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Isobaric versus hypobaric spinal bupivacaine for total hip arthroplasty in lateral position

Comparaison de l'anesthésie rachidienne isobare ou hypobare pour la chirurgie de la prothèse totale de la hanche en position de décubitus latéral

Thèse
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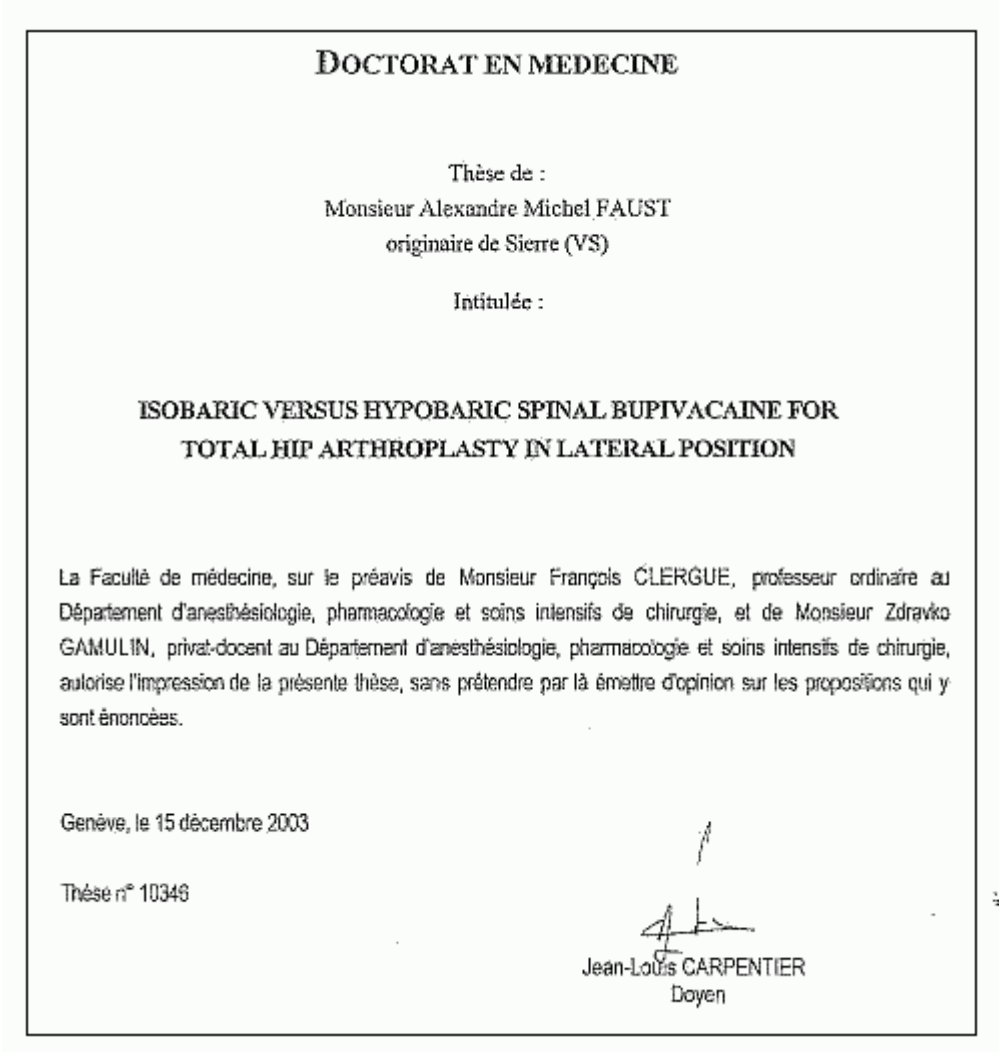

DOCTORAT EN MEDECINE

Thèse de :
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Intitulée :

ISOBARIC VERSUS HYPOBARIC SPINAL BUPIVACAINE FOR TOTAL HIP ARTHROPLASTY IN LATERAL POSITION

La Faculté de médecine, sur le préavis de Monsieur François CLERGUE, professeur ordinaire au Département d'anesthésiologie, pharmacologie et soins intensifs de chirurgie, et de Monsieur Zdravko GAMULIN, privat-docent au Département d'anesthésiologie, pharmacologie et soins intensifs de chirurgie, autorise l'impression de la présente thèse, sans prétendre par là émettre d'opinion sur les propositions qui y sont énoncées.

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Le but de cette étude est de comparer les effets anesthésiques et hémodynamiques de l'anesthésie rachidienne (AR) en utilisant des solutions d'anesthésiques locaux isobares ou hypobares, chez des patients bénéficiant de la mise en place d'une prothèse totale de la hanche en décubitus latéral.

L'anesthésie rachidienne (AR) consiste en l'injection d'un anesthésique local (AL) dans l'espace sous arachnoïdien (ou intrathécal) qui, en se mélangeant avec le liquide céphalo-rachidien (LCR) entraîne un blocage de la conduction des nerfs spinaux.

Se référant aux descriptions de Koller en 1884 concernant les propriétés anesthésiques topiques de la cocaïne, James Corning injecte cette substance dans la région médullaire chez un chien, ce qui entraîne une paralysie transitoire des membres postérieurs. En 1899, Augustus Bier est le premier à administrer de la cocaïne dans l'espace intrathécal en vue d'une chirurgie. Par la suite, l'AR va se répandre rapidement en Europe, aux USA ainsi que dans les pays du Tiers Monde en raison de sa simplicité, de son efficacité et de son bas prix. Alors que nombreux cliniciens considéraient durant la première moitié du siècle les bases scientifiques

et techniques de l'AR comme bien établies, un net regain d'intérêt s'est manifesté durant les dernières décennies grâce en particulier au développement de méthodes d'investigations modernes, comme les études randomisées en double aveugle.

Le canal spinal s'étend du foramen magnum jusqu'au hiatus sacré, mais l'espace sous-arachnoïdien se termine au niveau de la 2ème vertèbre sacrée (fig. 1a et 1b). La moelle épinière se terminant en général au niveau du bord supérieur de L2, la ponction de l'espace sous-arachnoïdien doit s'effectuer en dessous de L2 afin d'éviter tout traumatisme potentiel de la moelle épinière. Avant de pénétrer dans l'espace, l'aiguille de ponction doit traverser les ligaments spinaux (supra épineux, inter épineux, ligament jaune) (fig. 2) puis la dure-mère et l'arachnoïde qui lui est étroitement accolée (fig. 3). L'espace sous-arachnoïdien s'étend entre l'arachnoïde et la pie-mère qui adhère la moelle épinière. Il contient la moelle épinière, les racines nerveuses et le LCR. La moelle épinière donne naissance à 31 paires de nerfs spinaux, chacun composé d'une racine antérieure motrice et d'une racine postérieure sensitive. Ces deux racines se joignent pour former des fibres nerveuses sensitives et motrices. Chaque segment spinal pourvoit une région spécifique de la peau (dermatome), de muscles (myotome) et d'os (ostéome). Lors de l'AR un bloc moteur affecte le mouvement de différents articulations et muscles, le blocage sensitif entraîne une anesthésie cutanée dont la distribution est décrite dans la figure 5. L'AR peut également entraîner un blocage étendu des nerfs sympathiques et parasympathiques dont l'effet va se manifester principalement sur le contrôle de l'hémodynamique (pression artérielle et fréquence cardiaque).

Le liquide céphalo-rachidien (LCR) est clair et transparent, il entoure le cerveau et la moelle épinière dans un compartiment liquide appelé l'espace sous arachnoïdien. Il exerce ainsi un rôle protecteur contre les traumatismes et tous mouvements brusques.

Les nocicepteurs sont des récepteurs spécifiques à la douleur, activés par des stimulations intenses pouvant entraîner des lésions tissulaires. Ce sont des terminaisons nerveuses libres situées dans la peau ainsi que dans d'autres tissus sensibles à la douleur. Différents stimuli activent différents types de récepteurs (stimuli thermiques, mécaniques, chimiques). On différencie alors les nocicepteurs à leur modalité. Différentes fibres sensitives transmettent les impulsions afférentes en réponse aux stimulations douloureuses vers la corne dorsale de la moelle épinière. A partir des cellules de la corne dorsale, le stimulus douloureux est transmis vers les centres supérieurs.

L'hypotension artérielle est une manifestation physiologique fréquente du bloc spinal. Le blocage des fibres nerveuses sympathiques préganglionaires en est la cause principale. Ceci entraîne une dilatation des vaisseaux de résistance et de capacitance. Les fibres sympathiques qui émergent de T1 à T5 contrôlent la fréquence cardiaque. Un bloc spinal qui implique ces fibres va causer une dénervation sympathique centrainant une diminution de la fréquence et de la

contraction cardiaque se manifestant par une diminution du débit cardiaque. De l'étendue du bloc spinal, et donc du bloc sympathique, va dépendre le degré de l'hypotension artérielle. Des drogues à action vasoactive et chronotrope positive comme l'Ephédrine et l'Atropine peuvent être utilisées prophylactiquement ou lorsque l'hypotension apparaît.

La conduction de l'impulsion le long de la fibre nerveuse est due au gradient électrique à travers la membrane de l'axone. Celle-ci résulte du mouvement des ions, en particulier le sodium et le potassium à travers la membrane. Un stimulus adéquat réduit le potentiel électrique de la membrane, engendrant une phase de dépolarisation rapide (potentiel d'action) par entrée de l'ion sodium dans le milieu intracellulaire à travers des canaux membranaires spécifiques. Le flux de potassium de l'intérieur vers l'extérieur de la cellule nerveuse permet la repolarisation de la membrane. Les anesthésiques locaux (AL) empêchent la dépolarisation de la fibre nerveuse par interaction avec un récepteur spécifique du canal à sodium, inhibant ainsi la conductance du sodium et donc la conduction nerveuse.

Les fibres nerveuses sont classées suivant leurs tailles et leur degré de myélinisation. In vitro, des études récentes suggèrent que les gosses fibres myélinisées sont plus sensibles aux AL que les petites fibres non myélinisées. La quantité d'AL utilisée pour l'AR est un surdosage par rapport aux concentrations requises pour bloquer les différents types de fibres Toutefois, le niveau céphalique du bloc sympathique est supérieur de 2-3 segments par rapport au bloc sensitif, lui-même plus haut que le niveau du bloc moteur. Ce blocage différentiel de la conduction nerveuse s'explique par la baisse de la concentration de l'AL à mesure que l'on s'éloigne en direction céphalique du point d'injection. Pour les fibres myélinisées, au minimum trois noeuds de Ranvier consécutifs doivent être bloqués pour empêcher la conduction. Les petites fibres B des nerfs sympathiques préganglionaires possèdent de courtes distances internodales et sont donc plus sensibles aux AL. Les plus grosses fibres nociceptives A Delta ont des distances internodales plus grandes et requièrent des concentrations d'AL plus grandes pour être bloquées. Enfin, les plus grandes fibres A-Alpha ont les plus grandes distances internodales et sont bloquées seulement lorsque la concentration en AL est suffisante pour bloquer trois noeuds successifs.

