

Assessment of regional analgesia in clinical practice and research

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Assessment of pain and sensory function during regional analgesia contributes to a better understanding of the mechanisms underlying the action of drugs and techniques, and provides information on the effectiveness of regional analgesia in daily practice. Sensory tests only partially mimic clinical pain, mainly because they are artificial and reproduce only a part of the complex experience of pain. Therefore information gained by sensory tests should not be uncritically generalized to clinical pain conditions. Studies using experimental pain models are not in competition with studies performed under clinical conditions, but complement them. In order to mirror clinical pain, experimental models ideally stimulate muscles and viscera, induce peripheral and central sensitization, and evoke temporal and spatial summation. These methods are available, but are underused. Test modalities used in clinical practice have limited validity. In recent years almost no research has been performed to develop better test modalities that are suitable for daily practice.

Introduction

Regional analgesia is routine practice in anaesthesia and pain management. The possibility of providing analgesia at the site of pain, with minimal or no impairment of other functions, is the main rationale behind it. Surgical analgesia, postoperative pain management and the diagnosis and treatment of chronic malignant and non-malignant pain are the fields of application of regional analgesia techniques.

Two professional categories are interested in assessing pain and sensory function in regional analgesia: the clinician and the researcher. Basically, the clinician would like to know whether intervention will produce a good outcome for the patient. An example of outcome is the absence of pain during orthopaedic surgery performed under epidural block. In this case, assessment of sensory function is expected to provide predictive information on the success of the block and therefore to help

Accepted: December 14,
2004

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in making decisions on the anaesthetic management. The researcher is interested in investigating the analgesic effect of new drugs before they are used in patients, the combination of different drugs or the mechanisms of action of analgesics.

While there is little doubt about the usefulness of assessing the effects of regional analgesia on sensory function and pain, important questions need to be addressed. How can we measure pain? Can we predict a successful or unsuccessful block based on preoperative sensory assessments? Do experimental pain tests predict the efficacy of new drugs or techniques in the clinical settings?

The main aims of this review are to describe the methods used for sensory assessment and to evaluate the relationship between tests and clinical pain. Ultimately, we discuss what tests should be used, what applications are suitable and what information can be or cannot be expected from assessment of pain and sensory function. In this sense, this paper updates the information provided in a previous review.¹

General aspects

Pain and related parameters need to be assessed in all patients with acute and chronic pain. Patients undergoing regional analgesia are not an exception. Aspects related to pain include psychological evaluation,² assessment of function and disability³ and quality of life.⁴ Obviously, not all measurements need to be made in all patients after regional analgesia; the choice depends on the pain condition that is treated and the type of intervention. The assessment of ongoing pain and related factors⁵ is beyond the scope of this paper.

An extended and more advanced approach than simply assessing ongoing clinical pain is to impose additional standardized stimuli on the patient and evaluate the response under controlled conditions. This approach is termed quantitative sensory testing and allows a better understanding of the mechanisms involved in pain transduction, transmission and perception under normal and pathophysiological conditions.

Pain researchers and clinicians have a dream—to be able to measure pain objectively. However, this dream will probably never come true. Pain remains subjective in nature. Furthermore, it is a multidimensional sensory and emotional experience which is unlikely to be completely represented or described by a number. Thus any measurement or set of measurements will provide only partial information about the complex events and mechanisms that are involved in clinical pain. Nevertheless, important information can be gained by pain and sensory assessment, provided that the clinician and the researcher understand the characteristics of the tests that they employ.

Methods for assessing regional analgesia

An ideal test has the following characteristics:

- 1 it delivers reproducible stimuli;
- 2 it can be applied to all body parts without causing damage;
- 3 it selectively activates pain receptors;
- 4 it elicits different and discriminatory pain intensities and qualities;
- 5 it can induce temporal and spatial summation (see below);
- 6 it provides a positive correlation between stimulus intensity and pain intensity;
- 7 it is easy to apply and control in laboratory and clinical settings;
- 8 it mirrors clinical pain mechanisms.

Unfortunately the ideal test does not exist, and hence the most adequate test should be selected based on its characteristics and the specific application.

As pain is a multidimensional perception, the reaction to a single standardized experimental stimulus of a given modality can only represent a very limited fraction of the entire pain experience. Therefore it is mandatory to combine different stimulation and assessment approaches to gain advanced differentiated information about the nociceptive system under normal and pathophysiological conditions.

