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# Bupivacaine concentrations in lumbar cerebrospinal fluid in patients with failed spinal anaesthesia†

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**Background.** Spinal anaesthesia (SA) has high success rates. However, inadequate block after SA has been reported even in the absence of technical problems. Various mechanisms for failed SA (FSA) have been proposed, but reports of cerebrospinal fluid (CSF) concentrations of local anaesthetics (LA) after FSA are scarce. We report lumbar CSF concentrations of bupivacaine in 20 patients in whom adequate block after subarachnoid injection failed to develop.

**Methods.** All patients with inadequate block after subarachnoid injection of plain bupivacaine 0.5% and in whom a second subarachnoid injection of LA was to be performed as a rescue technique were eligible for entry into this study. A CSF sample was withdrawn immediately before injection of the second dose of LA. Patients in whom failure was obviously due to technical problems or inadequate dosage were excluded. Bupivacaine concentrations were assessed with high-performance liquid chromatography.

**Results.** During the study period of 15 months, 2600 spinal anaesthetics were performed. The failure rate was 2.7% (71 patients). In 20 patients (0.77%), CSF concentrations of bupivacaine were determined, which ranged from 3.36 to 1020 $\mu g\ ml^{-1}$.

**Conclusions.** Inadequate CSF concentration of LA is a common reason for FSA. However, in 12 of our 20 patients, concentrations were above 73 $\mu g\ ml^{-1}$, a concentration that should lead to an adequate block. In these patients, maldistribution of bupivacaine could be responsible for FSA. In view of the absence of sufficient block, despite adequate lumbar CSF concentrations of bupivacaine, concerns about neurotoxicity with repeat injections may be warranted.

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Spinal anaesthesia (SA) is a frequently used anaesthetic technique, and success rates and patient satisfaction are generally high.[1] However, there are numerous reports of failed SA (FSA), and published failure rates in large series of SA range from 0.46%[2] to 17%.[3] The reasons most commonly provided to explain failure are technical problems,[3] errors of judgement with respect to pharmacological factors, such as inadequate dose of local anaesthetic (LA), and inadequate positioning of the patient.[4] Proposed mechanisms for inadequate block despite correct dosing and injection technique are maldistribution,[5] variability in the anatomy of the lumbar subarachnoid space,[6] inadvertent subdural[7] or epidural injection,[8] and resistance to the effects of LA.[9]

Confronted with FSA, the anaesthesiologist can either administer general anaesthesia or repeat the subarachnoid injection with an identical or smaller dose of LA. However, choosing an adequate dose of LA for a second subarachnoid injection is difficult because the amount of LA already present in the subarachnoid space is unknown. A second dose may be too small, again resulting in an inadequately low sensory level of anaesthesia or too large,

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leading to an inappropriately high level of anaesthesia. In addition, reports of neurotoxic effects of LA[10] and a correlation between the dose of LA and the risk for neurotoxicity[11] call for cautious dosing when repeated subarachnoid injections of LA are performed. In fact, neurological deficits associated with repeated subarachnoid injection of LA after FSA have been reported,[12] and the safety of this practice has been questioned.[13]

We measured lumbar bupivacaine cerebrospinal fluid (CSF) concentrations in patients with FSA to test the hypothesis that the primary reason for FSA is an inadequate concentration of LA in the CSF.

## Methods

The study protocol was approved by the Regional Ethics Committee of the University Hospital Basel, Basel, Switzerland. The study lasted 15 months. For the purpose of this study, we defined FSA as inadequate sensory block for the planned procedure 15 min after subarachnoid injection of an adequate standardized dose of LA. All patients who fulfilled the definition of FSA and in whom SA had been performed with plain bupivacaine 0.5% (Carbostesin®, Astra Pharmaceutica AG, Dietikon, Switzerland) were eligible for entry into the study. However, of these patients, only those in whom the responsible anaesthesiologist decided to use a second subarachnoid injection of LA as a rescue technique were included, that is, no additional subarachnoid punctures were performed for study purposes only. Exclusion criteria were patient refusal, pregnancy, obvious technical difficulties during injection of the first dose, such as inability to aspirate CSF at the end of the injection, injection of less than the intended dose due to unexpected patient movement or inadvertent disconnection of the needle from the syringe during injection, and more than three attempts to enter the subarachnoid space.

