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Blood Gas and Electrolyte Changes after Tourniquet Application in Total Knee Replacement Surgery

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The tourniquet is widely used in upper and lower extremity surgery in orthopedic practice. However, safe working guidelines for the application of the tourniquet are not clearly defined. The use of a tourniquet is an important step in performing total knee arthroplasty, and it seems plausible that mechanical damage is directly related to the height and the duration of the pressure of the tourniquet applied. Even the tourniquet pressure which is widely accepted in clinical practice, if it is applied for several hours, would permanently damage not only tissues directly under the tourniquet but also the muscles and the nerves distal to the tourniquet. The resultant ischemia to limb produces local changes including hypoxemia, acidosis and hyperkalemia. Relatively little is known about the systemic effects of tourniquet release when the patient is undergoing total knee replacement surgery under a general anesthesia. Therefore, we studied the systemic effects. The results were as follows: 1) Approximately five minutes after the tourniquet was released there was a statistically significant increase in mean heart rate. : 2) Serum potassium levels tended to increase significantly until five minutes while the serum sodium level rose significantly only one minute, and the lactate level rose significantly for only two minutes after tourniquet released ; 3) $PaCO_2$ increased for five minutes after tourniquet release and remained elevatd for 30 minutes; 4) PaO_2 did not change significantly two minutes after tourniquet release; 5) The mean pH dropped to 7.34 and remained low for over five minutes.

Key Words: Tourniquet, electrolyte, blood gas

The use of a tourniquet for obtaining a bloodless field is an essential and accepted tool for extremity surgery. Postoperative complications with tourniquet use have been reported since the introduction of the Esmarch bandage. But they became rather rare after the advent of pneumatic touniquets. There is a need for information about the systemic and local effects of tourniquet use in persons of good physical status. For a proper functional state the peripheral tissues depend on an adequate supply of oxygen and an adequate microcirculation. An extensive study by Lundborg (1970) experimentally showed, however, that the intraneuronal microcirculation in the limbs of rabbits is completely reinstituted after 6-8 hours of ischemia and thus the nerves in the limb seems to have a wide margin of safety (Ocha *et al.* 1972). The cullular metabolic changes during clinical ischemia have often been indirectly approached by studies based on venous blood parameters in the ischemic extremity (Mullick 1978; Wilgis 1971). It is known from previous studies on reconstructive arterial surgery and tourniquet ischemia that there is an increased plasma potassium level after ischemia (Andersson *et al.* 1979). Potassium can also be released as a result of cellular damage. Therfore, quantitation of electrolyte changes and metabolic changes in man after prolonged operation in a bloodless field seems not only of theoretical interest but also of possible clinical importance. Therefore, we studied the systemic effects of

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a long term tourniquet application in patients who underwent a total knee replacement surgery.

MATERIALS AND METHODS

Twenty patients, 19 female and one male, between the ages of fourty-eight and seventy years (average 57.5), who underwent total knee replacement under tourniquet control were studied. Twenty patients with a mean body weight of 59.8 kg (range 49~70 kg), all ASA-physical status I or II without any neuromuscular disease or cardiovascular diseases except for mild hypertension, underwent the surgery for rheumatoid arthritis or osteoarthritis. Anesthesia was maintained with any combination of halothane and nitrous oxide, narcotics and nitrous oxide, enflurane and nitrous oxide, or narcotics, and halothane and nitrous oxide. Ventilation was controlled with a mechanical ventilator in order to maintain the end-tidal CO_2 between 30 and 40 mmHg. The end-tidal CO_2 was continuously monitored and ventilation adjusted as needed. The tidal volume, respiratory rate and FiO_2 were adjusted to maintain arterial oxygen saturation greater than 95%. Prior to the inception of the pneumatic tourniquet, rubber tourniquets were used for hemostasis. The cementless Miller-Galante three compartment total knee system was used in all patients. In all patients, the knee joint was exposed through a mid-patella incision medially, and the patella was then dislocated laterally. Soft tissue around the knee joint was handled carefully for the prevention of direct surgical cellular damage. Additional monitorings included an ECG for heart rate and detection of dysrhythmia and a percutaneous radial arterial line for continuous blood pressure monitoring and blood sampling. The pneumatic tourniquet was inflated to 400 mmHg at the thigh prior to skin incision and was released before wound closure. Tourniquet time duration ranged from 75 minutes to 90 minutes with mean duration 86.4 minutes.

