

Changes in Venous Blood Lactate, Venous Blood Gases, and Somatosensory Evoked Potentials after Tourniquet Application

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The effects of inflation of a 7-cm tourniquet applied to the upper arm of eight volunteers on venous lactate, venous blood gases, and ulnar nerve somatosensory evoked potentials (SSEPs) were investigated. The inflation pressure was 100 mmHg over the systolic pressure. Venous blood samples for lactate and blood gas determinations were withdrawn before tourniquet inflation; immediately and at 2, 5, 10, 15 min after tourniquet deflation; and additionally at 30, 45, and 60 min after deflation in the last four volunteers. SSEP stimulating surface electrodes were placed over the ulnar nerve at the wrist. Recording electrodes were placed over the ipsilateral ulnar groove of the elbow, Erb's point, and on the contralateral cortex. Averaged responses were acquired before inflation of the tourniquet, every 5-10 min during tourniquet inflation, and every 5-10 min for 45-60 min after tourniquet deflation. The tourniquet was inflated for 36 ± 11 min. After deflation of the tourniquet, post-deflation pain and paresthesias were felt by five volunteers; these occurred at 30-120 s after deflation and lasted for 75-120 s. The postdeflation pains were characterized as burning, cramping, paresthesias, buzzing, or severe expansion of the hand. The venous blood lactate levels were significantly elevated for 10 min, and the time course of its change did not correlate with reperfusion pain. The P_{O_2} and O_2Hb saturation in venous blood were significantly elevated for 10-15 min after deflation. The elevated lactate and P_{O_2} levels in the presence of a restored blood flow probably result from continued anaerobic muscle metabolism secondary to capillary closure from the tourniquet-induced ischemia. The Erb's and cortical SSEPs were abolished after inflation of the tourniquet; time to 100% depression of the amplitude was 22 ± 6 min for the Erb's and 25 ± 6 min for the cortex. These changes probably resulted from nerve compression and ischemia. The percentage depression of the ulnar nerve SSEP at the elbow was $84 \pm 16\%$, presumed to result solely from nerve ischemia. The SSEPs recovered after deflation of the tourniquet. (Key words: Metabolism: lactate. Monitoring: evoked potentials. Oxygen: tension. Pain: tourniquet.)

PAIN AFTER APPLICATION of a pneumatic tourniquet is a well-recognized phenomenon.¹ In a recent study in volunteers, Hagenouw *et al.* found that the degree of inflation pressure and the method of exsanguination did not influence the onset and severity of tourniquet pain.¹ It was

also noted that sensations of tingling, buzzing, or tightness occurred after deflation of the tourniquet. The investigators theorized that this "postdeflation or reperfusion pain" is related to washout of metabolites of anaerobic metabolism accumulating during the tourniquet inflation. This study evaluates blood lactate levels and pH, P_{CO_2} , and P_{O_2} values of venous blood after tourniquet deflation. The effect of tourniquet application on somatosensory evoked potentials (SSEPs) was also studied to examine the conduction of nerve impulse beneath the tourniquet² and to evaluate impulse transmission within the ischemic limb.

Materials and Methods

The study was approved by the Human Subjects Committee of Northwestern University and the Research Committee of Northwestern Memorial Hospital, and informed consent was obtained. Eight male, healthy volunteers, ages 26-39, were studied. All subjects refrained from eating, exercising, and taking any medication for at least 8 h before the study.

Each subject was placed in a quiet room with a constant temperature between 22 and 24° C. Baseline pain visual analog scale (VAS) score was determined with a 100-mm line at which the zero end stated "I have no pain at all" and the 100-mm end stated "I feel the worse pain imaginable."³ An 18-gauge intravenous cannula was inserted into an antecubital vein, and baseline blood samples for blood lactate and blood gases (pH, P_{CO_2} , P_{O_2} , O_2Hb saturation) were obtained. Baseline ulnar nerve SSEPs were determined.