After the injection of LA, the extent of the sensory block was assessed by cold-warm discrimination with an ether swab. If the patient fulfilled the entry criteria, oral informed consent for taking a CSF sample was obtained from the patient. The Regional Ethics Committee waived the need for written consent because no change in clinical management occurred and the volume of the test sample was less than that of the injected LA. During the second puncture, immediately before injection of the second dose of LA, a 1 ml sample of lumbar CSF was obtained and set aside. The sample was frozen immediately and stored at $-20^{\circ}C$ until analysis. CSF bupivacaine concentrations were measured after termination of the study period using high-performance liquid chromatography as previously described.[14] The lower limit for quantification with this method is 0.05 $\mu g\ ml^{-1}$. Patient position (sitting or lateral) and the lumbar segment for the first and second punctures were chosen according to the clinical judgement of the responsible anaesthesiologist. The needle types used were Sprotte 24 G in patients younger than 55 yr and Quincke 22 G or 25 G in patients older than 55 yr according to our departmental clinical practice at the time of this study. The bupivacaine doses were 15–20 mg for women and 17.5–22.5 mg for men.

## Results

During the study period, 2600 spinal anaesthetics were performed. FSA was observed in 71 patients (2.7%). No CSF sample was obtained in 45 patients because general anaesthesia was used as a rescue technique, obvious technical difficulties during injection, or patient refusal. Four additional patients were excluded because of administration of hyperbaric bupivacaine. In 22 patients, bupivacaine concentrations were measured. However, the data from two of these patients were excluded from analysis because the circumstances of the first and second taps were not sufficiently documented. The data of the remaining 20 patients (0.77%) are summarized in Table 1.

All CSF samples were obtained 15–45 min after the first injection (median: 25 min). The measured CSF concentration values were between 3.36 and 1020 $\mu g\ ml^{-1}$. Individual patient data, doses, technical details (needle type, site of first and second puncture, and patient positioning), and extent of sensory block before aspiration of the CSF sample are listed in Table 1. Six patients had a complete failure, that is, no sensory block at all, including the sacral dermatomes. The bupivacaine CSF concentrations measured in these six patients were between 3.36 and 106.0 $\mu g\ ml^{-1}$. The lowest measured concentration in a patient with partial but inadequate anaesthesia was 11.84 $\mu g\ ml^{-1}$. Figure 1 shows the relationship between the number of blocked segments and the corresponding lumbar CSF concentrations.

## Discussion

We found a wide range of bupivacaine concentrations in the lumbar CSF of patients with FSA. An important point for the interpretation of our data is defining a threshold for lumbar bupivacaine CSF concentrations above which a sufficient block should be observed. On the basis of the available data[15] and the fact that all our samples were obtained 15–45 min after the initial lumbar puncture, we assumed that a lumbar bupivacaine CSF concentration of $\geq 73\ \mu g\ ml^{-1}$ should lead to an adequate block. This concentration represents the 5th percentile of the concentrations sampled during the same time-span in 37 patients with adequate SA by Ruppen and colleagues.[15] In our series of 20 patients with FSA, eight patients had a lumbar CSF bupivacaine concentration $<73\ \mu g\ ml^{-1}$ and 12 patients a concentration $>73\ \mu g\ ml^{-1}$ (Table 1). Interestingly, only one of six patients with a completely FSA had a bupivacaine concentration above this threshold ($106\ \mu g\ ml^{-1}$). Resistance to bupivacaine, as suggested in

**Table 1** Characteristics of patients with FSA from whom CSF samples were obtained. BMI, body mass index; FSA, failed spinal anaesthesia; Patient position, patient position during performance of the initial spinal anaesthetic. Needle: Q, Quincke; S, Sprotte. Conc., bupivacaine concentration in the cerebrospinal fluid