Stimulus modalities

Stimuli can have phasic properties (short-lasting, milliseconds to few seconds) or tonic properties (long lasting, many seconds to minutes). The various pain induction techniques are summarized in Table 1. It can be seen that most methods have been developed for cutaneous applications and therefore may not reliably mimic clinical pain, which usually involves deeper structures. Only in more recent years have quantitative experimental methods for the assessment of muscle⁶ and visceral⁷ pain been developed.

Most studies have used electrical stimulation, but this technique is non-selective and bypasses the receptors by depolarizing the afferent nerve fibre. However, bypassing the nociceptors allows the exploration of pain reactivity without the influence of peripheral nociception. Thus investigations using electrical stimulation may be interesting when the excitability of the central nervous system is explored.⁸

The current perception threshold,⁹ which is based on the theory that different frequencies activate different nerve fibre populations, has been

Table 1 Pain induction modalities**Electrical**

Transcutaneous (through contact electrodes)
 Intracutaneous (through electrodes that penetrate the skin)
 Tooth pulp
 Intramuscular (through stimulating needles)
 Transmucosal (through contact electrodes: oral or visceral stimulation)

Heat

Radiant heat (laser, light, infrared)
 Contact thermode (skin stimulation)
 Circulating hot water (possible stimulation of skin, muscle or viscera)

Cold

Cold pressor test (immersion of hand in ice water)
 Contact thermode (skin stimulation)
 Evaporation of gas (e.g. ether applied to the skin)
 Menthol (applied to the skin)
 Circulating cold water (possible stimulation of muscle or viscera)

Mechanical

Brushing/stroking
 Pinprick (e.g. with needle or von Frey hair)
 Pinch
 Impact stimuli
 Pressure (generally with a pressure algometer)
 Distension (e.g. using a balloon to distend viscera)

Ischaemia

Tourniquet (applied to arm or leg to induce ischaemic pain and pressure pain)
 Exercise (e.g. lifting a weight repeatedly or exercising with a hand-grip trainer)

Chemical

Capsaicin (activation of nociceptor of skin, muscle or viscera)
 Mustard oil (induction of inflammatory reaction of the skin)
 Melittin (active substance of bee venom applied to the skin)
 Hypertonic saline (5% solution injected into muscle)
 Bradykinin, serotonin, substance P and other algogenic substances (injected into muscle)
 Carbon dioxide (stimulation of the nasal mucosa)
 Glutamate (injected into muscle to induce hyperalgesia)
 Nerve growth factor (injected into muscle)
 Glycerol (applied to viscera)
 Hydrochloride acid (applied to viscera by slow perfusion)

used for sensory assessment. The frequencies normally used are 5, 250 and 2000 Hz which have been claimed to activate C, A δ and A β fibres, respectively. However, there is no electrophysiological evidence that this differentiated nerve fibre activation can be achieved in humans.

Lasers are probably the most selective heat stimulators.¹⁰ The advantage is that the laser radiation can be applied without touching the skin and hence does not contaminate the stimulation by mechanosensitive input. The disadvantage is that lasers are usually difficult to operate, expensive and not very mobile.

Chemical stimuli are difficult to repeat over time. However, some of them (e.g. capsaicin) induce peripheral and central sensitization. Therefore

these tests may produce experimental conditions that are closer to clinical pain compared with methods that do not induce sensitization.¹

Assessment modalities

The methods used to assess experimentally induced pain are summarized in Table 2.

Psychophysical methods

Psychophysical tests require awake subjects who fully understand the instructions, which is usually the case during regional analgesia. Psychophysical methods can be divided into response- and stimulus-dependent methods.

Response-dependent methods rely on how the person evaluates intensity or unpleasantness of the stimulus on a given scale (e.g. the visual analogue scale).

Stimulus-dependent methods are based on adjustment of the stimulus intensity until a predefined response, typically a threshold, is reached. Thresholds are of great value because they can be assessed reliably, easily and simply. The pain threshold is the least amount of physical energy necessary to elicit pain, whereas the pain tolerance threshold is the least amount of physical energy that produces unbearable pain. Thresholds demonstrate where the pain range starts (pain threshold) and where it ends (pain tolerance threshold). However, there is no information about pain perception between these range delimiters as obtained using stimulus-response functions.

