The physiological responses to spinal and epidural anaesthesia have much in common. These responses are mostly due to the fact that the local anaesthetic solution blocks not only somatic, sensory and motor fibres, but also produces pre-ganglionic sympathetic blockade. Postganglionic sympathetic nerves play a role in controlling gastrointestinal tone and motility, cardiac function and vascular tone thereby regulating regional blood flow. Blockade of sympathetic nerve fibres and resulting loss of vasomotor tone causes changes in blood flow to various organs depending on the level of block achieved. The parasympathetic nervous system is also important in regulating cardiovascular and gastrointestinal function. This lecture will focus on comparison of the physiological responses to spinal and epidural anaesthesia.

MECHANISMS OF SPINAL AND EPIDURAL ANAESTHESIA

SPINAL ANAESTHESIA

The primary site of action of local anaesthetics injected into the subarachnoid space is on the spinal rootlets and in the case of afferent sensory fibres, on the dorsal root ganglia. The nerve roots leaving the spinal cord are not covered by epineurium and are readily exposed to local anaesthetics in the cerebrospinal fluid (CSF) [1]. This radicular blockade produces the well known segmental block pattern.

EPIDURAL ANAESTHESIA

In the epidural space possible sites of action include the spinal nerve trunks in the paravertebral space, the dorsal root ganglia, the dorsal and ventral spinal roots and the spinal cord itself [2]. In the epidural space the myelinated nerve sheath acts as a barrier to local anaesthetic diffusion, as do the meninges.

DIFFERENTIAL BLOCKADE

Differential sensitivities of nerves to local anaesthetics result in clinically important zones of differential blockade involving somatic motor fibres, somatic sensory fibres and pre-ganglionic sympathetic fibres [3-5]. The sequence of block varies according to the blocking agent, the size of nerve fibre axons, the frequency of nerve impulse transmission and the length of nerve fibre exposed to the local anaesthetic. Fink [4] suggested that fibre size is not as important as the length of exposed nerve fibre, i.e. the number of nodes (three consecutively) exposed. Measurements in isolated nerve fibres and anatomical considerations may explain the persistence of differential block after epidural and subarachnoid injection. According to this theory there may be differences in block following each technique.

SPINAL ANAESTHESIA

It has been assumed that sympathetic blockade extends two dermatomes beyond somatic blockade. However, discrepancies exist between studies and it has been shown that sympathetic block after spinal anaesthesia may extend up to six dermatomes beyond the sensory block [5]. Different techniques of indirectly measuring sympathetic function (infrared thermography, flowmetry, skin conductance responses) display wide discrepancies after spinal anaesthesia. The concentration of local anaesthetic in the CSF decreases as a function of the distance from the site of injection, and preganglionic nervous system fibres are anaesthetized by concentrations of local anaesthetics that are inadequate to affect sensory or motor fibres [1,5].

EPIDURAL ANAESTHESIA

The sensitivity of various nerve fibres to local anaesthetics is related to the size and type of the nerve fibre, the mass of local anaesthetic administered epidurally and the physicochemical properties of the local anaesthetic agent. With epidural anaesthesia there are zones of differential sensory blockade between pinprick and cold discrimination, with an increasing difference between levels of anaesthesia and analgesia in the cephalad direction.[3]
SYMPATHETIC DENERVATION

The sympathectomy that accompanies spinal and epidural anaesthesia is dependent upon height of block. As all sympathetic nerves derive from T1 through to L2, a block into the midthoracic region causes a considerable decrease in sympathetic activity, particularly in vasomotor control of the lower part of the body. It is unknown whether the efferent pre-ganglionic sympathetic nerves are distributed segmentally. Current methods do not show a clearly discernible boundary between blocked and unblocked segments and so the sympathetic block may appear patchy.

