Preoperative Evaluation of a Patient for Abdominal Aortic Aneurysm Repair

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(Received January 1, 1998; accepted April 20, 1998)

Coexistent cardiovascular disease is common in patients presenting for repair of aortic aneurysms. However, preoperative cardiac evaluation prior to abdominal aortic aneurysm (AAA) surgery remains contentious with significant variations in practice between countries, institutions and individual anesthetists. The following case report raises some everyday issues confronting clinical anesthetists.

CASE REPORT

A 77-year-old man presented for elective repair of a 6.5 cm abdominal aortic aneurysm (AAA). He had originally presented to his family physician complaining of low back pain of several months duration. He had been treated for hypertension for 10 years and had sustained a myocardial infarction (MI) five years previously. He had no history of angina pectoris and had good exercise tolerance, becoming breathless only on walking uphill briskly. He had no known allergies and was an ex-smoker for 20 years (35-pack/year history). The patient’s only previous anesthetic was uneventful. Preoperatively he was taking atenolol 100 mg, bendrofluazide 5 mg daily, nifedipine 20 mg daily and captopril 25 mg BD. The patient’s blood pressure was 140/80, and his pulse rate was 56/min. Examination of the airway, cardiovascular and respiratory systems was unremarkable.

An ECG showed sinus bradycardia of 56/min, left-axis deviation and left bundle branch block. The chest X-ray was normal. An echocardiogram showed an ejection fraction of 58 percent with regional wall dyskinesia in the left ventricle. Of note in the blood biochemistry was a low potassium of 2.9 mmol/l and a slightly elevated urea at 7.2 mmol/l; creatinine was 88 μmol/l.

On the morning of surgery, the patient received his usual medications (atenolol, bendrofluazide, nifedipine and captopril), and he was premedicated with diazepam 10 mg. Prior to induction, he was monitored with continuous ECG (leads II and V5), pulse oximetry and invasive blood pressure. A lumbar epidural catheter was sited after two attempts at inserting a thoracic epidural were unsuccessful. A test dose of bupivacaine 0.5 percent 2.5 ml with adrenaline 1:200,000 was uneventfully administered. At induction, the patient received fentanyl 250 μg, etomidate 16 mg and vecuronium 8 mg. Within minutes of induction, the patient’s pulse rate dropped from 60/min to 28/min with the development of complete heart block on the monitor. The blood pressure dropped from 150/80 mmHg to a systolic pressure of 30 mmHg. The patient was pale and clinically pulseless.

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\[b\] Abbreviations: MI, myocardial infarction; AAA, abdominal aortic aneurysm; CAD, coronary artery disease; ACE, angiotension converting enzyme.
Cardiopulmonary resuscitation was commenced, and the patient was intubated and ventilated.

Atropine 2.4 mg and adrenaline 2 mg (1 mg plus 1 mg) were given immediately, and the blood pressure increased to 79/50 with a sinus tachycardia. The patient required an adrenaline infusion to maintain his blood pressure. In the light of the patient’s previous MI, cardiovascular collapse and subsequent hypotension, development of another MI was considered a distinct possibility. The ECG was difficult to interpret because of the bundle branch block, but, following consultation with a cardiologist, the patient was given thrombolytic therapy (streptokinase 1.5 MU). At this stage, the vecuronium had worn off, and the patient had woken up and was extubated without any problems. The patient was admitted to the intensive care unit where he was mentally alert but required an adrenaline infusion for 20 hours following his collapse. Cardiac enzyme results were normal. Subsequent coronary angiography demonstrated triple vessel disease.

**DISCUSSION**

The main risk factor identified preoperatively in this patient was a previous myocardial infarction with corresponding dyskinesia demonstrated on two-dimensional echocardiography. His hypertension was controlled, and his exercise tolerance was judged to be satisfactory prior to surgery.