The diffuse noxious inhibitory control (DNIC) paradigm is based on the inhibition of pain that is produced by the application of another painful stimulus.¹¹ This is accomplished by measuring the pain response

Table 2 Pain assessment modalities

Psychophysical

Visual analogue scale (VAS), verbal rating scale (VRS) or numerical rating scale (NRS)

Pain detection and tolerance thresholds

Diffuse noxious inhibitory control (DNIC)

Electrophysiological

Microneurography

Excitatory or inhibitory reflexes

Evoked potentials

Electroencephalogram

Magnetoencephalogram

Imaging techniques

Positron emission tomography (PET)

Single-photon emission computed tomography (SPECT)

Functional magnetic resonance imaging (fMRI)

to a phasic stimulus before, during and after the application of a tonic painful stimulus. Impairment of the inhibition of the pain response is assumed to reflect altered endogenous pain modulation. Thus this test modality explores the complex phenomena of endogenous pain modulation, which is probably important in clinical pain. Indeed, DNIC may have a good correlation with clinical outcomes: the greater the DNIC, the lesser the pain and physical impairment.¹¹ To our knowledge, this paradigm has not yet been applied to questions related to regional analgesia.

Electrophysiological methods

Microneurography is the direct recording of nerve fibre activity via needle electrodes inserted percutaneously.¹² The method allows the identification of the different classes of fibres and nociceptors that are activated by different types of peripheral stimulation in humans.

The nociceptive withdrawal reflex is a spinal reflex of the lower limb that is elicited by a painful somatic stimulus.¹³ This stimulus can be applied as a single stimulus or repeated (temporal summation). Either the reflex threshold or the reflex amplitude to a fixed supra-threshold stimulus can be assessed. The stimulus threshold required to elicit the electromyographic response usually corresponds to the stimulus threshold eliciting a subjective pain perception.¹⁴ The generation of a withdrawal reflex is initiated by the nociceptive input, but extensive processing takes place within the spinal cord. Other afferent inputs, descending activity and the excitability of the neurons in this pathway modulate the generation of the spinal nociceptive reflex.

The potentials evoked by painful stimulation have been widely used in pain research. However, evoked potentials are the result of a general activation of the sensory system and are not specific correlates of pain perception.¹⁵ It is unclear whether they reflect the inhibition of nociceptive transmission after regional analgesia. Indeed, it has been repeatedly shown that there is poor correlation between analgesic effect and effect on evoked potentials.¹

The electroencephalogram and the magnetoencephalogram (measurement of the magnetic signals from the brain) reflect unspecific activity of brain structures, and until now have been of limited value in the evaluation of pain after regional analgesia.

Temporal and spatial summation

Temporal summation occurs when a stimulus causes exaggerated perceptions of pain when repeated at certain frequencies (Fig. 1). In animal studies, repeated stimulation increases the excitability of spinal cord neurons which persists after discontinuing the peripheral stimulation.

