



## Review

# Epidural Analgesia in the Dog and Cat

R.S. JONES

University Department of Anaesthesia, University Clinical Department, The University of Liverpool, The Duncan Building, Daulby Street, Liverpool L69 3GA, UK

### SUMMARY

A brief outline of the history of epidural analgesia is followed by a review of the anatomy of the epidural space with particular reference to epidural block. The technique of epidural injection in the dog is described as are the indications for the technique. These include the provision of anaesthesia for such procedures as orthopaedic surgery of the hind limb and caesarian section. The cardiovascular effects of epidural block are discussed and suggestions are made for the prevention of hypotension. The various drugs and their combinations which may be used for epidural administration are outlined. The commonest used local anaesthetic agents are bupivacaine and lidocaine. Epidural administration of opioid drugs is a relatively new technique which is used to provide intra- and post-operative analgesia. Morphine is the drug of choice for this indication.

The use of other classes of drugs, such as the alpha 2 agonists and ketamine, are also considered. A variety of side-effects, contra-indications and complications are described together with methods for reducing their incidence and effects.

© 2001 Harcourt Publishers Ltd

KEYWORDS: Epidural; analgesia; regional; dog; cat.

### HISTORY

Epidural analgesia was first administered to experimental dogs in 1885 (Corning, 1885). Some years later, Bier (1899) described the use of the technique of epidural anaesthesia on himself and in the dog. However, it was not until the classic work of Brook (1935) that the technique was investigated and evaluated in domestic animals, including the dog, in this country. Some years later, the technique was advocated for clinical use in the dog (Joshua, 1956; Spreull, 1958) and, experimentally, it was shown that the technique is effective in cats (Duce *et al.*, 1969). An excellent review by Bromage (1967) described the mechanisms of action of the technique, with particular reference to the human

subject. More recently, there has been a renewed interest in the technique with particular reference to the use of newer local anaesthetic solutions (Heath *et al.*, 1989) and the use of opioids in the epidural space to provide analgesia (Valverde *et al.*, 1989).

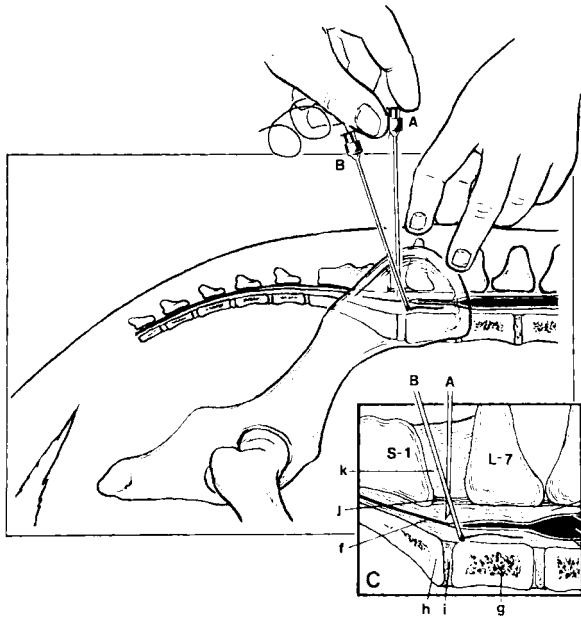
### ANATOMY

The anatomy of the epidural space in the lumbosacral region is described in standard textbooks of Veterinary Anaesthesia (Hall & Clarke 1991) and Anatomy (Miller *et al.*, 1964) (see Fig 1).

#### *Meninges*

The meninges are the fibrous membranes which surround and protect the spinal cord and the brain. They are composed of three membranes:

Correspondence to: Professor R. S. Jones University of Anaesthesia, University Clinical Department The University of Liverpool, The Duncan Building, Daulby Street, Liverpool L 69 3GA, UK, Fax: +44 151 706 5884; E-mail: [rsj@liv.ac.uk](mailto:rsj@liv.ac.uk)



**Fig. 1.** Needle placement in the lumbo-sacral space and catheter placement for continuous epidural analgesia. Inset (a) epidural space containing fat and connective tissue (b) *dura mater* (c) arachnoid membrane (d) spinal cord (e) cerebrospinal fluid (f) *cauda equina* (g) 7<sup>th</sup> lumbar vertebra (L7) (h) 1<sup>st</sup> sacral vertebra (S1) (i) intervertebral disc (j) interarcuate ligament (*ligamentum flavium*) (h) interspinous ligament.

