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Clinical experience

Hemodynamic collapse with cardiac arrest during a high subarachnoid block

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Abbreviations:
CSF LS – volume of cerebrospinal fluid in the lumbosacral spine segment
AP – antero-posterior diameter
LL – latero -lateral diameter

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Abstract

Background: *In this case report we tried to identify possible factors which could be related to developing an inappropriately high subarachnoid block and consequential hemodynamic collapse.*

Case report: *A 59 year old female patient was predicted for a lower extremity peripheral revascularization procedure.The progressive spread of spinal block with a decrease in blood pressure and slowing of heart rate ultimately resulted in a a bradycardia which lead to cardiac arrest. Since known risk factors were inapparent and CSF volume as a possible reason for the high block was excluded, we measured the segment length from the Th12 to the L5 vertebra with an MRI scan. Values for the respective parameters of lumbar segment length, angle of inclination and declination, lowest and highest points of the spinal canal which can be obtained from existing literature were compared to our results and we saw that the lumbar segment length and angle of inclination were not within the described ranges and that the lowest point of the spinal canal was at the cranial margin of the values described in literature. Thus we considered if the shorter lumbar segment and lowest spinal canal point at the Th7 level are responsible for the cranial distribution of the subarachnoid block.*

Conclusion: *Lumbar segment length, AP and LL diameters of the spinal canal and the lowest spinal canal point could help us identify higher risk groups which might require a modified dose of LA in order to assure optimal care for the patient.*

OBJECTIVE

Cardiac arrest as a consequence of an undesirably high subarachnoid block occurs in 6,4 out of 10 000 patients (0,06%), while bradycardia HR < 50/min is seen in 12 % of patients.

Perioperative risk factors for developing a bradycardia which may lead to cardiac arrest are: HR < 60 bpm at rest, prolonged PR interval, age < 50 years, ASA status, chronic therapy with β-blocking drugs and sensory block above the thoracic Th6 dermatome *(1)*. The achieved height of sensory block depends on the choice of local anaesthetic solution, the applied technique and anthropometric characteristics of the patient *(2, 3)*. Studies investigating the relationship between anthropometric variables and the distribution of SAB have shown a correlation between the volume of cerebrospinal fluid in the lumbosacral spine segment (CSF LS) with block distribution *(4)*, block regression *(5, 6)* and the physical status of the patient *(7)*. There is an inversely proportional relationship between body mass index (BMI) and CSF LS vol-

ume. The variations in anatomy and possible presence of pathological formations in the spinal and epidural space can influence the volume of CSF LS and thus influence the distribution of SAB *(8)*. In daily practice, it is impossible to measure the volume of CSF for each individual patient planned for SAB and as such, at present this method is not applied for the purpose of adjusting the dose of local anesthetic (LA) which is to be administered to a patient. Through this case report we tried to identify possible factors which could be related to developing an inappropriately high subarachnoid block and consequential hemodynamic collapse. The respective patient has consented to additional medical procedures and to the publication of this study.

Case report

A 59 year old female patient was predicted for a lower extremity peripheral revascularization procedure. Due to arterial hypertension and bronchial asthma she was classified as an ASA II patient in accordance to the ASA classification. Hypertension was managed with a calcium channel inhibitor while the symptoms of bronchial asthma were in remission and the patient was without therapy for the previous three months. Two preoperative ECG readings showed a HR of 71 and 68 bpm and PR intervals of 136 and 146 ms respectively. (The reference range for the PR interval is 0,20–0,40 ms). Body weight was 68 kg, height 172 cm and BMI = 25,51 kg/m^2 *(8)*. Thirty minutes prior to surgery, the patient was premedicated with midazolam 0.06 mg/kg and 0,5 mg atropine im. Preoperative volume replacement was managed with 8 mL/kg of Hartmann's solution. Intraoperative monitoring was standard nonvasive (ECG, NIBP, SpO_2 Infinity Delta, Dräger). Spinal puncture was conducted in the sitting position at the L3–4 interspace which was identified by ultrasound (Sonoline G50, Siemens, curved probe 2–5 MHz) and 15 mg of levobupivacaine (Chirocaine 0,5% Abbott lab.) was administered through a Whitacre 27G (Vygon) spinal needle. After anaesthetic administration, the patient was returned to the supine position. Sensory block spread was assessed by temperature sensation loss which was tested by using ice pillows from the fifth minute after anaesthetic administration in ten minute intervals. Table1. Shows the dynamics of the spread of sensory block, the patient's subjective condition and the dominant hemodynamic parameter.

