A DOUBLE-BLIND STUDY OF MOTOR BLOCKADE IN THE LOWER LIMBS

Studies During Spinal Anaesthesia with Hyperbaric and Glucose-free 0.5% Bupivacaine

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Lower abdominal surgery, vascular surgery and orthopaedic procedures on the lower limbs require good motor blockade. With certain local anaesthetics, however, the correlation between the intensity of motor blockade and the quality of sensory blockade seems to be unsatisfactory. During extradural anaesthesia 0.5% amethocaine and 1% etidocaine produce more profound motor paralysis than 0.5% bupivacaine (Bromage, 1969; Bromage, Datta and Danford, 1974; Engberg, Holmdahl and Edström, 1974). Glucose-free 0.5% bupivacaine provides a longer duration of motor blockade than 0.5% bupivacaine containing glucose following subarachnoid administration (Bengtsson, Edström and Lofström, 1983; Krüger et al., 1983; Axelsson, Edström and Widman, 1984). In these investigations the motor blockade in the lower extremities was estimated according to the scale proposed by Bromage (1965).

A new method of quantitating muscle strength (Axelsson et al., 1984) can be used to measure the isometric muscle strength in the lower extremities in non-anaesthetized patients, and in patients undergoing spinal anaesthesia.

The aims of this investigation were: (1) to test this method clinically, and to compare the degrees of sensory and motor blockade provided by the subarachnoid injection of 0.5% bupivacaine 4 ml with and without glucose; and (2) to compare the objectively recorded changes in muscle strength with the results using a modified Bromage scale, and with sensory blockade as assessed by pin-prick.

SUMMARY

Sensory and motor blockade were studied double-blind during spinal anaesthesia in 20 urology patients who received 0.5% bupivacaine solution 4 ml with or without glucose. Using a new method for determining muscle strength, motor blockade during anaesthesia was recorded quantitatively for flexion of the hip, extension of the knee and plantar flexion of the big toe. Movements of the lower part of the thoracic cage were recorded at the same time. Complete motor blockade of longer duration was observed for all three movements following the administration of the glucose-free solution compared with the solution containing glucose. During the regression phase, the muscle strength returned significantly later (knee extension and hip flexion) when glucose-free bupivacaine solution was given. There was no significant difference between the two anaesthetic solutions regarding plantar flexion of the big toe during this phase. For hip flexion (L1-L3) there was no noteworthy difference between the levels of analgesia and the motor blockade, whereas for plantar flexion of the big toe (L5-S2) the level of analgesia was 2-3 segments higher than the level of motor blockade. Thoracic movements (maximal inspiration to normal expiration) did not appear to be notably influenced by the level of analgesia. Complete regression of motor blockade was not observed for any of the movements at grade 0 of a modified Bromage scale. Not until 1.5–2 h after the attainment of this grade was the muscle strength of all movements restored (90% of control value).
PATIENTS AND METHODS

Measurement of motor and sensory blockade

The patients were studied before and after operation with the aid of an apparatus for determining muscle strength (Axelsson et al., 1985). The maximal isometric strength that was generated by the different movements of the lower limbs (see below) was transmitted to a force transducer (Alpha load beam 500 N BLH Electronics). The signals from the transducers were amplified and recorded on a four-channel Siemens-Elema Minograf 34. The error of the method was small (Axelsson et al., 1985).

To obtain supplementary information, mercury strain gauges (Medimatic) were used to measure the movements of the lower part of the thorax during respiratory movements which corresponded to the inspiratory capacity (tidal volume + inspiratory reserve volume). For technical reasons, respiratory movements could be recorded in only nine patients in the group that received glucose-free bupivacaine solution and in eight patients in the glucose-containing bupivacaine group.

The patient lay in the lithotomy position with his hands on his abdomen. The muscle strength in both legs was recorded simultaneously. The following movements were performed (fig. 1):

1. Flexion of the hip joint (femoral nerve L1–L3): The muscle force was taken up by a leather-covered plate and transferred via a lever to a freely movable metal rod; this in turn transmitted it to a force transducer.

2. Extension of the knee joint (femoral nerve L2–L4): the muscle force was transmitted to a transducer via a leather strap fixed around the ankle.

3. Plantar flexion of the big toe (tibial nerve

Fig. 1. Diagram of the apparatus for measuring muscle strength.
L5–S1, S2): this movement was performed against a rigid silicon plate which was connected to the force transducer.

A set of tests consisted of these three movements performed five times. The mean value of the measurements for each movement was calculated on both left and right sides. The set of tests included also five measurements of thoracic movement, and their mean value was calculated in a similar manner.