| Patient number | Age (yr) | Sex | Height (m) | Weight (kg) | BMI (kg $m^{-2}$) | Previous FSA | Dose (mg) | Patient position | Needle | First tap | Level of sensory blockade | Delay to sample (min) | Second tap | Conc. ($\mu g\ ml^{-1}$) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 86 | F | 1.64 | 56 | 21 | X | 17.5 | Lateral | Q 22 G | L4/5 | None | 40 | L5/S1 | 3.36 |
| 2 | 83 | M | 1.70 | 74 | 26 | | 20.0 | Sitting | Q 25 G | L4/5 | Right: L4, left: none | 20 | L5/S1 | 11.84 |
| 3 | 60 | M | 1.61 | 85 | 33 | | 20.0 | Sitting | Q 25 G | L3/4 | Patchy: right: S2–L1, left: L5–T10 | 45 | L3/4 | 19.02 |
| 4 | 67 | M | 1.70 | 58 | 20 | | 17.5 | Sitting | Q 25 G | L2/3 | None | 20 | L2/3 | 20.98 |
| 5 | 53 | F | 1.60 | 47 | 18 | | 20.0 | Lateral | S 24 G | L3/4 | None | 15 | | 30.17 |
| 6 | 63 | F | 1.60 | 60 | 23 | | 17.5 | Sitting | Q 25 G | L3/4 | Patchy T12 | 15 | L3/4 | 32.28 |
| 7 | 79 | M | | | | | 20.0 | Lateral | Q 25 G | L4/5 | None | 20 | L2/3 | 50.42 |
| 8 | 52 | F | 1.58 | 55 | 22 | | 15.0 | Lateral | Q 25 G | L3/4 | None | 15 | L3/4 | 55.66 |
| 9 | 66 | M | | 82 | | | 22.5 | Sitting | Q 25 G | L3/4 | None | 25 | L3/4 | 106.02 |
| 10 | 63 | M | 1.92 | 89 | 24 | | 20.0 | Lateral | Q 25 G | L3/4 | L1 | 35 | L2/3 | 114.48 |
| 11 | 70 | M | 1.65 | 66 | 24 | | 20.0 | Lateral | Q 25 G | L4/5 | L3 | 40 | L3/4 | 127.95 |
| 12 | 52 | M | 1.60 | 56 | 22 | | 20.0 | Lateral | Q 25 G | L3/4 | L5 | 40 | L3/4 | 137.50 |
| 13 | 78 | F | 1.51 | 63 | 28 | | 17.5 | Lateral | Q 22 G | L3/4 | Patchy T10 | 30 | L2/3 | 137.71 |
| 14 | 69 | M | 1.76 | 80 | 26 | X | 17.5 | Sitting | Q 25 G | L3/4 | Right: L3, left: none | 15 | L3/4 | 139.27 |
| 15 | 81 | F | 1.60 | 68 | 27 | | 17.5 | Lateral | Q 25 G | L3/4 | T12 | 35 | L4/5 | 160.16 |
| 16 | 56 | M | 1.65 | 65 | 24 | | 22.5 | Sitting | Q 25 G | L3/4 | L3 | 30 | L3/4 | 192.24 |
| 17 | 82 | F | 1.68 | 60 | 21 | | 15.0 | Lateral | Q 25 G | L3/4 | L4 | 30 | L3/4 | 194.74 |
| 18 | 42 | F | 1.57 | 47 | 19 | | 17.5 | Lateral | S 24 G | L4/5 | Patchy L1 | 20 | L3/4 | 204.49 |
| 19 | 65 | F | | | | | 17.5 | Lateral | Q 25 G | L4/5 | Right: L3, left: L4 | 20 | L3/4 | 253.65 |
| 20 | 22 | F | 1.75 | 54 | 18 | | 17.5 | Lateral | S 24 G | L4/5 | Right: T12, left: L3 | 30 | L3/4 | 1020.24 |