The blood pH, P_{CO_2} , and P_{O_2} were determined by an Instrument Laboratories (IL) Blood Gas Analyzer 813[®], whereas the O_2Hb saturation was determined by an IL Cooximeter 282[®]. The blood for lactate determination was collected in a test tube that contained sodium fluoride as a metabolic inhibitor and potassium oxalate as an anticoagulant.⁴ The specimen was chilled, centrifuged, and the lactate level in the plasma determined by a DuPont Automatic Clinical Analyzer[®] (aca). This method has a high specificity for lactate⁴; linearity of the assay was observed between 0 and 360 mg/dl.⁵ The technique has a 99% recovery of lactate in the blood and a 4% (± 0.09) coefficient of variation.^{5,†}

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† aca III[®] Chemistry Instruction Manual, DuPont Company, Instruments Products, Automatic Clinical Analysis Division, Wilmington, DE 19898.

A multichannel signal averager (Nicolet Pathfinder I®, Nicolet Biomedical, Madison, WI) was used to measure and record SSEPs; the technique has been described previously.³ A pair of stimulating surface electrodes were placed over the ulnar nerve at the wrist. Stimuli were applied at a rate of 8.7 Hz and an intensity level 1 mA above motor threshold. Recording electrodes were placed at the ipsilateral ulnar groove of the elbow, at Erb's point, and on the contralateral scalp over the neuronal generator areas (EEG International 10-20 locations C3' or C4'). The Erb's point is located at the neck, just above the clavicle, 5–6 cm from the suprasternal notch. C3' (left side) and C4' (right side) are located over the sensory parietal regions. The electrode at the ulnar groove recorded ulnar nerve evoked potentials before the site of tourniquet application, within the ischemic limb, whereas the Erb's and cortical electrodes recorded ulnar nerve evoked potentials beyond the site of tourniquet application. Three groups of 250 averaged responses were acquired from each recording site during each testing period. Composites were formed from the three averages. Peak-to-peak amplitudes and latencies of the composite responses were monitored and recorded.

Following baseline studies, the volunteer's arm was elevated then exsanguinated with an Esmarch bandage. A 7-cm tourniquet with a layer of soft-roll under it was applied at the mid-upper arm and inflated to 100 mmHg over the volunteer's baseline systolic blood pressure. After inflation of the tourniquet, VAS scores were recorded every 2–5 min and SSEPs were recorded every 5–10 min (each recording taking 5–10 min). The subject was encouraged to report any discomfort (tingling, warmth, numbness, or stiffness), and such complaints were noted. The tourniquet was deflated when pain became unbearable.

After deflation of the tourniquet, blood samples were obtained for lactate and venous blood gases immediately and at 2, 5, 10, and 15 min. For the last four volunteers, samples were also obtained at 30, 45, and 60 min. The SSEPs were recorded immediately after deflation and every 5–10 min for 45–60 min. VAS scores were recorded immediately after deflation, at 2 min, and then every 5 min until termination of the study.

Repeated measure analysis of variance (ANOVA) followed by Bonferroni corrected paired *t* tests compared posttourniquet lactate and venous blood gas values from baseline levels. A *P* value less than 0.05 was considered significant. The following SSEP changes were considered significant: 1) a decrease in the amplitude of 50% or more, and 2) a prolongation of the latency of 10% or greater.⁶

Results

There was a gradual increase in the VAS scores with inflation of the tourniquet, reaching a maximum of 90–

100. Tourniquet time ranged from 25 to 55 min, with a mean (\pm SD) duration of 36.2 (\pm 11.3) min. In all volunteers, the pain was felt at the site of the tourniquet and at the forearm and hand. The hand and arm pains were felt 5–10 min after inflation of the tourniquet. These pains were characterized as burning, cramping, tingling, and heavy sensations; they were more severe than the tourniquet pain in six of the eight volunteers. The hand and fingers later became numb and "paralyzed."

After inflation of the tourniquet, there was a progressive prolongation of the latencies and diminution of the SSEP amplitudes. The Erb's and cortical amplitudes were abolished in all subjects (table 1); time to 100% depression of the amplitude ranged from 15 to 30 min (mean \pm SD, 22.5 \pm 5.34) for the Erb's and 15–35 min (mean \pm SD, 25 \pm 5.98) for the cortical amplitude. The percentage depression of the ulnar nerve SSEP at the elbow ranged from 52 to 100% with a mean depression of 84.2 \pm 15.9%. The percentage prolongation of the latency of the ulnar nerve evoked potential at the elbow ranged from 52 to 112% (mean \pm SD, 91.8 \pm 20.8).

