INFLUENCE OF EXTRADURAL BLOCKADE AND EPHEDRINE ON TRANSCUTANEOUS OXYGEN TENSION

J. A. ODOOM, I. L. SIH, J. G. BOVILL, B. VAN DER BROEK AND J. OOSTING

Patients with incipient gangrene and severe rest pain often require continuous extradural, or regional, blockade (Smith, Fischer and Scott, 1984), with local anaesthetics to control their pain. Additionally, the technique improves limb blood flow, increases skin temperature and makes it possible for the dressing of wounds and physiotherapy to be carried out with less discomfort. Lumbar extradural blockade may also be used to predict whether subsequent surgical or chemical sympathectomy is likely to be effective.

However, extradural analgesia can cause systemic hypotension (Dagnino and Prys-Roberts, 1984), and influence lower limb blood flow. Transcutaneous oxygen tension (PtCO2) is influenced by variations in systemic arterial pressure (Eickhoff, Ishihara and Jacobsen, 1980; Eickhoff and Jacobsen, 1982) and blood flow (Tonnesen, 1978; Eickhoff and Jacobsen, 1980).

We have, therefore, investigated the effect of hypotension on PtCO2 in patients receiving extradural blockade.

PATIENTS AND METHODS

Twenty surgical patients (10 vascular and 10 urological) were studied before surgery. All patients gave informed consent to participate in the study. None had pulmonary disease, none was premedicated.

SUMMARY

The influence of lumbar extradural blockade with 0.5% plain bupivacaine on transcutaneous oxygen tension (PtCO2) and skin temperature was studied in 20 patients, 10 scheduled for vascular surgery and 10 for urological surgery. At the time of maximum extent of blockade, mean arterial pressure (MAP) had decreased significantly (P < 0.001) from 96.6 ± 18.8 mm Hg to 69.5 ± 10.1 (mean ± SD) in the vascular group and from 88.0 ± 14.7 mm Hg to 71.1 ± 12 mm Hg in the urological group. In the vascular group, PtCO2 decreased significantly in the ischaemic (P < 0.07) and non-ischaemic (P < 0.001) limbs. In the urological group, there was a significant (P < 0.001) decrease in PtCO2 in both limbs. There was no change in cutaneous temperature in the ischaemic limbs (vascular group), but the temperature in the non-ischaemic limbs increased significantly (P < 0.01). In the urological group, the cutaneous temperature increased significantly (P < 0.001) in both limbs. When ephedrine 10 mg was administered i.v., MAP increased significantly (P < 0.001) in both groups to pre-blockade values. This was accompanied in both groups by significant increases in PtCO2, but not by a change in skin temperature. There was a significant correlation between change in MAP and change in PtCO2 in both groups after ephedrine.

Selection of patients

Vascular group. Patients were included in the study if they had evidence of unilateral ischaemic vascular disease as determined by history, physical examination, translumbar aortography and standard vascular laboratory tests (Raines et al., 1976). These included evaluation of claudication...
by treadmill exercise, measurement of arterial pressures in arm, thigh, calf and ankle and pulse volume recording (PVR) using a Doppler ultrasonic technique (Yao, 1970). Ankle pressure index was calculated from the ratio of ankle to arm systolic arterial pressures. Ankle pressure index has been shown to correlate significantly with the degree of vascular occlusion found by arteriography (Yao, Hobbs and Irvine, 1969). All tests were performed before and after exercise.

Limbs with impending or frank gangrene, exercise-induced pain relieved by rest (claudication) or an ankle pressure index less than 1, were categorized as ischaemic. Patients with bilateral ischaemia were excluded.

**Urological group.** None of the patients in this group had a history of intermittent claudication. The absence of ischaemic vascular disease was further assessed by physical examination and Doppler ultrasonic flow measurements. Treadmill exercise tests and translumbar aortography were not performed.

Transcutaneous oxygen tension ($P_{tcO_2}$) in the lower limbs was measured using a TCM 1 TC oxygen monitor (Radiometer, Copenhagen) and recorded continuously (Gould Inc., Cleveland Ohio, U.S.A.). The skin temperature of the limbs was measured with Telethermometer probes (Yellow Springs Instrument Co. Inc., Yellow Springs Ohio, U.S.A.) applied 2–5 cm distant from the $P_{tcO_2}$ electrodes. The electrocardiogram (ECG) and the heart rate (HR) were displayed continuously. Mean arterial pressure (MAP) was recorded non-invasively every 5 min using an Omega 1000 automatic pressure recorder (Invivo Research Laboratories Inc., Tulsa Oklahoma 74145, U.S.A.).