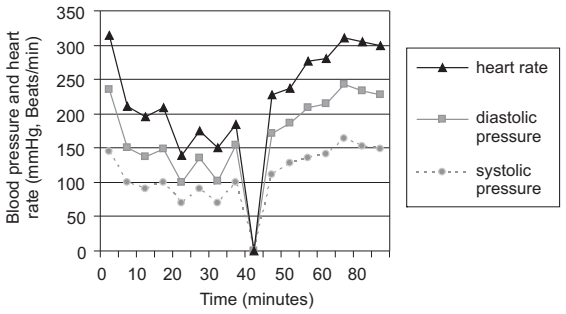

Figure 1. *Hemodynamic occurrences.*

A decrease in blood pressure and slowing of heart rate with a sensory block reaching the Th12 dermatome are visible 5 mins from anaesthetic application. Due to a ≤ 30 % fall in BP, volume replacement was carried out with 1500 mL of Hartmann's solution. A further spread of sensory block (Th 10) was followed by a further decrease in BP ≥ 30 %, refractory to colloid administration (500 mL 10% HAES) and bradycardia. Brief hemodynamic stability (systolic BP ≥ 100 mmHg and HR ≥ 60 bpm) was achieved by administering 1 mg Atropine and 25mg 1% Ephedrine-sulphate i.v.. Sensory block at the Th7 dermatome was accompanied by shivering, a subjective worsening of general condition, with a persisting hypotension despite a further volume replacement therapy of 1500 mL of Hartmann's solution. A bolus dose of norepinephrine 10 µg iv. regained a short-term hemodynamic stability. A maximum spread of block was determined at the Th1 dermatome level and was clinically accompanied by shivering of the whole upper body and deterioration in the general condition of the patient. The patient became dyspnoeic and was subjected to assisted ventilation with 100% O_2, maintaining a peripheral SpO_2 at 96–99%. Forty minutes after spinal puncture and just prior to converting to general anaesthesia (midazolam 0,1 mg/kg, fentanyl 0,3 µg/kg, pancuronium 0.9 mg/kg), the patient developed a bradycardia which lead to cardiac arrest. The response to atropine 1 mg and epinephrine 1 mg i.v. was ventricular tachycardia (VT) which deteriorated to ventricular fibrillation (VF). Conversion to sinus rhythm was achieved with 100 mg 2% lidocaine while ventilating with oxygen FiO_2. 100% Figure 1. Portrays the hemodynamic course of occurences. Central venous catheterization and arterial cannulation for invasive blood

TABLE 1

Spread dynamics of sensory block, the development of the clinical picture.

Time (min)	Height of sensory block	Subjective state of patient	Dominant hemodynamic parameter
5	Th 12	Satisfactory	Decrease in BP <30%
10	Th10	Satisfactory	Decrease in BP > 30%
20	Th7	Chills	HR < 40/min
30	Th5	Shivering	Refractory hypotension
40	Th4	Poor	VES
50	Th1		Cardiac arrest

pressure monitoring were performed. Hemodynamic recovery is achieved with a 100 μg epinephrine bolus, while stability was maintained with a continuous infusion of norepinephrine in the dose of 0,18 μg/kg/min via the CVC and surgery was continued. The depth of anaesthesia was maintained at 50–60 % with the aid of a BIS monitoring device using exclusively inhalational anaesthesia with 0,5 v % of Sevorane (Abbott lab) for the duration of the surgical procedure. The norepinephrine infusion was gradually decreased in accordance to systolic pressure being maintained at 130–150 mmHg. By the end of the surgical procedure (total time – 3 hr) the infusion of norepinephrine was discontinued. The patient was admitted to the surgical ICU for postoperative care and was extubated in the second postoperative hour. After extubation there was no residual sensoric or motoric blockade. Six hours after surgery, the patient was put on continued antihypertensive the rapy with NTG 0,6 μg/kg/min until the following morning. According to the visual analogue scale (VAS) the measured pain value exceeding the numeric factor 4 became apparent 14 hours from spinal puncture and the patient was put on continued analgesia (Tramadol 300 mg and 2,5 g metamizol iv./24h). On the second postoperative day the patient was discharged from the ICU and admitted to the vascular surgery ward.

