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REFERENCES

1. Shin B, Mackenzie CF, Helrich M: Hypokalemia in trauma patients. ANESTHESIOLOGY 65:90–92, 1986
2. D'Silva JL: The action of adrenaline on serum potassium. J Physiol (Lond) 82:393–398, 1934
3. Brown MJ, Brown DC, Murphy MB: Hypokalemia from beta$_2$-receptor stimulation by circulating epinephrine. N Engl J Med 309:1414–1419, 1983
4. Nordrehaug JE, Johannesen KA, von der Lippe G, Myking OL: Circulating catecholamine and potassium concentrations early in acute myocardial infarction: Effect of intervention with timolol. Am Heart J 110:944–948, 1985
5. Williams ME, Rosa RM, Silva P, Brown RS, Epstein FH: Impairment of extrarenal potassium disposal by α-adrenergic stimulation. N Engl J Med 311:145–149, 1984
6. McIntyre RW, Knopes KD, Ossey KD: The relationship between sympathoadrenal activity and extrarenal potassium regulation. ANESTHESIOLOGY 63:230–231, 1985
7. Hill AB, Nahrwold ML, Noonan D: A comparison of methods of blood withdrawal and sample preparation for potassium measurements. ANESTHESIOLOGY 53:60–63, 1980
8. Fell D, Derbyshire DR, Maile CJD, Larsson IM, Ellis R, Achola KJ, Smith G: Measurement of plasma catecholamine concentrations. Br J Anaesth 57:770–774, 1985

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Abolishing Pain on Injection of Etomidate

To the Editor:—We have been impressed with the rapid awakening and clear postoperative sensorium in adult outpatients undergoing anesthetic induction with etomidate. Pain on injection in these alert, unpremedicated patients can be a problem. This incidence is reported to be as high as 50–60%.[1,2]

We have found a simple technique which has so far reduced the incidence of pain on injection to zero. Just prior to induction, 25–100 mg of lidocaine is given through an injection port attached directly to the intravenous catheter. As soon as the injection is made, the intravenous drip is turned off for 30 s. Etomidate is then injected.

Using this technique, we have had no pain on injection through either spontaneous complaint or direct questioning in 30 consecutive patients.

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REFERENCES

1. Clarke RSJ: Comparative merits of intravenous anesthetic agents for out-patient surgery. Int Anesthesiol Clin 20:51–69, 1982
2. McCollum JSC and Dundee JW: Comparison of induction characteristics of four intravenous anaesthetic agents. Anaesthesia 41:995–1000, 1986

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Trunk Skin Temperature After Sympathetic Nerve Block—Is the Heat Really On?

To the Editor:—In a recent study, Chamberlain *et al.*[1] concluded, based on measurements of trunk skin temperature by infrared thermography, that sympathetic block can extend up to ten segments above the sensory block with spinal anesthesia. A surprising finding, indeed.

The authors can be congratulated for their provoking and stimulating paper, because it reminds us that we are blind when it comes to evaluation of the extent of sympathetic block with spinal or epidural anesthesia. Our assumption, based upon similar size of fibers carrying sympathetic and thermoreceptor traffic, that loss of temperature discrimination has the same level as loss of sympathetic outflow, may not be valid. Thus, the anesthesia community would receive with enthusiasm any monitor which reliably detects the level of sympathetic block, especially in the unconscious patient. Also, the

nagging physiological question of how trunk skin circulation is regulated is unresolved.

Unfortunately, however, the issue is not straightforward, and the conclusions reached by Chamberlain *et al.* may be premature.

1) Apart from tissue conductance and, thus, skin blood flow, skin temperature also depends on core and ambient temperatures.[2] Changes in skin temperature can qualitatively reflect skin blood flow only to the extent that both core and ambient temperatures are constant. It is questionable if skin temperature can simply be "corrected for cooling" by subtracting changes in oral temperature.