Despite high levels of epidural blockade (above T5) weak galvanic skin responses to arousal could still be elicited in the foot, indicating incomplete sympathetic blockade.[6] Even with subarachnoid local anaesthetic block to T5 sympathetic block in the foot was still incomplete.[7]

When comparing the physiological effects of spinal and epidural anaesthesia it is relevant to ask if spinal anaesthesia does result in a more complete sympathetic block than epidural anaesthesia. To answer this question Stevens et al.[8] studied healthy volunteers in a cross-over design and used a cold pressor test as a sympathetic stress. Plasma catecholamines and haemodynamic responses were similar following both techniques, indicating that spinal anaesthesia did not result in more profound attenuation of the sympathetic response than epidural anaesthesia. In addition neither technique produced total sympathetic blockade.

CARDIOVASCULAR EFFECTS

The cardiovascular effects observed with epidural and spinal anaesthesia are complex and are predominantly related to the extent of sympathetic denervation, autonomic balance, baseline blood volume and cardiovascular function. With epidural anaesthesia the pharmacological effect of systemically absorbed local anaesthetic agents and inclusion of adrenaline to the local anaesthetic solution may play a role. Individual cardiovascular response to different levels of sympathetic blockade varies widely, depending on the degree of sympathetic tone prior to the block.

The sympathetic trunk receives its preganglionic fibres from T1 to L2-3. The most important of the cardiovascular effects are related to blockade of vasoconstrictor fibres (below T4) with resulting dilatation of resistance and capacitance vessels and/or cardiac sympathetic fibres with loss of chronotropic and inotropic drive to the myocardium (T1-T5) (fig 1).

**Figure 1**

*Figure 1. Sympathetic blockade: “central” (cardiac) and “peripheral” components. These consist of T1-T4 cardiac sympathetic fibres and T1-L2 “peripheral” sympathetic fibres. Note important innervation of veins and venules. Vagal cardiac fibres are also shown. From Cousins MJ, Veering BT. Epidural Neural Blockade in Clinical Anaesthesia and Management of Pain, third Edition. Edited by Cousins MJ, Bridenbaugh PO. Philadelphia, Lippincott-Raven Publishers, 1998, p261*
The haemodynamic changes produced by spinal and epidural anaesthesia are believed to result from venous and arterial dilatation induced by sympathetic blockade. The venodilation effect predominates. A block below L3 does not affect the sympathetic system and the haemodynamic upset is minimal. Haemodynamic changes relate to the extent of involvement of groups of nerves supplying the peripheral vessels, the splanchnic bed (T5-L3) and the heart (T1-4). Compensatory vasoconstriction of capacitance vessels will occur in remaining unblocked areas. In addition circulating catecholamines released from the adrenal medulla due to increased activity in any unblocked fibres in the splanchnic nerves contribute to the increased sympathetic activity below and above level of block. In a block above T5 the cardioaccelerator nerves (T2-4) will be involved, resulting in a decrease in cardiac output, slowing of the heart rate and decreased contractility.

**THE CARDIOVASCULAR EFFECTS OF SPINAL COMPARED TO EPIDURAL ANAESTHESIA**

Comparing the cardiovascular effects associated with equal sensory levels of spinal and epidural anaesthesia (T-5) in volunteers who served as their own controls resulted in differential effects. The decrease in mean arterial blood pressure, cardiac output and stroke volume was greater following spinals. This is partly due to the fact that the onset of sympathetic blockade is slower following an epidural, which allows more time for compensation.

Relatively large amounts of local anaesthetic solutions are required to achieve satisfactory epidural anaesthesia. Local anaesthetic agents exhibit a direct action on vascular smooth muscle resulting in either vasoconstriction or vasodilatation, depending upon the local anaesthetic agent, its stereo chemical configuration and concentration, the type of blood vessel (capacitance or resistance) and pre-existing vascular tone. Lower doses of lidocaine, producing plasma concentrations up to 4 mg L⁻¹ have been shown to increase cardiac output by causing vasoconstriction. Conversely at higher doses the vasoconstrictor action of lidocaine changes to one of vasodilatation and hypotension may occur.

Epinephrine (5 µg.mL⁻¹) is commonly added to the local anaesthetic solution to slow systemic absorption and prolong the duration of action. Vascular systemic absorption of epinephrine results in beta-stimulation. In low doses, epinephrine produces β₂-receptor vasodilatation action leading to a reduction in peripheral resistance. In greater doses adrenaline stimulates the heart by β₁-receptor stimulation resulting in increased contractility and heart rate. The early cardiovascular changes observed with absorbed adrenaline are transient. Therefore the prolonged cardiovascular changes seen with local anaesthetics containing adrenaline are probably related to sympathetic blockade.

