Recovery of Storage and Emptying Functions of the Urinary Bladder after Spinal Anesthesia with Lidocaine and with Bupivacaine in Men

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Background: The aim of this study was to evaluate and compare the effects of spinal anesthesia with lidocaine and with bupivacaine on urinary bladder function in healthy men who were scheduled for minor orthopaedic surgical procedures.

Methods: Twenty men were randomly allocated to receive either bupivacaine or lidocaine. Before spinal anesthesia, filling cystometry was performed with the patient in the supine position and a pressure flow study was done with the patient in the standing position. After operation, cystometric measurements were continued until the patient could void urine spontaneously. The levels of analgesia and of motor blockade were recorded.

Results: The urge to void disappeared immediately after injection of the local anesthetics. There was no difference in the duration of lower extremity motor blockade between bupivacaine and lidocaine. Detrusor blockade lasted significantly longer in the bupivacaine group (means ± SD, 460 ± 60 min) than in the lidocaine group (235 ± 30 min). Total fluid intake and urine volume accumulated during the detrusor blockade were significantly higher in the bupivacaine group than in the lidocaine group. In the bupivacaine group, the total volume of accumulated urine (875 ± 385 ml) was also significantly higher than cystometric bladder capacity (505 ± 120 ml) with the risk of over distension of the bladder. Spontaneous voiding of urine did not occur until segmental sensory analgesia had regressed to the third sacral segment.

Conclusions: Spinal anesthesia with lidocaine and with bupivacaine causes a clinically significant disturbance of bladder function due to interruption of the micturition reflex. The urge to void disappears quickly and bladder function remains impaired until the block has regressed to the third sacral segment in all patients. With long-acting local anesthetics, the volume of accumulated urine may exceed the cystometric bladder capacity. With respect to recovery of urinary bladder function, the use of short-acting local anesthetics for spinal anesthesia seems to be preferable. (Key words: Bladder function; segmental sensory analgesia; urge.)

THE bladder has two functions: to collect and to expel urine. The coordination of bladder filling, urine storage, and voluntary micturition are under the control of supraspinal central, somatic, and visceral neurons in the thoracic, lumbar, and sacral spinal cord.

The walls of the bladder are composed primarily of smooth muscle and is called the detrusor muscle. Figure 1 shows a simplified representation of the lower urinary tract and its innervation. The trigone, the posterior part of the base of the bladder, extends between the ureteric orifices and the internal urethral meatus. The smooth muscle of the male bladder neck surrounds the preprostatic portion of the urethra and forms the internal urethral sphincter. The external sphincter is composed of striated muscle fibers and surrounds the distal portion of the prostate and the membranous urethra and forms a part of the urogenital diaphragm (fig. 1).1

The pelvic nerves (S2–S4) contain the sacral parasympathetic reflex arc, which leads the activity of the detrusor muscle and bladder neck.2–4 The pudendal nerve (S2–S4) innervates the skin, urogenital organs, and the external sphincter.5,6 Sympathetic innervation of the urinary bladder, the center of which is located in the thoracolumbar spinal cord (Th10 to L2), has an inhibitory effect on the detrusor and an excitatory effect on the base of the bladder, bladder neck, and urethra via the pelvic and hypogastric nerves.6–8

Although the precise neurologic pathways and functions concerned with urine storage and voiding have not been elucidated completely, it is generally accepted that the second, third, and fourth sacral nerves contain most of the fibers concerned with the control of the bladder and urethral sphincters.9 Filling of the bladder stimulates stretch receptors in the bladder wall that...
transmit sensory signals to the sacral spinal cord via the pelvic nerves. Impulses are then transmitted via the spinothalamic tracts to the frontal lobe. Voluntary micturition is initiated by efferent discharges from the cortex to the pontine micturition center which, via the reticulospinal tract, activates preganglionic parasympathetic motor neurons in the sacral (S2-S4) intermediolateral cell group. These motor neurons initiate contraction of the detrusor muscle. Descending efferent pathways produce a temporary inhibition of sympathetic firing via the hypogastric nerves. This inhibition promotes the opening of the bladder neck, a decrease in urethral pressure, and an increase in detrusor tone. At the same time, neuronal discharges in the pudendal nerve (S2-S4) to the striated muscle of the external sphincter are inhibited and voiding ensues.\textsuperscript{10}

The purpose of this study was to measure the effects of spinal anesthesia with bupivacaine and with lidocaine on the storage and emptying functions of the urinary bladder.