Nombreux sont les facteurs qui influencent l'étendue et la qualité du bloc spinal comme l'AL lui-même, le dosage, l'addition d'autres drogues (opiacés, vasoconstricteurs). L'un des facteurs le plus important est la baricité de la solution de l'AL.

Les solutions d'AL utilisées pour l'AR sont caractérisées selon leur baricité (hypobare, isobare et hyperbare). La baricité est définie comme le rapport entre la densité de la solution de l'AL à une température donnée et la densité du LCR à la même température. La densité est mesurée en gramme pour 1 mL de la solution étudiée, à une température donnée. Théoriquement selon cette définition, une

solution d'AL isobare a une baricité de 1, alors que les solutions hypobares et hyperbares ont une baricité respectivement inférieure et supérieure à 1. Les solutions d'AL utilisées pour la RA sont mélangées avec de l'eau pure, une solution salée isotonique ou des dextroses afin de les rendre respectivement hypo, iso ou hyperbares. Les propriétés physiques des solutions anisobares peuvent être utilisées avantageusement pour l'AR. Ainsi, une solution isobare tend à rester proche de la région du point d'injection tandis qu'une solution hypobare aura tendance à « flotter » vers le haut et une solution hyperbare à « couler » vers le bas (fig. 7). Il est donc théoriquement possible, en choisissant la baricité de la solution et la position du patient de diriger préférentiellement l'AL vers les nerfs qui doivent être anesthésiés. Ainsi, il doit être possible d'anesthésier préférentiellement l'hémicorps latéral supérieur avec une solution hypobare, injectée en position de décubitus latéral.

Dans notre institution, la mise en place d'une prothèse totale de la hanche en décubitus latéral est fréquemment effectuée sous AR en utilisant 15 à 17,5 mg d'une solution de bupivacaine-0.5 % isobare. L'utilisation d'une solution hypobare devrait en théorie produire une distribution sous arachnoïdienne de l'AL plus sélective vers le côté opéré, offrant ainsi un bloc moteur et sensitif plus long et plus profond, ce qui peut être un avantage certain en cas de prolongation inattendue du temps chirurgical.

La présente étude englobe 40 patients, âgés entre 40 et 75 ans, ASA I ou II, programmés pour bénéficier électivement de la mise en place d'une prothèse totale de la hanche en AR. Avec le coté opéré vers le haut, les patients ont été assignés au hasard et en double aveugle à recevoir une injection spinale de 3.5 mL de bupivacaine-0.5 % (17,5 mg) mélangée à 1.5 mL de solution saline 0.9 % (Groupe isobare) ou 1.5 mL d'eau distillée (Groupe Hypobare).

Le niveau maximal du bloc sensitif avec son temps d'installation et le degré maximal du bloc moteur avec son temps d'installation ont été évalués sur les côtés non dépendants (côtés opérés) et dépendants (côtés non opérés) jusqu'à régression du niveau sensitif à L2 et jusqu'à récupération complète du bloc moteur. Les changements hémodynamiques (pression artérielle et fréquence cardiaque) durant les 45 premières minutes après l'injection spinale et le temps entre l'injection spinale et le recours à une antalgie en raison de l'apparition d'une douleur supérieure à 3 (sur une échelle de 0 à 10) sur le site opératoire ont également été notés. La durée de l'anesthésie était définie comme le temps entre l'injection spinale et la fin de la chirurgie. La durée de l'analgésie chirurgicale était définie comme le temps entre l'injection spinale et le recours à une antalgie en raison de l'apparition d'une douleur de score supérieur à 3 sur une échelle visuelle analogique s'étendant de 0 à 10.

Les caractéristiques démographiques des deux groupes étaient comparables.

Le niveau maximal médian du bloc sensitif et le niveau maximal du bloc moteur étaient comparables entre les côtés opérés et non opérés dans chaque groupe et entre les côtés correspondants dans les deux groupes.

Comparé au groupe isobare, le temps entre l'injection spinale et la régression du bloc sensitif à L2 sur le côté opéré était plus long pour le groupe hypobare (287± 51 min vs 242± 36 min, $p<0.004$), ainsi que le temps entre l'injection spinale et le recours à une antalgie (290± 46 min vs 237± 39 min, $p<0.001$). Il n'y a pas eu de différence dans la qualité du bloc moteur à la fin de la chirurgie.

Les changements hémodynamiques observés durant les premières 45 min après l'injection spinale étaient comparables entre les deux groupes.

En conclusion, pour les patients devant bénéficier d'une prothèse totale de la hanche en position de décubitus latéral sous anesthésie rachidienne, 17.5 mg de bupivacaine hypobare comparée à une dose identique de bupivacaine isobare, prolonge la régression du bloc sensitif à L2 et retarde le recours à une antalgie, sans compromettre l'hémodynamique. Nous pensons qu'une augmentation de la durée du bloc sensitif de 45 min accroît la sécurité de l'AR pour ce type de procédure chirurgicale.

Total hip arthroplasty is frequently performed under spinal anesthesia using either isobaric or hypobaric anesthetic solution. However, these two solutions have never been compared under similar surgical conditions. The present study compares anesthetic and hemodynamic effects of isobaric and hypobaric bupivacaine in 40 ASA I-II patients undergoing total hip arthroplasty in the lateral decubitus position under spinal anesthesia. With operative side up, patients randomly received, in a double blind manner, a spinal injection of 3.5 mL (17.5 mg) of plain bupivacaine mixed with either 1.5 mL normal saline (Group Isobaric) or 1.5 mL distilled water (Group Hypobaric). Sensory level and degree of motor block were evaluated on non-dependent and dependent sides until regression to L2 and total motor recovery. Hemodynamic changes during the first 45 min after spinal injection, and the time between spinal administration and first analgesic for a pain score above 3 (on a 0 to 10 scale) were noted. Demographic characteristics of both groups were comparable. Upper sensory level and maximal degree of motor block were comparable between operative and nonoperative sides in each group and between corresponding sides in both groups. Compared to the isobaric, there was in the hypobaric group a prolonged time to sensory regression to L2 on the operative side (287 ± 51 min vs 242 ± 36 min, $p<0.004$) and a prolonged time to first analgesic (290 ± 46 min vs 237 ± 39 min, $p<0.001$). No difference in quality of motor block was noted at the end of surgery. Hemodynamic changes were comparable. We conclude that for total hip arthroplasty in the lateral position, spinal hypobaric bupivacaine appears to be superior to isobaric in that it prolongs

the sensory block on the operative side and delays the use of analgesics after surgery without further compromising hemodynamic.

Introduction

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1) Definition

Spinal anesthesia (SA) is the injection of a local anesthetic (LA) into the subarachnoid (or intrathecal) space, mixing with the cerebralspinal fluid (CSF) and creating a conduction blockade of the spinal nerves. The resultant nerve block provides surgical anesthesia as far cephalad as the upper abdomen.

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2) Historical

Following Koller's description of the topical anesthetic properties of cocaine in 1884, James Corning, a surgeon in New York City, injected cocaine into the region of the spinal cord of a dog, which resulted in transient hind limb paralysis. In 1899, Augustus Bier, a German surgeon, was the first individual to administer cocaine intrathecally for surgical anesthesia.

Following these initial reports, SA rapidly gained clinical acceptance in Europe and in the USA. It continues to be the most commonly used regional anesthesia in the USA and it is widely used in the Third World because it is simple, effective and cheap.

However, in the 1950s, its use almost stopped completely in the UK following the "Wooley and Roe" case in which two patients on the same operating list were permanently paralysed after spinal injection. It is now certain that these tragedies were the result of the antiquated antiseptic measures employed at the time.

In the last decade, there has been a considerable revival in the interest in, and use of SA. While many regarded the scientific basis of the technique to have been well established in the first half of the century, modern methods of investigation, especially the double-blind randomised trial, have shown that many of the earlier concepts were flawed (1-4)

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3) Anatomical considerations

3.1) Vertebral column

The spinal canal runs from the foramen magnum to the sacral hiatus, but the subarachnoid space usually ends at the level of the second sacral vertebra. The spinal cord usually ends at the upper border of the second lumbar vertebra though this can vary between T12 and L3 (figure 1a,b). So it is safest to enter the subarachnoid space below L2.

3.2) Spinal ligaments

Adjacent vertebrae are held together posteriorly by short tough ligaments. To insert a needle into the spinal canal between to vertebrae in the midline, it must traverse the supraspinous and interspinous ligaments before reaching the ligamentum flavum, the last barrier to the canal itself (figure 2a,b).

3.3) Spinal meninges

Within the spinal canal there are three sleeves of connective tissues, the dura mater, the arachnoid mater and the pia mater, which cover and protect the spinal cord. They form three spaces, the epidural space, the subdural space and the subarachnoid space (figure 3). The epidural space is that part of the spinal canal between its outer wall and the dura mater. On the inner side of the dura mater is the arachnoid matter, which is closely applied to the dura. The potential space between the dura and arachnoid mater is the subdural space. The subarachnoid space lies between the arachnoid and the pie mater which is adherent to the spinal cord. It contains the spinal cord, the nerve roots and the cerebrospinal fluid (CSF).

3.4) The spinal cord

The spinal cord is continuous with the medulla oblongata of the brain and runs from the foramen magnum to the upper border of the second lumber vertebra. (It extends to L3 in 10% of adults). The spinal cord gives rise to 31 pairs of spinal nerves each composed of an anterior motor root and a posterior sensory root (8 cervical, 12 thoracic, 5 lumbar, 5 sacral and 1 coccygeal). These two roots join to form both sensory and motor nerve fibres (figure 4). Because the spinal cord is shorter than the spine, the lower nerve roots become longer and more angulated caudally. The lower lumbar, the sacral and the coccygeal roots form, together with filum terminal, the cauda equina below the termination of the cord. It is in this region were spinal block are performed as the needle can not damage the spinal cord as the rootlets forming the cauda equina can move easily in the CSF.

3.5) Segmental distribution of the spinal nerves

Each spinal segment supplies a specific region of the skin (dermatome), of muscle (myotome) and of the bone (osteome). In spinal anesthesia, motor block affects the movement of various joints and muscles. The cutaneous distribution of the spinal nerves is shown in figure 5.