During the onset and regression of the spinal anaesthetic, the mean value of the isometric strength of each movement in every set of tests was calculated and was expressed as the percent deviation from the control value (mean value of four sets of tests before spinal anaesthesia). The quantitatively determined degree of motor blockade during spinal anaesthesia was presented graphically for each patient. In order to compare motor blockade in the two groups of patients who received 0.5 % bupivacaine 4 ml with and without glucose, respectively, a calculation was made for each movement in every patient of the time taken for 75, 50, 25 and 0% of the control value during onset, and for 0, 25, 50, 75 and 90% of the control value to be regained during the regression of blockade. The mean values of these times for the different levels were calculated for the two bupivacaine solutions, and are presented graphically (figs 3 and 4). The muscle strength of the lower extremities was considered to be restored completely when the strength of each movement had returned to 90% of its control value, and the patients were able to walk about 5 m.

Simultaneously with the tests of muscle strength (during onset of blockade), the degree of motor blockade was recorded in terms of a four-grade modified Bromage scale (0–3): 0 = no paralysis; 1 = inability to raise extended leg (just able to move knees); 2 = inability to flex knee (able to move feet only); and 3 = inability to flex ankle joint and first digit.

During the regression phase, grades 2 and 1 were given a modified definition, while grades 0 and 3 were unchanged: 3 = inability to flex ankle joint and first toe; 2 = just able to move knee; 1 = ability to raise extended leg but inability to flex ankle joint and first digit; and 0 = no paralysis.

During the onset of sensory blockade the level of analgesia was determined by pin prick until maximum cephalad spread of the analgesia had been attained. During regression, corresponding recordings were made every 15–30 min or, in certain patients, at every muscle strength test, until normal sensation had returned.

**Patients**

The double-blind study comprised 20 patients (ASA 2–3) undergoing urological surgery (TUR of cancer of the urinary bladder). They had neither spinal deformity, nor mental or neurological disorder. The patients were comparable in age, height and weight (table I). They were given verbal and written information about the study, which was approved by the Ethics Committee of the Örebro Medical Centre Hospital.

**Procedure**

The patients were allocated randomly to two groups of 10 patients each. One group received 0.5% bupivacaine solution 4 ml in 0.9% sodium chloride (density 1.000 at 37 °C, pH 6.2); the other group received 0.5% bupivacaine 4 ml containing 8% glucose monohydrate (density 1.021 at 37 °C, pH 5.0–6.0). No premedication was given, apart from atropine 0.5 mg i.v. just before the intrathecal injection.

With the patient in the sitting position, lumbar puncture (L3–L4) was performed (22-gauge needle) and the appropriate solution administered (0.5 ml s⁻¹). The patient remained sitting for 2 min, after which he was placed in the lithotomy position. All patients received 500 ml of Ringer's glucose solution just before the lumbar puncture. Any decrease in systolic arterial pressure by more than 30% was treated initially with etilefrine chloride (Etilefrinum) or dihydroergotamine (Orstanorm).

As soon as the patient had been placed in the lithotomy position, measurements of muscle power were undertaken: first, plantar flexion of the big toe, then extension of the knee and, finally, flexion of the hip. The measurements were repeated until motor blockade was complete or a steady state had been reached. Thoracic movements

<table>
<thead>
<tr>
<th>Table I. Patient characteristics (mean ± SEM)</th>
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<tr>
<td>Age (yr)</td>
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<tr>
<td>Glucose-free bupivacaine 20 mg</td>
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<td>Bupivacaine 20 mg + glucose 80 mg ml⁻¹</td>
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</table>
were recorded with the strain gauge during the onset of blockade once the level of analgesia had stabilized. Determinations of muscle strength were performed repeatedly during the regression of blockade.

One to two days after the operation the patients were questioned about possible post-spinal complications. In addition, they were asked to communicate with the authors should any complication arise following discharge from hospital.

Definitions

Regression time. The time from commencement of regression of blockade until 90% of the initial muscle strength had been regained for each movement.

Total regression time. The time from the commencement of regression of blockade until 90% of the initial muscle strength had been regained for all movements.

Statistics

Student’s t test for independent samples was used. The level of significance was P < 0.05.

RESULTS

Sensory blockade

There was a rapid onset of analgesia in both groups. Fifteen minutes after the intrathecal injection, the upper level of analgesia was T7–T8 and after 30 min it had reached T6–T8 irrespective of whether the bupivacaine was with or without glucose. There were no significant differences in the spread of the analgesia between the patients who had received the two different solutions (fig. 2). All patients in both groups had thoracic analgesia.