Arterial sodium, potassium, pH, $PaCO_2$ and PaO_2 were measured approximately 5 minutes before the tourniquet was released, and again at 1, 5, 15, 30 and 60 minutes after deflation. Bood gases were analysed with standard electrodes at 37 degrees Celsius, and values were corrected to measured body temperature. Values as to mean with standard deviations were recorded. Statistical evaluation was done with the MANOVA test at the significance level 0.05.

RESULTS

Arterial Pressure and Heart Rates

Approximately one minute after the tourniquet was released, there was a statistically significant increase in the mean heart rate from a mean of 104 ±58 bpm to 116±54 bpm. There was also a statistically significant decrease in the mean arterial pressure from 94±27 mmHg to 73±36 mmHg. These values remained statistically different from control levels for the full 5 minutes measured (Table 1). ECG monitoring revealed no significant arrhythmia in this group. The mean operative blood loss was 436cc.

Plasma Electrolytes

The pre-tourniquet release values were within the normal range. Upon release of the tourniquet an immediate increase in plasma potassium was observed. Fig. 1 shows the plasma K^+ values. One

Table 1. Hemodyanmic changes associated with tourniquet release

	Heart rate	Mean arterial blood pressure (mmHg)
Pre-release	104±58	94±27
Post-release	116±54*	73±36*
Release+5 min	112±58*	81±54*

*$p<0.1$

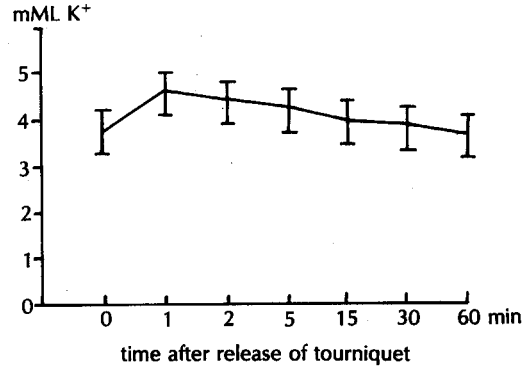

Fig. 1. *K^+ in mM/L in the arterial blood.*

minute after release, the mean value of potassium was 4.7 mM/L. In the arterial plasma potassium a significant increase was observed one to five minutes after the tourniquet was released. Even 15 minutes after reestablishment of blood flow, a statistically significant increase was seen ($p<0.1$). There was no statistically significant correlation between the potassium level and the duration of tourniquet inflation. The sodium values showed a small increase after reestablishment of blood flow in the arterial blood ($p<0.05$). Serum potassium levels returned to normal at 30 minutes after deflation and sodium levels returned to normal at five minutes.

The sequential Cl^- concentration in the blood showed no significantly different values. The CO_2 content did not change after deflation. Arterial plasma lactate concentration rose to a mean peak value of 2.45 mM/L two minutes after cessation of the tourniquet ischemia and remained significantly increased during the first five minutes (Table 2).

Blood Gas Analysis

The mean pH value decreased from a baseline of 7.45 ± 0.09 to 7.39 ± 0.09 at one minute after release of the tourniquet. After that the pH slowly increased but remianed below the baseline for 5 minutes (7.40 ± 0.22). The acid-base information demonstrated that bicarbonate was slightly below normal before tourniquet release. The mean standard bicarbonate decreased to 23.0 ± 2.2 mM/L at 2 minutes from an initial mean of 24.3 ± 2.2 mM/L. After 15 minutes, bicarbonate values returned to slightly above or below the base line. The mean P_aO_2 dropped from 147 ± 54 torr to 139 ± 45 torr at one minute. The mean P_aO_2 value at five minutes showed no significant difference from the baseline. Mean values of P_aCO_2 showed maximal increases from 36 ± 13 torr to 41 ± 9 torr at five minutes (Fig. 2). These values subsequently decreased but were

