal of the cross-clamp often results in severe hypotension, with sub-

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TABLE 1. Patient Information

	Age (Years), Sex	History*	Preoperative Electrocardiogram
Patient 1	76, M	Hypertension	Left axis, nonspecific ST-T wave changes
Patient 2	67, F	Hypertension, diabetes, COPD	Old anterolateral MI, left atrial enlargement, nonspecific ST-T wave changes
Patient 3	69, M	MI × 3, angina, COPD, alcoholic	Old inferoposterior MI
Patient 4	71, M	MI, congestive heart failure, hypertension, COPD, carotid stenosis	Old inferolateral MI, left ventricular hypertrophy
Patient 5	59, M	MI, COPD	Inferior MI, premature atrial contractions
Patient 6	63, F	Hypertension, COPD	Nonspecific ST-T wave changes
Patient 7	65, M	MI, congestive heart failure, atrial fibrillation, mitral stenosis, COPD	Atrial fibrillation, nonspecific ST-T wave changes
Patient 8	81, F	Hypertension	Nonspecific ST-T wave changes, premature atrial contractions
Patient 9	71, M	MI, COPD	Old anterior septal MI
Patient 10	78, M	—	Old anterior septal MI
Patient 11	67, M	MI, congestive heart failure, cerebral vascular accident	Left ventricular hypertrophy, ischemia, premature ventricular contractions
Patient 12	63, M	Hypertension, paroxysmal atrial fibrillation, COPD	Normal
Patient 13	67, M	Angina, congestive heart failure, aortic insufficiency, COPD	Left ventricular hypertrophy, left atrial enlargement, ischemia
Patient 14	67, F	Hypertension	Normal
Patient 15	66, M	MI × 2	Old inferior MI

* MI = myocardial infarction; COPD = chronic obstructive pulmonary disease.

sequent myocardial, cerebral, and/or renal ischemia.[2] Therefore, we undertook this study to demonstrate that precise monitoring of cardiovascular function will document the nature of cardiovascular decompensation and allow for its immediate intraoperative correction.

METHODS

The hemodynamic changes that occurred when the aortic cross-clamp was applied infrarenally and later released were studied in 15 patients, ASA Classes III and IV (table 1), scheduled for elective abdominal aortic aneurysmectomy or bypass procedures for arteriosclerotic occlusive disease. All patients were premedicated with morphine sulfate, 0.1 mg/kg, and scopolamine, 0.4 mg, im, an hour prior to arrival in the operating room. This premedication, along with lidocaine, 1 per cent, by infiltration, was usually sufficient to allow the insertion of at least one large-bore peripheral intravenous cannula for volume infusion, a #18 radial-artery cannula, and a 7-Fr thermodilution pulmonary-artery catheter, inserted percutaneously via the internal jugular vein. The patients were then anesthetized with morphine, 1 mg/kg, iv, given as 5-mg boluses every 60 sec, nitrous oxide, and oxygen, often supplemented with halothane 0.5–1 per cent. Endotracheal intubation was facilitated with an infusion of succinylcholine, 0.2 per cent. Subsequent muscle relaxation was achieved with *d*-tubocurarine. Heart rate (HR), electrocardiogram lead V_3 or V_4, and systolic, diastolic, and mean (MAP) arterial, pulmonary arterial (PAP), pulmonary capillary wedge (PCWP), and central venous (CVP) pressures were recorded continuously on a Hewlett-Packard eight-channel recorder. Cardiac output (CO) was determined using thermodilution technique, and data were examined immediately before and 1–3 min after application of the aortic cross-clamp, and immediately before and 1–3 min after its release. Arterial blood was sampled for Pa_{O_2}, Pa_{CO_2}, *p*H and lactate determinations prior to aortic cross-clamping and 1 min after unclamping. From the outset of the surgical pro-

cedure, the anesthesiologist attempted to establish and maintain a PCWP between 10 and 20 torr, either with volume infusion (an average of 1,000 ml of lactated Ringer's solution/hr) or with sodium nitroprusside (0.01 per cent infusion), as needed throughout the operation. Aortic cross-clamping times averaged 80 min. The clamp was removed abruptly to permit observation of the stepwise changes in hemodynamic function.