2) Skin temperature may be increased by heat convection from underlying active muscle.[3] Could detectable or nondetectable shivering of the upper trunk above the level of motor block explain the observed increase in skin temperature?[2]

3) Sympathetic block not only prevents neuronally mediated vasoconstriction, but may also block nerve traffic to the adrenal medulla,[4] thereby possibly influencing blood catecholamine levels. Indeed, studies in both humans[5] and dogs[6] have shown that epidural block can lower profoundly plasma norepinephrine concentrations and also influences vasopressin and renin levels.[5,6] Can these pharmacological effects explain the observed increase in skin temperature, rather than the nerve block itself?

4) Chamberlain *et al.* do not provide the reader with any hemodynamic data, *e.g.*, blood pressure, except for the fact that seven of their 20 patients needed treatment with ephedrine and/or volume after data collection. To what extent can changes in blood pressure or cardiac output alter regional skin blood flow and temperature, either passively or through reflexes?

5) Despite an upper sensory level of only T-5, the authors found increased skin temperatures in all dermatomes (T-1 through L-2) studied. This is surprising, because upper limb vasoconstriction has been demonstrated by whole body scintigraphy[7] and plethysmography[8] after epidural block with a level as high as T2-T3 (pinprick). Only after extension of the block above T-1 did arm blood flow increase.[8] While differences between epidural and spinal anesthesia probably exist, a drop in trunk skin temperature and a variable skin blood flow have been reported after spinal anesthesia with a sensory block of T-10 or above.[9] In our experiments in conscious dogs,* in which complete sympathetic block (sensory block of T-1, Horner's sign, absence of blood pressure increase after brief clamping of chronically exteriorized carotid arteries) was induced by epidural lidocaine, we have observed a decrease in trunk skin temperature, while the skin temperature in the upper and lower limb simultaneously increased. This was true with constant ambient temperatures both above and below the dog's thermoneutral temperature and with core temperature unchanged.

6) Finally, Chamberlain *et al.*, unfortunately, do not provide us with statistical data showing that trunk skin temperature does indeed significantly increase in the various dermatomes.

Skin blood flow and temperature are influenced by a variety of mechanisms, including those unrelated to the sympathetic nervous system. Accordingly, skin temperature is, at best, an indirect means for assessment of sympathetic function, with no golden standard available for clinical comparison.

While data as such provided by Chamberlain *et al.* are useful for broadening our understanding of the physiology of the autonomic nervous system, caution must be exercised when relating changes in skin temperature directly to sympathetic function.

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REFERENCES

1. Chamberlain DP, Chamberlain BDZ: Changes in the skin temperature of the trunk and their relationship to sympathetic blockade during spinal anesthesia. ANESTHESIOLOGY 65:139–143, 1986
2. Thauer R: Circulatory adjustments to climate requirements, Handbook of Physiology, Circulation, volume 3. Washington, American Physiological Society, 1965, pp 1921–1966
3. Cooper T, Randall WC, Hertzman AB: Vascular convection of heat from active muscle to overlying skin. J Appl Physiol 14: 207–211, 1959
4. Malmejac J: Activity of the adrenal medulla and its regulation. Physiol Rev 44:186–218, 1964
5. Ecoffey C, Edovard A, Pruszczynski W, Taly E, Samii K: Effects of epidural anesthesia on catecholamines, renin activity, and vasopressin changes induced by tilt in elderly men. ANESTHESIOLOGY 62:294–297, 1985
6. Stanek B, Schwartz M, Zimpfer M, Raberger G: Plasma concentrations of noradrenaline and adrenaline and plasma renin activity during extradural blockade in dogs. Br J Anaesth 52: 305–311, 1980
7. Arndt JO, Hock A, Stanton-Hicks M, Stuhmeier KD: Peridural anesthesia and the distribution of blood in supine humans. ANESTHESIOLOGY 63:616–623, 1985
8. Bonica JJ, Berges PV, Morikawa K: Circulatory effects of peridural block. ANESTHESIOLOGY 33:619–626, 1970
9. Bengtsson TI: Changes in skin blood flow and temperature during spinal analgesia evaluated by laser doppler flowmetry and infrared thermography. Acta Anaesthesiol Scand 28:625–630, 1984

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