The duration of analgesia was significantly longer in the lower thoraco–lumbar segment when bupivacaine without glucose had been given. With the glucose-free solution the analgesia at the level of T11–T12 lasted for 210 min, compared with 150 min for the solution containing glucose. The corresponding durations for the L3–L4 level were 270 and 210 min, respectively.

Onset of motor blockade

As there was no significant difference in the degree of motor blockade between the left and right sides, descriptions of motor block have been based on the mean result from the two sides. All patients in the glucose-free group had complete motor paralysis. In the glucose-containing group one patient had complete motor blockade in the muscles performing plantar flexion of the big toe, while knee extension and hip flexion remained at muscle strength values of 19 and 10% of the control values 30 min after spinal injection. For
FIG. 3. Onset (left and the circle) and regression (to the right) of motor blockade following spinal injection of 0.5% glucose-free bupivacaine 4 ml. The arrows indicate the times at which different grades on the Bromage scale were reached. There was no significant difference between the three types of movement.

At Bromage 0 there was a considerable degree of motor blockade.

FIG. 4. Onset (left and the circle) and regression (to the right) of motor block following spinal injection of 0.5% bupivacaine 4 ml with 8% glucose. The time of onset and regression at 0% level is based on nine patients (knee extension and hip flexion). The arrows indicate the Bromage grading. At Bromage 0 the strength of knee extension had largely returned, while the motor block still remained in the other movements.
this reason, the time of onset and regression at 0% level is based on nine patients.

The onset of motor blockade was rapid in both groups (figs 3 and 4). Only 50% of the initial muscle strength of the three muscle movements remained 3-4 min after injection. After 5 min in the glucose-free group and 5-8 min in the glucose group, the muscle strength was 25% of its control value. Fifteen minutes after injection, complete motor blockade was attained with both solutions. During the onset of motor blockade there were no significant differences between the two groups.

Regression of motor block

The patients receiving 0.5% bupivacaine without the addition of glucose could begin to flex their hips 225 min after the injection, and knee extension and plantar flexion of the big toe began to return about 20 and 25 min later, respectively (fig. 3). During regression there was no significant difference between the restoration of muscle strength in the three types of movement. Regression time was 120-150 min for each movement (fig. 3), and total regression time was almost 160 min. Ninety per cent of the muscle strength had returned for plantar flexion of the big toe and extension of the knee, after 390 and 375 min, respectively.

In the patients receiving 0.5% bupivacaine with the addition of glucose, ability to flex the hips began to return 130 min after the spinal injection, and about 10 min later knee extension became possible (fig. 4). Plantar flexion of the big toe recovered about 60 min after hip flexion. The muscle strength of plantar flexion of the toe took significantly longer to return to the levels of 0, 25 and 50% of the initial value than that of the other two movements. At the 75% and 90% levels there were significant differences in the duration between extension of the knee and plantar flexion of the big toe. The regression time for the different types of movement varied between 120 and 180 min. The total regression time was just over 200 min. The muscle strength of plantar flexion of the big toe and of extension of the knee returned 340 and 255 min, respectively, after the spinal injection (fig. 4).

On comparing the glucose and glucose-free groups, the duration of complete motor blockade was found to be significantly longer for all movements in the latter group.

During the regression phase, on the average, hip

![Graph showing motor blockade in spinal anaesthesia. Comparison of 0.5% bupivacaine 4 ml with and without added glucose. Plantar flexion of big toe: The glucose-free solution gave significantly longer complete motor block (0% level) than the solution containing glucose.](https://academic.oup.com/bja/article-abstract/57/10/960/399896/fig-5)
flexion could be performed earlier than knee extension and plantar flexion of the big toe, irrespective of local anaesthetic. This order of sequence was noted in seven of 10 patients in the glucose-free group and in three of 10 in the glucose group. Hip flexion and knee extension returned simultaneously in one of 10, and in six of 10 patients in these two groups, respectively. Plantar flexion of the toe returned last in eight of 10 in the glucose-free group and in all patients of the glucose group. Regression of motor blockade commenced significantly later for plantar flexion of the toe when glucose-free solutions had been given, compared with the solution containing glucose. The lengths of time required to reach the levels of 25, 50, 75 and 90% of the control value did not differ significantly between the two groups (fig. 5). For extension of the knee (fig. 6) and flexion of the hip (fig. 7), except for hip flexion at the 90% level, the isometric strength was restored significantly more slowly in the glucose-free group than in the group that received the glucose-containing solution.