3.6) Autonomic nervous system

Spinal anesthesia can cause a widespread blockade of both sympathetic and parasympathetic nerves. This has considerable effect, mainly on the control of the circulation (arterial blood pressure and heart rate).

3.6 1) Sympathetic nervous system

Efferent impulses from the central nervous system to the blood vessels and organs supplied by sympathetic nerves must travel along both pre and postganglionic nerves. The ganglia are located in the sympathetic chain, and in the large plexuses within the thorax and the abdomen. Preganglionic nerves fibres arise from the lateral column of the grey mater of the spinal cord and live it through the ventral nerve roots from T1 to L2. The preganglionic nerve fibres, which are lightly myelinated, live the spinal nerve to become the white rami communicantes which run to the sympathetic chain. The sympathetic chains run the length of the spinal column on the anterolateral aspect of the vertebral bodies. The unmyelinated postganglionic fibres arising from ganglia are widely distributed to all the organs receiving a sympathetic nerve supply. Many run back to the spinal nerves via the grey rami communicante and reach structures within the distribution of those spinal nerves.

3.6 2) Parasympathetic system

Efferent and afferent nerves of the parasympathetic system run either in the cranial nerves (cranial outflow) or in the 2^{nd}, 3^{rd} and 4^{th} sacral nerves (the sacral outflow). Unlike all other autonomic nerves, the cranial parasympathetic nerves cannot be affected by spinal blockade unless the LA diffuses into the cranium.

3.7) Cerebral spinal fluid (CSF)

CSF is a clear colourless fluid. Both the brain and the spinal cord are enclosed in a fluid compartment, namely the subarachnoid space. This serves as protection against trauma due to sudden movement. It also supports the brain and cord and maintains a uniform pressure upon them. The major portion of the CSF is produced from blood in the choroid plexuses in the four ventricules of the brain and is absorbed back into the blood by the arachnoid granulations found in the superior sagittal and transverse venous sinus. The two lateral ventricules communicate with the third one through the interventricular foramina. Connecting the third and fourth ventricule is the cerebral aqueduct of Sylvius. From the fourth ventricule, CSF reaches the cranial subarachnoid space and rises over the surface of the brain (figure 6). Apart from the physical protection provided to the brain, CSF provides a chemically stable environment. It probably takes part in nourishing the brain, assists in removing the product of neuronal metabolism and is important in the distribution and elimination of drugs injected into the CSF. Recently, Schiffer et al. (5) reported the density of the human CSF of 1.000529 ± 0.000107 at 37°C (5). Interestingly, they found a significant difference between women and men in CSF density, protein and glucose contents. The impact of mass represented by higher levels of glucose and proteins in men's CSF could explain the higher values of CSF density measured in men. In addition, they found a significant correlation between CSF density and upper sensory block level after injection of 15 mg spinal plain bupivacaine (considered slightly hypobaric in most patients but in clinical practice as isobaric (6)) in patients turned supine and kept in horizontal position all along the surgical procedure. Though the exact mechanism for this relation remains to be elucidated, it could allows a better understanding of the unpredictability of the extend of plain bupivacaine spinal anesthesia.

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4) Physiological considerations of spinal anesthesia

4.1) Nociceptors and peripheral pain mechanisms

Nociceptors are specific pain receptors activated only by intense, tissue-damaging activity. They are free nerve endings located in skin and other pain-sensitive tissues. Mechanical stimulation activates*mechanical nociceptors*. *Mechanothermal nociceptors* respond to noxious mechanical or thermal stimulation. *Polymodal nociceptors* respond to mechanical, thermal or chemical stimuli. Fast A-delta or slow C-fibres conduct afferent impulses in response to painful stimuli to the dorsal horn of the spinal cord, via dorsal spinal nerve roots. From the cell of the dorsal horn, the painful stimulus is transmitted to higher centres via several tracts of which description is beyond the scope of this introduction.

4.2) Cardiovascular effects of spinal anesthesia

Arterial hypotension is a common physiological manifestation of a spinal block. The primary cause of arterial hypotension during SA is the blockade of preganglionic sympathetic nerve fibres. This produces dilatation of resistance and capacitance vessels. Sympathetic fibres from T1-T5 control cardiac rate. SA that blocks these fibres causes cardiac sympathetic denervation that causes a moderate decrease in heart rate as well as a decrease in cardiac contractility with the consequence of a diminution of the cardiac output. The degree of hypotension relates to the spread of the local anesthetic within the subarachnoid space and the extend of the sympathetic blockade. A low block limited to the lumbar or sacral dermatomes causes little or no change in blood pressure. In opposite, high spinal anesthesia results in sympathetic blockade of fibres innervating the heart as well as those controlling peripheral vascular beds and may produce profound hypotension. Both the incidence and the degree of hypotension are reduced by limiting the height of the block and by keeping it below the sympathetic supply to the heart. Placement of the patient in a slight head-down position (5-10°) also limits the hypotension by improvement of the venous return. Vasoconstrictors drugs to increase cardiac output and peripheral resistance can be used prophylactically or when hypotension occurs. Ephedrine is the drug of choice.

5) Pharmacological considerations

5.1) Mechanism of action of local anesthetics

The conduction of impulses along nerve fibre is due to changes in the electrical gradient across the nerve membrane. This result from movement of ions, particularly sodium and potassium, across the membrane. An adequate stimulus that reduces the membrane potential produces a spontaneous rapid phase of depolarization (action potential). Depolarization is due to the inward passage of sodium from the extracellular to the intracellular space via specific channels in the membrane. The flow of potassium from the interior to the exterior of the nervous fibre causes repolarization. Local anesthetic (LA) agents prevent depolarization of the nerve membrane by blocking the flow of sodium ions. LA, such as lidocaine apparently penetrate the lipoprotein membrane matrix to reach the axoplasm and then enter the sodium channel where they interact with a specific receptor to block it and inhibit the sodium conductance resulting in the blockade of the conduction. The anesthetic profile of a local anesthetic is related to the lipid solubility, the

protein binding, the pKa and the intrinsic vasodilatator activity. From this parameters depend the potency of LA, the duration and the onset of action.

5.2) Mechanism of spinal anesthesia

Nerve fibres are classified on the basis if size and degree of myelination, which in turn determine the conduction velocity. In vitro recent studies suggest that large myelinated fibres are more sensitive to LA blockade than smaller unmyelinated fibres. LA injected intrathecally bind to a greater extent in spinal nerve roots and the periphery of the spinal cord, where the small-diameter fibres are located. Less drug diffuses into the dorsal root ganglion and centre of the spinal cord. As a result, sensory anesthesia occurs more rapidly than motor blockade. Ultimately sufficient drug reaches the large myelinated fibres to causes motor block.

5.3) Differential sensory/motor blockade

The amount of LA used for spinal anesthesia is an overdosage in relation to the minimum concentration required to block the various nerve fibres types. This is the reason for the relatively rapid blockade of sensory, sympathetic and motor blockade during clinical spinal anesthesia. In general, the cephalad level of sympathetic blockade is 2-3 segments higher than the level of sensory blockade, which, in turn is higher than the level of motor blockade. This differential level of nerve blockade may be related to several factors: the anesthetic concentration within the CSF declines as the cephalad distance from the site of injection increases; in myelinated nerve fibres at least three consecutive nodes of Ranvier must be completely blocked to prevent conduction; decremental conduction block occurs when greater than three nodes are exposed to subminimal blocking concentration of LA. The small B fibres in preganglionic sympathetic nerves possess short internodal distances and are most susceptible to conduction block, especially at upper spinal levels where CSF concentration of LA are low. The larger A-delta nociceptor fibres have longer internodal distances and require a higher LA concentration for blockade. The larger A-alpha fibres have the greatest internodal distances and are blocked only when the LA concentration is sufficient to inhibit three successive nodes.

5.4) Factors influencing spinal anesthetic activity

The factors that influence the spread and quality of SA include: the LA drug, the dosage of LA, the addition of a vasoconstrictor to the solution, the baricity of the solution, the addition of opioids to LA and pregnancy. We will concentrate on the baricity of the AL solution.

5.4.1) The baricity of the anesthetic solution

LA used for SA are characterised according to their baricity (hypobaric, isobaric, or hyperbaric). Baricity is the ratio of the density of a LA solution at a specified temperature to the density of cerebral spinal fluid at the same temperature. Density is the weight in grams of 1 mL of a solution at a specified temperature. Specific gravity is the ratio of the density of a solution at a specified temperature to the density of water at the same temperature.

Theoretically and according to these definitions, an isobaric LA solution has a baricity of 1 while hypobaric and hyperbaric solutions have baricities less than or greater than unity, respectively. Clinically, hypobaric intrathecal solutions have been defined as those with a density less than tree standard deviations below mean human CSF density (7). Green stated that the density of a LA solution must be far enough below the mean density of CSF (1.0003) to take into account the small but important normal variation in density of CSF about the figure of 1.0003 if the solution is to be hypobaric in all patients, not just in some patients. LA solutions with baricities less than 0.9990 are predictably hypobaric in all patients (6). Using more accurate and precise techniques in the measurements of the density of human CSF, Richardson and Wissler (8) determined values for the density of the CSF with ranges much narrower than previously reported, defining limit of hypobaricity of intrathecal solutions greater than previously proposed (between 1.00016 and 1.00037 g/mL according to the variations of CSF density in different subgroup of patients). It is important to note that density varies inversely with temperature: therefore, a LA which has the same density as CSF at 37°C is more dense (hyperbaric) at room temperature (the temperature at which LA are usually injected) than CSF at 37°C. However, the clinically important density of a LA solution is that which is measured at 37°C because, during SA, LA solutions rapidly equilibrate with the temperature of CSF.

LA solutions used for SA are formulated with water, normal saline or dextrose to make them hypobaric, isobaric or hyperbaric respectively.

The significance of baricity lies in the fact that isobaric solutions tend to remain in the area where they are injected into the CSF, while hypobaric solutions "float" upwards and hyperbaric solutions "sink" (figure 7). Therefore, it is possible, by selecting the appropriate baricity and patient position to "direct" the LA to nerves that need to be anesthetized. For example, it is possible to anesthetize the sacral nerves by injecting a hyperbaric solution with the patient in the upright position or a hypobaric solution with the patient in the prone jack-knife position (figure 8). Similarly it should be possible to anesthetize preferentially the hemilateral upper part of the body with a hypobaric solution injected in lateral decubitus position (1-4).

