Title: SYSTEMIC AND METABOLIC RESPONSES TO TOURNIQUET ISCHEMIA IN CHILDREN

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Introduction. Tourniquets are used every day to minimize bleeding in orthopedic surgery. Yet, the metabolic effects of tourniquet ischemia have not been described in children.

Methods. We studied eleven patients with a mean age of 7.4 years (range 2-10) all ASA I for elective surgery of an extremity. Informed consent was obtained in all cases under guidelines established by our Clinical Investigation Committee. Anesthesia was induced with halothane/N2O/O2 via mask. All patients were intubated, either with succinylcholine, halothane or pancuronium. Ventilation was controlled with a nonrebreathing system to produce a slight hyperventilation (pCO_2 33-38 mmHg). Anesthesia was maintained with halothane/N_2O, and narcotics given as needed. At the end of the case, patients were weaned from the ventilator no sooner than five minutes after the tourniquet was deflated. A radial artery cannula was inserted percutaneously and used for continuous blood pressure monitoring and blood sampling. The end tidal pCO_2 was monitored with a capnograph. Arterial blood samples were analyzed for lactate, myoglobin, sodium and potassium, acid base data and blood gases. Blood samples were drawn before the tourniquet was inflated, immediately before the tourniquet was released and 1, 5, 15, 30 and 60 minutes after the tourniquet release. For nine of the patients arterial lactate levels were drawn before the tourniquet was applied and 15 and 30 minutes later. The significance of the difference was determined using students t-test for paired values, where $P \leq 0.05$ was considered significant.

Results. The mean duration of tourniquet occlusion was 55.7 min (range 25-125). About 30 seconds after release of the tourniquet, the average systolic blood pressure had fallen from 113±9 mmHg to 98±4 mmHg (mean ± sd), while the heart rate increased from 105±4 to 117±11 beats/min. The blood pressure and heart rate returned to control values within 15 minutes. No cardiac arrhythmias were noted on continuous EKG monitoring.

Arterial Blood Samples

	Prerelease	Postrelease 1-Minute	5	15	30	60
K+	3.6±0.4	3.95±0.4*	3.87±0.3*	3.7±0.29*	3.6±0.37	3.6±0.28
pH	7.38±0.03	7.32±0.04*	7.35±0.04*	7.35±0.05*	7.27±0.05*	7.34±0.03*
pCO_2	36±2	44±5*	39±4*	47±7*	43±4*	40±5*
HCO_3^-	23±0.8	21.7±0.9*	22±1.3*	20.5±3*	21.8±1*	22.5±1.5
BE	-3.4±1.6	-4.5±1.6*	-4.5±1.4*	-6±3*	-5±2*	-3.9±1.6*
Lactate	1.84±0.3*	1.95±0.32*	2.71±0.02*	2.21±0.02*	1.57±0.43*	1.24±0.04

	Pretourniquet inflation	15 min on tourniquet	30 min on tourniquet
Lactate	1.07±0.2	1.44±0.3*	1.84±0.3*

- All these values are mean ± sd. K+, HCO_3^-, BE, Lactate are mmol/l; CO_2 mmHg
- * $P \leq 0.05$ when compared to initial values

The serum myoglobin was not changed by the use of the tourniquet, but dependent upon the use of succinylcholine. Mean postintubation level of myoglobin was 1007±712 ng/ml in patients who received succinylcholine as opposed to 63±81 ng/ml in those who did not (normal value up to 85 ng/ml).

Discussion. The major changes were a transient relative hyperkalemia and an acidosis. The increase in potassium was not associated with disturbances in cardiac rhythm. This increase was presumably the result of potassium release from hypoxic muscle cells. The metabolic acidosis began with the inflation of the tourniquet. Although the pCO_2 was maintained below 40 torr, the nonvolatile acids began to accumulate before the tourniquet was released. This may be due to a deep component of the circulation that allowed these wastes to seep into the general circulation despite good arterial occlusion. With the release of the tourniquet, the rapid washout of these metabolites caused a more severe acidosis. Without increasing the minute ventilation, this acidosis began to be corrected. However, once the children were taken off the ventilator, there was a rebound where the acidosis became more pronounced before resolving. This was not the case in adult studies.[1-2] Also, the metabolic acidosis was more severe in children. This may be a reflection of the young child's higher metabolic rate accumulating more wastes during the hypoxic period. The lack of increase in myoglobin demonstrates that local circulatory arrest of up to 125 minutes duration does not cause a significant ischemia muscle damage. The serum myoglobin was elevated only in those cases where succinylcholine was used. These changes, which are consistent with previous findings,[3] remained below potentially dangerous levels.[4]

References.

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