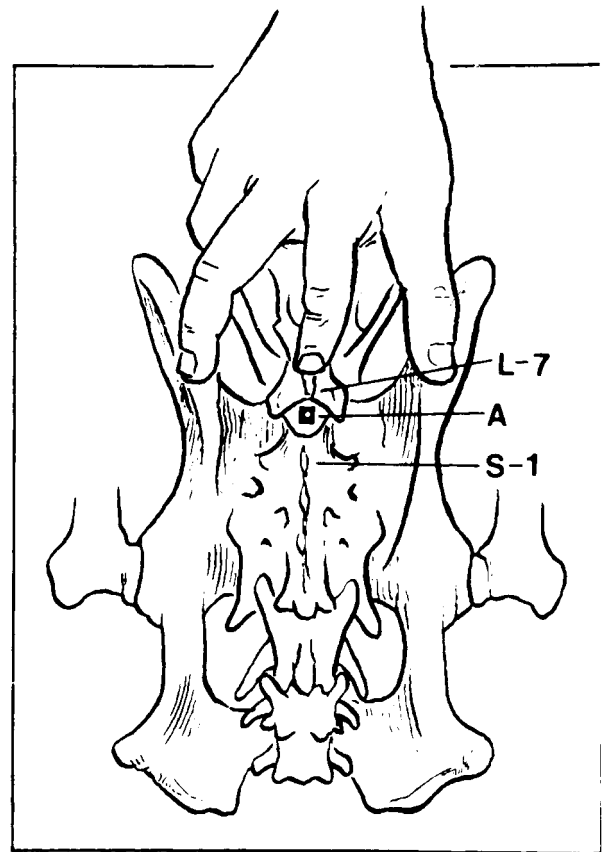
the *dura mater*, the arachnoid, and the *pia mater*. The *dura mater* is sometimes referred to as the pachymeninx, because of its tough, fibrous nature. The combined arachnoid and *pia mater* is called the leptomeninx because of its thinness.

#### *Spinal meninges*

The spinal *dura mater* consists of only one layer, the meningeal layer. It is separated from the periosteum of the vertebrae by the epidural cavity which is filled by a semifluid fat (at body temperature) and by the vertebral venous sinuses. The spinal *dura mater* is continuous with the meningeal layer of the cranial *dura mater* at the *foramen magnum*.

The spinal *dura mater* is in the form of a long tube surrounding the spinal cord. It has lateral tubular extensions which cover the spinal nerve roots and accompany them to the intervertebral foramina. As the dorsal and ventral roots join to form the spinal nerve, the *dura mater* blends to form a single sheath which continues as the epineurium of the spinal nerve.

The capillary space between the *dura mater* and the arachnoid is the subdural cavity, which contains



**Fig. 2.** Dorsal view showing palpation of the lumbar spinous process (L7) and the external angles of the ilia.

a small amount of fluid.

Caudally, the spinal *dura mater* tapers to a point and forms a part of the *filum terminale* (*filum durae matris spinalis*). The *dura* surrounds the *filum terminale* of the spinal *pia mater*, which fuses to it, and then extends caudally to attach to the periosteum of the spinal canal at the seventh or eighth coccygeal vertebra. It serves to attach the dural sac and spinal cord caudally.

#### *Spinal arachnoid* (*arachnoidea spinalis*)

The spinal arachnoid is a thin, almost transparent tube which envelops the spinal cord and has, like the spinal *dura mater*, tubular extensions surrounding the dorsal and ventral spinal nerve roots.

The subarachnoid cavity is the space between the spinal *pia mater* and the arachnoid membrane. It is filled with cerebrospinal fluid which pushes the arachnoid peripherally and holds it in contact with the spinal *dura mater*.

The lumbar cistern of the spinal subarachnoid

cavity envelopes the spinal nerves of the *cauda equina*. The cistern is narrow at the level of the lumbosacral foramen, gradually tapers to a point, and ends at the level of the first sacral vertebrae.

The spinal *pia mater* is a tough, highly vascularized membrane that intimately adheres to the spinal cord and roots of the spinal nerves, forming parts of the epineurial sheaths.