Table 2. Electrolyte chagnes of arterial blood

mM/L	Before inflation	After release of tourniquet					
		1 min	2 min	5 min	15 min	30 min	60 min
Na	142±13	146±18	143±13	141±13	141±13	142±13	141±13
K	3.8±1.8	4.7±2.7*	4.5±1.8*	4.3±1.8*	4.0±2.7	3.9±0.9	3.8±1.8
Cl	102±22	100±18	102±13	101±18	102±13	101±18	102±22
CO_2	22±13	21±9	20±18	21±9	21±13	20±13	21±9
Lactate	1.93±1.21	2.44±3.84*	2.45±3.30*	2.04±2.15	1.90±2.01	1.67±2.15	1.80±3.67

*$p<0.05$

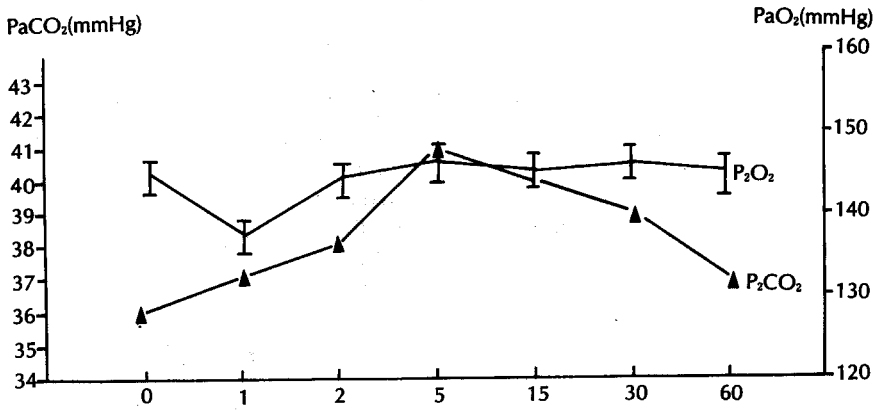

Fig. 2. PaO_2, P_aCO_2 changes of arterial blood.

Table 3. Metabolic changes associated with tourniquet release

	pH	PaO_2 mmHg	$PaCO_2$ mmHg	HCO_3 mM/L	BE mM/L
Pre-release	7.45±0.09	147±54	36±13	24.3±2.2	-4±9
Release+ 1 min	7.39±0.09*	139±45*	37±18	24.0±3.6	-3±9
Release+ 2 min	7.38±0.40*	146±45	38±13	23.0±2.2*	-4±9
Release+ 5 min	7.40±0.22*	148±36	41± 9*	24.7±3.1	-4±13
Release+15 min	7.44±0.09	147±45	40±13*	24.3±2.7	-3±13
Release+30 min	7.45±0.13	148±45	39±18*	24.8±4.5	-4±9
Release+60 min	7.44±0.22	147±45	37±13	24.3±2.7	-4±9

*$p<0.05$

still significantly elevated at 30 minutes (Table 3).

DISCUSSION

The tourniquet plays an indispensable and vital part in operations upon the extremities. It is, therefore, imperative that anyone associated with its use should have an intimate knowledge of this tool which could either be a beneficial instrument or a deleterious weapon depending on how it is used. Complete arrest of circulation of an extremity is obviously unphysiologic. During the ischemic phase, there is a fall in the temperature of the skin and the muscles of the limb, which become pulseless and pale. In two hours of tourniquet ischemia, the venous pH falls in a linear fashion (Adams 1971). At the same time, the venous PO_2 falls and the venous PCO_2 rises. And then irreversible muscle fatigubility develops. Striated muscles rendered ischemic for two hours show evidence of cellular damage and atrophy of muscle tissue, while decreases in the content of myosin, water-soluble protein and nonprotein nitrogen are also observed (Benzan *et al.* 1988; Haljamäe and Enger 1975; Solonen and Hgelt 1968). Various complications were reported on the use of the Esmarch bandage. The pneumatic tourniquet is now the best available method of applying uniform pressure to a wide area if it is correctly padded, inflated to a known pressure and monitered by an interested anesthetist. The cause of complications has been postulated as either a consequence of direct pressure of the tourniquet on a nerve or due to ischemia-induced tissue changes. It is well known that the risk of complications increases with the time of ischemia and that continuous tourniquet time of less than two hours is considered to be safe (Bruner 1951; Griffiths and Heywood 1973). The use of an intramedullary alignment rod in the distal part of the femur and cementing procedure are an important step in performing total knee replacement arthroplasty. The use of cement in total knee replacement might affect the level of the blood chemistry and gas study. On the basis of that observation, we did not use cemented total knee replacement system.