6) Clinical considerations

6.1) Clinical rational for spinal anesthesia

Regional anesthesia such as SA has been shown to of benefit in several areas:

The metabolic stress response to surgery and anesthesia is much more effectively reduced by SA than by general anesthesia (9, 10). Many studies (especially in elective hip surgery) have shown a reduction in blood loss of 20-30% in-patients receiving spinal anesthesia versus general anesthesia (11). Several studies demonstrated that regional techniques decrease the incidence of venous thromboembolic complications by as much as 50%, especially in lower extremity procedures (12). Data regarding pulmonary complication are mixed; however, pulmonary compromise appears to be less in peripheral procedure performed under regional anesthesia. Other area of benefit include avoidance of endotracheal intubation in patients with a difficult airway or reactive airway disease and decreased risk of gastric aspiration.

6.2) Spinal anesthesia for surgical operations

Spinal anesthesia produces intense blockade of those spinal nerves, and the spinal cord, which are exposed to the injected LA. Somatic pain and motor function are abolished in the blocked segments. Visceral pain is also prevented if the appropriate afferent nerves are blocked. SA is more suited to surgery below the ombilicus and in this situation the patient may remain awake. Surgery above the ombilicus using SA is less appropriate and would necessitate a general anesthetic in addition, in order to abolish unpleasant sensations from visceral manipulation resulting from afferent impulses transmitted by the vagus nerves.

6.3 Contraindications

Contraindications include hypovolaemic shock, septiceamia or bacteraemia, pre-existing neurological disease, increased intacranial pressure and heamorrhagic diathesis (1-4).

7) Background and aim of the study

In our institution total hip arthroplasty (THA) in the lateral position is frequently performed using single-shot spinal anesthesia with 15 to 17,5 mg of plain bupivacaine 0.5% which provides a surgical anesthesia for 3-4 hours. In this particular position, with the non-dependent (or operative) side up during the spinal injection and all along the surgery, hypobaric bupivacaine by "floating" upwards to the hemilateral upper part of the body could in theory provide a more selective subarachnoid distribution of local anesthetic on the operative side. This can result in a more profound sensory and motor block of longer duration in favor of the operative side which could be advantageous in the case of an unexpectedly prolonged surgery, since the induction of general anesthesia in the lateral position is not only uneasy to perform but presents an increased risk for the patient. Besides, a more selective or unilateral block on the operative side should diminished the extend of the blockade of the preganglionic sympathetic fibres and then diminished the degree of systemic arterial hypotension often observed after spinal anesthesia. Consequently, the use of hypobaric bupivacaine should theoretically increase the reliability of spinal anesthesia for this type of surgical procedure.

The use of hypobaric local anesthetic has already been reported for single-shot injection (13, 14) and continuous spinal anesthesia (15, 16). However, possible advantages of hypobaric over isobaric solutions have not been tested in these specific surgical conditions. The aim of the present study is to compare the anesthetic and hemodynamic effects of isobaric (plain bupivacaine mixed with normal saline) and hypobaric bupivacaine (plain bupivacaine mixed with distilled water) solutions for THA performed with patients in the lateral decubitus position.

Patients and Method

After approval from the Ethics Committee of our institution and written informed consent, 40 orthopedic patients, between 40 and 75 years old, ASA physical status I or II, scheduled to undergo elective THA under single-shot spinal anesthesia were enrolled in the study. Exclusion criteria were coagulation disorders, local infection and obvious spinal postural abnormalities (cyphosis) as well as inability to comprehend basic aspect of the study.

Preoperative medication consisted of morphine 0.1 mg/kg SC administered one hour before arrival in the operating room, in order to ameliorate discomfort of dependent shoulder during prolonged lateral decubitus position. Standard noninvasive monitoring consisted in a continuous electrocardiogram, peripheral pulse oxymetery, and automatic noninvasive blood pressure measurement on the non-dependent arm. More invasive monitoring (i.e. central venous pressure invasive arterial pressure or an indwelling urinary catheter) was used only if required by the patient's clinical conditions. After placement of a peripheral intavenous (IV) catheter on dependent forearm, preanesthetic hydration consisted

of 10 ml/kg of a crystalloid solution. During the first hour after spinal injection 5 ml/kg of the same solution were further infused. Thereafter fluids were given on the basis of changes in arterial pressure and estimated blood loss, replaced with a crystalloid solution on a 3:1 mL basis. When available, autologous blood was given if hematocrit dropped under 30% and homologous blood was only administered if hematocrit dropped under 26%.

Using a sealed envelope system the patients were randomly assigned to receive either hypobaric or isobaric bupivacaine solutions, which were prepared as follow :

Isobaric bupivacaine : 3.5 mL (17.5 mg) of plain bupivacaine 0.5% (5 mL vial Carbostesine® 0.5%, Astra Zeneca, Grafenau, Zug, Switzerland) diluted with normal saline to a total of 5 ml; measured density at 37 °C was 0.999406 g/mL.

Hypobaric bupivacaine : 3.5 mL (17.5 mg) of plain bupivacaine 0.5% diluted with distilled water, to a total of 5 mL; measured density at 37 °C was 0.997302 g/mL.

According to the randomization an anesthesiologist not participating in patient's care or data collection prepared 10 mL of distilled water or normal saline. The anesthesiologist in charge of the patient withdrew 3.5 mL of plain bupivacaine 0.5% from a 5 mL vial and added 1.5 mL of the prepared. The density measurements of study solutions were performed using an Anton Paar densitometer (DMA 4500, Anton Paar GmbH, Graz, Austria). For each solution three measurements were performed and mean value considered.

With the operating table horizontal, the patients were placed in the lateral decubitus position with the operated hip up. Lumbar puncture was performed at the L2-L3 interspace with a 25-Gauge Whitacre needle. After observing a free cerebrospinal fluid (CSF) reflux, the needle aperture was oriented upwards and 5 mL of the study solution injected at the rate of approximately 0.5 mL/s. The patients remained in the lateral position until the end of surgery, at which time they were turned supine.

The following variables were measured throughout the study :

Evolution of upper sensory block level on non-dependent (operated) and dependent sides. A pinprink test (24 gauge needle) was performed on the midthoracic line every 5 min during the first 45 min after spinal injection, and then every 15 min until sensory regression to L2 (level of the surgical incision). Maximal upper sensory block level, its onset time and time to regression to L2 on both sides were recorded.

Evolution of degree of motor block using a modified Bromage scale (17) ranging from 0 to 4 (0 = able to move hip, knee, ankle and toes; 1 = unable to move

hip, able to move knee, ankle and toes; 2 = unable to move hip and knee, able to move ankle and toes; 3 = unable to move hip, knee, ankle, able to move toes; 4 = unable to move hip, knee, ankle and toes) on both limbs, every 5 min during the first 45 min after spinal injection. In order not to interfere with the surgical procedures, the degree of motor block was not determined during the operation. At the end of surgery, degree of motor block was determined again for both limbs and tested every 15 min until total motor recovery. Maximal degree of motor block, its onset time, and time to total motor recovery of both limbs were recorded.

Mean arterial pressure (MAP) and heart rate (HR) were recorded every 2.5 min during the first 45 min after spinal injection, every 5 min during surgery and every 15 min in the recovery room until the end of the study (defined as sensory regression to L2 on both sides and/or total motor recovery of both limbs). Maximal decrease in MAP and HR from baseline value (determined with patients in the lateral decubitus position just before spinal injection) were recorded for the first 45 min after spinal injection. Ephedrine 5-10 mg IV was given if MAP decreased by more than 20% from baseline value or if systolic arterial pressure dropped below 90 mmHg. Atropine 0.5 mg IV was given if HR decreased less than 45 beats/min.

Duration of anesthesia was defined as time between spinal injection and the end of the surgery.

Duration of surgical analgesia was defined as the time between spinal injection and the first analgesic requirement for a pain score at the operated site above 3 on visual analog scale ranging from 0 to 10.

All variables above were determined during anesthesia by the anesthesiologist in charge of the patient, and in the recovery room by nurses in charge who were trained to report accurately these variables. Discomfort related to the lateral position during surgery was treated with fentanyl 1mcg/kg IV (maximum 2 doses) and anxiety with midazolam 1 mg IV.

Statistical analysis

Prospective power tests defined the sample size using sensory block level regression time to L2 of 157 ± 37 min using 3 mL of plain bupivacaine 0.5%. (18). The sample size was computed to detect a 25% difference in favour of the hypobaric group i.e., a longer duration of block with a power of 80% and a two-tailed significance level of 5% (β = 0.2; α = 0.05). A minimal sample of 14 patients for each group met these criteria. This study enrolled 20 patients per group. Results are expressed as mean ± SD or median (ranges) for discrete variables. Comparisons between groups or between both sides in the same group were performed using the Student *t* test for unpaired or paired data, the Mann-Whitney U test and the chi-square test as required. A P value < 0.05 was considered statistically significant.

Results

Twenty patients were allocated to each group. One isobaric spinal anesthesia failed. This patient was not considered for further analysis. Patient's demographic and preanesthetic hemodynamic data were comparable between the two study groups (table 1).

Median upper sensory levels of both non-dependent (operated) and dependent sides in both groups along with onset times are illustrated in figure 9. There was no difference between corresponding sides in the two groups or between operated and non-operated sides in the same group.

Maximal degree of motor block achieved and onset times are presented in table 2 and are also comparable between the two groups and in the same group between both sides.

Duration of anesthesia defined as the time between the spinal injection and the end of the surgery was comparable between the two groups, 170 ±± 25 min for isobaric and 168 ±± 23 min for hypobaric.

Sensory regression times to L2 are presented in table 3. When comparing non-dependent and dependent sides, patients in both groups show significantly prolonged sensory regression to L2 on non-dependent (or operated) side. When comparing both groups (figure 10), regression to L2 on the non-dependent side was significantly prolonged in the hypobaric group (287 ± 51 min vs 242 ± 36 min, $p < 0.004$); likewise, time to first analgesic requirement was significantly longer in the hypobaric group (290 ± 46 min vs 237 ± 39 min, $p < 0.001$).

Since the degree of motor block was not tested during surgery and since at the end of surgery complete motor recovery of one or both limbs were observed in some patients, only relevant data are available after surgery was completed in all patients, i.e. 225 min after spinal injection. At this time in the hypobaric group, 5 patients had a complete motor recovery on the non-dependent side compared to 15 on the dependent side ($p< 0.0001$). In the isobaric group, the data (8 vs 11) were not statistically different between the two sides (table 3). In addition, contrary to the sensory block (figure 10), there were not statistical difference when corresponding sides between the two study groups were compared.