Results (Table 2)

Within 1–3 min after application of the clamp across the aorta, systolic pressure rose 17 per cent, while diastolic pressure and MAP were similar to control values. The surprising statistical finding that MAP did not change significantly while systolic pressure rose and diastolic pressure stayed the same may have been due to the use of vasodilator therapy for eight of our patients. CO decreased 21 per cent and stroke work (SW = stroke volume × MAP × 0.0134) decreased 12 per cent. Rate pressure product (RPP), which is the product of systolic pressure and HR, was not significantly altered. As expected, total peripheral resistance (TPR = MAP − CVP/CO × 80) increased 36 per cent. Pulmonary vascular resistance (PVR = PAP − PCWP/CO), however, increased insignificantly. HR, CVP, and PCWP did not change. Evidence of new arrhythmias or increased myocardial ischemia was not observed on the continuous EKG printout. All changes manifested within the first 3 min after clamping and, although data were examined for more than 30 min thereafter, no further change occurred unless a sudden rapid blood loss ensued.

Upon removal of the aortic cross-clamp, systolic pressure, diastolic pressure, and MAP fell rapidly 26, 21, and 20 per cent, respectively (table 3). However, the lowest MAP observed was 55 torr. CO actually increased 16 per cent, while RPP decreased 25 per cent and TPR, 28 per cent. HR, SW, CVP, PCWP, and PVR did not change. Average arterial lactate concentration increased from 0.95 to 2.08 mEq/l, but *p*H was not affected. Pa_{O_2} and Pa_{CO_2} did not change appreciably throughout the study, and again, no evi-

Table 2. Cardiovascular Effects of Aortic Cross-clamping (Means ± SE)

	Pre-clamp	Post-clamp	Per Cent Change	Significance of Change*
Heart rate	80.5 beats/min 4	76.5 beats/min 3.9	↓5	NS
Systolic arterial pressure	124.6 torr 5.2	146.4 torr 5.6	↑17	$P < 0.0025$
Diastolic arterial pressure	71.4 torr 3.6	73.2 torr 3.4	↑3	NS
Mean arterial pressure	91.3 torr 3.9	97.7 torr 3.6	↑7	NS
Rate pressure product	99.6/10² 5.5	111.1/10² 8.1	↑12	NS
Cardiac output	4.7 l/min 0.4	3.6 l/min 0.4	↓21	$P < 0.001$
Stroke work	72.9 gm-m 12.7	64.5 gm-m 9.9	↓12	NS
Central venous pressure	6.9 torr 1.3	5.9 torr 1.6	↓14	NS
Total peripheral resistance	1,583 dynes·sec/cm^{-5} 158	2,159 dynes·sec/cm^{-5} 232	↑36	$P < 0.001$
Mean pulmonary arterial pressure	16.4 torr 1.6	14.6 torr 1.9	↓11	NS
Pulmonary capillary wedge pressure	11.0 torr 1.5	9.3 torr 2.6	↓15	NS
Pulmonary vascular resistance	120.5 dynes·sec/cm^{-5} 27	140.1 dynes·sec/cm^{-5} 19	↑16	NS
*p*H	7.52 0.02	7.48 0.03		NS

* NS = not significant.

TABLE 3. Cardiovascular Effects of Aortic Unclamping (Means ± SE)

	Pre-unclamp	Post-unclamp	Per Cent Change	Significance of Change*
Heart rate	78.7 beats/min 4.3	79.7 beats/min 4.1	↑1	NS
Systolic arterial pressure	142.3 torr 6.1	104.6 torr 4.5	↓26	$P < 0.001$
Diastolic arterial pressure	70.4 torr 2.4	55.4 torr 3.2	↓21	$P < 0.001$
Mean arterial pressure	94.0 torr 3.2	74.7 torr 2.7	↓20	$P < 0.001$
Rate pressure product	111.1/10² 6.9	82.7/10² 6.6	↓25	$P < 0.001$
Cardiac output	3.8 l/min 0.3	4.4 l/min 0.2	↑16	$P < 0.05$
Stroke work	60.8 gm-m 7.0	55.3 gm-m 3.8	↓9	NS
Central venous pressure	6.8 torr 1.2	4.8 torr 0.9	↓29	NS
Total peripheral resistance	1,933 dynes·sec/cm^{-5} 158	1,392 dynes·sec/cm^{-5} 149	↓28	$P < 0.025$
Mean pulmonary arterial pressure	16.9 torr 1.6	13.6 torr 1.2	↓20	NS
Pulmonary capillary wedge pressure	12.0 torr 1.2	8.7 torr 1.2	↓28	NS
Pulmonary vascular resistance	117.5 dynes·sec/cm^{-5} 13.7	91.4 dynes·sec/cm^{-5} 10	↓22	NS
Arterial blood lactate	0.95 mEq/l 0.096	2.08 mEq/l 0.23	↑120	$P < 0.001$
*p*H	7.44 0.03	7.42 0.02		NS

* NS = not significant.

dence of myocardial ischemia or new arrhythmias was recorded.