**BRADYCARDIA**

Epidural and spinal anaesthesia (with or without involvement of cardiac segments) may be associated with profound bradycardia and, in some patients, with transient cardiac arrest. This appears to be a vagal reflex. The blockade of sympathetic cardiac accelerator fibres and the failure to block the vagus nerve may cause vasovagal episodes. The normal reflex tachycardia through stimulation of baroreceptors in response to a decreased arterial pressure is often absent during epidural and spinal anaesthesia when sympathetic blockade extends to the lower thoracic segments. Factors thought to contribute to the development of bradycardia include: decreased cardiac sympathetic tone, decreased venous tone, reflex decrease in heart rate resulting from decreased degree of pacemaker stretch, and reflex decrease in heart rate mediated via ventricular mechanoreceptors.

Bradycardia is probably caused by an enhancement of cardiac vagal activity secondary to decreased venous return. Jacobsen et al. described echocardiographic and circulatory findings, along with changes in vagally mediated plasma hormonal parameters, in volunteers given lumbar epidurals from T8-10 to L3-L4. Hypotension was accompanied by a 30% decrease in heart rate and a decrease in end-diastolic left ventricular cross-sectional diameter, indicating decreased preload. There was a marked increase in plasma pancreatic polypeptide, an index of vagal activation. This increase in pancreatic polypeptide indicated that organs other than the heart are also exposed to increased vagal activity. The results of this study indicate that during lumbar epidural anaesthesia, presyncope symptoms are preceded by a reduction of central volume and of left ventricular diameters by approximately 13%. The aetiology of profound bradycardia or asystole during epidural anaesthesia remains unknown. The observations support an activation of the Bezold-Jarisch reflex, a cardio-inhibitory vasodepressor reflex elicited during central volume depletion from mechanoreceptors in the inferoposterior wall of the left ventricle. Vagal activation with bradycardia is thus a protective reflex that prevents the heart from contracting when relatively empty.
RESPIRATORY EFFECTS

The lungs are innervated from the sympathetic system from T2 to T7 and from the parasympathetic system via vagal afferent and efferent pathways.

Epidural or spinal anaesthesia per se have little effect on respiration in patient without pre-existing lung disease. Most of the respiratory changes associated with spinal and epidural anaesthesia are due to motor block of the muscles of respiration. Healthy unpremedicated patients receiving high-thoracic dermatome neuraxial blocks are able to maintain normal arterial blood gas tensions and the hypercapnic response is unchanged or increased.[15,16] However, preoperative or intra operative sedation during spinal anaesthesia can result in abnormal gas exchange. Thus, oxygenation should be monitored with pulse oximetry whenever sedative drugs are employed.

Since epidural anaesthesia induces segmental block of spinal nerves, an adequate extension of the block of motor nerves can selectively affect respiratory muscles in the rib cage. In healthy awake volunteers thoracic epidural anaesthesia (TEA) caused a reduction of ventilatory response to CO₂ during spontaneous respiration principally because of decreased contribution of the rib cage to tidal breathing.[17] This probably reflects blockade of the efferent or afferent pathway (or both) of the intercostal nerve roots. The respiratory effects of TEA are: modest reduction in vital capacity (VC) and forced expiratory volume in 1 second (FEV₁), reduction in total lung capacity (TLC) and maximal mid-expiratory flow rate (MMF) with high thoracic epidural block (T1-6). These alterations in expiratory function are probably of minor clinical importance in healthy patients. In severely compromised pulmonary patients however high TEA may impair the ability to cough effectively.