Deflation of the tourniquet relieved the pain in all volunteers. Reperfusion pain occurred in five of eight volunteers. These painful sensations were characterized as burning, cramping, paresthesias, buzzing, or severe expansion of the hand ("blowing up"). Reperfusion pain started 30–120 s after deflation of the tourniquet and lasted for 75–120 s. The mean tourniquet time of the five experiencing postdeflation pain was 41 (\pm 11) min; the mean tourniquet time of the three volunteers who did not was 28 (\pm 6) min.

All the SSEPs appeared within 5 min after deflation of the tourniquet, except in the volunteer who tolerated 55 min of inflation. His elbow SSEP appeared immediately, but his cortical and Erb's potentials appeared at 30 and 70 min, respectively. The ulnar nerve amplitude at the elbow completely recovered in six volunteers; time to 100% recovery ranged from 5 to 115 min (mean \pm SD; 26.7 \pm 43.4). The Erb's SSEP amplitude completely recovered in seven volunteers; time to 100% recovery ranged from 1 (immediate) to 70 min (mean \pm SD, 15.9 \pm 24.3). The cortical SSEP amplitude regained its baseline value in seven volunteers; time to 100% recovery ranged from 1 to 30 min (mean \pm SD, 13 \pm 12.4). The elbow, Erb's, and cortical SSEP amplitudes of the volunteers with less than 100% recovery were 72–88% of their baseline values (table 1). They attained these values within 10–45 min after tourniquet deflation; there was no further amplitude increase after this time. There was progressive improvement (decrease) of the latencies with time; the latencies of the three amplitudes were all within 9% of their baseline values at the conclusion of the study (table 1). These SSEP changes with tourniquet inflation and deflation are demonstrated in figure 1. Seven of the eight

TABLE 1. SSEP Amplitude Changes after Tourniquet Application

Volunteer	Inflation Time (min)	Percent Block, Elbow	Time to 100% Block (min)		Time to 100% Recovery (min)		
			Erb's	Cortex	Elbow	Erb's	Cortex
1	25	52	25	25	5	(72% at 15 min)	15
2	55	83	20	20	115	70	30
3	35	93	30	30	10	10	(83% at 10 min)
4	45	100	15	35	15	15	30
5	45	73	25	25	10	1	1
6	25	100	25	25	(88% at 45 min)	5	5
7	25	90	15	15	(85% at 15 min)	5	5
8	35	83	25	25	5	5	5
Mean percentage depression of amplitude		84 ± 16	100 ± 0	100 ± 0	-	-	-
Mean percentage recovery of amplitude		-	-	-	97 ± 6	96 ± 10	98 ± 6
Mean percentage prolongation of latency		92 ± 21	—*	—*	-	-	-
Mean percentage recovery of latency		-	-	-	96 ± 3	97 ± 4	99 ± 2

* Latency cannot be determined; the amplitude was abolished.

volunteers had single SSEP amplitude in all three recording sites during the period of recovery. The volunteer who had a 55-min duration of tourniquet application had temporal dispersion of the SSEP at his elbow 70 min after tourniquet deflation. These two components later merged into a single, well-defined response (fig. 2).

The blood lactate levels immediately at 2, 5, and 10 min after tourniquet deflation were significantly ($P < 0.05$) higher than the baseline value (table 2). The levels then progressively decreased and returned to the baseline values at 30–60 min. Blood lactate levels ranged from 15 to 28 mg/dl (mean ± SD, 21 ± 6) in those with reperfusion pain compared with 16–22 mg/dl (mean ± SD, 19 ± 3) in those without reperfusion pain. There was no signifi-

cant decline in lactate from 2 min when reperfusion pain was maximal to 5 min when reperfusion pain was not felt any longer.