**Comparison between modified Bromage scale and method for measuring muscle strength**

Using the modified Bromage scale, the duration of motor blockade was found to be significantly longer in those patients receiving glucose-free solution than in the group that received the glucose-containing solution (table II).

In the glucose-free group, of the initial strength of knee extension and hip flexion, 5% and 16% respectively, had returned when the modified Bromage grade changed from 3 to 2 (fig. 3). At a modified Bromage grade of 0, the muscle strength of plantar flexion of the big toe, knee extension and

**Table II. Duration of motor block (min) as recorded by the Bromage scale. A significant difference between the two anaesthetic solutions is indicated by the t value. **P < 0.01; ***P < 0.001**

<table>
<thead>
<tr>
<th>Bromage grade</th>
<th>Glucose-free bupivacaine 20 mg</th>
<th>Bupivacaine 20 mg + glucose 80 mg ml⁻¹</th>
<th>t</th>
</tr>
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<tbody>
<tr>
<td>3</td>
<td>257 ± 20.3</td>
<td>148 ± 18.3</td>
<td>4.09***</td>
</tr>
<tr>
<td>2</td>
<td>285 ± 20.3</td>
<td>170 ± 16.9</td>
<td>4.36***</td>
</tr>
<tr>
<td>1</td>
<td>307 ± 21.3</td>
<td>222 ± 14.6</td>
<td>3.32**</td>
</tr>
</tbody>
</table>
Fig. 7. Motor blockade in spinal anaesthesia. Comparison of 0.5% bupivacaine 4 ml with and without added glucose. Flexion of the hip: The time of onset and regression at 0% level is based on nine patients (0.5% bupivacaine 4 ml with glucose). *P < 0.05; **P < 0.01; ***P < 0.001.

Fig. 8. Sensory and motor blockade in spinal anaesthesia. The level of analgesia correlated with motor blockade curve after spinal injection of 0.5% glucose-free bupivacaine 4 ml. The isometric strength of hip flexion returned at the same time as the analgesia (pin-prick) changed from L1 to L2. When the level of analgesia changed from L5 to S1, the strength of knee extension had largely returned, while the strength of plantar flexion of the big toe was still reduced.
hip flexion had regained 16%, 32% and 38% of their control values, respectively (fig. 3). In the glucose group, grade 3 on the modified Bromage scale changed to grade 2 when 5% and 13% of the initial muscle strength had returned for knee extension and hip flexion, respectively. Modified Bromage grade 1 changed to 0 when the corresponding figures for plantar flexion of the big toe, knee extension and hip flexion were 8%, 75% and 58%, respectively (fig. 4).

Relation between segmental level of analgesia and motor blockade

The strength of hip flexion and knee extension (L1–L4) began to return when the level of analgesia (to pin-prick) was around L1–L2 in the glucose-free group (fig. 8) and at T12 in the group that received the glucose-containing solution (fig. 9). For plantar flexion of the big toe (L5–S2), the muscle strength returned when the level of analgesia was around L3–L4 for both solutions. When grade 3 on the modified Bromage scale changed to grade 2, the upper level of analgesia lay at around L3 in the glucose-free group and at T12 in the glucose-containing group. At modified Bromage grade 0 the upper level of analgesia lay at around L4 for both solutions (figs 8, 9).

Patients were mobilized once they had regained 90% of the initial muscle strength at each movement bilaterally, and after the heart rate had been assessed in the sitting position. This occurred after about 420 min in the glucose-free group and after 395 min in the other group. This difference was not significant.

Thoracic movements did not appear to be influenced by the extent of the spinal anaesthesia. Even when the upper level of analgesia lay at about T3, the deflections showed a pattern similar to that obtained under control conditions.

Complications

A decrease in systolic arterial pressure of more than 30% was noted in two patients in the glucose-free group and in three in the glucose
MOTOR BLOCKADE IN SPINAL ANAESTHESIA

Table III. Frequency of reductions in systolic arterial pressure (percentage of control)

<table>
<thead>
<tr>
<th>Decrease in arterial pressure (%)</th>
<th>Bupivacaine 20 mg</th>
<th>With glucose 80 mg ml⁻¹</th>
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<tbody>
<tr>
<td>20-30</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>≥ 30</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

group (table III). Two patients in each group were treated with etilefrine chloride and one patient was given dihydroergotamine. One patient in each group received additional atropine. In one of the five patients the duration of both sensory and motor blockade was longer than the average. One patient in the glucose-free group complained of nausea in connection with hypotension. No post-spinal headache occurred.