A catheter was inserted to the lumbar extradural space at the L2–3 space and the initial (baseline) measurements made. Fifteen millilitre of 0.5% plain bupivacaine was then injected through the catheter and the various indices measured continuously until maximum blockade was established. Ephedrine 10 mg was given i.v. and the measurements continued for a further 30 min. MAP, HR, $P_{tcO_2}$ and temperature during maximum extradural blockade, and at the peak effect of the ephedrine, were noted. All measurements were obtained in an induction room with the ambient temperature maintained at 22 °C. Four patients in whom blockade was unilateral or incomplete were excluded from further study.

**RESULTS**

Patient characteristics are summarized in table I. The groups were comparable with respect to age, weight, height and sex distribution.

The mean changes in MAP, $P_{tcO_2}$, and cutaneous temperature before and after extradural blockade, and after the administration of ephedrine, are shown in tables II (vascular group) and III (urological group).

**Changes after extradural blockade**

The average times to maximum extradural blockade, as judged by motor blockade, were $30\pm 8.5$ min and $31.6\pm 9.2$ min in the vascular and urological groups, respectively. At this time, MAP had decreased significantly from $96.6\pm 18.8$ mm Hg to $69.5\pm 10.1$ mm Hg in the vascular group (table II) and from $88\pm 14.7$ mm Hg to $71.1\pm 12$ mm Hg in the urological group (table III). This was associated with a significant decrease in $P_{tcO_2}$ in the ischaemic ($P < 0.01$) and non-ischaemic ($P < 0.001$) limbs in the vascular group. There was a similar decrease ($P < 0.001$) in both limbs in the urological group. In the vascular group, there were no significant changes in cutaneous temperature in the ischaemic limb. There was a significant ($P < 0.01$) increase in temperature in the non-ischaemic limb. In the urological group, there was a significant ($P < 0.001$) increase in cutaneous temperature.

**Changes after administration of ephedrine**

Ephedrine significantly increased MAP from $69.5\pm 10.1$ mm Hg to $96.9\pm 9.5$ mm Hg in the vascular group (table II) and from $71.1\pm 12$ mm Hg to $89.8\pm 12.4$ mm Hg in the urological group (table III).

**TABLE I. Patient characteristics (mean±SD)**

<table>
<thead>
<tr>
<th></th>
<th>Vascular group</th>
<th>Urological group</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Male/female</td>
<td>8/2</td>
<td>9/1</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>$69.1\pm 6.7$</td>
<td>$68.7\pm 9.3$</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>$72.7\pm 10.8$</td>
<td>$72.6\pm 11$</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>$173.5\pm 8.8$</td>
<td>$174.6\pm 5.1$</td>
</tr>
</tbody>
</table>
In the vascular group, $P_{tcO_2}$ increased significantly in both the ischaemic ($P < 0.02$) and non-ischaemic limbs ($P < 0.001$). In the urological group, $P_{tcO_2}$ increased significantly ($P < 0.001$) in both limbs. There was no significant change in cutaneous temperature in either limb of either group after the administration of the ephedrine.

In the vascular group, the change in MAP correlated well with changes in $P_{tcO_2}$ in the ischaemic limb ($r = 0.64$; $P < 0.01$), and the non-ischaemic limb ($r = 0.76$; $P < 0.001$) (fig. 1). A similar correlation between change in MAP and change in $P_{tcO_2}$ ($r = 0.77$; $P < 0.001$) was found in the urological group (fig. 2).

**DISCUSSION**

Vascular tone is controlled by the level of sympathetic activity. In the normal subject, lumbar sympathetic blockade, whether produced by surgical or chemical denervation, causes vascular dilatation, a decrease in regional vascular resistance and, in the absence of a large decrease in arterial pressure, increases in capillary blood...
Fig. 1. Relationship between changes in mean arterial pressure (ΔMAP) and transcutaneous oxygen tension (ΔPtco₂) in ischaemic and non-ischaemic limbs after maximum extradural blockade (▲ = ischaemic; ■ = non-ischaemic) and after the administration of ephedrine (△ = ischaemic; O = non-ischaemic) in 10 vascular patients.

Fig. 2. Relationship between changes in mean arterial pressure (ΔMAP) and combined transcutaneous oxygen tension (ΔPtco₂) of the lower limbs after maximum extradural blockade (●) and after administration of ephedrine (○) in 10 urological patients.

flow to the skin (with a concomitant increase in skin temperature) (Mellander and Johansson, 1968; Herman, Dworecka and Wisham, 1970; Gillespie, 1973; Uhrenholdt, 1973). Skin capillary oxygen tension and saturation have been shown to increase following sympathetic denervation (Lofstrom and Zetterquist, 1967; Bridenbaugh, Moore and Bridenbaugh, 1971; Hatangdi and Boas, 1985).