The phenomena which accompany paralysis of the spinal nerves are more complex than with peripheral nerves. This is due to the varying types of nerves which make up the spinal nerve. Sensory fibres are paralysed more readily and rapidly than motor fibres and the sympathetic fibres are even more susceptible. Spinal nerves result from the union of two roots—a dorsal, ganglionic or sensory root and a ventral motor root. In the dog, union occurs within the foramina except in the lumbar and coccygeal regions where it takes place within the vertebral canal.

## TECHNIQUES

The technique of epidural injection in the cat and dog is almost invariably carried out at the lumbosacral space although, in larger dogs, it may occasionally be performed at the sacro-coccygeal space.

Whilst the procedure is sometimes performed under general anaesthesia in the dog, it is usually considered to be mandatory in the cat. Obviously, if the technique is being used to produce analgesia, it is more likely to be performed under general anaesthesia in both species. However, under some circumstances in the dog, epidural injection may well be used to provide anaesthesia for procedures such as orthopaedic operations in the hind-limb or caesarean section. In this situation, it is desirable, much more humane and convenient to sedate the dog heavily. The drugs of choice for sedation are a combination of acepromazine and morphine although other combinations have been used. In order to prevent an overdose of morphine when it is administered by the epidural route, it is advisable to ensure that, if morphine is also used for premedication, the total dose is not excessive. Hence, it may be preferable to use another opioid drug such as pethidine or an alpha 2 agonist such as medetomidine, for premedication.

One important aspect of epidural block which is sometimes neglected is the prevention and/or treatment of the associated hypotension produced by the sympathetic blockade which occurs as a

result of the epidural injection of local anaesthetic agents. Animals undergoing caesarean section would appear to be particularly susceptible to this problem. This subject has been reviewed in the dog by Nolte *et al.* (1983) and has also been investigated in the human subject (Hallworth *et al.*, 1982; Lewis *et al.*, 1983). It is recommended that up to 20 mL/kg of Hartmann's solution is administered to both the dog and cat as a vascular preload. In practice, this means as soon as it is possible to place a catheter in the cephalic vein, fluid administration should be commenced. The solution can be administered whilst the site is being prepared and the epidural block is being performed.

In order to carry out the procedure, the animals are placed either on their sides in right or left lateral recumbency or positioned on their sterna. If in lateral recumbency, the hind limbs are pulled forward or, if in sternal recumbency, the hind limbs should be 'tucked' under the animal. This ensures that there is a maximum gap between the last lumbar vertebra and the sacrum. The site is located by using the external angles ('wings') of ilia and the dorsal spinal processes of the seventh lumbar vertebra and the sacrum as anatomical landmarks (see Figs 1 & 2). For preference, the animals are placed in sternal recumbency which makes it easier to be absolutely certain that the needle is inserted exactly in the mid-line. The external angles of the ilia are palpated with the thumb and middle finger of one hand and the index finger is directed caudally. The lumbosacral space is located by palpation of the depression immediately caudal to the dorsal spinous process of the seventh lumbar vertebra. The spinal needle is inserted slowly at an angle of 90° to the animal's skin and care must be taken to ensure that it is in the mid-line. A skin weal of local anaesthetic solution should be used in the conscious dog but care must be taken not to distort the tissues by over enthusiastic use of local anaesthetic. The choice of the size of needle varies with the size of the animal. A 2.5 cm 22 Gauge spinal needle is recommended for cats and small dogs, a 3.8 cm 20 Gauge needle for medium sized dogs and a 7.5 cm 18 Gauge needle for large dogs. As the needle is advanced and pierces the interarcuate ligament, a distinct 'popping' sensation is felt in the fingers. If this is not felt and the needle strikes bone, then it should be withdrawn and redirected as appropriate.

The best method of ensuring that the needle is in the epidural space is to use the 'loss of resistance test'. Up to 2 mL of air or saline may be injected

and the absence of resistance confirmed. In the conscious dog, it is not unusual to witness movement of the tail as the needle comes into contact with nervous tissue. Once the needle is considered to be in the epidural space, it should be carefully examined for the presence of clear cerebro-spinal fluid (CSF) or blood before injection is made. The presence of CSF indicates that subarachnoid puncture has occurred. A number of different actions may then be taken. The technique of epidural block may be abandoned altogether. Alternatively, the needle may be removed and an attempt made to repeat the technique and the dose of local anaesthetic may be reduced by 50% (Skarda, 1996). If blood is observed issuing from the needle, then it should be removed and the procedure repeated as it is important that local anaesthetic solutions are not injected intravenously since this may precipitate signs of acute toxicity which include convulsions and/or cardiopulmonary depression and even cardiac arrest. Regional (epidural) anaesthesia will not be produced.

