Changes in ionized calcium concentrations and acid–base status during abdominal aortic vascular surgery

S. JANKOWSKI, J. KNIGHTON, R. DUNNILL and D. DICKSON

Summary
Abdominal aortic surgery may produce significant haemodynamic instability (from a combination of factors: hypovolaemia, acid–base disturbances, vasoactive metabolite release from ischaemic tissues and hypocalcaemia). Calcium is often given after aortic unclamping to attenuate this instability. We studied 20 patients undergoing elective abdominal aortic surgery and observed a triphasic change in ionized calcium concentrations and acid–base status. Initially, during the cross-clamp period (when patients were cardiovascularly stable), ionized calcium concentrations decreased significantly (mean 1.06 (0.08) to 0.91 (0.13) mmol litre\(^{-1}\); \(P<0.01\)), while a significant metabolic acidosis developed (pH 7.38 (0.05) to 7.30 (0.05); \(P<0.05\)). Second, release of the aortic cross-clamp resulted in further acidosis (pH 7.27 (0.05) (\(P<0.05\)) mixed respiratory and metabolic) with a decrease in mean arterial pressure, with no change in ionized calcium concentrations. The third phase was associated with spontaneous restoration of acid–base status and ionized calcium concentrations to normal over 2 h. There was no correlation between units of blood given, volume of blood lost, fluid volume given or duration of aortic cross-clamping and degree of ionized hypocalcaemia. We conclude that ionized hypocalcaemia occurred during the cross-clamp period of aortic surgery, was unrelated to the volume of blood given and did not appear to be responsible for the changes in arterial pressure during surgery. (Br. J. Anaesth. 1998; 81: 965–967).

Keywords: ions, calcium; surgery, vascular; acid–base equilibrium, metabolic acidosis.

Unclamping of the abdominal aorta during vascular surgery may produce cardiovascular instability (arterial hypotension, tachycardia and changes in cardiac output).\(^1\) Several factors may contribute: sudden reduction in left ventricular afterload, blood loss, reactive hyperaemia, metabolic effects, lactic acidosis, hyperkalaemia, hypercapnia and release of toxic, vasoactive products of anaerobic metabolism.\(^2\)

Aggressive fluid management, volume loading before aortic unclamping, sodium bicarbonate or calcium (particularly with large blood transfusions)\(^1,2\) are used to minimize these changes. The role of exogenously administered calcium in maintaining cardiovascular function is unclear and disputed.\(^3\) Further, the practice of giving calcium during blood transfusions is empirical and the rationale for its use dates back to the period when whole blood was given rather than the current practice of giving specific blood products.\(^4\)

In this study we have investigated the changes in ionized calcium concentrations [Ca\(^{2+}\)] (physiologically active fraction of blood calcium) and acid–base status during abdominal aortic surgery.

Methods and results
After obtaining approval for the study from the local Ethics Committee and verbal consent, we studied 20 patients (16 males, ASA II or III, mean age 72 (range 51–89) yr), undergoing elective abdominal aortic surgery. All patients were anaesthetised by one of two anaesthetists (R.P.H.D./D.M.D.) using a standard anaesthetic technique. Surgery was performed by the same two surgeons.

All patients were given oral temazepam and metoclopramide as premedication. Before operation a large bore i.v. cannula and radial arterial catheter were inserted under local anaesthesia. Arterial oxygen saturation, heart rate and invasive mean arterial pressure (MAP) monitoring was started. A low thoracic–high lumbar (T12–L1) epidural was sited under local anaesthesia. A test dose of 0.5% bupivacaine 3 ml was given, followed by a loading dose of 8–10 ml of 0.125% bupivacaine with fentanyl 50 g and an infusion of 0.125% bupivacaine with fentanyl 4 g ml\(^{-1}\) at 10–15 ml h\(^{-1}\). General anaesthesia was induced in all patients with fentanyl 5 g kg\(^{-1}\) and thiopental 3–5 mg kg\(^{-1}\). Vecuronium or pancuronium was used to facilitate tracheal intubation. Anaesthesia was maintained with 1–1.5% sevoflurane and 60% nitrous oxide in oxygen. A triple-lumen central venous catheter was then inserted into the right internal jugular vein.

All patients received 10% mannitol 500 ml before clamping of the aorta, glyceryl trinitrate 3–5 mg min\(^{-1}\) and dopexamine 3 g kg\(^{-1}\) min\(^{-1}\) infusions. Levels of inotropic support were unchanged during aortic cross-clamping. No patient received sodium bicarbonate or calcium. Fluid replacement consisted of crystalloid and colloid infusions (with blood as...
required) to maintain a central venous pressure of at least 12–15 mm Hg.

Blood-gas analysis and ionized [Ca\textsuperscript{2+}] measurements were performed before surgery, before aortic unclamping, at 3, 15 and 30 min after aortic unclamping, and immediately on arrival in the intensive care unit. All blood sample were obtained using 1 ml of arterial blood in 50 u. of dry heparin and analysed using an ABL 500 Radiometer blood-gas and ion-selective analyser for ionized [Ca\textsuperscript{2+}], (normal range = 1.05–1.25 mmol litre\textsuperscript{-1}).

