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Use of a pneumatic tourniquet induces changes in central temperature†

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Summary

Twenty-six patients requiring orthopaedic surgery were anaesthetized and oesophageal and rectal temperature were monitored continuously. Twenty patients requiring a pneumatic tourniquet were allocated prospectively to one of two groups: passive group (Pg) with reflective insulation on all available skin surface ($n=10$) and forced group (Fg), with active warming by a forced air system ($n=10$). Six patients without a tourniquet were used as a reference group (Rg). The pneumatic tourniquet time was similar in the tourniquet groups. During tourniquet inflation, oesophageal temperature increased with time. The difference was significant compared with the reference group at approximately 20 min. At about 30 min, oesophageal temperature in group Fg was significantly higher than that in group Pg. After tourniquet deflation, temperature decreased transiently. Changes in rectal temperature were similar but delayed significantly. A mechanism to explain the increase in core temperature during pneumatic tourniquet use remains unclear. A redistribution mechanism by cooling of the blood in a cold and vasodilated limb could explain the decrease of temperature after tourniquet deflation. (*Br. J. Anaesth*. 1996; **77**: 786–788)

Key words
Surgery, orthopaedic. Equipment, tourniquets. Temperature, monitoring.

The use of a pneumatic tourniquet for "bloodless-field" surgery is common in orthopaedic practice. However, its use is known to cause many adverse effects[1]: it has been noted that central temperature increases progressively in paediatric patients while the limb tourniquet is inflated,[2 3] but not in adults. On the other hand, a significant decrease in core temperature occurs abruptly after tourniquet release.[4]

In order to clarify temperature changes during tourniquet use, we have compared oesophageal and rectal temperatures during and after a prolonged period of tourniquet inflation in adults.

Methods and results

We studied 26 male adult patients (range 18–45 yr), ASA PS I–II, undergoing orthopaedic surgery. The study was approved by the local Ethics Committee and informed written consent was obtained from each patient. Patients were healthy and none was taking medication. They had no evidence of infection and were not given blood or blood products.

Twenty of 26 patients were scheduled prospectively for fixation of a single lower extremity fracture using the pneumatic tourniquet for a prolonged period (>60 min). Patients with multiple trauma or short periods of tourniquet inflation were excluded. After induction of general anaesthesia, an 8-cm tourniquet cuff was applied around the upper thigh of the operative limb. For exsanguination, the leg was elevated for 5 min at 45° before tourniquet inflation. Arterial flow was occluded with a pressure of 350 mm Hg. For ethical reasons, the reference group (Rg) was studied without randomization and served only as a standard reference. Successively, six patients were included in group Rg: three patients undergoing the same surgery but with tourniquet contraindications (skin lesion or excessive oedema) and three patients where a tourniquet was not indicated (intramedullary nailing of shaft fractures of the tibia).

Premedication was not given and anaesthesia was induced with thiopentone 4–6 mg kg^{-1} i.v. and fentanyl 3 μg kg^{-1} i.v. No neuromuscular blocking agents were used. After tracheal intubation, the lungs were ventilated mechanically with 60% nitrous oxide in oxygen in a semi-closed system without any volatile anaesthetic. Additional doses of fentanyl (5–10 μg kg^{-1} h^{-1}) or thiopentone (1.3–2.5 mg kg^{-1} h^{-1}) were given as needed. A minimal infusion of lactated Ringer's solution at room temperature was given. Ambient temperature was controlled at 20 °C. For all patients, routine monitoring included electrocardiography, pulse oximetry, end-tidal carbon dioxide and arterial pressure measurements.

Twenty patients were allocated randomly to one of two groups using a sealed envelope technique. In

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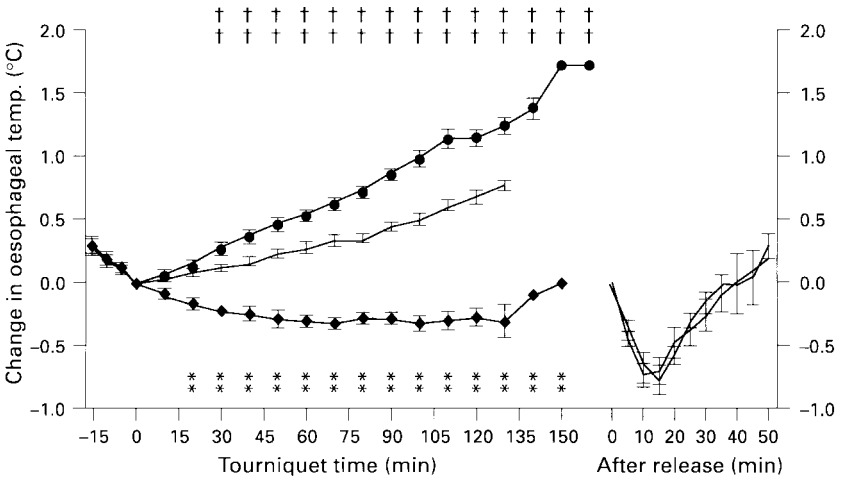

Figure 1 Change in oesophageal temperature (mean, SEM) in patients with a tourniquet undergoing active warming (group Fg) (●) or passive insulation (group Pg) (–) and in patients without a tourniquet (group Rg) (◆). Time zero for temperature reference was tourniquet inflation time whereas core temperature was measured continuously as soon as possible after induction (for the control group time zero was surgical incision). After tourniquet release a new time zero for temperature reference was the time of release of the tourniquet. **$P<0.01$ group Rg *vs* group Pg; ††$P<0.01$ group Pg *vs* group Fg.

the passive group (group Pg, $n=10$) the available skin surface was covered with passive reflective insulation material. In the forced group (group Fg, $n=10$) patients were covered by an active warming system (forced air system: Bair Hugger 500E Augustin medical SA at 38 °C with full body 300 blanket). Group Rg was treated with the same active warming procedure. As soon as possible after induction, core temperature was measured simultaneously in the lower third of the oesophagus and rectally with thermistor probes (Hewlett Packard 21075 A probe). Both temperatures were monitored (Hewlett Packard 78364 C monitor) continuously until the end of surgery. There was no intervention which could have changed the temperature.