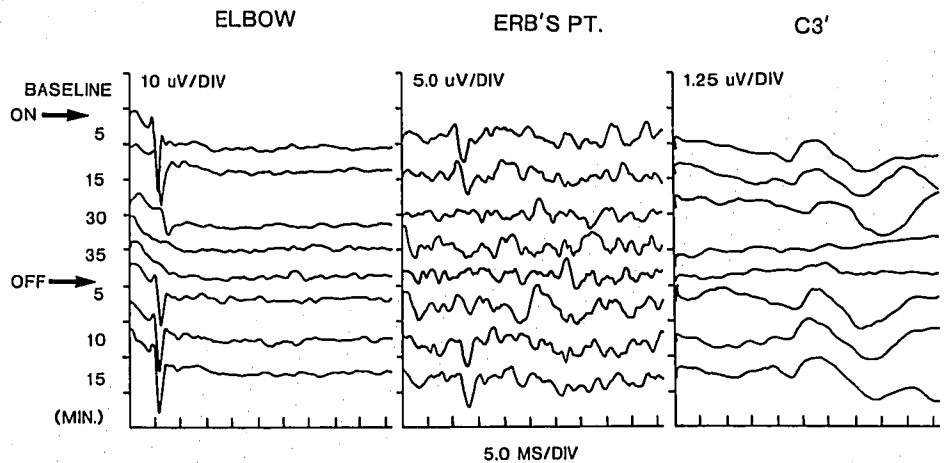
As shown in table 2, immediately after tourniquet deflation the venous blood pH decreased compared with the baseline value but returned to normal value at 2 min. The venous P_{O_2} at 2, 5, and 10 min and the oxyhemoglobin saturation at 2 and 5 min postdeflation were significantly higher ($P < 0.05$) than baseline and were seen in all volunteers.

Discussion

The occurrence of paresthesia after deflation of the tourniquet has been described previously.^{1,7,8} Percuta-

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FIG. 1. Ulnar nerve SSEP changes in a volunteer after tourniquet inflation and deflation. Fifteen minutes after tourniquet inflation, the SSEP amplitude at the elbow and Erb's were depressed by 60% and at the cortex by 30%. The Erb's amplitude was abolished at 30 min and the cortical amplitude at 35 min. Percentage depression at the elbow at 35 min, just before tourniquet deflation, was 93%. After tourniquet deflation, the elbow and Erb's amplitude completely recovered at 10 min, whereas the cortical amplitude stabilized at 83% recovery.



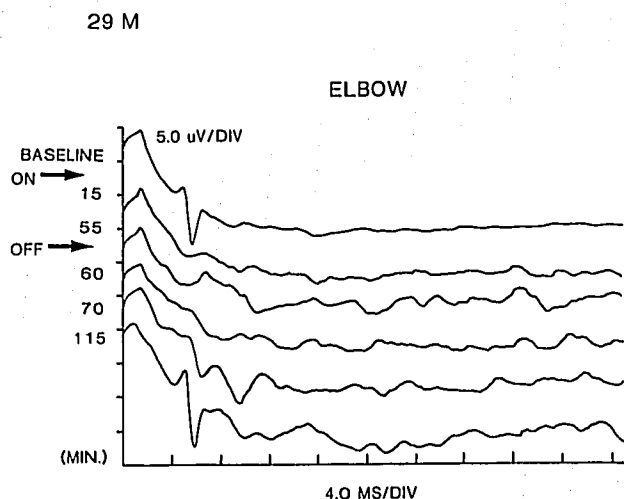


FIG. 2. Ulnar nerve SSEP tracings at the elbow, showing temporal dispersion during recovery, 70 min after tourniquet deflation. This volunteer had 55 min of tourniquet inflation.

neous intraneural microelectrode recordings from sensory nerve fascicles in awake subjects correlated these paresthesias with spontaneous increases in neural activity, including bursts of high-frequency impulses (200–300 impulses/s).⁸ The cessation of neural firing coincided with disappearance of the paresthesia. Repeated tourniquet application exhausted the paresthesia and the paroxysmal neural discharges. Local anesthetic block significantly decreased the paresthesia even though the spontaneous neural activity was still present.⁸ This observation probably explains the absence of paresthesia after tourniquet release in patients who had spinal anesthesia.⁹