*Should this patient have had a more thorough cardiac work-up prior to surgery in view of this history and echocardiographic finding?*

This patient was at intermediate risk of developing a perioperative cardiac event according to guidelines from both the American College of Physicians [1] and the American College of Cardiology /American Heart Association [2]. A preoperative dipyridamole-thallium scan or dobutamine stress echocardiography was indicated according to these guidelines. In general, a less invasive approach is taken in Ireland and in the United Kingdom to preoperative assessment and treatment of patients with coronary artery disease (CAD) presenting for AAA repair. The rationale for this is that the benefits of an aggressive approach have not been proven to date, even among patients with preoperative mild stable angina [3-5]. Nevertheless, there are reports showing that prior MI is associated with adverse outcomes (death or MI) following AAA repair [6, 7]. Furthermore, among patients with left bundle branch block, those with left-axis deviation have a greater incidence of myocardial dysfunction, more advanced conduction disease and greater cardiovascular mortality than those with a normal axis [8]. Hence this finding should have increased suspicions that the patient might have extensive CAD, as later confirmed by coronary angiography, even if he didn’t have troublesome angina.

*Should this patient have been paced preoperatively?*

The bradycardia shown on the ECG was thought to be the result of beta-blockade. An overview of studies examining the perioperative risks in patients with preoperative conduction disturbance concluded that fewer than two percent of patients with chronic bifascicular block (either left bundle branch block or right bundle branch block combined with left anterior or posterior hemiblock) progressed to complete heart block during surgery [9]. The presence also of a prolonged P-R interval may be suggestive of increased risk of complete heart block, but this is controversial and, in any event, was not relevant to our patient. A significant incidence of perioperative bradyarrhythmias in patients with left bundle branch block has recently been reported [10]. The bradyarrhythmias in this study responded to pharmacotherapy, and the authors did not recommend prophylactic insertion of a temporary pacemaker.
There is no good evidence that our patient warranted prophylactic pacing. Nevertheless, placement of a central line before induction of anesthesia would have been appropriate, to aid the insertion of a temporary pacing wire if needed. A transcutaneous pacemaker was available for use in the operating theatre had our patient not responded to atropine. The patient was anesthetized and so would have been a good candidate for transcutaneous pacing until transvenous pacing was established.

Should one anesthetize a patient for AAA repair with a serum potassium of 2.9 mmol/l?

There is little clear evidence one way or the other. This patient’s records indicated that the hypokalemia was chronic. Treatment with a thiazide diuretic was the most likely cause for this finding. It should be noted that the dose of bendrofluazide was 5 mg daily rather than the more usual 2.5 mg daily. There is no evidence of increased efficacy but good evidence for increased metabolic disturbance at higher doses of thiazide diuretics. The clinical significance of hypokalemia in thiazide-treated patients is disputed [11]. Indeed, there is even controversy as to whether diuretics really cause significant depletion of total body potassium [12, 13].

It is not surprising that anesthetists also vary in their responses to hypokalemia. Some would accept this low potassium level arguing that the chronic nature of the hypokalemia would allow restoration of equilibrium at cellular level and maintenance of transmembrane potential. A prospective study of outcome in patients who were chronically hypokalaemic prior to surgery did not show an increase in dysrhythmias intraoperatively [14]. Others argue for respecting a cut-off point for potassium of 3.0 mmol/L—the point below which U waves occur with increasing frequency. Several large studies have shown a correlation between diuretic induced hypokalemia and ventricular ectopic activity [15-17]. Use of thiazide diuretics is also associated with hypomagnesemia, which may contribute to arrhythmogenesis [18]. Inotropes, such as adrenaline, and bronchodilators, such as salbutamol, have been shown to lower serum potassium [19, 20]. AAA repair is a major procedure expected to take many hours and requiring infusion of large volumes of crystalloid, colloid and blood. Inotropes may be required in the course of surgery or afterwards in the intensive care unit. For these reasons, it is preferable to have normal electrolyte levels preoperatively. There is consensus that rapid correction of chronic hypokalemia by intravenous infusion is undesirable and is associated with significant morbidity [11]. Potassium should ideally be replaced orally over three to four days.