Temporal and Spatial Summation

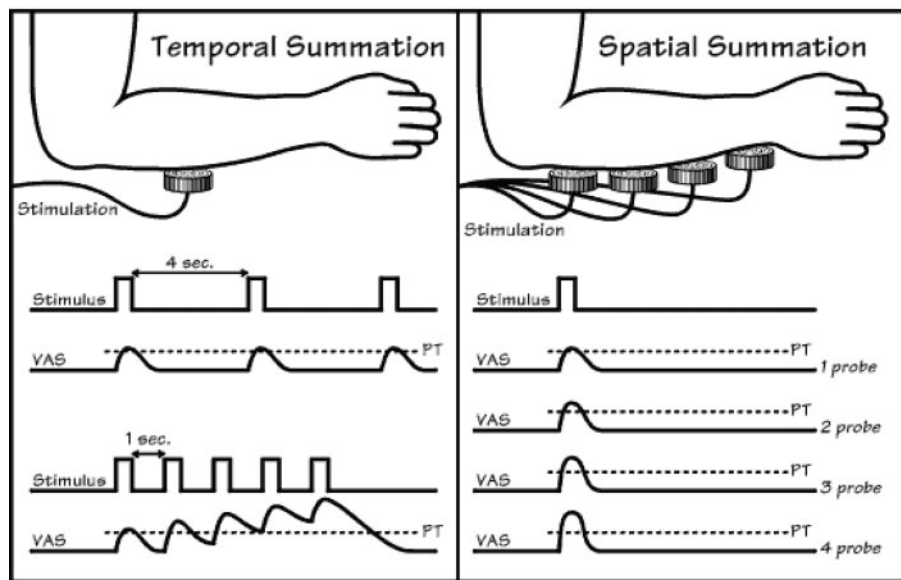


Fig. 1 Temporal and spatial summation induced by heat stimulation. For temporal summation, repetition of the stimulus every 4 s results in a pain sensation of constant intensity (VAS, visual analogue scale for pain intensity). However, repetition of the stimulus at higher frequency (every 1 s) produces a progressive increase in pain sensation as the result of central summation phenomena. For spatial summation, increasing the stimulation area by increasing the number of stimulating probes results in increased pain intensity, although the intensity of the stimulus remains constant. Reproduced with permission from Curatolo *et al. Anesthesiology* 2000; **93**: 1517–30.

This phenomenon is probably an important mechanism for the induction and maintenance of acute and chronic pain syndromes in humans.¹⁶

Temporal summation can be induced by repeated thermal¹⁷ or electrical¹³ stimulation of the skin. It has also been elicited by stimulating muscles⁶ or viscera¹⁸ in humans. Temporal summation can be measured by either psychophysical or electrophysiological responses. In the latter case, electromyographic recordings of the nociceptive reflex after repeated stimulation of the sural nerve are performed.¹³ Five electrical stimuli at a frequency of 2 Hz are applied through surface electrodes to the innervation area of the sural nerve. An increased reflex amplitude is observed during the stimulation at a current intensity that corresponds to the stimulus intensity causing a subjective increase in pain perception.¹³

Temporal summation is a very potent mechanism which is difficult to block pharmacologically and cannot be adequately blocked by epidural analgesia¹⁹ or general anaesthesia²⁰ (Table 3).

Table 3 Pharmacological modulation of temporal summation in humans

Drug	Administration	Effect
Isoflurane	Inhalation	Slight attenuation ³⁶
Ketamine	Intravenous	Attenuation ^{37,38}
Dextromethorphan	Oral	Attenuation ³⁹
Alfentanil	Intravenous	Attenuation ¹⁵
Propofol	Intravenous	No effect ¹⁵
Bupivacaine	Epidural	Attenuation ¹⁹
Bupivacaine	Intrathecal	Complete inhibition ³⁴
Lidocaine	Epidural	Attenuation ⁴⁰
Epinephrine	Epidural	No effect ⁴¹
Clonidine	Epidural	Attenuation ⁴¹

Spatial summation occurs when pain perception increases with increasing area at which the stimulus is applied, although the stimulus intensity remains constant.²¹ It is observed after application of heat, cold, pressure or pinprick.¹ The mechanisms underlying spatial summation are poorly understood. The *N*-methyl-D-aspartate (NMDA) receptor antagonist ketamine inhibits heat pain arising from a large area more effectively than it inhibits heat pain arising from a small area.²² This suggests that the NMDA receptor may be involved in spatial summation.

Imaging techniques

Functional neuroimaging has fundamentally changed our knowledge about the cerebral representation of pain. For the first time it has been possible to delineate the functional anatomy of different aspects of pain in the brain.

Positron emission tomography (PET)²³ and single-photon emission computed tomography (SPECT)²⁴ require the injection of water or glucose labelled with a radio-isotope into the blood stream flowing towards the brain. Radio-isotopes with a short half-life accumulate for a brief period of time in the active areas of brain and can be localized by scanners sensitive to the transient increase in radio-activity (Fig. 2). The temporal and spatial resolution is not as good as with functional magnetic resonance imaging (fMRI) (see below), but PET and SPECT also allow assessment of the concentration of ligands and receptors that are relevant to pain transmission in the brain.

fMRI²⁵ is based on the different magnetic properties of the different tissues in the brain. Red blood cells loaded with oxygen (oxygenated haemoglobin) present with different magnetic properties than unloaded ones (deoxygenated haemoglobin), and active areas in the brain have higher levels of oxygenated haemoglobin. This blood oxygenation level dependent signal allows the detection of active areas with good spatial and temporal resolution by magnetic field scanners.

