Laboratory Investigations

Baricity and the distribution of lidocaine in a spinal canal model

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The role of the baricity of local anaesthetic solutions in determining the distribution of local anaesthetics injected into the subarachnoid space (and hence the level of anaesthesia) has been challenged. A recent study found no difference in the extent of cephalad spread of hyperbaric and isobaric solutions and concluded that density had no effect on the spread of local anesthetics. The present study, to determine the validity of this conclusion, utilized a spinal model filled with a "cerebrospinal fluid equivalent." Following the injection of hyperbaric lidocaine, the local anaesthetic was most concentrated at the lower end of the column, whereas following the injection of isobaric solution the local anaesthetic was most concentrated around the site of injection. Therefore, baricity is an important determinant of local anaesthetic distribution in the subarachnoid space.

On a récemment mis en doute l'influence de la baricité sur la distribution (et le niveau anesthésique) des solutions d'anesthésique local injectées dans l'espace sous-arachnoïdien. Ainsi, après avoir observé la même extension céphalade de l'effet de solutions hyperbare et isobare d'anesthésique, un auteur concluait à l'absence d'effet de la densité sur leur distribution. Avec notre étude, nous avons voulu vérifier cette conclusion en employant un modèle de canal rachidien rempli de pseudo LCR. Nous avons pu mesurer qu'après l'injection de lidocaïne hyperbare, cette dernière se concentrait au fond du canal alors que la lidocaine isobare se distribuait de part et d'autre du site d'injection. La baricité joue donc un rôle important dans la distribution des anesthésiques injectés dans l'espace sous-arachnoïdien.

Key words
ANAESTHETIC TECHNIQUES: spinal;
PHYSICS: baricity.

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Anaesthetists routinely use hyperbaric local anaesthetic solutions to control the level of spinal anaesthesia. This practice is based on the assumption that when a local anaesthetic is mixed with dextrose and injected into cerebrospinal fluid, the local anaesthetic molecules will follow the heavy dextrose molecules. As a result the anaesthetic level can be controlled by controlling the position of the patient. To our knowledge, the veracity of this assumption has not been documented scientifically.

Hyperbaric anaesthetic solutions in spinal anaesthesia were introduced in 1907 by Barker[1] who developed a glass replica of the vertebral column with several injection ports. When a hyperbaric local anaesthetic solution mixed with methyl violet dye was injected into this glass column, which was filled with "mock spinal fluid," the methyl violet always spread to the lowest point or points in the tube. Thus, when the tube was placed in the upright position (simulating the spinal canal in a sitting patient), the injected dye gravitated to the caudal end of the tube. On the other hand, when the tube was placed in a horizontal position (simulating the spinal canal in a supine patient) and the dye was injected at the level of the lumbar lordosis, it moved both cephalad and caudad as far as the thoracic and sacral curves. Ever since Barker's demonstration of this phenomenon, the baricity of a local anaesthetic and the position of the patient have been considered to represent the two major factors that determine the distribution of local anaesthetics in the subarachnoid space.[2] This concept is consistent with many clinical studies, all of which have shown the differences in

dermatomal levels following the injection of hyperbaric and hypobaric solutions.[3–6]

Recently this concept has been challenged by Bengtsson,[7] who injected bupivacaine with and without glucose in patients in the sitting position and found no difference in the extent of cephalad spread between the hyperbaric and isobaric solutions. He felt that this refuted the presumed influence of baricity on the distribution of local anaesthetics injected into the cerebrospinal fluid. The present study was undertaken to document whether the addition of glucose to a local anaesthetic alters the distribution of that local anaesthetic when injected into the spinal canal.

Methods

A replica of the spinal canal was made (Figure 1) using as close to the dimensions of the human spinal canal as possible: the Tygon tubing, which was 100 cm long with an internal diameter of 2.7 cm, was curved appropriately to simulate the lumbar and thoracic curves. Throughout the length of the tubing 25-gauge needles were inserted at 10 cm intervals, with a three-way stopcock attached to each needle. The needles were labeled "A" to "H" sequentially in a cephalad to caudad direction, so that each 10 cm level represented approximately 2.5 spinal segments. Finally, the tubing was filled with "cerebrospinal fluid equivalent," which was prepared by adding 12 ml, 5% dextrose and 7 ml, 5% albumin to one litre of lactated Ringer's solution. The resultant solution had a glucose concentration of 54 $mg \cdot dl^{-1}$, an albumin concentration of 38 $mg \cdot dl^{-1}$, and a specific gravity of 1.004 at 24° C. Thus, the solution approximated human cerebrospinal fluid, which has a glucose content of 50–80 $mg \cdot dl^{-1}$,[8] protein content of 23–38 $mg \cdot dl^{-1}$,[9] and specific gravity ranging from 1.003 to 1.009.[9]