Should this patient have received his antihypertensive medications preoperatively?

It is common practice to continue beta-blockers preoperatively on the basis of their cardioprotective effects [21-23] and to prevent beta-blocker withdrawal syndrome [24]. Calcium channel blockers are also usually continued preoperatively, although the resultant myocardial depression, systemic vasodilation and slowing of atrioventricular conduction may be potentiated by inhalational agents [25]. There has been more concern about whether or not to continue angiotensin converting enzyme (ACE) inhibitors perioperatively [26]. Colson et al. documented significant blood pressure decreases at induction in patients treated with ACE inhibitors [27]. Hemodynamic results indicated that hypertension in this group of patients was due to the inability of the heart to maintain cardiac output during acute changes in ventricular volume. They suggested that crystalloids as well as inotropes were required to control blood pressure in such patients. However, a subsequent study by Sear et al. showed no difference between hemodynamic responses at induction between patients treated with different antihypertensive agents including ACE inhibitors [28]. There is still a lack of documentation in the literature of the effects of multiple antihypertensive therapy on patients at induction.
Was appropriate monitoring in place prior to induction?

The intention had been to insert a central venous line after induction; this did happen but under more stressful conditions than had been envisaged! As previously stated, in view of the ECG findings, it would have been advisable to have had central vein access prior to induction. The benefits of inserting a pulmonary artery flow directed catheter for monitoring during AAA surgery are uncertain [29] and may even be detrimental in critical care patients [30].

Were suitable induction agents used in this patient?

Fentanyl in low to moderate doses can be associated with a mild bradycardia, which is probably central in origin as it does not occur after vagotomy [31]. Vecuronium is usually stable as regards cardiovascular effects, but bradycardias and some dysrhythmias have been reported. Because vecuronium lacks vagolytic effects, any opioid-induced bradycardia is unopposed when fentanyl and vecuronium are used in combination. Similarly, bradycardia and asystole have been reported following the administration of sufentanil with vecuronium, interestingly in patients who were receiving both beta-blockers and calcium channel blockers [32]. Etomidate in the dose used in this patient has minimal cardiovascular effects, although there is experimental evidence of an increase in central vagal tone [33]. On balance, it must be stated that the combination of fentanyl, vecuronium and etomidate was not appropriate in this case because of the pre-existing conduction disturbance and bradycardia. Despite wishing to maintain a relatively slow heart rate, it might have been more appropriate to administer a vagolytic agent such as pancuronium instead of vecuronium at induction. We do not believe that the epidural bupivacaine contributed to the collapse in this patient since only a test dose had been administered and a suitable time period had elapsed between this and induction. Also, on emergence from anesthesia, there was no evidence of subarachnoid block.

What lessons can be learned from this case?

This patient presented with a number of problems including a history of previous MI, hypertension requiring quadruple therapy, hypokalemia and an ECG showing conduction defects. The patient required more thorough cardiac investigation preoperatively. Non-invasive testing such as dipyridamole thallium imaging or stress echocardiography would have provided useful information on risk. Left bundle branch block can be associated with perioperative bradyarrhythmias, but the literature does not support perioperative prophylactic cardiac pacing. A transcutaneous pacemaker was available, but central venous access should have been established prior to induction of anesthesia to aid the insertion of a temporary pacing wire if required.

Treatment of hypokalemia preoperatively is contentious, but on balance we would argue that the potassium level should have been corrected over several days prior to surgery in order to optimize the patient for such a major procedure. Antihypertensive medications were given preoperatively. There is a clear rationale for continued treatment with beta-blockers, but there are potential disadvantages associated with calcium channel and ACE inhibitors. It would have been reasonable to omit the ACE inhibitor or calcium channel blocker preoperatively on the basis that the effects of multiple antihypertensive therapy at induction are unknown but most likely are additive.

