What to do if a neuraxial block in a patient spreads too cephalad soon after injection?

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When a segmental block is intended, the appearance of signs of a block where it was not expected may be quite troublesome. It speaks for itself that thoracic and cervical epidurals are excluded because numbness in one or both upper extremities is to be expected. Before discussing the possible explanations the definition of 'shortly' needs to be defined. It absolutely excludes patients becoming numb after hours or days of treatment particularly with infusions. I suggest to assume a delay not exceeding 30 minutes after the administration of a bolus dose.

- Pre-existing diseases, clinical conditions or other contributing factors
  - Patients in labor may tend to hyperventilate. This may cause paraesthesias all over the body but more specifically in the extremities. It may cause difficulty in determining the upper sensory level.
  - Due to swelling and oedema numbness in the fingers may be explained by median nerve entrapment causing a transient carpal tunnel syndrome.
  - Placing the patient in a specific position which she has not assumed during the last month, thus of pregnancy may cause cervico-brachial nervous compromise.
  - Some pre-existing (an unknown) diseases may be unmasked by neuraxial techniques such as multiple sclerosis and ALS. Diabetes may cause polineuropathy. Repeated blood pressure measurements (every minute) may cause more harm in the patients with pre-existing peripheral neuropathy such as in diabetic patients or sclerotic disease.

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Accompanying symptoms of excessive spread

Accompanying symptoms may be a Horner syndrome (more frequently in parturients ?) because the sympathetic block tends to spread more cephalad than the sensory block (1). Whereas Horner syndrome was thought to be related to the use of potent local anaesthetics concentrations only (2), similar symptoms are equally possible with weak concentrations. Also trigeminal nerve palsy, hemifacial numbness and flushing may be concurrent symptoms. In the absence of other symptoms (Horner syndrome is possible as a single event) no further action should be undertaken besides careful monitoring. More dramatically the occurrence of motor impairment may compromise respiration although the intercostal nerves contribute less to it than the diaphragm requiring a block up to C4.

Too cephalad spread of a block may increase the risk of haemodynamic instability and bradycardia especially with blocks above T4.

Subdural blockade

More attention is given during last years to the occurrence of subdural blocks. The onset is rather slow (>20 min. for a presumed epidural block) and may cause either failing blocks (3) mostly characterized by sensory blocks without motor impairment (restricted spread possible in 17%) or bizar and excessive extension of the blocks (74%) affecting motor capacity and sensory function of areas where the dose given would not be expected to cause any effect (4). Excessive distribution may also cause patchy assymetrical spread, facial/head involvement while Horner syndrome but also respiratory arrest (larger volumes injected?), bradycardia and loss of consciousness have been reported. Surprisingly haemodynamic stability may be preserved (5,6). Although subarchnoidal injection may cause subdural spread to a lesser extent, this will also be characterized by no or slow onset and failure of the presumed spinal block with unexpected motor sparing. Such a mostly partly injection may theoretically be possible with the classical Sprotte needles as with such needles the orifice may cover the three spaces i.e. subarachnoid, subdural and epidural.

The subdural space is not a potential space but rather the result of trauma, back surgery or tissue damage creating a cleft within the meninges. Usually after a few hours, longer lasting than the expected 'presumed epidural' effect of the injected LA, the block wears off. Subdural blocks may occur with low doses of LA such as those used for epidural infiltration (injection through the needle while mostly failed back surgery). As the symptoms become obvious after at least 20 minutes, it is clear that test doses will not
reveal subdural catheter placement. If a catheter is present, the injection of contrast medium may evidence the catheter location and the confusing spread of the LA. Meanwhile 70 radiographically confirmed cases have been reported (4).

**Excessive spread of spinal, combined spinal-epidural or epidural**

Effects of spinal anaesthesia tend to occur faster than those of epidurals, unless the latter is not correctly placed. If a spinal with a correct dose appears to induce numbness of an upper extremity, it might be hoped that the block may not spread more cephalad. In most cases the patient will also become hypotensive and bradycardic which may further increase patient discomfort and anxiety.

Treatment is symptomatic with oxygen, atropine and vasopressors. Only rarely a spinal block will cause a total spinal or such a cephalad spread that intubation is required (unless the patients has respiratory disease).