The Tygon "spinal canal" was filled with 400 ml of "CSF equivalent." Such large volumes were utilized to minimize the concentration effect of withdrawing samples totalling 32 ml per experiment. Since the volume of CSF in the human spinal cord ranges from 25–35 ml,[9] and since 1 ml of 5% lidocaine (50 mg) in 7.5% glucose represents a lower dose clinically, the equivalent dose for our 400 ml spinal canal would be 12 ml of 5% lidocaine (600 mg). Thus, 12 ml of each of the local anaesthetic preparations were injected at level F with the column in the upright ("sitting") position, after which 1 ml samples were withdrawn simultaneously from levels A through H at 5, 10, 15 and 20 min.

The following experiments were carried out:

1 *Hyperbaric lidocaine with glucose:*
Commercially prepared 5% lidocaine in 7.5% glucose, 12 ml (600 mg) were injected at level F at a rate of 0.2 $ml \cdot sec^{-1}$ at 24° C.

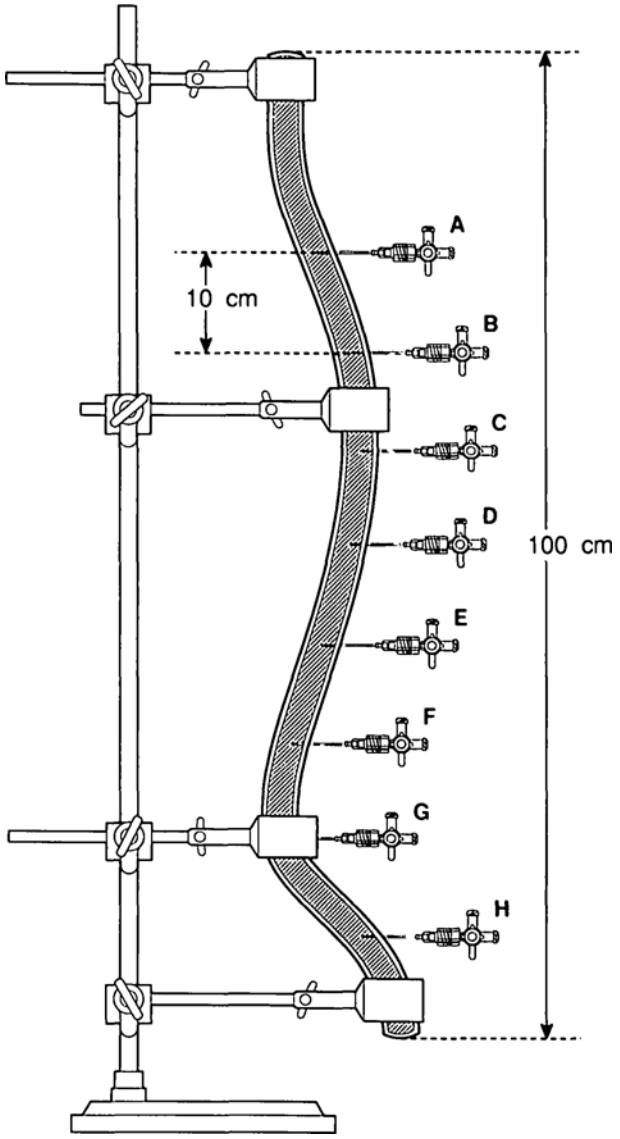

FIGURE 1 Spinal canal model (see text for specifications).

2 *Hyperbaric lidocaine without glucose:*
A 5% lidocaine solution prepared by diluting one part 20% preservative-free lidocaine (marketed for ventricular arrhythmia) with three parts "CSF equivalent" to give a solution with a specific gravity of 1.030 was also injected in 12 ml at level F at a rate of 0.2 $ml \cdot sec^{-1}$ at 24° C.

3 *Isobaric lidocaine:*
This solution was prepared by mixing 1% lidocaine with equal volumes of distilled water to give a solution with a specific gravity of 1.004. Again 12 ml of solution were used and injected at level F at a rate of 0.2 $ml \cdot sec^{-1}$ at 24° C.