Hemodynamic changes, observed during the first 45 min after spinal injection, were comparable between the two groups. Maximal decrease MAP was 32 ± 13% vs 31 ± 16% and for HR 14 ± 11% vs 14 ± 10% for isobaric and hypobaric group respectively (NS) (table 4). Ten patients in the isobaric and nine in the hypobaric group received ephedrine; one patient in the isobaric group received atropine.

Finally, two patients in the isobaric group received fentanyl for discomfort and 4 received midazolam for anxiety. In the hypobaric group three patients received fentanyl and 5 midazolam.

Discussion

The results of the present study demonstrate an advantage of hypobaric over isobaric spinal anesthesia in patients undergoing THA in the lateral decubitus position. Although both solutions provide satisfactory analgesia overcoming the main surgical duration, the benefit of hypobaric bupivacaine is evidenced by a significantly delayed sensory regression to L2 on the non-dependent side, thus postponing the need for first analgesic.

Sensory regression to L2 on the non-dependent side lasted 45 min longer in the hypobaric group compared to the isobaric group and we believe that this difference is clinically relevant, particularly in the context of single-shot spinal anesthesia performed in patients undergoing surgery in the lateral position. Because surgical time can sometimes be longer than expected and exceed anesthesia time, heavy sedation or even induction of general anesthesia can be required, which is often hazardous in the lateral position.

Although described as a possible anesthetic technique (6) there are only a few studies reporting the use of hypobaric spinal anesthesia (13-16, 19). Before the present study two other reports compared hypobaric and isobaric bupivacaine. Van Gessel et al.(20) reported during continuous spinal anesthesia for hip surgery fewer failures with isobaric vs hypobaric bupivacaine. However both injection of local anesthetic and surgery were performed with patients . Kuusniemi et al. (21) tested small doses (6 mg) of hypobaric and plain bupivacaine in the lateral position for knee arthroscopy; twenty min after spinal injection the patients were turned supine for surgery. A differential spread for both sensory levels and motor block between non-dependent and dependent sides was demonstrated for each solutions. However no difference was found between the two solutions, when comparing same sides.

To our knowledge, the present study is the first comparing isobaric and hypobaric bupivacaine in patients undergoing surgery in the lateral position. During progression of spinal anesthesia both solutions demonstrate qualities of isobaricity (no difference in upper sensory level and maximal degree of motor block between non-dependent and dependent sides.) The results of other studies investigating subarachnoid distribution of hypobaric local anesthetic in lateral position suggest a dose-related-effect in favor of non-dependent side. As previously said, Kuusniemi et al. (21) reported a differential sensory and motor blocks with hypobaric solution (6 mg bupivacaine in 3.4 mL). Van Gessel et al. (16) observed a differential spread only for motor block when using hypobaric solutions of tetracaine or bupivacaine

(7.5 mg in 3 mL). Atchison et al. (13) studying the effects of speed of injection documented a differential sensory spread between non-dependent and dependent sides with 10 mg of hypobaric tetracaine in 5 mL when injected over 250 sec with an electrically driven syringe pump and through a Whitacre needle with the aperture oriented upward. When the same solution is injected rapidly over 10 sec, which approximates usual clinical spinal injection speeds, no differential sensory block was found. Using an identical methodology Horlocker et al.(14) on the other hand did not show any difference in sensory levels between non-dependent and dependent sides in either slow or fast injection groups, using 15 mg of hypobaric bupivacaine in 5 ml. Consequently the appearance of a differential block seems to be favored by using low dose of hypobaric solution injected in a very slowly.

In our study, the absence of early clinical signs of preferential distribution in favor of the non-dependent side in the hypobaric group can be explained essentially by the following mechanism. Unlike to hyperbaric solutions (22), there is a relatively small difference in density between the hypobaric bupivacaine solution used in the present study (0.997302 g/mL) and CSF using measurements previously made (5) in our institution (1.000529 ± 0.000107 g/mL). This slight difference associated with an elevated dose (17.5 mg) and volume (5ml) of hypobaric bupivacaine, moreover injected rapidly over 10 sec favorised an initial bilateral subarachnoid distribution. Thus, despite injecting the anesthetic solution through a directional Whitacre needle, there was no early evidence of a preferential spread.

However, qualities of hypobaricity evidently appear during regression of spinal anesthesia in both groups, but is clinically more relevant in the hypobaric group:

The regression time to L2 between non-dependent and dependent sides is significantly different in the two groups;

Unlike to the isobaric group, significantly less patients receiving hypobaric bupivacaine show a complete motor block recovery on the non-dependent compared to the dependent side (table 3).

The appearance of this delayed asymmetrical block can be attributed to the differences in densities between anesthetic solutions and CSF, associated with prolonged lateral position of about 3 hours in the present study. It has also been shown that part of a local anesthetic injected intrathecally remains free in CSF at least 60 min, since one hour after administration, variations in upper sensory level were found when changing patient position (23, 24). We speculate that in the present study 3 hours of lateral decubitus allows more neural fixation on the non-dependent roots of hypobaric than isobaric bupivacaine. These arguments could explain the more pronounced differential spread of hypobaric over isobaric bupivacaine observed during regression of the spinal anesthesia.

In the present study, some degree of differential block is documented for isobaric bupivacaine (table 3). Similar findings are reported by others for patients receiving plain bupivacaine and tested in the lateral position (21, 25), questioning if plain bupivacaine is isobaric or hypobaric (25). Greene (6) stated that the limit between hypobaric and isobaric local anesthetic solutions is a baricity of 0.9990 which is calculated by dividing density of local anesthetic with that of CSF. In 1954, Davis and King (7) stated that this limit is a density of local anesthetic lower than 3 standard deviations below mean human CSF density. Using more precise techniques of measurement of CSF density, Richardson and Wisler (8) determined upper limits of hypobaricity as density of local anesthetic between 1.00016 to 1.00037 g/mL according to the variations of CSF density in different subgroup of patients and considers mixture plain bupivacaine-morphine with density of 0.99941 as hypobaric (26). Recently we reported the density of plain bupivacaine 0.5% of 0.999343 ± 0.000004 g/mL at 37°C (20). Compared to this value, the density of hypobaric bupivacaine investigated in the present study was lower (0.997302 g/ml) and that of the isobaric solution higher (0.999406 g/mL). Their baricities calculated with density of CSF of 1.000529 g/mL (5) were 0.996774 and 0.998876 for hypobaric and isobaric solutions respectively. Thus, hypobaric bupivacaine appears to be hypobaric according to the definition of Greene, Davis and Richardson, whereas isobaric bupivacaine is at the limit between hypobaric and isobaric for Greene but remains hypobaric for Davis and Richardson. It should be noted that these different limits of baricity are given arbitrarily and that, besides baricity, there are more than 20 demonstrated or theoretical factors influencing subarachnoid distribution of local anesthetics (6). Nevertheless, it is admitted that plain bupivacaine 0.5% administered at usual volume of 3 mL in patients placed supine after injection behave clinically as isobaric (6). However, the result of the present study suggest that local anesthetic solutions considered as isobaric, with a density even higher than that of plain bupivacaine but lower than that of the CSF, can show some signs of hypobaricity in patients kept in prolonged lateral position.

Hemodynamic changes were comparable between the two studied groups after the fist 45 min initial 3.5 mL spinal injection (table 4). Maximal fall in mean arterial pressure of about 30 % can be attributed to the high dose of bupivacaine (17.5 mg) used. This dose was given to guarantee success of spinal anesthesia but may be associated with a greater degree of hemodynamic derangement in both groups. The absence of an early difference in maximal MAP decrease between the two groups can be the reflect of the absence of early clinical signs of preferential distribution of LA in favor of the non-dependent side in the hypobaric group as discussed previously. Nevertheless, as we discussed before, clinical signs of preferential block appear during regression of spinal anesthesia in both groups, but are clinically more relevant in the hypobaric group. We can speculate that this preferential block could have been correlated with a better hemodynamic in favor of the hypobaric group. However this hypothesis is difficult to demonstrate since after 45 min of spinal injection and particularly during the surgical procedure,

many other factors such as surgical psychological stress, blood loss, could make the interpretation of systemic hemodynamic data less accurate.

Concerning the possible influence of the site of injection, we assumed in our study that the spinal puncture was performed at the L2-L3 level. It has been shown that the clinical determination of spinal level puncture was wrong in 50% of cases (27). Accordingly, we can speculate that in the current study that some spinal bupivacaine injection were performed at one higher or one lower level. We believe that this fact cannot influence our results since the same error would have been repeated in both groups and it seems difficult to imagine that a difference by one or two segments in site of lumbar puncture can be responsible for the wide variation in upper spread of spinal block observed (8 segments for the non-dependent side of both groups and 14 and 12 segments for the dependent side of isobaric and hypobaric group respectively (fig. 9).

Conclusion

For patients undergoing THA in lateral position under spinal anesthesia, 17.5 mg of hypobaric bupivacaine, compared to identical dose of isobaric bupivacaine, prolongs sensory regression to L2 and delays the use of first analgesic, without further compromising systemic hemodynamics. We believe that 45 min longer duration of spinal anesthesia is clinically relevant and increases the reliability of hypobaric spinal anesthesia for this type of surgical procedure.

Further investigations could be performed by adding for example adrenaline in the hypobaric solution. This could theoretically increase the duration of the block. Thus it could be possible to diminish the dose local anesthetic to obtain an early more selective block and to insure a better hemodynamic.