DISCUSSION

Aortic cross-clamping depressed myocardial function, manifested by reduced cardiac output, while PCWP, CVP and RPP (a generally accepted indirect expression of myocardial oxygen consumption[3]) were unchanged. We believe that depression of myocardial function reflected the inability of hearts compromised by coronary-artery disease to respond to an acute increase in TPR, and thus in afterload, imposed by the aortic cross-clamp. The increased afterload (*i.e.*, the increase in myocardial wall tension) necessitated an increased myocardial oxygen consumption. However, because of diseased coronary arteries, perhaps the myocardium was unable to receive a greater supply of oxygen to accommodate this need; hence, the ventricle moved to a less favorable function curve.

Attia *et al.*[1] described three patients with severe coronary-artery disease who became ischemic during aortic clamping. In addition, hemodynamic changes indicative of left ventricular (LV) failure were observed. Carroll *et al.*[5] found similar deterioration in two of 14 patients during aortic clamping. In our study, similar hemodynamic changes occurred in the majority of patients whether or not there was a history of severe coronary-artery disease. Eight of 15 patients needed intraoperative intervention with sodium nitroprusside. The crucial point is that none of our patients had decompensation severe enough to cause overt LV failure, irritability, or increased myocardial ischemia. We actively prevented an increase in myocardial oxygen demand by preventing large increases in PCWP, and presumably in LV end-diastolic volume and myocardial wall tension, by use of sodium nitroprusside.[4] Indeed, RPP remained unchanged, though this measure of myocardial oxygen consumption is admittedly indirect at best, especially

where changes in contractility or ventricular volume occur, as they may have here.

Aortic unclamping in our patients proved to be beneficial rather than dangerous, even though the cross-clamp was removed abruptly, a practice that we would not routinely recommend. Severe hypotension did not occur, and again, there was no evidence of additional myocardial ischemia or arrhythmia. Upon unclamping, the sudden and profound decreases in TPR and afterload improved myocardial function, as evidenced by significantly increased CO, decreased RPP, and unchanged SW. PCWP, CVP, and HR were also unchanged. These results point to improvement in myocardial performance. The same work was performed, a better cardiac output was obtained, while the myocardial oxygen cost decreased, perhaps allowing our patients' diseased hearts to operate on a more favorable ventricular function curve.

We did not observe a significant role for circulating myocardial depressant factors that may have been released into the circulation from ischemic extremities or gut at the time of unclamping, as described by Fahmy *et al.*[¶] In addition, although arterial blood lactate levels did double, *p*H and Pa_{CO_2} remained unchanged. Thus, despite the presence of potential myocardial depressants at the time of unclamping, myocardial function in our patients actually improved. Similar improvement was reported by Bush *et al.*[6]

We feel that the major factor allowing our patients to tolerate sudden unclamping was volume loading. About 1 l of lactated Ringer's solution/hr was infused, in addition to replacement of blood loss under careful supervision to maintain PCWP at 10–20 torr prior to unclamping. The information gleaned from pulmonary-artery catheterization is crucial to titrate volume, vasodilators, or vasopressors for optimal control of each patient's hemodynamic status in the presence of coronary-artery disease. Since the changes observed with both aortic clamping and unclamping occurred within 3 min, rapidity of the operation does not obviate the necessity for invasive monitoring of these fragile patients.

Like the patients of Carroll *et al.*,[5] many patients (73 per cent) were hypertensive and/or tachycardic in the immediate postoperative period. Half of these patients needed vasodilator therapy and, occasionally, propranolol, iv.

In summary, these data indicate that the severe consequences of infrarenal aortic cross-clamping and unclamping can be mitigated or entirely avoided in patients with coronary-artery disease by the judicious use of volume loading and rapid-acting vasodilators such as sodium nitroprusside in conjunction with the monitoring of PCWP and, wherever possible, cardiac output.

[¶] Fahmy NR, Attia RR, Ebeid AM, et al: Hemodynamic effects of aortic unclamping: Possible role of vasoactive intestinal polypeptides. Abstracts of Scientific Papers, Annual Meeting of the American Society of Anesthesiologists, 1977, p 217.

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