Positron Emission Tomography (PET) and Pain

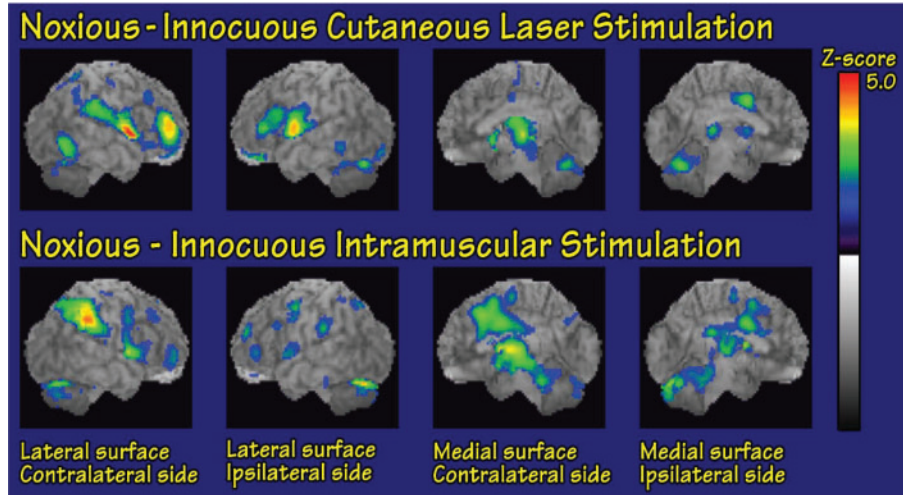


Fig. 2. PET after cutaneous laser stimulation (top) and electrical intramuscular stimulation (bottom) in healthy volunteers.⁴² The maps are superimposed on a magnetic resonance image. Note the different, although overlapping, activation of brain structures after cutaneous and intramuscular stimulation. Reproduced with permission from Svensson *et al. J Neurophysiol* 1997; **78**: 450–60.

Implications for research

The main applications of sensory testing for research are to investigate mechanisms of action of regional analgesia techniques, to study new drugs and combinations of drugs, and to compare drugs and techniques.

Given regional analgesia techniques modulate different pain pathways and mechanisms. If we assume that different stimulus paradigms activate different mechanisms or pathways, we should be able to determine which mechanisms or pathways are involved and to what degree. For instance, if the activation of mechanism/pathway A is predominantly inhibited by the drug or technique employed, the target of the specific intervention can be clarified.

While the above assumption is the framework for a mechanism-based approach of sensory testing, this concept can be implemented only partially; selectivity is still a major problem of sensory tests. Most stimulation modalities activate different nerve fibres and probably involve multiple pain mechanisms,¹ and many of these mechanisms are still poorly understood. Experimental pain studies are frequently performed on healthy volunteers. However, the nociceptive system of healthy subjects is very different from that of pain patients. Indeed, pain states are associated with plasticity changes in both the peripheral and the central nervous system which profoundly affect the processing of applied sensory stimuli.¹⁶ Even when sensory tests are performed on pain patients, the pain stimuli applied

are artificial and may not resemble clinical pain. Most of the experimental measurements are short, harmless, predictable, easily tolerated and involve little stress. Therefore they exhibit marked differences to clinical pain.

Thus it is important to employ human experimental models that can simulate pathophysiological conditions. An example may be the use of experimental stimuli after previous application of capsaicin²⁶ or induction of burn injury²⁷ (experimental hyperalgesia), inducing temporal and spatial summation,^{10,19} or stimulating muscle⁶ or viscera.¹⁸ Experimental investigations of such mechanisms in relation to regional analgesia have not yet been performed extensively, probably because of the sophisticated technology involved and their invasive nature.

Despite the above limitations, the experimental approach to the assessment of regional analgesia has important positive features which can be summarized as follows.

- 1 The same stimulus can be repeated over time. This allows the assessment of the time course of the drug action. Clinical pain is not repeatable and there is a large time variation.
- 2 The same stimulus can be applied to different body areas, thus allowing the assessment of the spread of block.
- 3 The variability associated with the type of painful stimulus is avoided. Variability is usually large in studies performed under clinical conditions.
- 4 The volunteer can be used as his or her own control and so studies can be performed with a much smaller number of participants.
- 5 Differentiated responses to the activation of different structures (skin, muscle, viscera) can be recorded (multitissue sensory testing approach).
- 6 Differentiated responses to different stimulus modalities can be assessed (multimodal sensory testing approach).
- 7 Pain sensitivity at normal and injured regions can be compared quantitatively.