two reports,[16 17] cannot explain the completely FSA in these six patients because they all developed adequate anaesthesia after the repeated injection. A more likely cause for completely or partially FSA is the failure to inject a sufficient dose of LA into the CSF as a result of unrecognized technical problems. Alternatively, an unanticipated large lumbar CSF volume could also explain inadequately low bupivacaine concentrations.[18 19] A large variability (43–81 ml) of lumbosacral CSF volumes calculated from MRI sequences has been suggested as the most important factor contributing to the variability in spread of SA.[6] However, this interpretation is questioned by the fact that exclusion of one of the 10 volunteers in that study would eliminate the statistical significance of the correlation between lumbosacral CSF volume and spread of SA.[20] Inadvertent (partial) subdural or epidural injection could also explain low CSF bupivacaine concentrations. In an unfixed anatomic preparation of a human spinal column, Mollmann and colleagues[7] were able to reproduce injection of LA into the subdural space in all preparations with a Sprotte needle but not with a Quincke needle. In our series, only three injections were performed with Sprotte needles, and only one of these patients had a CSF concentration below 73 $\mu g\ ml^{-1}$. Hence, this was not a major cause of FSA in our series. Maldistribution of LA and sampling at the 'wrong' anatomical level could be another explanation for low CSF concentrations of bupivacaine. In one of the eight patients with CSF bupivacaine concentrations $<73\ \mu g\ ml^{-1}$, the CSF sample was obtained one interspace higher, and in two patients one interspace lower than where the primary injection of LA had taken place (Table 1).

Maldistribution could be the most important explanation for the FSA in the 12 patients who had CSF bupivacaine concentrations $>73\ \mu g\ ml^{-1}$ (106–1020 $\mu g\ ml^{-1}$). Maldistribution needs to be discussed with an understanding of the factors determining intrathecal drug spread. The distribution of plain bupivacaine is somewhat unpredictable.[21] In an extensive review, Hocking and Wildsmith[21] discuss characteristics of the injected LA, clinical

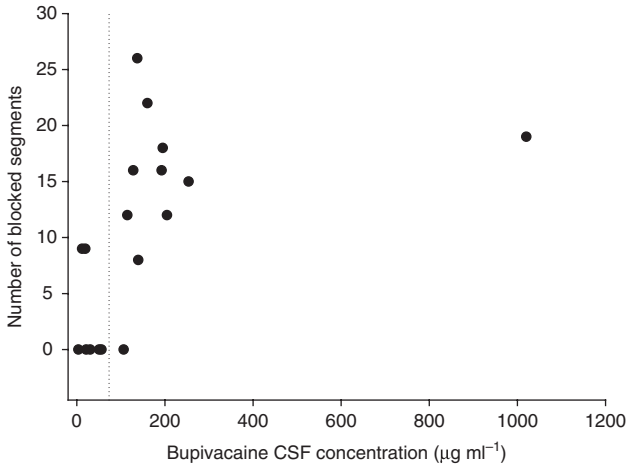


**Fig 1** Bupivacaine concentrations in the lumbar CSF and the number of blocked segments. Bilateral segments are counted separately and were counted as follows: starting from S5, each blocked segment was added to the number of blocked segments, for example, a patient with a block level of L5 on the right side and a level of L3 on the left side would be represented by 14 blocked segments (right: S5, S4, S3, S2, S1, L5 and left: S5, S4, S3, S2, S1, L5, L4, L3, i.e. 6+8 segments). Dotted line: 73 μg ml$^{-1}$.

technique including patient position, the level of injection, type of needle and direction of the needle opening, fluid currents in the CSF generated by injection of the LA, and patient characteristics as factors that may influence intrathecal distribution of LA. All our patients received plain bupivacaine that is slightly hyperbaric at room temperature and slightly hypobaric at body temperature.[21] As the LA was always stored at room temperature and all patients were normothermic, the slightly changing baricity of bupivacaine cannot explain the heterogeneity of our results. The injected volumes of bupivacaine ranged from 3 to 4.5 ml in our patients. This large range should also not have had a major impact on intrathecal drug spread as an increase of 50% in volume has been shown to increase the mean spread only by one dermatome.[21] The clinical technique used is a more important factor influencing distribution of bupivacaine, particularly the level of injection. With plain solutions, a higher level of injection leads to greater cephalad spread even if the difference in injection level is only one interspace.[21] Results of studies investigating the speed of injection of LA into the subarachnoid space are conflicting, but faster injections of plain bupivacaine probably lead to greater spread.[21] As we did not control for speed of injection, this factor may have contributed to the variability of bupivacaine in the CSF. Weight, height, and vertical length of the vertebral column of patients correlate with the distribution of LA after subarachnoid injection of plain bupivacaine. However, the predictive value of these variables is low.[21] Patient positioning during injection of bupivacaine was not standardized in our study but should not have an influence on the subarachnoid distribution of plain bupivacaine 0.5%.[21] Effects of needle type and direction of needle opening have, to our knowledge, not been investigated with plain bupivacaine. Although these factors may explain some of the heterogeneity of our data, they are unlikely to fully explain FSA. In contrast, individual anatomical factors including the configuration of the spinal column (kyphosis, lumbar lordotic curvature) may influence distribution of the LA or even cause maldistribution.[21] If SA with plain bupivacaine is repeated on a second occasion, using exactly the same technique, a block with an extent similar to the first SA results.[22 23] Two of our patients had a history of FSA. Other anatomical factors that may limit the distribution of the injected LA are fibrous attachments and membranous structures in the subarachnoid space. Fibrous attachments were found between nerve roots, between nerve roots and the arachnoid membrane at one or more spinal levels in 16 of 26 autopsy subjects studied using endoscopy, or both. In another three subjects, a membranous structure was identified in the posterior midline of the subarachnoid space.[24] By limiting the distribution of the injected LA, these fibrous attachments and membranes may be associated with a variation in the extent of SA.