The factors that incite the reperfused ischemic nerves to spontaneously discharge and cause paresthesias are not known. It has been postulated that reperfusion pains and paresthesias may be related to the washout of the intracellularly accumulated products of anaerobic metabolism as reperfusion of the tissues resumes.¹ Our study showed

that the time course of change in blood lactate did not correlate with reperfusion pain. The postdeflation pain and paresthesias in our volunteers were maximal at 2 min postdeflation and disappeared by 5 min, yet the mixed venous blood lactate levels were not significantly different during these periods. Paresthesias were also not felt immediately upon release of the tourniquet when the blood lactate levels were the highest. The mixed venous blood lactate levels do not directly reflect intraneural lactate levels, however, and it is possible that a decrease in neural lactate levels triggered the abnormal increase in nerve activity.

Lactate, derived mainly from skeletal muscle and erythrocytes with normal venous blood concentration from 4 to 12 mg/dl, is the end product of anaerobic metabolism.⁴ Tissue hypoxia secondary to circulatory, hematologic, respiratory, and cellular dysfunction leads to increased production of lactate.¹⁰ Lactate levels are elevated after tourniquet inflation and return to baseline values within 5–30 min after deflation, depending on the duration of tourniquet application.^{11,12} We initially determined the lactate levels up to 15 min after deflation in the first four subjects and found that the levels remained elevated at this time. We therefore lengthened our sampling to 1 h. The persistent elevated lactate levels, after restoration of blood flow, may result from slow washout of accumulated lactate from constricted tissue compartments or from continued anaerobic muscle metabolism.^{13,14} Muscle biopsies from the lateral vastus muscle of the leg (where the circulation is first restored¹⁴) showed continued production of lactate up to 4 h after aortic declamping.¹⁴

Venous P_{O_2} and oxyhemoglobin saturation increased, almost approaching arterial values, after deflation of the tourniquet. This surprising phenomenon has been observed and ascribed to arterial-to-venous shunting.¹⁵ A recent study that recorded arterial rather than venous blood gas levels did not note these changes.¹⁶

Yamada *et al.* noted abolition of the Erb's potential and

TABLE 2. Lactate and Venous Blood Gas Changes after Tourniquet Deflation

	Baseline (Before Inflation)	Minutes after Tourniquet Deflation							
		Immediate	2	5	10	15	30	45	60
Lactate (mg/dl)	9 ± 4	28 ± 9*	20 ± 5*	18 ± 2*	18 ± 3*	15 ± 3	10 ± 1	9 ± 1	7 ± 1
pH	7.37 ± 0.03	7.30 ± 0.04*	7.38 ± 0.02	7.40 ± 0.02	7.40 ± 0.02	7.40 ± 0.02*	7.40 ± 0.02	7.40 ± 0.02	7.41 ± 0.01
P_{CO_2} (mmHg)	50 ± 4	63 ± 9*	47 ± 4	43 ± 3*	44 ± 2*	44 ± 4*	48 ± 3	45 ± 3	45 ± 3
P_{O_2} (mmHg)	33 ± 6	41 ± 15	63 ± 15*	63 ± 14*	54 ± 12*	48 ± 13	44 ± 17	50 ± 18	44 ± 16
O_2 Hb saturation (%)	62 ± 11	63 ± 22	90 ± 6*	91 ± 4*	82 ± 13	74 ± 20	66 ± 18	73 ± 17	69 ± 6

Values are mean ± SD.

* $P < 0.05$ versus baseline.

almost complete disappearance of the later cortical SSEP components after 24 min of tourniquet inflation.² We noted abolition of the Erb's and cortical potentials after 15–35 min of tourniquet application (table 1). The inflation pressures in Yamada's and our study were 80 and 100 mmHg, respectively, above the subject's systolic blood pressure. The differences in the time to 100% block of the Erb's and cortical potentials result from differences in the susceptibility of the nerves to ischemia and compression. Although a uniform amount of tourniquet pressure was given, the actual amount of pressure to the nerve varied because of differences in the amount of muscle and fat covering the nerve. Yamada *et al.* ascribed the abolition of the Erb's and cortical SSEPs to nerve ischemia. However, the effect of mechanical compression by the tourniquet could be another factor. Changes in the ulnar nerve potentials at the elbow, seen in our study and not measured by Yamada, must result solely from nerve ischemia, whereas changes in the Erb's and cortical SSEPs result from nerve ischemia and compression by the tourniquet. Another study showed disappearance of the ulnar nerve (wrist to elbow) action potential after 20–25 min of tourniquet application.¹⁷ This study, however, did not follow transmission of the nerve impulse beyond the tourniquet.

