PvO₂ changes in cutaneous veins during regression of spinal anaesthesia

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Following transurethral resection of the prostate under spinal anaesthesia, 26 patients were divided into two groups according to the sensory levels measured in the recovery room: Group A: 20 patients with sensory level T₁₀ or above, and Group B: six patients with sensory level T₁₁ or T₁₂. Through an intravenous catheter placed preoperatively in a superficial vein of the foot, serial blood samples were drawn to measure the PvO₂. In Group A, the PvO₂ increased significantly after spinal anaesthesia compared with preoperative control values, then decreased when the sensory level receded to T₁₁ or T₁₂. In Group B, the changes in PvO₂ from control to postspinal and to motor recovery were not significant. The authors postulate that sympathetic denervation induced by spinal anaesthesia increases the PvO₂ by the opening of arteriovenous anastomoses in the cutaneous circulation, and the PvO₂ decreases with sympathetic recovery. These findings confirm that significant sympathetic denervation of the lower limbs can be expected at a sensory level of T₁₀ or above, and sympathetic recovery begins when the sensory level recedes below T₁₀.

Key words

ANAESTHETIC TECHNIQUES: spinal; OXYGEN: venous oxygen pressure.

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We previously demonstrated that increased cutaneous blood flow to an affected digit following sympathetic denervation increased the pulse volume amplitude of a photoelectric plethysmograph.¹ The increased pulse volume amplitude of a toe following spinal anaesthesia began to decrease when the sensory level receded below T₁₀, suggesting sympathetic recovery initiates at this sensory level.² In a further study of circulatory kinetics, the pulse volume amplitude was shown to result from pulsating venous flow generated by shunting of the arterial pulse via open arteriovenous (AV) anastomoses.³ Since cutaneous blood flow, pulse volume amplitude, and AV shunting seem to be commonly controlled by sympathetic tone, sympathetic denervation and recovery during the course of spinal anaesthesia should accordingly change PvO₂ in the superficial vein of the foot. Our present study seeks to identify whether such changes in PvO₂ occur as the sensory level recedes below T₁₀ during regression of spinal anaesthesia.

Methods

The study was approved by the institutional review board for human studies. The subjects were 26 consenting patients who underwent transurethral resection of the prostate (TUR-P) under spinal anaesthesia. The mean age was 72 (range 55–87). Patients with documented neurologic disease or with vascular insufficiency of the lower limb were excluded. Also excluded were patients whose foot vein could not be cannulated preoperatively, or whose mental status was not clear enough to allow evaluation of the sensory level with a pinprick method. In the preoperative holding area, in addition to a regular intravenous cannula, a superficial vein of the foot or the saphenous vein at the ankle was cannulated with a 22-gauge catheter with the tip directed distally. The first preoperative control PvO₂ was measured from the blood sample taken through the catheter. The cannulation and sampling were accomplished without applying a tourniquet and with the patient in the supine position. The catheter was then plugged with a rubber pad (Injection Site®), after flushing with a heparin solution, until the next sample was obtained. Special anaesthetic or surgical

restrictions were not imposed for the study, except that all patients were treated in a similar manner, as used most commonly for TUR-P in our hospital. Spinal anaesthesia was administered with the patient seated by injecting 10–12 mg of hyperbaric tetracaine, without epinephrine.

None of the patients were premedicated, but most were sedated during the procedure, with diazepam, 5–10 mg IV. All breathed room air throughout the study period. The bladder was continuously irrigated with 1.5 per cent glycine solution (approximately 200 mOsm·L⁻¹) intra-operatively and normal saline postoperatively. Operative time, intraoperative blood loss, and crystalloid replacement were variable. The patients arrived in the recovery room in the supine position and were alert enough to allow measurement of the sensory level with a pinprick method. Depending upon the initial sensory level measured, the patients were assigned to two groups: the sensory levels of Group A (20 patients) were T₁₀ or above, and Group B (six patients) were T₁₁ or T₁₂. The PvO₂ was measured three times for Group A and twice for Group B in the recovery room at the following times: when the sensory level was at T₁₀ or above (Group A), when the sensory level receded to T₁₁ or T₁₂ (Group A), or after initial measurement of sensory level (Group B), and when the patients in both Groups A and B were able to lift their hip by their own power (motor recovery).