The combination of agents used to induce anesthesia was not appropriate because of their cumulative vagotonic effects. At least one agent with sympathomimetic properties, such as pancuronium, should have been used. The cumulative effects of this patient's problems resulted in complete heart block and cardiovascular collapse when agents likely to cause a mild bradycardia were administered.
REFERENCES

1. American College of Physicians. Guidelines for assessing and managing the perioperative risk from coronary artery disease associated with major non-cardiac surgery. Ann. Int. Med. 127: 309-12, 1997.
2. Eagle, K.A. Surgical Patients with Heart Disease: Summary of the ACC/AHA guidelines. Am. Fam. Phys. 56:811-8, 1997.
3. Hertzer, N.R., Young, J.R. Beven, E.G., and O'Hara, P.J. Late results of coronary bypass in patients with infrarenal aortic aneurysms. Cleveland Clinic Study. Ann. Surg. 205:360-367, 1987.
4. Hertzer, N.R., Beven, E.G., Young, J.R., and O'Hara, P.J. Coronary artery disease in peripheral vascular patients: a classification of 1000 coronary angiograms and results of surgical management Ann. Surg. 199:223-233, 1984.
5. Taylor, L.M., Yeager, R.A., and Moneta, G.L. The incidence of perioperative myocardial infarction in general vascular surgery. J. Vasc. Surg. 15:52-59, 1992.
6. Yeager, R.A., Weigel, R.M., Murphy, E.S., and McConnell, D.B. Application of clinically valid cardiac risk factors to aortic aneurysm surgery. Arch. Surg. 121:278-281, 1986.
7. Lachapelle, K., Graham, A.M., and Symes, J.F. Does the clinical evaluation of the cardiac status predict outcome in patients with abdominal aortic aneurysm? J. Vasc. Surg. 11:650-658, 1992.
8. Dhringra, R.C., Amat-Y-Lyon, F., Wyndham, C., Sridhar, S.S., Wu, S.S., Denes, P., and Rosen, K.M. Significance of left axis deviation in patients with chronic left bundle branch block. Am. J. Cardiol. 42:551-556, 1978.
9. Roizen, M.F. Anaesthetic implications of concurrent diseases. In: Miller, R.D., ed. Anaesthesia, fourth edition. New York: Churchill Livingstone; pp. 903-1006.
10. Gauss, A., Hubner, C., Radermacher, P., Georgieff, M., and Schutz, W. Perioperative risk of bradycardiac in patients with asymtomatic chronic bifascicular block or left bundle branch block. Does an additional first-degree atrioventricular block make any difference? Anesthesiology 88:679-87, 1998.
11. Harrington, J.T., Isner, J.M., and Kassirer, J.P. Our national obsession with potassium. Am. J. Med. 73:155-159, 1982.
12. Singh, B.N., Hollenberg, N.K., Poole-Wilson, P.A., and Robertson, J.I. Diuretic-induced potassium and magnesium deficiency: relation to QT prolongation, cardiac arrhythmias and sudden death. J. Hypertens. 10:301-316, 1992.
13. McIntosh, G.T., Yeo, W.W., Ramsey, L.E., and Moser, M. Cardiotoxicity and diuretics: much speculation—little substance. J. Hypertens. 10:317-335, 1992.
14. Vitez, T.S., Soper, L.E., Wong, K.C., and Soper, P. Chronic hypokalaemia and intraoperative dysrhythmias. Anaesthesia 63:130-133, 1985.
15. Holme, I., Helgeland, A., Hjermann, I., Leren, P., and Lundlarsen, P.G. Treatment of mild hypertension with diuretics: the importance of ECG abnormalities in the oslo study and in MRFIT. JAMA 251: 1298-1299, 1984.