The persistence of the ulnar nerve SSEP at the elbow in contradistinction to the Erb's and cortical potentials results from the ability of nerve fibers distal to the tourniquet to conduct impulses.¹⁸ Tourniquet inflation does not necessarily mean complete ischemia because blood vessels through the bone remain open and so are the small blood vessels in the interstices between the nerve bundles.¹⁹ Demyelination changes have been found to be restricted to the area under the tourniquet.^{18,20} The differences in the percentage block of the SSEP at the elbow result from differences in the degree of nerve ischemia, the amount of blood supply, and variations in nerve susceptibility to ischemia.

There was rapid recovery of the SSEP amplitudes after tourniquet deflation.^{2,17} We noted almost immediate reappearance of the SSEPs, except in the volunteer who had 55 min inflation of the tourniquet. His ulnar nerve SSEP at the elbow, which reappeared immediately, showed temporal dispersion at the 70-min postdeflation time (fig. 2). Temporal dispersion of evoked muscle action potentials have been noted after prolonged tourniquet application²¹ and may result from differences in the rate of recovery of the nerve fibers. His cortical response initially appeared at 30 min, whereas his Erb's potential did not reappear until 70 min after tourniquet deflation. Once the cortical and Erb's SSEPs appeared, they attained 100% of their baseline amplitudes. The delayed appearances of the cortical and Erb's potentials may result not only from their prolonged recoveries but also from increased noise

(from involuntary muscle activity) masking small SSEP signals. The differences in the rate of recovery of the nerve fibers, as shown by the temporal dispersion on figure 2, may have resulted in diminished and unrecognizable SSEP signals until at 30 and 70 min, when the cortical and Erb's potentials became clearly recognizable. The earlier appearance of the cortical amplitude results from the ability of the cortex to amplify nerve impulses.^{2,3,22}

There was no further increase in the amplitudes of the four volunteers with less than 100% recovery of the SSEPs (table 1). Inconsistent increases in amplitude are not unusual during periods of recovery. It is for this reason that a change of 50% or greater is necessary before it is considered significant.⁶ In all the volunteers, the latencies progressively decreased with time. Changes in SSEP latencies are more consistent and are considered better indices of nerve recovery.

Skin temperature was not measured in our study because the ambient temperature was maintained in a very narrow range and because it does not necessarily reflect temperature in the nerve. Castaigne *et al.* believed that the changes in peripheral conduction after tourniquet ischemia were not related to temperature because the average temperature reduction in the ischemic limb after 30 min of tourniquet application was only 2.5° C.¹⁷ In addition, the reduction in the amplitude began to appear after 3–5 min of ischemia and reappeared 15–30 s after release of the cuff.¹⁷ Another study showed only a slight decrease of the posterior tibial nerve SSEP amplitude from 1.7 to 1.2 uV when the nasopharyngeal temperature decreased from 36 to 27° C and the lower limb muscle temperature decreased from 33 to 30° C during cardiopulmonary bypass.²³

In summary, we confirmed the observation of Hagenouw *et al.* on the recurrence of pain after deflation of a tourniquet. We found no correlation between venous lactate levels and reperfusion pain. We also noted that anaerobic muscle metabolism continues for several minutes after restoration of blood flow; this may result from persistent capillary closure secondary to tourniquet-induced ischemia. With tourniquet inflation, the Erb's and cortical SSEPs were abolished, assumed secondary to nerve compression and ischemia. The significant depression of the ulnar SSEP at the elbow and significant prolongation of its latency are secondary to nerve ischemia. The evoked potentials recovered after deflation of the tourniquet.