Results are expressed as means $\pm$ standard deviations. One-way analysis of variance was performed to compare the changes in PvO₂ in each of the two groups A and B. A $p < 0.05$ was considered statistically significant.

Results

The results are summarized in the Table. In Group A the PvO₂ rose from the preoperative control value, 4.9 ± 1.5 , to 8.7 ± 1.1 kPa when the sensory level was at T₁₀ or above, then decreased to 5.3 ± 1.3 kPa when the sensory level receded to T₁₁ or T₁₂. A further decrease (4.6 ± 0.9 kPa) was observed with motor recovery. These changes in PvO₂ were statistically significant. In Group B the changes in PvO₂ from the control value (4.5 ± 0.7 kPa), to postspinal (4.5 ± 1.2 kPa) and to motor recovery (3.8 ± 1.0 kPa) were not statistically significant. Despite motor paralysis of the feet at a sensory level of T₁₁ or T₁₂, clinical signs of sympathetic denervation (venodilation, increased skin temperature, and hyperaemia) were not observed in patients in Group B.

Discussion

Cutaneous blood flow can vary from 1 to as much as 150 ml/100 grams of skin/min. These variations are possible because high pressure arteriolar blood can be shunted through AV anastomoses into highly compliant low pressure subdermal venous plexuses. The AV anasto-

TABLE PvO₂ (kPa) during regression of spinal anaesthesia (Mean $\pm$ SD)

	Group A (N = 20)	Group B (N = 6)
Control	4.94 ± 1.51	4.49 ± 0.72
T ₁₀ or above	8.71 ± 1.14	—
T ₁₁ or T ₁₂	5.33 ± 1.25	4.52 ± 1.24
Motor recovery	4.63 ± 0.91	3.77 ± 1.04

Changes in PvO₂ in Group A are significant (one-way analysis variance).

moses, unique structures of the cutaneous circulation, have little myogenic tone, and are innervated by sympathetic vasoconstrictor fibers.^{4,5} Using a microsphere technique, Cronenweil *et al.*⁶ measured blood flow through AV anastomoses in dogs. Sympathectomy increased flow through anastomoses from 7 ml·min⁻¹ (control) to 30 ml·min⁻¹. Electrical stimulation of the distal end of the transected sympathetic chain immediately and reversibly decreased in flow rate.

Superficial veins in the foot receive their blood mainly from the skin; therefore, opening of the AV shunts associated with sympathetic denervation greatly increases the venous flow. Clinical correlates of enhanced AV shunting would include hyperaemia resulting from elevated PvO₂, venodilation to accommodate extra blood coming through AV shunts, and increased skin temperature due to filling of subdermal venous plexuses. With respect to the PvO₂, Stevens *et al.*⁷ reported a 14 per cent increase in venous oxygen saturation in the lower extremity following spinal anaesthesia. This rise in oxygen saturation was however attributed to maldistribution of cardiac output.

According to known anatomy, the sympathetic innervation of the lower extremity originates in the preganglionic fibres from T₁₁ through L₂. After passing through the dura some preganglionic fibres end in the nearest paravertebral ganglia, but the majority either ascend or descend two or three spinal segments in the sympathetic chain before forming synapses with multiple postganglionic fibers. Thus sympathetic motor pathways are not distributed in a segmental manner as are somatic sensory fibres, and stimulation or paralysis of a single preganglionic fibre produces a diffuse response. Therefore, to achieve a complete sympathetic denervation of the lower limb, the preganglionic fibers T₁₁ through L₂ should be blocked. This should occur with spinal anaesthesia that yields a sensory level at T₁₀ or above. As the level recedes to T₁₁, recovery of some of the T₁₁ preganglionic fibres should produce a partial but diffuse sympathetic recovery of the lower limb. As the spinal anaesthesia continues to recede, the more pronounced and ultimately complete sympathetic recovery will follow.