There are two investigative approaches based on blood parameters in the ischemic extremity after tourniquet inflation and on direct monitoring of cellular metabolic parameters. Tissue gas tensions are more sensitive indicators of tourniquet hypoxia than the blood gases within the ischemic extremity. However, the blood gas measurements do not monitor tissue metabolism adequately when the patient is in shock. Several authors have reported that despite inflation of a tourniquet to levels above systolic blood pressure, blood flow continues through the soft tissue vessels distal to the tourniquet. It seems reasonable to postulate that the biochemical data from venous blood during tourniquet ischemia express the sum of events within diverse tissues having differing metabolic rates and end products. However in this study, the metabolic consequences of complete tourniquet ischemia during human extremity operations are indirectly studied through the systemic (i. e. radial) arterial blood analysis of parameters such as pH, PCO_2, PO_2, standard bicarbonate, potassium and sodium as well as blood pressure and heart rate during and after ischemia (Larsson and Bergastrom 1978; Paletta *et al.* 1972). Intracellular metabolism requires a very narrow range of free hydrogen ion concentration (pH) within which enzymatic and biochemical processes

function efficiently and appropriately. Deflation of a pneumatic tourniquet following total knee replacement is considered a crisis point surgery because sudden hypotension, electrolyte and blood gas changes are likely to occur. These deflation phenomena can be minimal and temporary, but on occasion the fall of blood pressure and the elevation of potassium are profound and refractory (Fahmy *et al.* 1990; Terhan *et al.* 1971). Significant deviations from these normal ranges could be poorly tolerated and may be life-threatening. The declamping shock has been attributed to reactive hyperemia with pooling of blood in the leg, metabolic acidosis following lactate accumulation in the ischemic leg muscles, and hyperkalemia. A local collection of metabolites, tissue acidosis and potassium release can lead to localized cellular damage, and it can be deleterious to the conduction system of the heart provoking ventricular fibrillation. Therfore, a better understanding of these hemodynamic homeostatic mechanisms is important in clinical practice (Solonen *et al.* 1968). In our study, the arterial plasma potassium level was significantly increased one to five minutes after tourniquet release. However, the elevated pottasium did not affect the function of myocardial electrophysiology.

Enger *et al.* (1977) showed a significant change of transmembrane potentials during the three hours of tourniquet ischemia. In that study, in spite of restitution of blood flow after deflation of the tourniquet, normalization of the metabolic state was not seen for at least 20 minutes after release of the tourniquet. Modig's group (1978) demonstrated a higher peak lactate value, and decrease in arterial pH was observed to be less in patients undergoing knee arthroplasty with tourniquet under epidural anesthesia. In our study the maximal decrease in pH in the systemic circulation was observed about five minutes after deflation. After that the pH slowly increased but the pH did not completely correct for 15 minutes.