Materials and Methods

After obtaining approval from the Committee on Medical Ethics of the University Hospital, Utrecht, and the informed consent of the patients, the study was performed in 20 men classified as American Society of Anesthesiologists’ physical status I who were aged 19-50 yr and scheduled for elective minor orthopedic surgery of the lower limb during spinal anesthesia. Patients with a history of mental, hepatic, renal, urinary tract, spinal, or neurologic disorders, and those who were taking any medication were not accepted for the study. The patients were randomly allocated to the bupivacaine or to the lidocaine group. No premedication was used.

When the patient arrived in the urodynamic room, electrocardiographic monitoring was initiated and baseline blood pressure, heart rate, and pulse oximetry were measured and recorded. An intravenous cannula for infusion was inserted, through which Gelofusine (a gelatin solution; Vifor Medical SA, Switzerland) was slowly infused.

Cystometry is a method by which the pressure-volume relation of the bladder is measured (fig. 2) and used to assess detrusor activity, sensation, capacity, and compliance. Before cystometry, a urethral catheter is placed and residual volume is measured. The bladder is then filled. During filling and voiding, the pressure in the bladder is measured using a second urethral catheter. To measure the pressure in the abdomen, a catheter is inserted in the rectum. In clinical practice, intrarectal pressure appears to be a fair approximation of abdominal pressure. Bladder and rectal measuring catheters are connected to pressure transducers. All systems are zeroed at atmospheric pressure and the reference point is the superior edge of the symphysis pubis. The intravesical pressure is the pressure within the bladder. The detrusor pressure is that component of intravesical pressure that is created by forces in the bladder wall. It is estimated by subtracting abdominal pressure (rectal pressure) from intravesical pressure. The urinary flow rate during voiding is defined as the volume of fluid expelled via the urethra per unit of time and is expressed in milliliters per second. The urinary flow rate is measured using a rotating disc uroflowmeter (standard equipment in general urologic practice). Flow rate is registered simultaneously with the pressures. The maximum flow rate is the maximum measured value of the flow rate during voiding.\textsuperscript{11}

In this study, the bladder pressure was measured using a 5-French urethral catheter and the rectal pressure was measured using a 14-French catheter, and both were expressed as centimeters of water. After emptying via the catheter, the bladder was filled with saline at 37°C, through a second 5-French urethral catheter, at
was lower extremity motor blockade using the Bromage score. (0, no motor block; 1, inability to raise the extended leg; 2, inability to flex the knee; 3, inability to flex the ankle). After the bolus, the Gelofusine infusion was reduced to a rate of 1 ml·kg⁻¹·h⁻¹ and allowed to run until the end of the operation.

When spinal anesthesia was considered sufficient for surgery, the operation was started. After surgery, sensory segmental analgesia and lower extremity motor blockade were recorded every 30 min. At the same time, the sensation of urge at previous recorded cystometric capacity was estimated by repeated cystometrograms. Before filling the bladder every 30 min, the urine volume accumulated in the bladder was measured and recorded. The urinary bladder was refilled up to the cystometric capacity and spontaneous voiding of the bladder was attempted. If there was no detrusor activity (that is, no spontaneous micturition), the bladder was emptied. These periods of detrusor inactivity are shown in figure 3B and 3C.

The duration of motor blockade was defined as the time from the injection of the local anesthetics until total recovery of hip, knee, and ankle motility. The investigation was concluded when the sensation of urge at cystometric capacity had returned and the patient could empty his bladder (fig. 3D). The duration from spinal injection until total recovery of bladder function (return of urge at cystometric capacity together with the ability to empty completely the bladder only with the help of the detrusor) is defined as the detrusor block. After operation all patients were allowed free oral fluids, and this intake and urine production was recorded. The volume of urine accumulated in the bladder and measured before refilling the bladder every 30 min, plus the difference between filling volume and emptied volume at these time points were recorded as total urine volume produced by the patients during the detrusor block.

The methods, definitions, and units of the pressure flow study were those proposed by the International Continence Society, except when specifically noted.¹¹

The results were recorded as means ± SD. Nonparametric tests were used to evaluate differences (Mann–Whitney U test for unpaired differences, Wilcoxon signed rank test for paired differences). Probability values <0.05 were considered significant.