Data are expressed as mean (SEM) and analysed using ANOVA with repeated measures. Regression analysis and the Mann–Whitney *U* test, and the Kruskal-Wallis test for non-parametric data, as appropriate, were used to determine significance ($P<0.05$).

Patient characteristics were comparable for all three groups. Tourniquet times were similar (106 (7) min and 115 (7) min for groups Pg and Fg, respectively). Initial temperatures after induction of anaesthesia were comparable. All patients had an initial rectal temperature (37.1 (0.1) °C) significantly higher ($P<0.01$) than the oesophageal temperature (36.6 (0.2) °C).

Changes in oesophageal temperature before, during and after tourniquet inflation are shown in figure 1. Before tourniquet inflation, temperature decreased similarly. Exsanguination had no significant effect. Patients in group Rg remained nearly normothermic throughout surgery. In contrast, core temperature increased significantly in both tourniquet groups and exceeded reference values at all times after 20 min had elapsed. Increases in group Fg were significantly greater than those in group Pg at all times after 30 min had elapsed. This central temperature increase correlated ($P<0.001$) with tourniquet time ($r=0.98$ and $r=0.99$ for groups Pg and Fg, respectively). Immediately after tourniquet deflation, core temperature decreased similarly in both tourniquet groups. The maximum mean decrease, 10–15 min after tourniquet deflation, was 0.7 (0.1) °C. This decrease did not correlate significant with tourniquet duration and was transient.

Changes in rectal temperature were similar but with a significant delay compared with oesophageal changes. In the tourniquet groups, pelvic temperature increased and was significantly higher than that in group Rg at all times after 50 min had elapsed. Increases in group Fg were significantly higher than those in group Pg at all times after 70 min had elapsed. This increase correlated with time ($r=0.93$ and $r=0.96$ for groups Pg and Fg, respectively; $P<0.001$). After tourniquet deflation, rectal temperature did not change.

Progressively, in both tourniquet groups, arterial pressures increased significantly (systolic arterial pressure before tourniquet 111 (18) mm Hg and before release 150 (18) mm Hg, diastolic arterial pressure before tourniquet 59 (13) mm Hg and before release 89 (12) mm Hg; $P<0.01$). After release, pressures decreased significantly to a minimal level obtained 3–10 min after tourniquet release ($P<0.01$). There was a significant difference in end-tidal carbon dioxide concentration between the level just before tourniquet release (4.3 (0.3) kPa) and the peak 3–8 min after tourniquet release (5.9 (0.4) kPa; $P<0.01$). There was no difference between tourniquet groups and no correlation with tourniquet time. All of these variations returned to normal 20 min after tourniquet release.

Comment

In this study the duration of tourniquet inflation appeared to be the main precipitating factor for the increase in core temperature. This increase correlated with duration of tourniquet inflation. It was

similar to that reported in previous paediatric studies where there was no significant difference between smaller and larger children,[2 3] but it has not been described previously in adults. We have found similar changes in adults, with the greatest changes in patients who were actively warmed.

Oesophageal temperature is monitored commonly in patients receiving general anaesthesia. It is considered a true measure of core temperature and it responds rapidly to changes in body temperature. Conversely, rectal temperature correlates poorly with core temperature and has a significantly delayed response because of cooling by the legs. In our study, in all patients, initial rectal temperature was significantly higher than oesophageal temperature because of two possible mechanisms: delay in perioperative hypothermia developing and warming by blood as it returned from the traumatized leg. There was a delay when comparing changes in rectal temperature with oesophageal temperature but hyperthermia was not greater with rectal measurements.

We postulate that the increase in core temperature during tourniquet inflation was caused by slow release of ischaemic metabolites from the extremity into the systemic circulation, using vascular bone as a tourniquet bypass.[3] However, when heat loss to the environment decreases because of reduced surface area while the level of heat production remains stable, core temperature may increase. But, as observed in children, hyperthermia was greater than predicted from the reduction in effective surface area.[2] In fact, if the surface area of each leg constitutes approximately 18% of the total body surface area, the estimated mass of the leg was approximately 16% in adults.[5] It has been suggested that tourniquet ischaemia may initiate a reaction similar to that of malignant hyperthermia.[6]

After tourniquet release, core temperature decreased rapidly but transiently. This significant decline has been described previously in adults[4] and was explained by redistribution with cooling of blood in a cold and vasodilated limb. Further studies may confirm that postoperative hyperthermia could be explained, in part, by tourniquet use. We also observed other adverse effects such as an increase in arterial pressure during tourniquet use and an increase in end-tidal carbon dioxide at tourniquet deflation.

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