The single patient with a very high (1020 μg ml$^{-1}$) bupivacaine CSF concentration warrants special attention. This concentration is higher than the concentrations reported by Ruppen and colleagues[15] and led to an incomplete block (L3 left, T12 right). Injection into a low lumbar CSF volume could be assumed: 17.5 ml would be appropriate to achieve this concentration with the used dose of 17.5 mg. However, such a CSF volume is far below the values that were found in an MRI study.[6] Moreover, such a small CSF volume alone would not explain the FSA. Some other factor, probably anatomical, leading to maldistribution must have been present. Injection into a (congenital) spinal arachnoid[25] or s.c. cyst[26] has been proposed but seems unlikely in view of the normal neurological presentation of the patient.

Our findings might support concerns about neurotoxicity after repeated injections of bupivacaine into the subarachnoid space. Although our data do not allow drawing any conclusion on this issue, a correlation between bupivacaine dose and neurotoxicity has been demonstrated in dogs.[11] Considering that more than half of the measured CSF concentrations in our patients were in a range that should have led to an adequate block, we agree with Drasner and Rigler[13] that if a second dose is to be considered, careful testing including the sacral dermatomes should precede the injection. We suggest that if any sensory anaesthesia is present, the second injection should not be performed as maldistribution may be present. On the other hand, some of the doses we used were rather large and it may be inappropriate to extrapolate our suggestion to patients who receive lower doses of bupivacaine. An alternative approach would be to use repeat injections, provided the total amount administered

does not exceed a dose that the clinician would consider reasonable as a single injection.

Two limitations of our protocol need consideration: in order to avoid unnecessary lumbar punctures for study purposes only, sampling of CSF did not always take place at the same interspace as the primary injection of LA had taken place. On the basis of experimental data from an upright spinal canal model, LA concentrations should not be markedly influenced by choosing a modestly distant interspace.[27] However, models of the spinal canal lack a representation of the spinal cord and the cauda equina, which may act as baffles to the generation of fluid currents.[21] We cannot exclude that some of our measurements represent local values. On the other hand, inadequately high local concentrations of LA in the subarachnoid space are not influenced by the interspace at which the sample was obtained. The second problem is the variable time-lag between the injection of bupivacaine and sampling for determination of the CSF concentration, as bupivacaine concentrations change during this time.

In summary, we report CSF concentrations of plain bupivacaine in 20 patients with FSA. Patients in whom obvious technical problems or insufficient dosage of LA could explain FSA were excluded. We found a wide range of CSF concentrations with more than half of the values in a range where adequate block should have been present. Inadequately low CSF concentration of bupivacaine due to failure to realize technical problems during injection of the LA is the most likely explanation for FSA when low concentrations are present. Maldistribution due to anatomical factors is probably the most frequent cause of FSA in cases with adequate CSF concentrations, but further studies are needed to clarify the cause of FSA in such patients. Although our data provide no evidence for a relationship between repeated subarachnoid injections of LA and neurotoxicity, we suggest that a second injection of LA after FSA should only be performed in patients with complete failure or if a repeat injection is performed in a patient with incomplete failure, the total amount of LA administered should not exceed a dose that the clinician would consider reasonable as a single injection.

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