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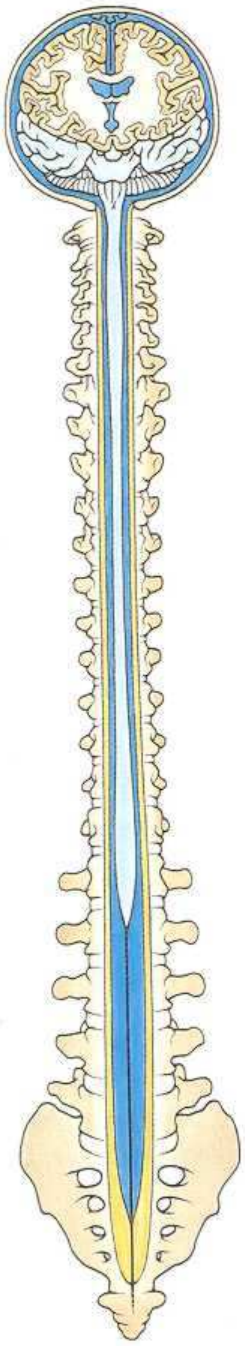
Figures



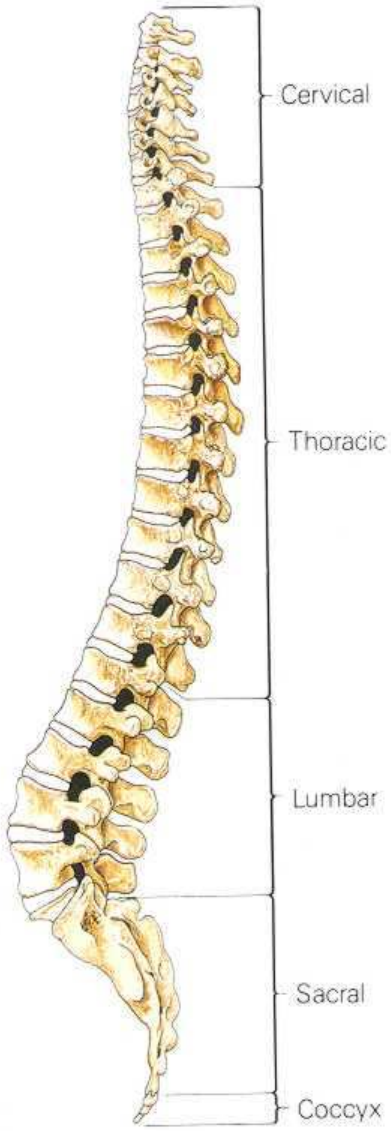

Cervical
Thoracic
Lumbar
Sacral
Coccyx

Fig. 1.6. Cross-section of a lumbar vertebra showing the attachment of the spinal ligaments.

1. Supraspinous ligament
2. Interspinous ligament
3. Ligamentum flavum
4. Posterior longitudinal ligament
5. Anterior longitudinal ligament

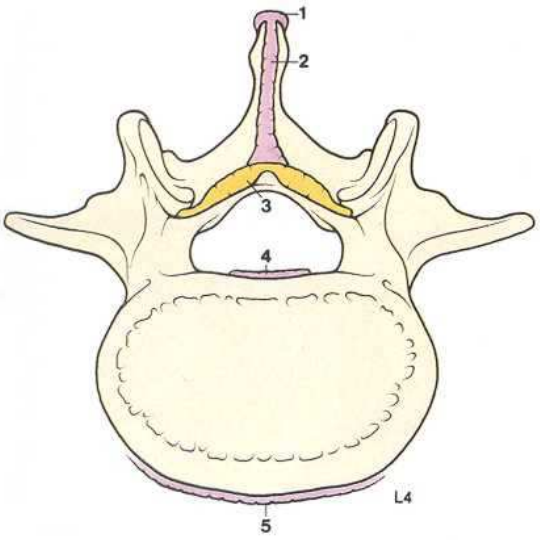


Fig. 1.7. Sagittal section through the second and third lumbar vertebrae showing the ligaments attached to adjacent laminae and spinous processes.

1. Supraspinous ligament
2. Interspinous ligament
3. Ligamentum flavum

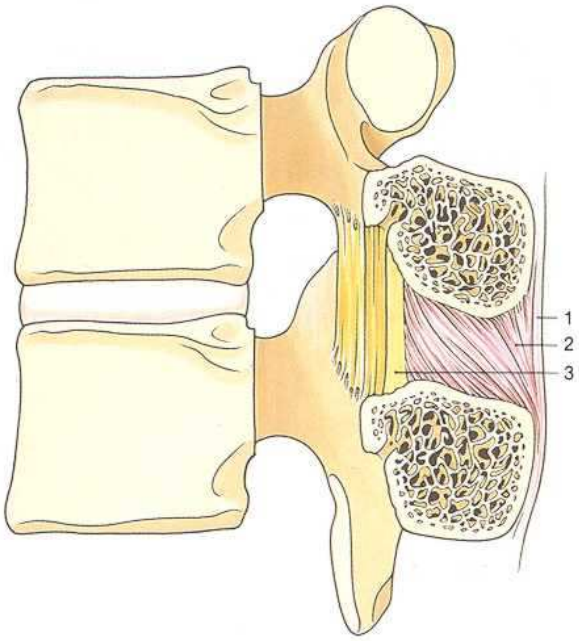


Fig. 1.9. Cross-section of the spinal canal in the thoracic region showing the spinal ligaments and the contents of the dural sac.

1. Posterior longitudinal ligament
2. Periosteum
3. Nerve root
4. Subarachnoid space
5. Epidural space
6. Pia mater
7. Arachnoid mater
8. Subdural space
9. Subarachnoid septum
10. Dura mater (inner layer)
11. Dura mater (outer layer)
12. Ligamentum flavum
13. Ligamentum denticulatum
14. Dorsal nerve root
15. Ventral nerve root
16. Dorsal root ganglion
17. Spinal nerve

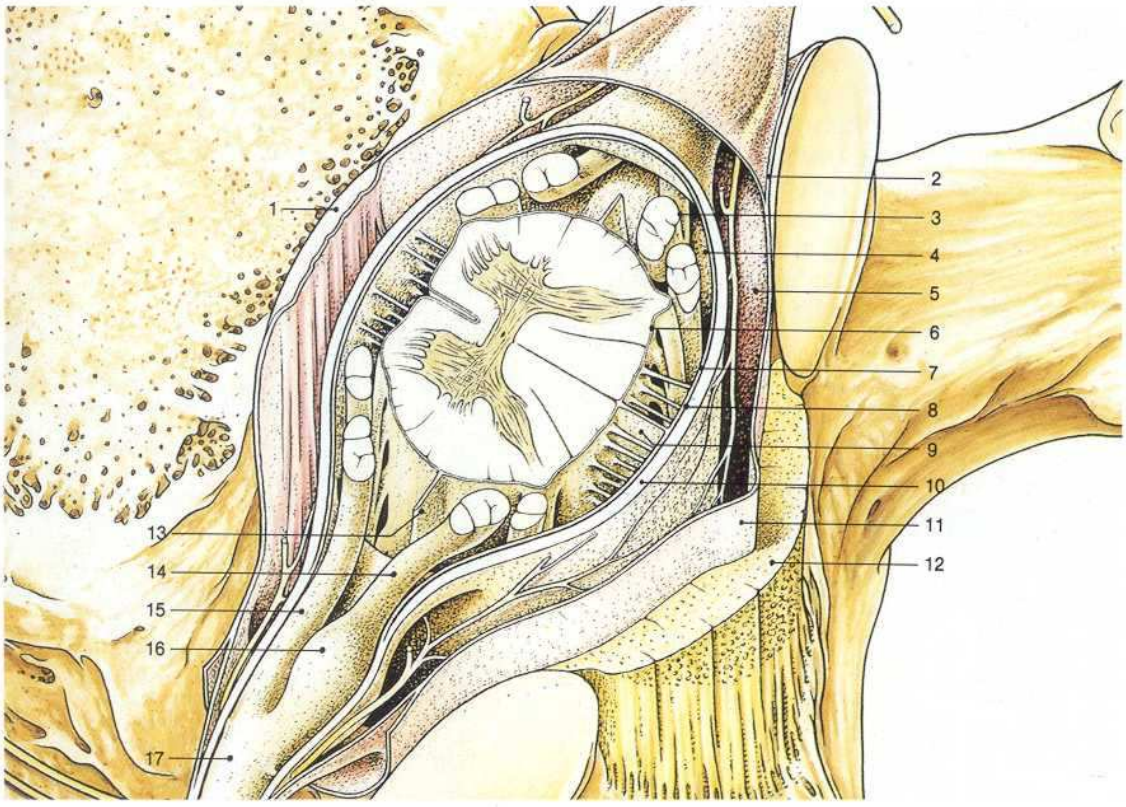


Fig. 1.18. Cross-section of the spinal canal at T11.

1. Ligamentum denticulatum
2. Posterior median septum
3. Anterior median fissure
4. Posterior longitudinal ligament
5. Pia mater
6. Dura mater
7. Subdural space
8. Arachnoid mater
9. Subarachnoid space
10. Ventral nerve root
11. Dorsal nerve root
12. Dorsal root ganglion
13. Anterior meningeal branch to epidural and subarachnoid space
14. Epinerium (continuation of dura mater)
15. White ramus communicans
16. Gray ramus communicans
17. Spinal nerve T 11
18. Intercostal nerve (ventral ramus)
19. Perineurium (continuation of arachnoid mater)
20. Dorsal ramus of spinal nerve
21. Posterior meningeal branch to epidural and subarachnoid space
22. Dorsal root of T 11
23. Dorsal root of T 12
24. Dorsal root of L1
25. Dorsal root of L2
26. Dorsal root of L3
27. Trabecula
28. Subarachnoid septum