In patients with peripheral vascular disease, the effects of sympathetic blockade are unpredictable, and published results of studies on skin blood flow after sympathectomy are conflicting (Uhrenholdt et al., 1971; Delaney and Scarpino, 1973; Morrice et al., 1975). Since accurate measurement of skin blood flow in the clinical situation is technically difficult, skin temperature is frequently used as an indicator of skin blood flow, on which it is largely dependent (Joly and Weil, 1969; Wright and Cousins, 1972). It has also been reported that variations of temperature following sympatheticotomy are related to arterial pressure at the ankle (Thulesius, Gjöres and Mandaus, 1973; Uhrenholdt, 1973).
EXTRADURAL BLOCKADE AND \( \text{Pt}Co_2 \)

In our study, we attempted to distinguish between the effects of extradural analgesia on ischaemic limbs in patients with unilateral vascular disease so that the unaffected limb could serve as a control. In addition, we had a further control group of patients (urological) without arteriosclerotic vascular disease. Although advanced arteriosclerotic vascular disease is often widespread, it may be more severe in one limb than the other. Thus, it is possible that, in our vascular patients, some degree of arteriosclerotic vascular disease may have been present in the "non-ischaemic limb". However, the finding that \( \text{PtCo}_2 \) in this limb (table II) was the same as that in the urological group (table III) suggests that the degree of arteriosclerotic disease in the non-ischaemic limb was minimum. In the non-ischaemic limb \( \text{PtCo}_2 \) was higher (66.2 ± 5.5 mm Hg) than that of the ischaemic limb (32.7 ± 18.5 mm Hg). The clinical diagnosis of unilateral ischaemia was based on history, physical examination and haemodynamic measurements (Yao, 1970; Raines et al., 1976), supplemented by angiographic findings.

We found that the hypotension associated with maximum extradural blockade correlated significantly with the decrease in \( \text{PtCo}_2 \), indicating a decrease in blood flow. In the non-ischaemic limb, this reduction was unlikely to be of clinical significance, but in the ischaemic limb in which blood flow was already critical, a further decrease in blood flow could cause further deterioration in tissue oxygenation.

Cutaneous temperature increased significantly in the non-ischaemic limb after the extradural blockade, but did not change in the ischaemic limb. The finding of increased temperature, together with a decrease in \( \text{PtCo}_2 \) (an indication of a decreased blood flow) would suggest that the increase in skin temperature after extradural blockade was the result of cutaneous vasodilatation and hyperaemia (Uhrenholdt et al., 1971). The difference between our findings and those reported by Wright and Cousins (1972) may be explained by differences in the magnitude of the decrease in MAP. In the vascular bed of an ischaemic limb, blood supply is reduced, transmural pressure loss and the vessels are maximally dilated because of the accumulation of vasodilator metabolites and loss of myogenic tone. Under these circumstances, sympathectomy may be unable to dilate the vessels of the ischaemic limb further, but may do so in other regions and induce a decrease in perfusion pressure and a reduction in blood flow to the ischaemic limb (Mellander and Johansson, 1968). Our finding supports the view that often sympathectomy does not benefit those limbs that most need an increase in blood flow (Froysaker, 1973; Uhrenholdt, 1973).

Previous studies (Modig, Malmberg and Karlstrom, 1980; Modig et al., 1983) have demonstrated that extradural analgesia increases blood flow to the large vessels of the lower limb, but appears to decrease local blood flow in the small vessels. This may explain the reduction in intraoperative blood loss during extradural analgesia. Extradural blockade can affect the cutaneous circulation in several ways. It causes arteriolar vasodilatation, leading to a decrease in MAP and pulmonary artery pressure (Modig and Malmberg, 1975). Arteriovenous anastomoses in the skin are opened. The large blood flow through these anastomoses results in a steal of blood from the superficial cutaneous capillaries (Cronenwett et al., 1983), and both of these effects result in decreases in the blood flow to the skin and in \( \text{PtCo}_2 \) (Davis and Greene, 1959).

The present study has demonstrated that extradural blockade decreased \( \text{PtCo}_2 \) in limbs of both groups of patients. When ephedrine was given to increase the arterial pressure to pre-blockade values, \( \text{PtCo}_2 \) increased. This finding is consistent with the previously reported results in animals (Tremper, Waxman and Shoemaker, 1979) and man (Tremper and Shoemaker, 1981) that \( \text{PtCo}_2 \) increases with an increase in cardiac output. It has been shown that, in the presence of either subarachnoid (Ward et al., 1966) or extradural blockade (Engberg and Wiklund, 1978), the administration of ephedrine causes an increase in MAP but has minimal effect on peripheral vascular resistance in the area of the block. The increase in arterial pressure with induced vasodilatation in the lower limbs may beneficially increase flow to the legs and thus \( \text{PtCo}_2 \) (Hatangdi and Boas, 1985).

In conclusion, sympathectomy, if it is to be of value in patients with ischaemic limbs, should be combined with measures designed to prevent any decrease in mean arterial pressure.

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REFERENCES