16. Cohen, J.D., Neaton, J.D., Prineas, R.J., and Daniels, K.A. Diuretic induced ventricular tachycardia: diuretics, serum potassium and ventricular arrhythmias in the multiple risk factor intervention trial. Am. J. Cardiol. 60: 548-554, 1987.
17. Medical Research Council Working Party on mild to moderate hypertension. Ventricular extrasystoles during thiazide treatment: substudy of MRC mild hypertension trial. Br. Med. J. 287:1249-1253, 1983.
18. Swales, J.D. Magnesium deficiency and diuretics. Br. Med. J. Clin. Res. 285:1377-1378, 1982.
19. Hastwell, G. and Lambert, B.E. The effect of oral salbutamol on serum potassium and blood sugar. Br. J. Obstet. Gynaecol. 85:767-769, 1978.
20. Brown, M.J., Brown, D.C., and Murphy, M.B. Hypokalaemia from beta 2-receptor stimulation by circulating epinephrine. N. Engl. J. Med. 309:1414-1419, 1983.
21. Leone, B.J., Lehot, J.J., and Francis, C.M. Beta-blockade reverses regional dysfunction in ischaemic myocardium. Anesth. Analg. 66:607-611, 1987.
22. Prys-Roberts, C., Foxe, P., and Biro, G.P. Studies of anaesthesia in relation to hypertension. V. Adrenergic beta-receptor blockers. Br. J. Anaesth. 45: 671-680, 1973.
23. Mangano, D.T., Layug, E.L., Wallace, A., and Tateo, I. Effect of atenolol on mortality and cardiovascular morbidity after noncardiac surgery. Multicenter study of perioperative ischaemia research group. N. Engl. J. Med. 335: 1713-20, 1996.
24. Alderman, E.L., Col tart, D.J., and Wetlock, G.E. Coronary artery syndromes after sudden propranol withdrawal. Ann. Intern. Med. 81:625-628, 1974.
25. Reves, J.G., Kissin, I., Lell, W.A., and Tosone, S. Calcium entry blockers: uses and implications for anesthesiologists. Anesthesiology 57:504-518, 1982.
26. Coriat, P., Richer, C., Douraki, T., Gomez, C., Hendricks, K., Giudicelli, J.F., and Viars, P. Influence of chronic angiotensin-converting enzyme inhibition on anesthetic induction. Anesthesiology 81:299-307, 1994.
27. Colson, P., Saussine, M., Seguin, J.R., Cuchet, D., Chaptal, P.A., and Roquefeuil, B. Hemodynamic effects of anaesthesia in patients chronically treated with angiotensin-converting enzyme inhibitors. Anesth. Analg. 74:805-808, 1992.
28. Sear, J.W., Jewkes, C., Tellez, J.C., and Foex, P. Does the choice of antihypertensive therapy influence haemodynamic responses to induction, laryngoscopy and intubation? Br. J. Anaesth. 73:303-308, 1994.
29. Isaacson, I.J., Lowdon, J.D., and Berry, A.J. The value of pulmonary artery and central venous monitoring in patients undergoing abdominal aortic reconstructive surgery: a comparative study of two selected, randomized groups. J. Vasc. Surg. 12:754-760, 1990.
30. Connors, A.F., Speroff, T., and Dawson, N.V. The effectiveness of right heart catheterization in the initial care of critically ill patients. JAMA 276:889-897, 1996.
31. Reitan, J.A. Central vagal control of fentanyl induced bradycardia during halothane anesthesia. Anesth. Analg. 57:31-34, 1978.
32. Starr, N.J., Sethna, D.H., and Estafanous, F.G. Bradycardia and asystole following the rapid administration of sufentanil with vecuronium. Anesthesiology 64:521-523, 1986.
33. Inoue, K. and Reichelt, W. Efferent vagal discharge and heart rate in response to methohexitone, althesin, ketamine and etomidate in cats. Br. J. Anaesth. 54:1105-1109, 1982.