An ideal approach to peripheral tissue changes after tourniquet inflation would be direct measurement of tissue metabolites and cellular metabolism. During ischemia, energy is liberated through utilization of ATP giving rise to ADP and AMP. Since breakdown products of AMP diffuse across the intracellular membrane, these constituents may be lost to the blood. The decrease in the creatine pool on the other hand supports a loss throught disrupted cellular membranes. It is therefore, considered important to study direct cellular metabolic parameters such as adenosine triphosphate, and glucose-6-phosphate of skeletal muscles for more exact evaluation of the effect of tourniquet ischemia. We have not made such a direct study. In this indirect study, release of leg tourniquet ischemia seems to cause no myocardial or pulmonary disturbance, and induces only moderate and reversible changes in the acid-base status and the serum potassium level in the arterial blood. This is presumably due to the low metabolic rate of the ischemic limb and to rapid volme replacement upon release of the tourniquet, restoring a stable circulatory condition. These data seem to indicate that there was no local ischaemic muscular injury. Understanding of the systemic as well as the local effects of the pneumatic tourniquet on the limb is important for the surgeon and anaesthetist. In the study we observed that the systemic changes after up to two hours of tourniquet ischemia were moderate and reversible, even in the elderly. However, awareness as well as the ability to regulate the time and pressure of tourniquet inflation, should help minimize complications.

REFERENCES

Adams JP, Wiglis EFS: Discussion. Observation on the effects of tourniquet ischemia. *J Bone Joint Surg Am 53: 1343, 1971*

Andersson J, Eklöf B, Neglen P, Thomson D: Metabolic changes in blood and skeletal muscle in reconstructive aortic surgery. *Ann Surg 189: 283-1979*

Benzan HJ, Toleikis JR, Meagher LL, Shapiro BA, Tsa'o CH, Avrm MJ: Changes in venous blood lactate, venous blood gases, and somatosensory evoked potentials after tourniquet application. *Anesthesiology 69: 677, 1988*

Bruner JM: Safety factors in the use of the pneumatic tourniquet for haemostasis in surgery of the hand. *J Bone Joint Surg 33: 221, 1951*

Enger E, Jennische E, Medegard A, Haljamäe H: Cellular Restitution after 3 hours of complete tourniquet ischemia. *Acta Chir Scand Suppl 481: 57, 1977*

Fahmy NR, Criandler HP, Danylchuk K, Matta EB, Sunder N, Siliski JM: Blood gas and circulatory changes during total knee replacement. *J Bone Joint Surg 72-A: 19, 1990*

Griffiths JC, Heywood OB: Biomechanical affects of the tourniquet. *Hand 5: 113, 1973*

Haljamäe H, Enger E: Human skeletal muscle energy metabolism during and after complete tourniquet ischemia. *Ann Surg 182: 9, 1975*

Larsson J, Bergstrom J: Electrolyte changes in muscle tissue and plasma in tourniquet ischemia. *Acta Chir*

Scand 144: 67, 1978

Lundborg G: Ischemic nerve injury. Experimental studies on intraneuronal' microvascular patho-physiology and nerve function in a limb subjected to temporary circulatory arrest. *Scand J Plast Reconstruct Surg Suppl: 6, 1970*

Modig J, Kolstad K, Wigren A: Systemic reactions to tourniquet ischemia. *Acta Anaesthesiol Scand 22: 609-614, 1978*

Mullick S:. The tourniquet in operations upon the extremities. *Surg Gynecol Obstet 1546: 821, 1978*

Ocha J, Fowler TJ, Gilliat RW: Anatomical changes in peripheral nerves compressed by a pneumatic tourniquet. *J Anat 113: 433, 1972*

Paletta FX, William V, Ship AG: Prolonged tourniquet ischemia of the extremities; an experimental study in dogs. *J Bone Joint Surg Am 73: 433, 1972*

Solonen KA, Hgelt L: Morphological changes in striated muscles during ischemia. *Acta Orthop Scand 39: 13, 1968*

Solonen KA, Tarkkanen L, Narvanen S, Gordon R: Metabolic changes in the upper limb during tourniquet ischaemia. *Acta Orthop Scand 39: 20, 1968*

Tarhan S, Fulton RE, Moffitt EA: Body metabolism during general anesthesia without superimposed surgical stress. *Anesth Analg 50: 915, 1971*

Wilgis EF: Observations on the effect of tourniquet ischemia. *J Bone Joint Surg 53A: 1343, 1971*