References

1. Hagenouw RRP, Bridenbaugh PO, Van Egmond J, Steubing R: Tourniquet pain: A volunteer study. *Anesth Analg* 65:1175–1180, 1986
2. Yamada T, Muroga T, Kimura J: Tourniquet-induced ischemia and somatosensory evoked potentials. *Neurology* 31:1524–1529, 1981

3. Benzon HT, Toleikis JR, Shanks C, Ramseur A, Sloan TB: Somatosensory evoked potential quantification of ulnar nerve blockade. *Anesth Analg* 65:843-848, 1986
4. Tietz NW: *Fundamentals of Clinical Chemistry*, third edition. Philadelphia, WB Saunders, 1987, pp 442-443
5. Westgard JO, Lahmeyer BL, Birnbaum ML: Use of the DuPont "Automatic Clinical Analyzer" in direct determination of lactic acid in plasma stabilized with sodium fluoride. *Clin Chem* 18:1334-1338, 1972
6. Grundy BL, Nash CL, Brown RH: Deliberate hypotension for spinal fusion: Prospective randomized study with evoked potential monitoring. *Can Anaesth Soc J* 29:452-461, 1982
7. Merrington WR, Nathan PW: A study of postischaemic paraesthesia. *J Neurol Neurosurg Psychiatry* 12:1-18, 1949
8. Ochoa JL, Torebjork HE: Paraesthesia from ectopic impulse generation in human sensory nerves. *Brain* 103:835-853, 1980
9. Bridenbaugh PO, Hagenouw RRP, Gielen MJM, Edstrom HH: Addition of glucose to bupivacaine in spinal anesthesia increases incidence of tourniquet pain. *Anesth Analg* 65:1181-1185, 1986
10. Oliva PB: Lactic acidosis. *Am J Med* 48:209-225, 1970
11. Haljamae H, Enger E: Human skeletal muscle energy metabolism during and after complete tourniquet ischemia. *Ann Surg* 182:9-14, 1975
12. Larsson J, Hultman E: The effect of long-term arterial occlusion on energy metabolism of the human quadriceps muscle. *Scand J Clin Lab Invest* 39:257-264, 1979
13. Andersson J, Eklöf B, Neglén P, Thomson D: Metabolic changes in blood and skeletal muscle in reconstructive aortic surgery. *Ann Surg* 189:283-289, 1979
14. Eklöf B, Neglén P, Thomson D: Temporary incomplete ischemia of the legs induced by aortic clamping in man. Effects on central hemodynamics and skeletal muscle metabolism by adrenergic block. *Ann Surg* 193:89-98, 1981
15. Mansberger AR, Cox EF, Flotte CT, Buxton RW: "Washout" acidosis following resection of aortic aneurysms: Clinical metabolic study of reactive hyperemia and effect of dextran on excess lactate and pH. *Ann Surg* 163:778-787, 1966
16. Brustowicz RM, Moncorge C, Koka BV: Metabolic responses to tourniquet release in children. *ANESTHESIOLOGY* 67:792-794, 1987
17. Castaigne P, Cathala HP, Beaussart-Boulengé L, Petrover M: Effect of ischaemia on peripheral nerve function in patients with chronic renal failure undergoing dialysis treatment. *J Neurol Neurosurg Psychiatry* 35:631-637, 1972
18. Mayer RF, Denny-Brown D: Conduction velocity in peripheral nerve during experimental demyelination in the cat. *Neurology* 14:714-726, 1964
19. Denny-Brown D, Brenner C: Paralysis of nerve induced by direct pressure and by tourniquet. *Arch Neurol Psychiatry* 51:1-26, 1944
20. Ochoa J, Danta G, Fowler TJ, Gilliat RW: Nature of the nerve lesion caused by a pneumatic tourniquet. *Nature* 233:265-266, 1971
21. Fowler TJ, Danta G, Gilliat RW: Recovery of nerve conduction after pneumatic tourniquet: Observations on the hind limb of the baboon. *J Neurol Neurosurg Psychiatry* 35:638-647, 1972
22. Elsen A, Purves S, Hoirch M: Central nervous system amplification: Its potential in the diagnosis of early multiple sclerosis. *Neurology* 32:359-364, 1982
23. Leyssius AT, Kalkman CJ, Bovill JG: Influence of moderate hypothermia on posterior tibial nerve somatosensory evoked potentials. *Anesth Analg* 65:475-480, 1986