Each experiment was carried out three times at 24° C and at an injection rate of 0.2 ml·sec^{-1}. Finally, to determine the influence of injection rate and temperature, two additional experiments were carried out with hyperbaric lidocaine with glucose, one at an injection rate of 0.5 ml·sec^{-1} (at 24° C) and the other at 37° C at an injection rate of 0.2 ml·sec^{-1}. The samples were assayed for their lidocaine and glucose concentrations, using the Abbott TD_x fluorescent polarization[10] and glucose oxidase[11] methods respectively.

All specific gravity determinations were made using a Reichert refractometer at 24° C. All data are presented as means ± SEM. The data were analyzed using an independent Student's t test and one-way analysis of variance. Adjustments for multiple comparisons were made using the Bonferroni's method. Therefore, the nominal probability value was 0.01.

Results

In all the experiments, there were no significant differences among levels F, G, and H, nor were there differences between levels C, D, and E (analysis of variance). Similarly, there were no differences among the samples drawn at 5, 10, 15, and 20 min following injection. Thus, lidocaine and glucose concentrations "above the level" of injections include the mean obtained from all three trials at levels F, G, H and all the samples drawn at 5, 10, 15 and 20 min, whereas lidocaine and glucose concentrations below the level of injection refer to all three trials with samples drawn from levels C, D, and E at 5, 10, 15 and 20 min. The concentrations represented on the graphs were the means of each separate level from the 3 trial at 5, 10, 15, and 20 min. Levels A and B were excluded from the assays because they represented high thoracic and cervical levels (approximately T_2 and above) and because random sampling at these levels revealed glucose and lidocaine concentrations and specific gravities similar to the values obtained at Level C.

Hyperbaric lidocaine with glucose

Figure 2 depicts the mean concentrations of glucose and lidocaine using a logarithmic scale following injection of lidocaine 5% with glucose 7.5%. The mean lidocaine concentration below the site of injection was 3872 ± 252 μg·ml^{-1}, whereas above it was 7.34 ± 4.8 μg·ml^{-1}. The values above the level of injection were different from the values below the site of injection ($P < 0.0001$, t test with Bonferroni correction). There was an abrupt increase in glucose concentration from 54.03 ± 0.29 mg·dl^{-1} above to 458 ± 7.89 mg·dl^{-1} below the site of injection, concomitant with the change in lidocaine concentration. The mean specific gravity above the site of injection was

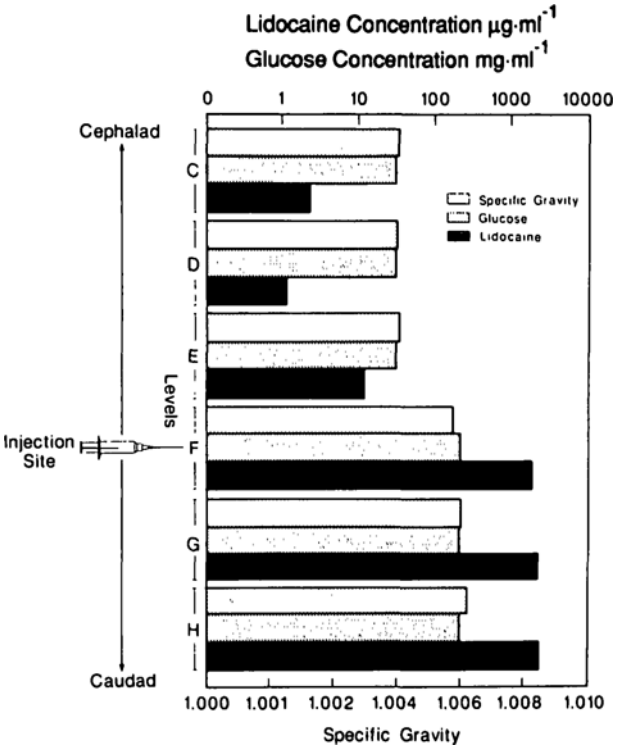

FIGURE 2 Mean concentration of lidocaine and glucose and SG after injection of lidocaine 5% with glucose 7.5% (hyperbaric lidocaine with glucose).

1.004, while below it was 1.0077, a difference which was statistically significant ($P < 0.0001$, t test).