Results

The groups were not significantly different with respect to height and weight, but the men in the lidocaine
Fig. 3. Registration of intravesical pressure, abdominal pressure, detrusor pressure (intravesical pressure minus abdominal pressure), and flow rate in one patient. (A) Pressure flow study before anaesthesia. (B) Pressure flow study during detrusor blockade (no detrusor activity and no flow is recorded). Because of movement artifacts transmitted along the connections to the pressure transducers, artificial pressure spikes are created on the pressure registrations. (C) After regression of segmental sensory analgesia to the first sacral segment, the patient could void a part of the bladder content only by extreme abdominal straining. Because of movement artifacts and to small differences between rectal and bladder pressure response, artificial pressure spikes are created on the detrusor pressure registration during extreme straining. (D) Pressure flow study on return of detrusor function. Absolute intravesical and abdominal pressures are determined by the position of the external pressure transducers. Thus, intravesical and abdominal pressure are artificially high.

Group were significantly older (38 ± 8 yr) than those in the bupivacaine group (29 ± 9 yr, table 1). Urodynamic parameters before anesthesia were not significantly different between the groups (table 2). No patient appeared to have residual volume after voiding before operation.

The urge to void had disappeared within 60 s after start of injections of local anesthetics. One patient given bupivacaine had no motor blockade despite adequate analgesia. Four patients (two in each group), in whom the segmental sensory analgesia had regressed to the first sacral segment, experienced abdominal tension but no urge to void at cystometric capacity. Two of these patients could void a part of the bladder content only with the help of extreme abdominal straining (fig. 3C). Recovery of the ability to void normally (fig. 3D) did not occur until the segmental sensory analgesia had regressed to the second sacral segment. Fourteen patients, seven in each group, in whom the segmental analgesia had regressed to the second sacral segment, experienced urge at cystometric capacity and were able to empty their bladders. The remaining six patients were able to void when segmental analgesia had reached the third sacral segment. During anesthesia, no bradycardia or arterial hypotension was recorded.

Urodynamic parameters after anesthesia did not differ from those before anesthesia in both groups (table 2). The duration of lower extremity motor blockade did not differ between the groups (table 3, fig. 4), but detrusor blockade lasted significantly longer in patients given bupivacaine than in patients given lidocaine (table 3, fig. 4). Although the groups differed in age, mean durations of detrusor blockade and motor blockade did not change when adjusted for age. Total fluid intake and total accumulated urine volume was significantly greater in the patients given bupivacaine than in patients given lidocaine (table 3), reflecting the longer duration of sensory block in men in the bupivacaine group (fig. 4). In the patients given bupivacaine, total accumulated urine volume was significantly ($P < 0.01$) greater than the cystometric bladder capacity (table 3).
Table 1. Demographic Data

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Lidocaine Group (n = 10)</th>
<th>Bupivacaine Group (n = 10)</th>
<th>Significance (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>38 ± 8</td>
<td>29 ± 9</td>
<td>0.03</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>180 ± 9</td>
<td>182 ± 6</td>
<td>NS</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>85 ± 13</td>
<td>83 ± 14</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS = not significant.

Discussion

All the patients in this study had normal lower urinary tract function, which was unlikely to have been influenced by the type of surgery performed. Although there was a significant difference in mean age between both groups, this did not influence the results. Disappearance of the urge to void occurred immediately and completely at the end of spinal injection of the local anesthetics in all patients. This observation differs from those of Axelsson et al., who reported "a dull feeling of tension," a delay of 2–5 min before the disappearance of urge, and only a partial disappearance of urge in some patients. These differences could have been caused by the selection of the patients (Axelsson’s group studied patients scheduled for urologic surgery) and differences in urodynamic methods.

The duration of absence of the urge coincided with the time required for the sensory blockade of the spinal cord to regress to the S3 segment. Axelsson et al. however, found that detrusor activity returned when the level of analgesia was "at or caudal to L5."

Table 2. Urodynamic Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Lidocaine Group (n = 10)</th>
<th>Bupivacaine Group (n = 10)</th>
<th>Significance (P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cystometric capacity (ml)</td>
<td>565 ± 162</td>
<td>505 ± 119</td>
<td>NS</td>
</tr>
<tr>
<td>Detrusor pressure at maximum flow before anesthesia (cmH₂O)</td>
<td>43 ± 10</td>
<td>50 ± 16</td>
<td>NS</td>
</tr>
<tr>
<td>Maximum flow before anesthesia (ml/s)</td>
<td>20 ± 8</td>
<td>20 ± 7</td>
<td>NS</td>
</tr>
<tr>
<td>Detrusor pressure at maximum flow on return of detrusor function (cmH₂O)</td>
<td>43 ± 6</td>
<td>49 ± 18</td>
<td>NS</td>
</tr>
<tr>
<td>Maximum flow on return of detrusor function (ml/sec)</td>
<td>19 ± 7</td>
<td>17 ± 5</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS = not significant.