Because of these characteristics, quantitative sensory tests have an important role in regional analgesia research, provided that one is fully aware of the information that can and cannot be gained by them. Experimental pain research is not an alternative to research performed under clinical conditions; rather, it complements it.

Clinical implications

Traditional clinical testing

The main aims of sensory tests for clinical purposes are to assess the spread of sensory block and to predict the effectiveness of the regional analgesia technique in individual patients.

Assessing the spread of sensory block provides information on the site of effect of the technique employed, which is important for deciding further management.¹ For instance, normal sensation of pinprick or cold after injection of a local anaesthetic clearly indicates inadequate position of the needle or catheter and warrants either supplemental block or change to general anaesthesia. Unilateral spread of block or inadvertent intrathecal injection after epidural analgesia can be detected, thereby avoiding pain during surgery or severe complications resulting from injection of high doses in the subarachnoid space, respectively.

Most of the assessment methods that have been described in this paper require expensive apparatus and substantial expertise. Moreover, there is very little scientific data and clinical experience on the value of these tests for clinical purposes. Thus, despite the major technological improvements and the substantial increase in experimental data on sensory assessments, simple methods such as pinprick, cold and touch are still used in clinical practice.

There is evidence that block of touch, pinprick or cold sensation does not necessarily imply block of nociception during regional analgesia.²⁸ The occurrence of pain after a regional block that produces complete absence of touch, pinprick or cold sensation may puzzle the anaesthesiologist. However, in view of the characteristics of these sensory tests, this phenomenon is hardly surprising. First, different nociceptors and fibres are activated by these tests and the surgical stimulus. For instance, while absence of touch sensation implies an effect of the block on A β fibres, pain is transmitted by A δ and C fibres. Moreover, although block of pinprick sensation indicates an effect on A δ fibres, this does not mean that all the functions that are mediated by these fibres are blocked. The intensity of the stimulus is a determinant of the efficacy of regional block. The simple sensory tests are weak stimuli that are easier to block than strong surgical stimuli. As mentioned above, analgesia to brief stimuli, applied to small areas, does not imply analgesia to long-lasting stimuli, applied to wide areas (temporal and spatial summation). Finally, testing the skin does not give information on drug effects on deep structures that contribute to pain in clinical conditions.

After epidural analgesia, the larger the number of dermatomes that are hyposensitive to pinprick, the lower is the incidence of pain during²⁹ and after²⁸ surgery. Therefore the spread of sensory block may be a useful clinical indicator of the efficacy of the regional block. However, the spread, as assessed by pinprick or cold stimulation, can explain only a limited part of the variability of the efficacy of epidural analgesia.^{28,29} In a study of postoperative pain, the slope of the curve describing the relationship between number of dermatomes that were hyposensitive to pinprick and relief of postoperative pain was very shallow, indicating a modest

quantitative impact of spread on pain relief.²⁸ Moreover, only 2–5% of the variability of postoperative pain intensity could be explained by the spread of sensory block in a multivariate analysis.²⁸ This means that adequate analgesia cannot be predicted based only on the determination of the spread of sensory block. Other factors, partly unknown, play an important role. For instance, the depth of the sensory block at the site of surgery, which cannot be quantified in clinical practice, may play an important role.

An important limitation of the tests used in clinical practice is that the response is usually confined to either block or no block of sensation, i.e. the response is not quantitative. The same response to pinprick stimulation (e.g. absence of sensation) is obtained at dermatomes displaying different degrees of sensory block as measured by pain threshold after laser stimulation.¹⁰ Therefore these simple qualitative tests have a low discriminative power with regard to the depth of sensory block after regional analgesia.

Assessing the effects of opioids

Epidural fentanyl injected at the lumbar level produced a higher degree of analgesia to heat at the lumbar dermatomes than at sacral or thoracic dermatomes.³⁰ This suggests that the effect of epidural fentanyl on the peripheral nerves or dorsal root ganglions may be more important than is usually believed. In fact, if penetration through the meninges and action at the spinal cord receptors were the main mechanisms explaining regional analgesia after epidural fentanyl, the depth of analgesia at the sacral dermatomes would be at least equally strong. Conversely, lumbar epidural morphine attenuates heat pain up the trigeminal dermatomes, reflecting rostral spread of this hydrophilic drug after penetration into the subarachnoid space.³¹

It is clinically relevant that opioids frequently have no effect on pinprick or cold sensation.³² Thus opioid-based epidural analgesia cannot be assessed reliably by these tests.