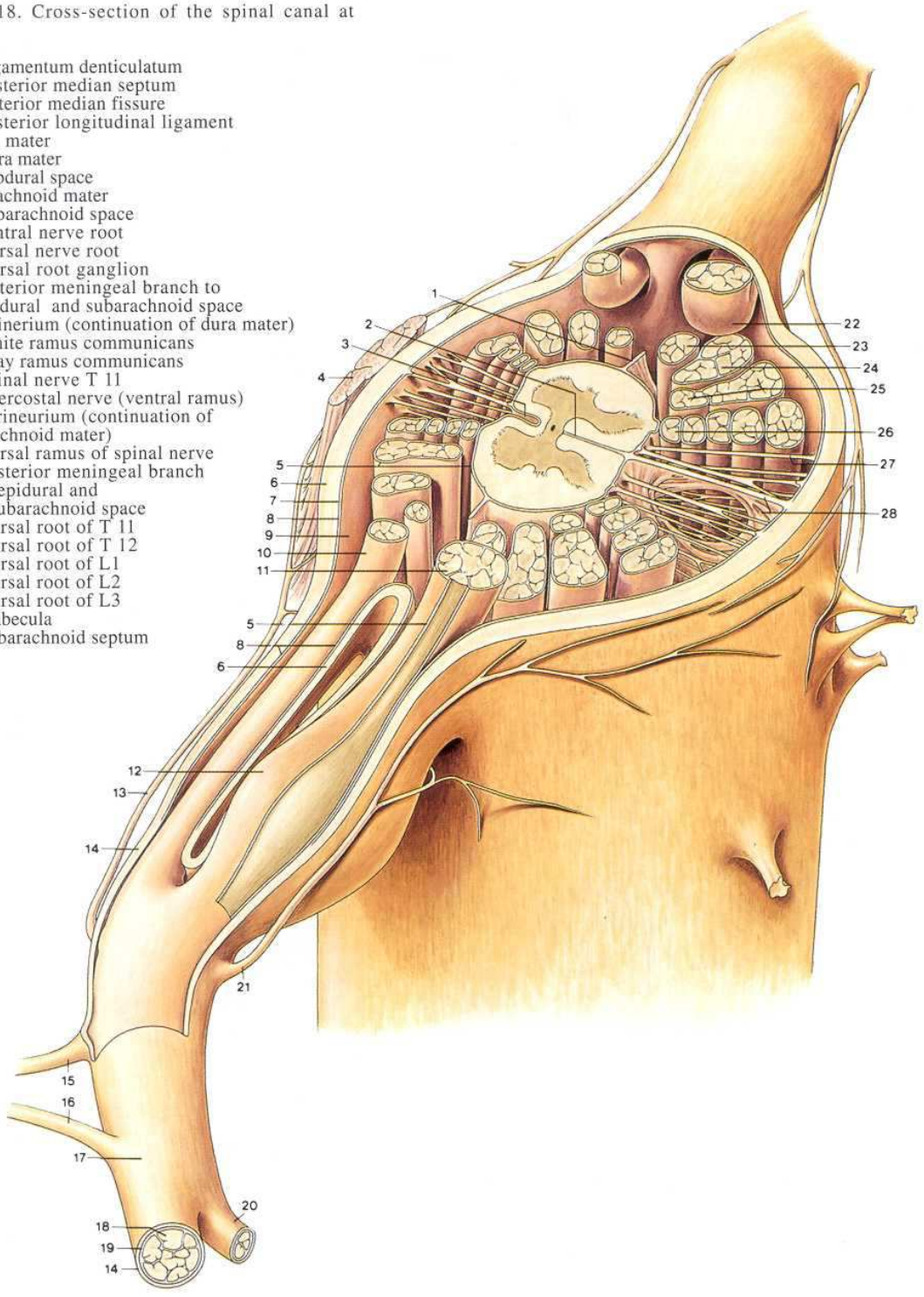


Fig. 1.29. Dermatomal distribution of spinal nerves. This fig is derived from several authorities and should be considered as a guide only.

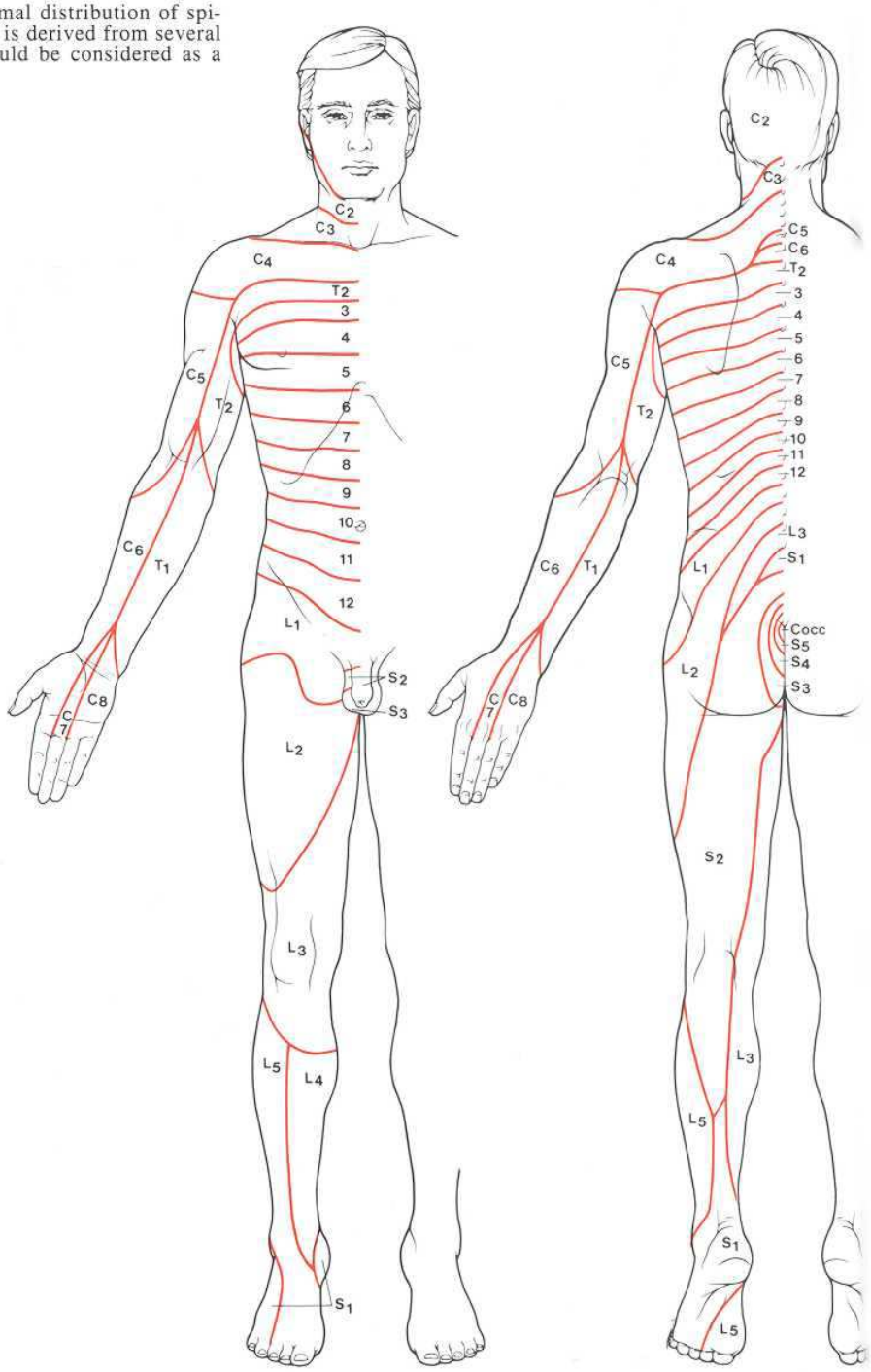


Fig. 1.36. Cerebrospinal fluid circulation. Arrows indicate direction of flow of the cerebrospinal fluid.

1. Arachnoid granulation
2. Dura mater (outer layer)
3. Dura mater (inner layer)
4. Subdural space
5. Arachnoid mater
6. Subarachnoid space
7. Superior sagittal sinus
8. Pia mater
9. Choroid plexus of the 3rd ventricle
10. Great cerebral vein
11. Cisterna cerebellomedullaris
12. Interventricular foramen
13. Interpeduncular cistern
14. Aqueduct of Sylvius
15. Cistern of the great cerebral vein (cisterna ambiens)
16. Choroid plexus of the 4th ventricle
17. Foramen of Magendie

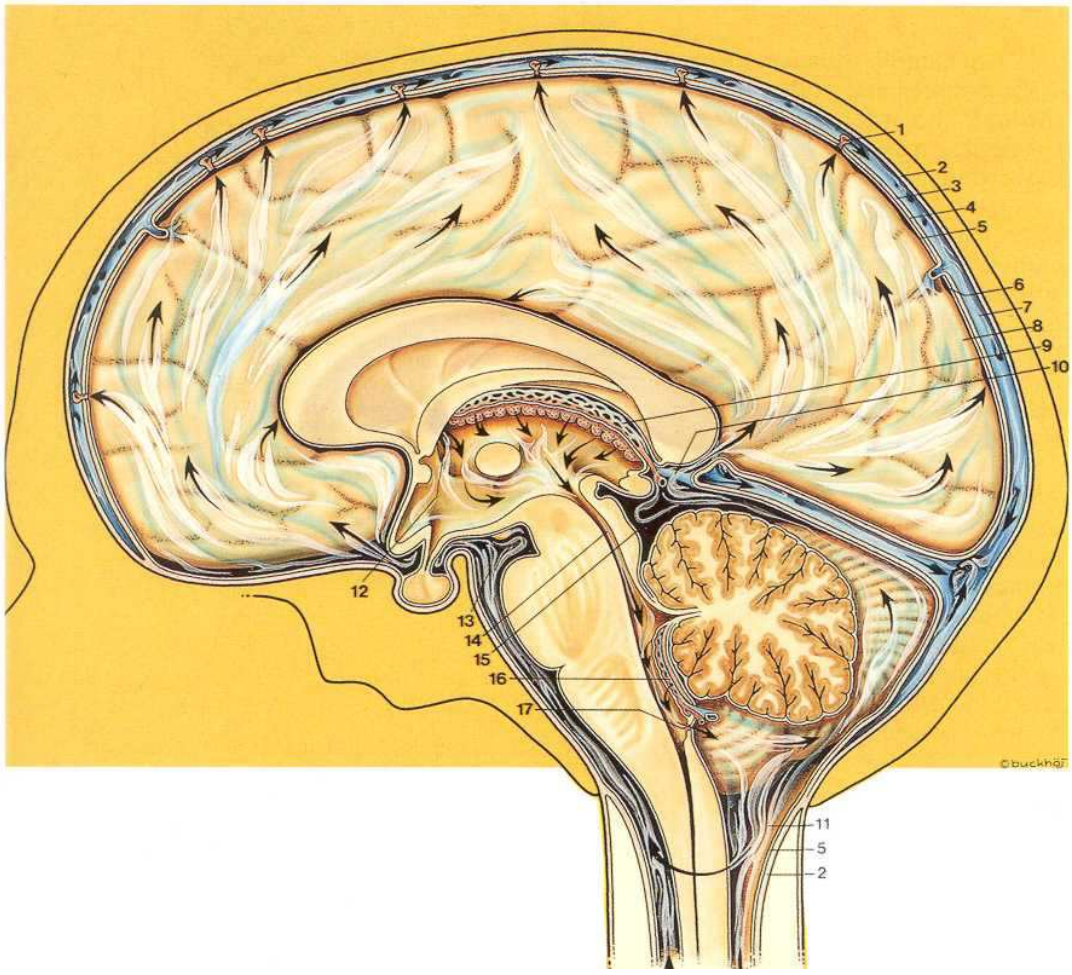


Fig. 3.20. Effect of baricity on the diffusion of solution injected into the subarachnoid space in the upright position.