The risk may be higher with plain than with hyperbaric solutions as the upper spread of the latter will be restricted to the lowest point of the thoracic kyphosis whereas plain solutions may spread unlimited. With hyperbaric solutions extreme head down positions should be avoided to limit the rostral spread. With plain solutions the opposite will be the case but care should be taken to keep both legs in an elevated position to ensure adequate venous return.

The need for intubation may be more troublesome for C-section patients because of the risk of aspiration. The patient should be reassured and especially if the hand grip shows a reasonable strength there is no reason for panic.

An intrathecal overdose as with intended epidural anaesthesia with inadvertant intrathecal injection (no test dose given) will cause a total spinal. This will require immediate treatment as there will be central nervous, respiratory and haemodynamic implications. The patient will recover after few hours of ventilation. The cardiovascular effects are not always the most dramatical ones.

Finally it should be stipulated that symptoms of inadvertant subarachnoid injection of an epidural bolus dose are not always full-blown as with multi-hole catheters only one of the orifices may have entered the CSF.

**Does CSE cause higher blocks than a single dose spinal?**

The difference between single dose spinal (SSS) and CSE is that in the former the epidural space is not identified and that the dura is not displaced anteriorly. This has been thought to reduce the intradural diameter pus-
hing the local anesthetic, injected spinally, more cephalad. The literature on this issue is rather controversial. In a first study the group of Sia found no difference between spinal, CSE with or without epidural catheter placement in minor gynaecological procedures (7). Subsequently they found no difference during C-section after established labour (8) but a higher spread with CSE during elective C-section (9). Most recently Horstman et al found no difference in upper sensory level, CSF pressure or hypotension when SSS or CSE in the lateral decubitus position were compared for C-section but with rather high hyperbaric bupivacaine doses i.e. 12mg plus fentanyl and morphine (10). When the epidural catheter is used shortly after the spinal to inject air, saline or a local anaesthetic, the block may spread more cephalad (see next paragraph).

The role of a dural hole

When a dural hole is made, either intended or inadvertently (larger hole) epidurally injected substances may cause the block to spread more cephalad. The explanation may be a mechanical one as the pressure in the epidural space may cause anterior displacement of the dura pushing the injected spinal drug more cephalad. Otherwise the creation of a dural hole may increase the block level by intrathecal migration of epidurally injected LA or in case of sufficient leakage (as after major wet taps) the local anaesthetic being taken to higher levels by the CSF entering the epidural space.

Spinal after failing epidural

At least 12 cases have been reported during the early 90’s describing a high spinal block after spinal injection to rescue a failing epidural during labour which had to be converted into a block for a C-section (11,12). Especially during the early 90’s high and total spinals have been caused by the existence of a residual block while the selected intrathecal LA dose was inappropriate. The incidence was assumed to be as high as 11% (12) but recent case series have been more optimistic but some aspects should be taken into consideration (13). Actually it is recommended to lower the doses, to wait until the block wears off until it has reached T12 or to wait at least 30 min. after the last top-up dose. The most recent cohort study by Visser et al. compared the block characteristics of 128 patients receiving a spinal after removal of the epidural with another 508 patients receiving spinal anaesthesia without any epidural pre-treatment (14). None of all these patients developed a total spinal while also the incidence of a high spinal (0.8% vs 0.2%), incidence of hypotension, ephedrine requirements and Apgar scores were comparable.

Nevertheless and despite these reassuring reports anaesthetists should be
alert at all times. Even this year we had a high spinal, up to C2, affecting the respiratory function (but endotracheal intubation could be avoided) after a repeat low-dose spinal after a failed CSE and subsequently failed epidural supplementation.

**Less common causes**

The loss of resistance to air may cause air bubbles to enter the subarachnoid space. These bubbles are taken more cephalad and cause nerve root compression. Theoretically, this may occur at the cervico-thoracic area.

Dural tap has been described to cause C8-paraesthesia without a good explanation (15).

**In conclusion**

Numbness in the upper extremity after a lumbar block is mostly due to excessive cephalad spread related to the position of the patient, the dose given or injection in the wrong compartment. Presumed epidural injection may enter the subarachnoid or subdural space. Other explanations have been described but occur rather rare.

**REFERENCES**

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