The study by Eichenberger and colleagues,³⁰ mentioned above, also employed an intramuscular pain model (injection of hypertonic saline) which proved sensitive for detecting the analgesic action of epidural fentanyl. A study of intravenous remifentanyl found a much deeper analgesic effect of this opioid on intramuscular than on cutaneous electrical pain.³³ This suggests that opioids may have a stronger effect on deep pain than on skin pain in clinical conditions, and this difference could also apply to epidural opioids. Translated to surgical pain, one can assume that pain on surgical incision does not necessarily imply pain during surgical stimulation of deep tissues.

Differences between different anaesthetic techniques

Do different regional anaesthetic techniques work to the same extent? To our knowledge, there is no direct comparison of different techniques in terms of depth of sensory block, as assessed experimentally in a dose-response manner. We performed two different studies analysing the effects of 20 ml bupivacaine 0.5% epidurally¹⁹ and 18 mg bupivacaine intrathecally³⁴ on repeated electrical stimulation. The median pain threshold in the epidural study increased from 6 mA at baseline to 23 mA at 60 min. In the intrathecal study, the median pain threshold increased from 4 mA at baseline to 60 mA, which was the maximum current intensity allowed, at 40 min. Despite the limitations regarding the comparison between two different studies and the use of single doses, the data suggest that the sensory block is stronger with spinal than with epidural analgesia, an impression that is shared by most anaesthesiologists. It is noteworthy that the measurements were made at the dermatome S1, which is thought to be particularly resistant to epidural blockade.

Importance of temporal and spatial summation

An important concept in regional analgesia is that the duration of the applied stimulus (temporal summation) and the area where the stimulus is applied (spatial summation) are determinants of the effects of regional block.^{10,19} Analgesia to a brief stimulus is not necessarily a guarantee of analgesia to a long-lasting stimulus. This finding is clinically relevant, since clinical pain is mostly ongoing rather than brief in duration. The different effect on the two stimulation modalities is probably due to the fact that brief stimuli, although not perceived as painful, are not completely blocked by epidural local anaesthetics and arrive at the spinal cord. At this level, they sum and eventually evoke pain if they are applied for a longer time. In contrast, spinal anaesthesia with 18 mg bupivacaine inhibited pain after both single and repeated electrical stimulation, indicating complete inhibition of temporal summation.³⁴ This may be the result of a strong block of the sensory input, which is therefore unable to sum in the spinal cord to evoke a pain sensation.

Spatial summation after epidural¹⁰ and intrathecal³⁵ administration of bupivacaine 0.5% was studied by comparing the response to stimulation using one needle with the response to stimulation using 10 needles applied simultaneously. In both studies, pain was evoked with 10 needles in patients in whom stimulation with one needle did not evoke pain. Therefore a stimulus applied to a small area, although not evoking a pain sensation, may not be completely blocked and will arrive at the spinal cord. When the same stimulus is applied to a wider area, it can spatially

sum in the spinal cord and be perceived as painful. This finding also has clinical relevance, since clinical pain is rarely confined to very small areas. It is noteworthy that many sensory tests used for clinical and experimental purposes are of brief duration and are applied to small areas. This probably contributes to the discrepancy between sensory tests and clinical pain.

Conclusions

Sensory tests have an important role in regional anaesthesia and pain research. They have substantially increased our understanding of the laws regulating the action of drugs and techniques that are used daily in clinical practice. Studies conducted using experimental pain cannot be expected to provide the same information as clinical trials. They have limitations, mostly due to their artificial nature and the unidimensional assessment procedure. However, experimental tests can provide information that cannot be gained in clinical trials and can reduce the gap between animal research and studies conducted under clinical conditions. Thus sensory assessment is not an alternative to clinical pain assessment. Rather, the two approaches complement each other. Tests that more closely mimic clinical pain are available, but are still underused.

In clinical practice, sensory tests provide useful information on the efficacy of regional analgesia, but have limited validity. Unfortunately, almost no progress on assessment of regional analgesia for clinical purposes has been made in the last decade, largely because of the paucity of published research.

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