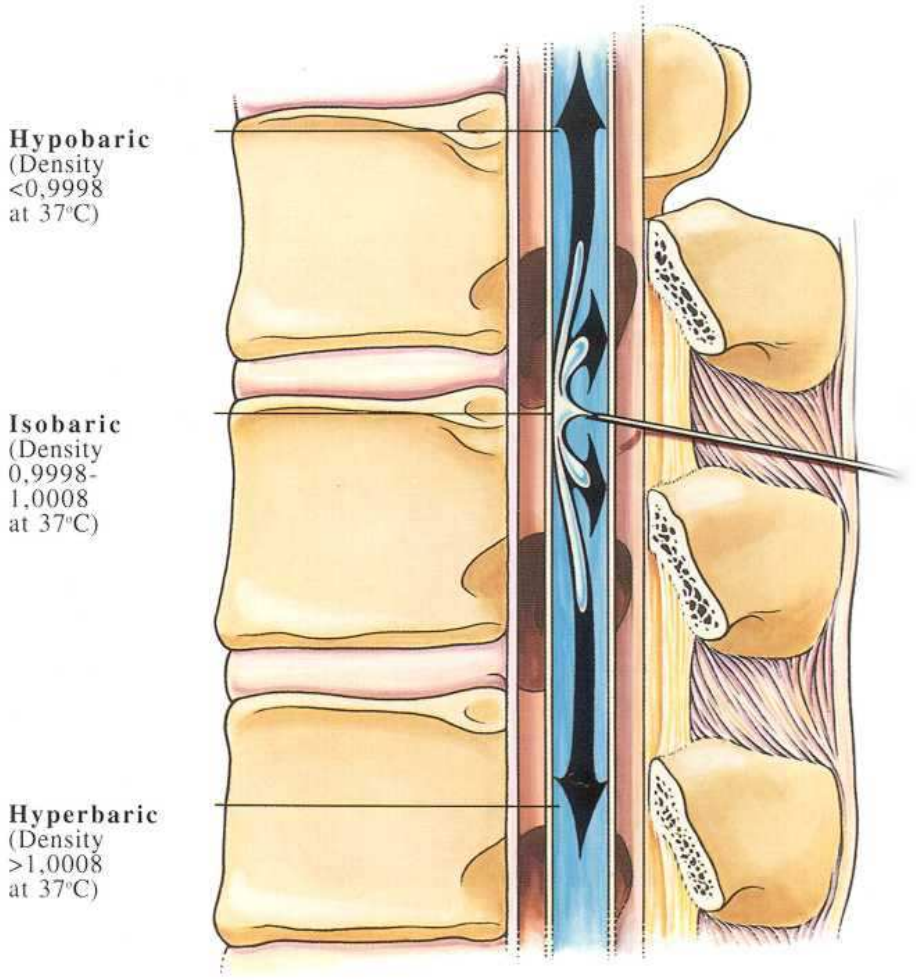
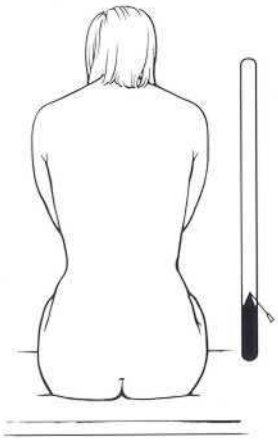
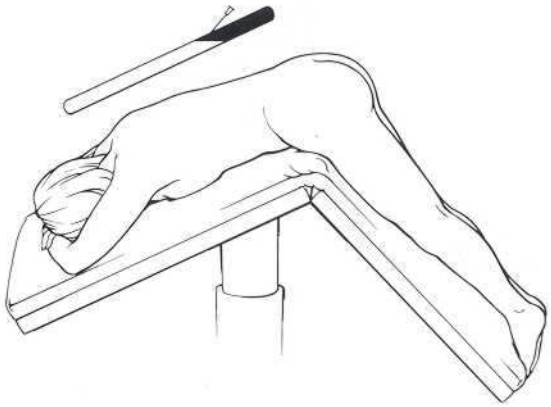


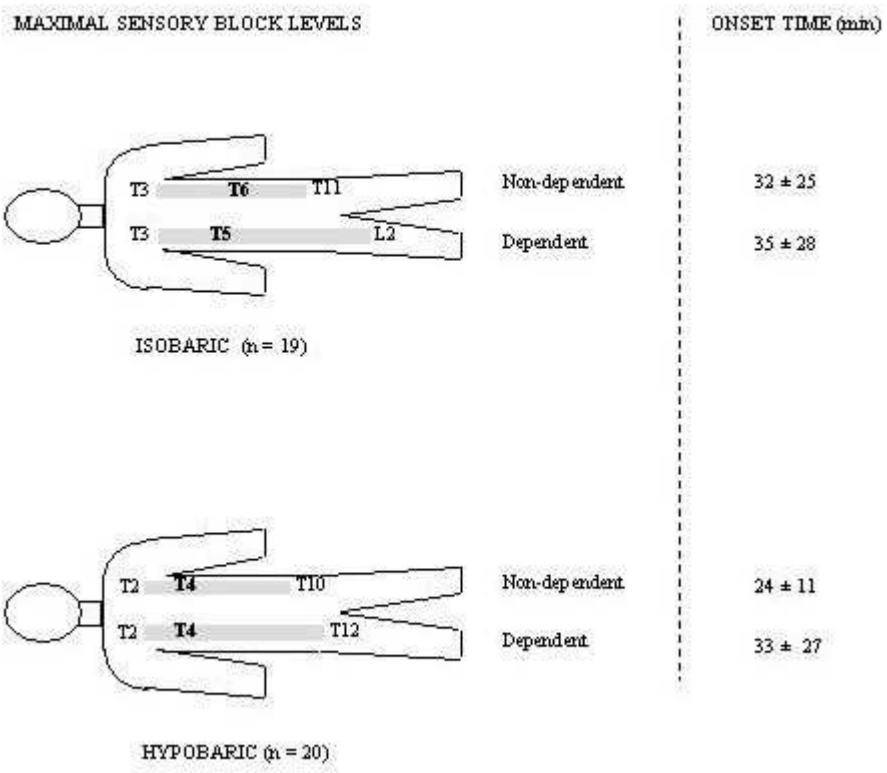
Fig. 3.21. The effect of position and baricity on the distribution of a local anaesthetic solution in the spinal subarachnoid space. The tubular figures represent the dural sac with CSF. The black areas show the local anaesthetic solution.

A. Upright position for saddle block. A hyperbaric solution, "sinks" to the most dependent area.

B. Prone jack-knife position for rectal surgery. A hypobaric solution, "floats" to the upper most area.

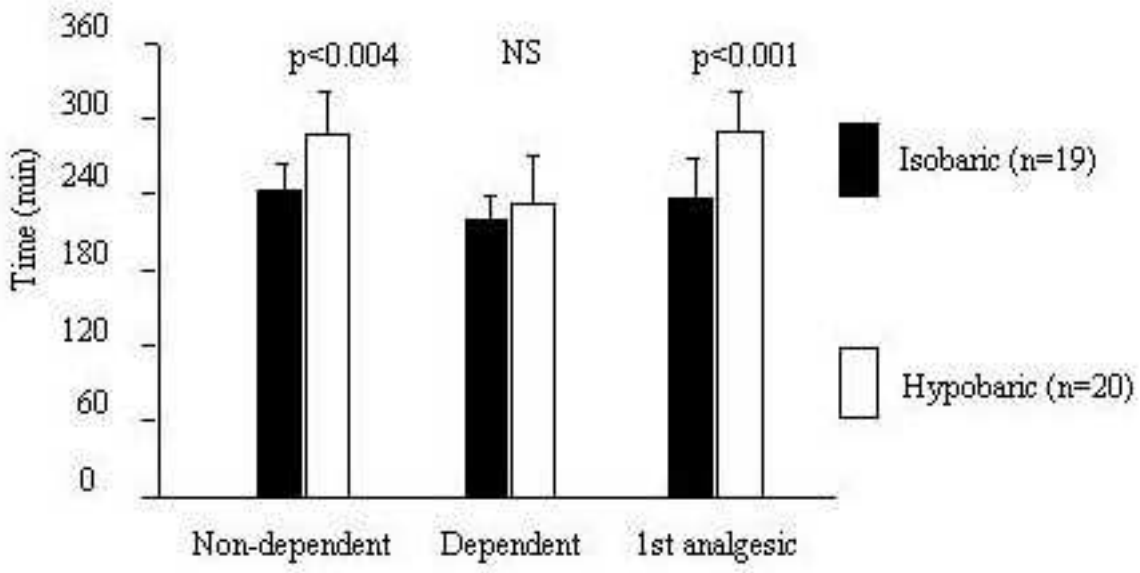






Maximal sensory block level expressed as median with ranges and onset time expressed as mean ± SD for non-dependent and dependent sides of isobaric and

hypobaric groups. There was no statistical difference in any variables between both sides of the same group and between corresponding sides of the two groups



Comparison of times of sensory regression to L2 between non-dependent sides and first analgesic for pain score > 3 (0-10) at operated side between the two groups. Regression to L2 on the non-dependent side and time of administration of first analgesic is significantly prolonged in hypobaric compared to isobaric group.

	Isobaric n = 19	**Hypobaric** n = 20
Age (yr)	63 ± 8	61 ± 10
Weight (kg)	72 ± 12	81 ± 15
Height (cm)	166 ± 8	166 ± 10
ASA status (I/II)	6/13	2/18
Female/Male ratio	11/8	11/9
MAP (mmHg)	103 ± 16	104 ± 13
Heart rate (beats/min)	73 ± 15	76 ± 16

ASA: American Society of Anesthesiology
MAP: Mean arterial pressure

Table 1: Patients characteristics and preanesthetic hemodynamic variables (mean ± SD)

	ISOBARIC (n = 19)		HYPOBARIC (n = 20)	
	Non-dependent	Dependent	Non-dependent	Dependent
Maximal degree of motor block	4 (4)	4 (3-4)	4 (4)	4 (1-4)
Onset time (min)	16 ± 4	22 ± 9	13 ± 7	16 ± 10

Table 2: Motor block characteristics (median with range in brackets or mean ± SD)

	ISOBARIC (n = 19)		HYPOBARIC (n = 20	
	Non-dependent	Dependent	Non-dependent	Dependen
Regression to L2 (min)	242 ± 36 *	219 ± 30	287 ± 51**	233
Nb of patients with complete motor recovery (0 degree of motor block) at 225 min	8	11	5 **	15

* p < 0.005, ** p < 0.0001 between non-dependent and dependent sides

Table 3: Times of sensory regression to L2 (mean ± SD) and motor block caracteristic at 225 min after spinal injection

	ISOBARIC n = 19	HYPOBARIC n = 20
Maximal decrease from preanesthethic baseline value in		
MAP (%)	32 ± 13	31 ± 16
HR (%)	14 ± 11	14 ± 10
Time to reach maximal decrease in		
MAP (min)	24 ± 11	21 ± 10
HR (min)	29 ± 14	27 ± 18

MAP: Mean arterial pressure
HR: Heart rate

Table 4: Hemodynamic changes observed during the first 45 min after initial 3.5 mL spinal injection (mean ± SD)