This sequence of sympathetic recovery during regression of spinal anaesthesia was well reflected by changes in PvO_2 in our present study. However, sympathetic responses in a partially recovered sympathetic system in our patients could be enhanced by postoperative bladder filling and/or spasm caused by continuous bladder irrigation with normal saline.

Using a sensitive Thermographic Imager, Chamberlain and Chamberlain⁸ measured an increase in skin temperature at the dermatomal level as an indicator of truncal sympathetic blockade during the onset of spinal anaesthesia. They found that mean sympathetic-sensory differential was more than four spinal segments higher than the conventionally recognized two to three segmental differences. This extensive sympathetic blockade was explained by a partial sympathetic blockade resulting from overlapped sympathetic innervation. In an editorial comment to this study, Greene⁹ argued that such an extensive differential blockade could not be expected even though one preganglionic fibre ascends and descends three spinal segments. If peripheral responses to preganglionic stimulation (or blockade) are thus diffuse, a discrete dermatomal level should be impossible to measure but sympathetic-sensory differential can be easily explained. For the same anatomic reason, partial sympathetic recovery should occur much earlier than the sensory recovery.

A study by Bengtsson *et al.*¹⁰ used a similar protocol as in our present study, except that the degree of sympathetic blockade was evaluated by skin blood flow change using a laser doppler flowmeter. They found that when a stable sensory level (mean T_8) receded to a T_{10} mean level in the recovery room, the elevated skin blood flow associated with spinal anaesthesia declined to a preblock level at the upper and lower thighs, and that the blood flow of the foot declined somewhat, but did not reach preblock levels. This finding indicates that a partial sympathetic recovery of the lower limbs has occurred at a mean sensory level of T_{10} . The early sympathetic recovery in the lower limb was also reported during regression of epidural anaesthesia.^{2,11}

In summary, the PvO_2 of a superficial vein of the foot significantly increased following spinal anaesthesia if the sensory level obtained was T_{10} or above. However, when the sensory level obtained was only T_{11} or T_{12} , there were no significant changes in the PvO_2 over control values. It is postulated that the increases in PvO_2 are due to increased AV shunting secondary to profound sympathetic denervation produced at sensory levels T_{10} or above and furthermore, that sympathetic recovery begins when the sensory level recedes below T_{10} .

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Résumé

A la suite d'une résection transurétrale de la prostate sous anesthésie péridurale, on a divisé 26 patients en deux groupes selon les niveaux sensoriels mesurés en salle de réveil: Groupe A: 20 patients ayant un niveau sensoriel de T_{10} ou plus, et Groupe B: six patients ayant un niveau sensoriel de T_{11} ou T_{12} . On a obtenu des échantillons sanguins sériés par un cathéter intraveineux placé en préop dans la veine superficielle du pied, afin de mesurer la PvO_2 . Dans le Groupe A, la PvO_2 augmentait de façon significative après l'anesthésie péridurale en comparaison des valeurs de contrôle préopératoires, et elle diminuait quand le niveau sensoriel revenait à T_{11} ou T_{12} . Dans le Groupe B, les changements qui se produisirent dans la PvO_2 à partir des valeurs-témoins jusqu'aux valeurs postrachidiennes et aux valeurs du rétablissement moteur ne sont pas significatifs. Les auteurs considèrent que la dénervation sympathique induite par anesthésie péridurale augmente la PvO_2 en ouvrant les anastomoses artério-veineuses dans la circulation cutanée, et que la PvO_2 diminue avec le rétablissement sympathique. Ces constatations confirment qu'on peut s'attendre à une dénervation sympathique significative des membres inférieurs à un niveau sensoriel de T_{10} ou plus, et que le rétablissement sympathique commence quand le niveau sensoriel revient en-dessous de T_{10} .