When the hyperbaric lidocaine with glucose was injected in a separate experiment at a more rapid rate (0.5 ml·sec^{-1} instead of 0.2 ml·sec^{-1}), there was an increase in the lidocaine concentration to 3325 ± 588 mg·dl^{-1} at Level E, one level above the site of injection, with a corresponding increase in the glucose concentration (227 ± 48 mg·dl^{-1}), resulting in a specific gravity of 1.006.

In a separate experiment carried out at 37.7° C, the distribution of hyperbaric lidocaine was not significantly different from the distribution at 24° C ($P < 0.0001$, analysis of variance).

Hyperbaric lidocaine without glucose

In the absence of glucose, the 5% lidocaine had a specific gravity of 1.030. The majority of the lidocaine was distributed below the site of injection (Figure 3). The mean lidocaine concentration in all the samples below the site of injection was 5952 ± 302 μg·ml^{-1}. Above the site of injection the mean lidocaine concentration was 135 ± 47 μg·ml^{-1}. The glucose concentrations at all levels were that of the cerebrospinal fluid equivalent. The mean specific gravity above the site of injection (1.004) was

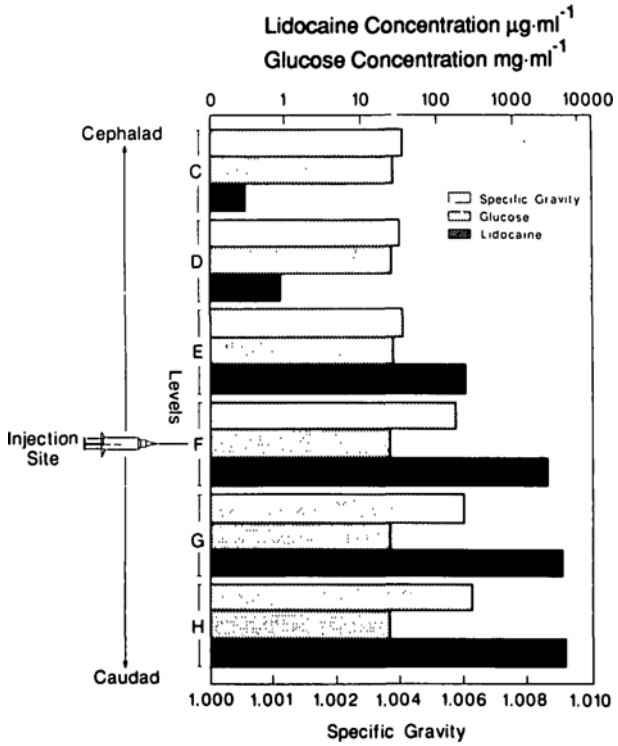

FIGURE 3 Mean concentration of lidocaine and glucose and SG after injection of lidocaine 5% (hyperbaric lidocaine without glucose).

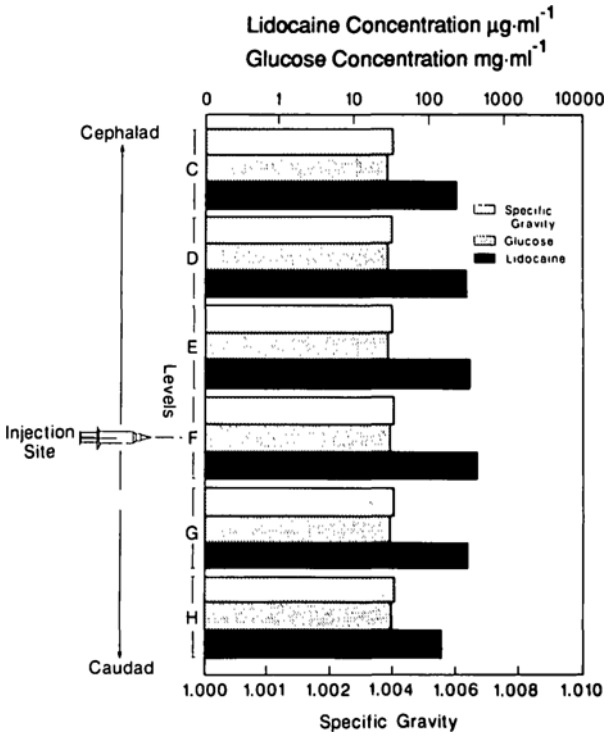

FIGURE 4 Mean concentration of lidocaine and glucose and SG after injection of lidocaine 0.5% (isobaric lidocaine).

different ($P < 0.0001$, t test) from the mean specific gravity below the site of injection (1.006). Similarly, the lidocaine concentration above the injection site was different ($P < 0.0001$) from the mean lidocaine concentration below the injection site.