In our study, the duration of detrusor blockage was much longer than the motor blockage in both groups. The identical length of the motor blockage in both groups may be related to the relatively high dose of lidocaine. The total accumulated urine volume in the men in the bupivacaine group was greater than the cystometric bladder capacity. Under these conditions and without bladder catheterization, the bladder can be significantly distended. This problem has been described by Tattersal, who warned that large urine volumes can be produced during spinal anesthesia with long-acting local anesthetics.

All patients studied in this trial were able to void completely when the level of sensory anesthesia reached the S3 segment. There was no difference between the flow before compared with after anesthesia in both groups, which is possible only if the urinary outflow tract also is recovered. The bladder neck is a part of the urinary outflow tract and also affects the flow.

Four patients, who had no urge to void, experienced a sensation of abdominal tension as the bladder was filled, and two of these were able to void a small volume of urine using abdominal muscle strain when the level had reached S1. Scott reported that voiding by abdominal strain is commonly seen during unilateral or saddle block anesthesia. It seems that the whole act of micturition remained uncoordinated until motoneuron function at S3 had fully recovered. Although it is known that many factors can predispose to urinary retention after anesthesia, the frequency of this complication remains controversial. Tammela et al. reported an incidence ranging from 4% to 25% of urine retention and
SPINAL ANESTHESIA AND URINARY BLADDER FUNCTION

Fig. 4. (Top) Time course of segmental level of analgesia in ten patients after spinal anesthesia with 2 ml lidocaine, 5% hyperbaric, until spontaneous voiding. (Bottom) Time course of segmental level of analgesia in 10 patients after spinal anesthesia with 2 ml bupivacaine, 0.5% hyperbaric, until spontaneous voiding. Seven patients in each group, in whom the segmental analgesia had regressed to the second sacral segment, experienced urge at cystometric capacity and could empty their bladders. The last six patients could void urine spontaneously when segmental analgesia had reached the third sacral segment. Arrow 1: Average motor block time in the lidocaine (144 ± 35 min) and in the bupivacaine (148 ± 76 min) groups. Arrow 2: Average detrusor time block in the lidocaine (233 ± 31 min) and in the bupivacaine (462 ± 61 min) groups.

bladder dilatation in 8% of patients. Orkoh and Rosenberg recorded an incidence of 18% of disturbed micturition after elective surgery under general anesthesia. The frequency of micturition complications after spinal anesthesia with local anesthetics is probably higher but, as yet, unknown. Stricker and Steiner reported a 26% incidence of urinary retention after spinal anesthesia with tetracaine and lidocaine with or without adrenaline in patients with and without a history of urinary tract problems, whereas other authors found a frequency of 36%,18,19. The frequency of urinary retention after spinal anesthesia is increased if large volumes of fluids are administered to prevent hypotension.

The urodynamic changes during and after peridural and spinal anesthesia with local anesthetics may last for many hours and depend partly on the physicochemical properties of the drug. In most cases, no severe long-term complications develop. However, the physician should be concerned about the patient in whom regres-

sion of the somatic blockade is slow and who has not passed urine. Potentially harmful urinary retention should be suspected in the presence of severe pain, bradycardia, hypotension or hypertension, heart dysrhythmias, or vomiting. Urinary retention can produce irreversible detrusor damage leading to incontinence and recurrent urinary infections.20

In conclusion, spinal anesthesia with lidocaine and with bupivacaine causes a clinically significant disturbance of bladder function due to interruption of the micturition reflex. This study shows that the urge to void disappears quickly after spinal injection of the local anesthetics and that bladder function remains impaired until the sensory block has regressed to the S3 segment. The extremely long-lasting recovery of the urinary bladder function may imply that the contents of the urinary bladder may easily exceed the cystometric capacity of the urinary bladder before the normal function has reappeared, thus leading to acute postoperative distension. This justifies the need to accurately monitor the filling condition of the urinary bladder, to apply voluntary abdominal strain in case a full bladder is suspected, and ultimately single bladder catheterization if voiding is not achieved. Spontaneous voiding may not be expected until regression of the sensory blockade has reached the S3 level. This may not happen until many hours after disappearance of the motor blockade. With the increasing number of patients now being treated under spinal anesthesia, it is important to recognize the effect of this type of anesthesia on urinary bladder function.

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References


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