DISCUSSION

In most recent investigations of motor paralysis during spinal blockade, blockade has been assessed by the method proposed by Bromage (1965). However, the Bromage method is qualitative and does not permit of exact comparisons. Hitherto no method has been available for the quantitative determination of the degree of motor blockade. The apparatus used in this study was constructed to measure the maximal isometric strength in the lower extremities during spinal anaesthesia and so determine motor blockade quantitatively. At the same time, the thoracic respiratory movements could be recorded to assess the motor blockade of the lower intercostal muscles.

A comparison of the two groups of patients who had received 0.5% bupivacaine 4 ml, with or without the addition of glucose, found that complete motor blockade was of significantly longer duration (in all movements) when the glucose-free solution had been given. When all movements were included, in the glucose-free group the total regression time was significantly shorter than the duration of complete motor blockade. The opposite was found in the other groups. Thus, a glucose-free solution should be preferable for operations requiring prolonged complete motor blockade.

With regard to hip flexion and knee extension, the duration of motor blockade was significantly longer in the glucose-free than in the glucose-containing group. As the regression time for each movement was about 2–3 h irrespective of anaesthetic solution, the longer duration of motor blockade with the glucose-free solution must be attributable to a high concentration in the region L1–L4 which innervates the muscles for hip flexion and knee extension. The difference in the duration of motor blockade between the two anaesthetic solutions was greatest in the muscles performing knee extension. This indicates that the glucose-free solution was present at a higher concentration than the glucose-containing solution in the motor roots around the L2–L4 region, that is the site of the spinal injection.

The motor level during spinal anaesthesia is said to lie 2–3 segments below the sensory level (Freund et al., 1967). In the present study the sensory and motor levels differed by slightly less (by 0–2 segments) in the upper lumbar segments. In the sacral segments the difference was in accord with previously reported findings.

The use of the modified Bromage scale for recording motor blockade did not permit an equally reliable evaluation of the level of analgesia. When grade 3 on the modified Bromage scale changed to grade 2, there was some difference in the level of analgesia between the two solutions. At modified Bromage grade 0 the level of analgesia was the same for both solutions.

The original Bromage scale is based on the assumption that the onset of motor blockade takes place first in the hip and knee, followed by the foot and toe. According to our previous findings, motor blockade regresses last in the foot and toe and, consequently, we have altered the order of sequence of the scale so that it better fits with our clinical practice. In the present study, during regression, only seven of the 10 patients who received the glucose-free solution and three of the 10 in the glucose group matched the modified order of sequence (hip followed by knee and foot/toe last) when it was assessed by our method of measuring muscle strength.

Irrespective of which anaesthetic solution had been given, when motor blockade changed from grade 1 to 0, the muscle strength had not regained the normal value for any of the types of movement studied. In the group which received glucose in the solution, knee extension and hip flexion had returned to a greater extent (75% and 58%, respectively) than the corresponding movements in the glucose-free group (36% and 38%). Thus, modified Bromage grade 0 does not represent...
equal degrees of regression of motor blockade in patients receiving 0.5% bupivacaine 4 ml with and without glucose. Not until 1.5–2 h had elapsed after Bromage grade 0 had been reached had the strength returned in all movements irrespective of anaesthetic solution.

Had the foot movement been tested alone (cf. Lanz et al., 1983), the difference in the duration of complete motor blockade between the two solutions would have persisted. No difference would have occurred during the regression, as was obtained in the present study when two other types of movements were investigated also. In seven of our patients motor blockade remained in one of the other two movements when the strength of plantar flexion of the big toe had returned to normal. Thus, to obtain more information on motor blockade associated with spinal anaesthesia, testing of three movements of the lower extremities is to be preferred to testing of the movement of the foot alone.

CONCLUSION

Glucose-free bupivacaine solution gave significantly longer complete motor blockade than bupivacaine solution without glucose. The muscle strength of hip flexion and knee extension returned significantly later after administration of the glucose-free anaesthetic solution. A shorter total regression time (all movements) in the glucose-free anaesthetic group than in the group that received glucose in the anaesthetic solution meant that the patients could be mobilized after almost the same length of time, 6.5–7 h, irrespective of which solution had been given — that is, not until 1.5–2 h had elapsed after Bromage grade 0.

ACKNOWLEDGEMENTS

This study was supported by grants from the Örebro County Council. The help and guidance of Bertil Lofström, Professor of Anaesthesiology, Linköping University, throughout the study, are greatly appreciated. The authors also thank Eric Leander, Lecturer in Statistics at the University of Linköping, for helping with the statistical analysis. The assistance of Ing-Marie Ackheim, Kerstin Larsson, Håkan Karlsson, Ulf Larsson and Rolf Johansson is gratefully acknowledged.

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