Isobaric lidocaine (no glucose)

Following the injection of the isobaric solution, the lidocaine distributed itself throughout Levels "C" through "H" (Figure 4) with no differences in the mean lidocaine concentration above and below the site of injection (496 ± 64.8, $\mu g \cdot ml^{-1}$ vs 441 ± 79.4 $\mu g \cdot ml^{-1}$).

Discussion

The results of the present study demonstrate that hyperbaric solutions, with or without glucose, are distributed by gravity at and below the site of injection. Isobaric solutions, on the other hand, are distributed throughout all of the levels measured at 24° C. There was no difference in the distribution of the hyperbaric solutions at 24° C versus 37.7° C but a faster injection rate of 0.5 $ml \cdot sec^{-1}$ created a barbotage effect that carried the lidocaine 10 cm above the site of injection. This was not seen with an injection rate of 0.2 $ml \cdot sec^{-1}$.

Bengtsson[7] injected bupivacaine 0.5% and 0.75%, with and without glucose, into the subarachnoid space of patients sitting for two minutes after the injection. He found no difference in the maximal cephalad spread; all solutions reached a T_6–T_8 level. He concluded that density did not affect the distribution of the local anaesthetic in spinal anaesthesia. Our *in vitro* study contradicts Bengstsson's conclusion. A more logical explanation of Bengtsson's findings is that, although much of the hyperbaric anaesthetic will flow into the sacral portion of the spinal canal with the patient sitting, as soon as the patient is placed in the supine position, some of the hyperbaric solution will course cephalad until it reaches the deepest point in the thoracic concavity, namely, T_6.[12] Since the specific gravity of bupivicaine 0.5% without glucose is 1.004 compared with the average specific gravity of human CSF of 1.0068, the solution is hypobaric. During the two minutes following injection, while the patient is still in the sitting position, this hypobaric solution rises (moves cephalad), reaching the level of T_6–T_8 *before* the patient is returned to the supine position. Hence the level of anaesthesia with the two solutions under the clinical conditions of Bengtsson's study resulted in levels of anaesthesia that were not significantly different.

The present study using a spinal canal model showed that baricity was a major factor in determining the distribution of lidocaine in the CSF.

References

1 *Barker AE*. A report on clinical experiences with spinal analgesia in 100 cases. BMJ 1907; 1: 665–74.

2 *Greene NM*. Physiology of Spinal Anesthesia, 3rd ed. Baltimore: Williams & Wilkins 1981.

3 *Axelson K, Widman B*. Clinical significance of specific gravity of spinal anesthetic agents. Acta Anaesthesiol Scand 1979; 23: 427–34.

4 *Brown DT, Wildsmith JAW, Covino BG, Scott DB*. Effect of baricity on spinal anaesthesia with amethocaine. Br J Anaesth 1980; 52: 589–95.

5 *Chambers WA, Edstrom HH, Scott DB*. Effect of baricity of spinal anaesthesia with bupivacaine. Br J Anaesth 1981; 53: 279–82.

6 *Moller IW, Fernandes A, Edstrom HH*. Subarachnoid anaesthesia with 0.5% bupivacaine: effects of density. Br J Anaesth 1984; 56: 1191–5.

7 *Bengtsson M*. Spinal analgesia, assessment of sensory, motor and sympathetic blockade with special reference to bupivacaine – a clinical study. (Thesis) Linkoping University, Sweden, 1984; 25.

8 *Lund PC*. Principles and Practice of Spinal Anesthesia. Springfield: Charles C. Thomas Publishers 1971.

9 *Cousins MJ, Bridenbaugh PO*. Neural Blockade, 2nd ed., Phildelphia: J.B. Lippincott 1988.

10 *Kaplan LA, Pesce AJ*. Methods in Clinical Chemistry. St. Louis: C.V. Mosby Co. 1987; 911–6.

11 *Kaplan LA, Pesce AJ*. Methods in Clinical Chemistry. St. Louis: C.V. Mosby Co. 1987; 105–12.

12 *Lee JA, Atkinson RS, Watt MJ*. Macintosh's Lumbar Puncture and Spinal Analgesia, 5th ed. London: Churchill Livingstone, 1985; 192.