The diaphragm is the principle muscle of inspiration. Diaphragmatic dysfunction is a major determinant of the impaired respiratory function observed after upper abdominal and thoracic surgery. Diaphragmatic activity increases following TEA possibly by the interruption of an inhibitory reflex of phrenic nerve motor activity, either related to direct deafferentation of visceral sensory pathways, or by a diaphragmatic load reduction due to increased abdominal compliance.[18]

GASTROINTESTINAL EFFECTS

Epidural and spinal block extending from T6-L1 effectively denervates the splanchnic sympathetic supply to the abdominal viscera. Spinal and epidural anaesthesia are both capable of blocking all of the sympathetic fibres innervating the bowel and the hypogastric nerves (S2-4). The vagus nerve, of course, will remain unblocked and as a consequence sympathetic blockade results in a small contracted gut owing to parasympathetic dominance by the vagus and hypogastric nerves. In general, parasympathetic outflow increases gut activity, including tonic contraction, sphincter relaxation, peristalsis and secretion. Gut transit time is reduced and gastric emptying is increased. These results suggest that intraoperative and postoperative epidural anaesthesia with local anaesthetic agents may be useful in preventing and relieving postoperative ileus.[19] Blood flow to the gastrointestinal tract is significantly increased, which may benefit anastomotic sites.[20]

Nausea and vomiting may be associated with spinal and epidural anaesthesia. The aetiology of nausea during central neural blockade is poorly understood, but an increased incidence of nausea was associated with hypotension, unopposed vagal activity, sensory levels above T5 and a history of motion sickness.[12]

ENDOCRINE-METABOLIC PHYSIOLOGY

Significant metabolic and endocrine alterations are associated with the trauma of all surgical procedures. These changes are termed the surgical stress response.

Spinal and epidural anaesthesia are able to block the sympathetic stimulation caused by surgical stress that leads to catecholamine and hormonal release. The inhibition is related to the level of neural blockade, particularly in operations on the lower abdomen and lower extremities. There is however little inhibition of endocrine-metabolic response by epidural anaesthesia in upper abdominal surgery, probably due to a failure to block the vagus nerve.
Few studies have been performed comparing endocrine metabolic responses to similar surgical procedures performed either during spinal or epidural anaesthesia, except during hysterectomy, where no additional metabolic effect was seen with the denser block of a spinal.[21] In contrast, studies in major abdominal (colonic) surgery showed continuous spinal (T4-S5) to be more effective in reducing the stress response than epidural anaesthesia (T4-S5).[22] Continuous spinal anaesthesia attenuated, but did not abolish, the increase in plasma cortisol associated with colonic surgery.

**THERMOREGULATORY RESPONSE**

Hypothermia (a decrease in core temperature) is common in patients undergoing surgery with epidural and spinal anaesthesia. Three major factors contribute: (1) internal redistribution of body heat from central to peripheral regions, (2) heat loss to the environment, and (3) inhibition of central thermoregulatory control. Shivering-like tremor during epidural anaesthesia is triggered by core hypothermia and preceded by vasoconstriction above the level of the block.

Comparable sweating, vasoconstriction and shivering thresholds during spinal and epidural anaesthesia have been demonstrated, suggesting that thermoregulatory processing is similar during each type of regional anaesthesia, although the amount of local anaesthetic administered usually differs by an order of magnitude.[23]

**CONCLUSION**

The physiological responses to spinal and epidural anaesthesia result from three effects (1) autonomic blockade with effects on both the vascular beds and cardiac function; (2) abolition of somatic pain and the reflex responses associated with it, and (3) from the effects of motor block.

When comparing the physiological effects of spinal and epidural anaesthesia it is important to understand that epidural anaesthesia is slowed by the dural and epineurial barriers. Therefore the onset of sympathetic blockade with epidural anaesthesia is slower than with spinal anaesthesia. In general the fall in blood pressure will be relatively mild and more gradual with epidural with comparable spread. In addition the spread of epidural anaesthesia can be more carefully controlled and the block can be performed segmentally, restricting the haemodynamic side effects.

The question arises as to whether there will exist any difference with regard to differential block between spinal and epidural anaesthesia. Neither technique provides total sympathetic blockade. Further studies are however needed before clinically relevant conclusions can be drawn. This summary has shown that the physiological effects of spinal are similar to those of epidural anaesthesia, with the exception that local anaesthetic blood levels following epidural anaesthesia may produce systemic effects on their own.
REFERENCES

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