Changes from baseline values for metabolic and [Ca\textsuperscript{2+}] data were evaluated using analysis of variance for repeated measurements (ANOVA) with Bonferroni correction for multiple comparisons. Statistical significance was defined as $P<0.05$.

We observed a triphasic response in acid–base status and ionized [Ca\textsuperscript{2+}] (fig. 1). The first phase (aorta cross-clamped) was associated with metabolic acidosis and a significant reduction in ionized [Ca\textsuperscript{2+}] (mean 1.06 (sd 0.08) to 0.91 (0.13) mmol litre\textsuperscript{-1}; $P<0.01$) with no significant changes in MAP. The second phase (aortic cross-clamp release) was associated with a transient decrease in MAP and further increase in acidosis (metabolic and respiratory) (without further significant changes in ionized [Ca\textsuperscript{2+}]). The third phase was associated with spontaneous restoration of acid–base status and ionized [Ca\textsuperscript{2+}] over 2 h.

There was no significant correlation (Pearson product moment) between degree of reduction in ionized [Ca\textsuperscript{2+}] and aortic cross-clamp time ($r=0.309$, $P=0.189$) (mean 43.4 (sd 11.3) (range 20–65) min), blood loss ($r=0.361$, $P=0.129$) (1280 (569) (range 350–2500) ml), fluid volume given (crystalloid and colloid) ($r=0.149$, $P=0.529$) (2090 (584) (range 1000–3500) ml, units of blood transfused ($r=0.397$, $P=0.083$).

Comment

During aortic cross-clamping we observed metabolic acidosis with ionized hypocalcaemia with no change in MAP. Reduction in ionized [Ca\textsuperscript{2+}] was not related to the number of units of blood transfused, measured blood loss, volume of fluid given or cross-clamp time. Patients not given blood still had significant reductions in ionized [Ca\textsuperscript{2+}]. Metabolic acidosis has been reported previously with lactic acidosis\textsuperscript{1} while ionized hypocalcaemia (under life-threatening circumstances) has been reported only once.\textsuperscript{5} Hypovolaemic shock leads to ionized hypocalcaemia and is related to duration of shock.\textsuperscript{3} The decrease in ionized [Ca\textsuperscript{2+}] in our patients may reflect hypovolaemia or indicate ischaemic tissues. In patients after cardiorespiratory arrest, ionized hypocalcaemia is common and thought to reflect ischaemia within peripheral tissues as a result of intracellular influx of calcium.\textsuperscript{3} In aortic surgery, blood flow to the lower limbs may still occur during the cross-clamp period because of well developed collateral circulations,\textsuperscript{1} and ionized hypocalcaemia may indicate washout of blood from ischaemic tissues.

On release of the aortic cross-clamp, a reduction in MAP occurred with no significant change in ionized [Ca\textsuperscript{2+}] but with a further acidosis and hypercapnia. Although reported previously, these metabolic abnormalities are not thought to be entirely responsible for haemodynamic instability during this period. During this type of surgery, the importance of avoiding hypovolaemia and tissue hypoperfusion and preventing depression of the cardiovascular system with aggressive volume replacement has been emphasized.\textsuperscript{1,2}

The role of calcium in the maintenance of cardiovascular function is complicated and depends on many factors (including whether or not a patient is hypo- or normocalcaemic).\textsuperscript{3} Previous work suggests that hypocalcaemia (from citrate used as an anticoagulant) may cause haemodynamic instability, but this is disputed because giving calcium may not always restore cardiovascular stability. Hypocalcaemia with blood transfusion is transient and stopping the transfusion results in a spontaneous increase in calcium concentrations.\textsuperscript{3} Calcium administration during aortic surgery is empirical and is used to reverse cardiovascular instability (thought to be caused by...
large (>5 u.) blood transfusions. Rebound hypertension has been documented and indiscriminate use of calcium has resulted in cardiac arrest. Further, it is unclear if giving calcium to patients with ischaemic tissues is detrimental as it may worsen ischaemic injury. Over the next 2 h there was a spontaneous restoration of ionized $[\text{Ca}^{2+}]$ and acid–base status with no significant change in MAP. Therefore, giving calcium to patients during aortic surgery may be unnecessary.

In summary, we observed a decrease in ionized $[\text{Ca}^{2+}]$ during aortic cross-clamping without change in arterial pressure. The cause of this ionized hypocalcaemia is unclear and probably multifactorial but it may reflect peripheral tissue ischaemia and a degree of hypovolaemia. Blood transfusion did not appear to cause ionized hypocalcaemia. Treatment of ionized hypocalcaemia during abdominal aortic surgery should primarily be volume replacement and calcium given only during severe hypocalcaemia and life-threatening circumstances (preferably while monitoring ionized $[\text{Ca}^{2+}]$). These findings do not relate to emergency aortic surgery.

References