DISCUSSION AND CONCLUSION

Risk factors which may be observed during an anaesthesiological exam and which may be related to the potential development of bradycardia and cardiac arrest during spinal anaesthesia were not observed on this patient. The progressive spread of the spinal block during a 50 minute period completely correlates to the deterioration of the patient's general condition and level of hemodynamic instability. The fact that known risk factors were inapparent compelled us to investigate the anatomical parameters which could be related to developing an adversely high SAB. The anatomical variability in patients is generally considered responsible for a wide distribution of the attained level of SAB spread, however, which anatomical parameter has most significance in this respect is still unclear *(2)*. Sharma R *et al*. have described two cases of an unexpectedly high spinal block in which lumbar stenosis of the spinal canal was consequently found *(9)*. The CSF volume in the lumbosacral region varies between studies: 28,0–81,1 mL (n=25) *(7)*, 42.7–81,81 mL (n=9) *(6)*, 20.5–61.6 mL (n=41) *(5)* and 10,6–61,3 mL(n=10) *(8)*. Spinal canal stenosis is indicative of a smaller CSF LS volume *(7)*. Carpenter RL has confirmed an inverse correlation between the volume of CSF LS and the height of achieved sensory block $\rho = -0,65$ *(6)*. To exclude a pathological substrate which could have influenced the volume of CSF in our patient and thus have affected the block distribution, we performed an MRI scan of the patient's entire vertebral column and there was no anomaly detected. The patient's position during scanning was identical to the patient's positioning when the block was being performed. Picture 1. MRI of the spine. As a possible reason for the high block, with an MRI scan, we measured the segment length from the upper border of the Th12 vertebra to the lower edge of the L5 vertebra, which measured 17,88 cm. The lowest point of the spinal canal was at the level of the Th7 vertebra and the highest at the L4 vertebra. The angle of inclination was 30°, while the angle of declination was 20,5°.

The values for the respective parameters of lumbar segment length, angle of inclination and declination, lowest and highest points of the spinal canal which can be obtained from existing literature are L5/Th12 – 18,8 to 22,2 cm *(6)*, 18,5° (15–22,5) and 13,4° (8–18,5), Th9-Th7 and L3 – L4 *(10)*. When we compare these results we can see that the lumbar segment length and angle of inclination are not within the described ranges and that the lowest point of the spinal canal is at the cranial margin of the values described in literature. It is known that the level at which spinal puncture is performed directly affects the attained level of sensory block *(11, 12)*. Thus it may be asked whether the shorter lumbar segment and lowest spinal canal point at the Th7 level are responsible for the cranial distribution of the subarachnoid block using a conventional plain solution of 0,5% levobupivacaine. It may also be asked whether and how the angles of inclination and declination affect the block distribution with the administration of hypo- or hyperbaric solutions of LA. Ultrasound is a noninvasive technique, available in the OR, which can be used to determine the diameter of the spinal canal and intervertebral space of the lumbar segments, thus providing us with an insight into the relation of these parameters to subarachnoid block distribution when administering conventional doses of local anaesthetics. Even though Arzola C had not substantiated the relationship between ultrasonically determined AP diameters of the dural sac with block distribution in pregnant women *(13)*, due to increased intraabdominal pressure, specific CSF density, diminished lumbar lordosis and a cranial shift of the lowest spinal canal point in pregnancy, we nevertheless consider that a similar investigation should be conducted on the general population of patients. There is very little chance that one anatomic variable will ultimately determine the choice and quantity of the administered local anaesthetic. Information about lumbar segment length, AP and LL diameters of the spinal canal and the lowest spinal canal point could help us identify higher risk groups which might require a reduced or increased dose of administered LA in order to assure optimal care for the patient. No pathoanatomical or pathophysiological substrate could be established as a causative factor for the extensive block which occured in our patient. The measured anatomical parameters (LS segment length, lowest point of the spinal canal and angle of inclination) are near the endpoints of established anatomical variability and it is possible that with a conventional dose of levobupivacaine these factors contributed to the development of the extensive SAB accompanied by hemodynamic collapse.

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