The injection of solution into the epidural space should be carried out over a period of some 30 to 60 s and solutions should be at body temperature.

### **SOLUTIONS FOR EPIDURAL ADMINISTRATION**

The various drugs, their properties and the effects which they are likely to produce when administered epidurally have been reviewed by Pascoe (1997). The expectation, when a drug is injected epidurally, is that it will have a localized and more intense effect than when it is given systemically. Local anaesthetics affect conduction in nervous tissue and this effect is usually related to the volume and concentration of the drug. However, the vast majority of other drugs which are injected into the epidural space act at specific receptors and their effect will depend on the density of the receptor population and the cell types in which the receptors are located. Following the epidural administration of any drug, it must diffuse into the neuronal tissue to exert its effect. It may also 'leak' through the foramina in the vertebral canal, may get taken up into fat or be removed by the blood. It may also diffuse into nerve roots beyond the meningeal sleeve or through the dural cuffs at the dorsal roots or directly through the meninges to the CSF and spinal cord. It has been demonstrated, by Bernard and Hill (1992), that there is an optimal

range of solubility for the meningeal penetration of chemical agents. The *dura* itself appears to be fairly permeable and there is no difference between morphine and alfentanil which are compounds with very different lipid solubilities. The main barrier appears to be the *pia* arachnoid with a complex mixture of water (extracellular fluid and CSF) and lipid (cell membranes). If a drug is hydrophilic, it will only pass slowly across the meninges due to the lipid. Conversely, if it is highly lipophilic, the passage will be delayed by water. The most rapid diffusion appears to occur with molecules which have an octanol:buffer distribution coefficient between 129 (alfentanil) and 560 (bupivacaine).

#### *Local anaesthetic solutions*

The site of action of local anaesthetic agents, administered by the epidural route, is still somewhat controversial. The main sites are considered to be the intradural spinal nerve roots and the periphery of the spinal cord. The various factors involved in the production of the block have been discussed in an excellent review by Bromage (1967). The final effect of a local anaesthetic is related not only to its lipid solubility but also to physico-chemical properties i.e. the pKa, the pH of the solution and of the tissues and the protein binding capacity of the drug. The pKas of amide local anaesthetics are similar so that the cationic form slightly predominates at physiological pH but it is the base form which is considered to be responsible for penetration of the lipid membrane. However, it is the ionic form which is thought to be responsible for penetration of the lipid membrane. The permeability of lidocaine (lignocaine) and bupivacaine is similar, despite the differences in their lipid solubility. The onset of action was similar for the two drugs: 3–4 min to the onset of motor block (Lebeaux, 1973). Clinically, it would appear that the time to peak effect with bupivacaine is much slower (Heath *et al.*, 1985). The effectiveness of the block was also affected by the concentration of the drug. A slightly higher success rate was achieved with 0.75% as opposed to that with 0.5% solutions.

The duration of block appears to be related to the protein binding capacity of the drug. Agents, such as lidocaine and mepivacaine which are less highly protein bound (65–75%), have a duration of action of 1.5 to 4 h, whereas bupivacaine and ropivacaine are highly protein bound (99%) and have a prolonged duration of action of 3–6 h (Feldman

*et al.*, 1996). The duration of block can be affected by the addition of vasoconstrictor drugs such as epinephrine (adrenaline). It appears to prolong the duration of block with lidocaine and mepivacaine but not with bupivacaine or ropivacaine. Epinephrine decreases the vascular uptake of the drugs and, hence, it reduces the likelihood of systemic toxicity. There has recently been a renewed interest in attempting to use local anaesthetic drugs to produce a sensory block without interfering with motor function. This has received considerable attention in man and it is achieved with dilute solutions of the agents. A continuous infusion of the agent may be used to provide analgesia and the person is still able to walk. Bupivacaine at a concentration of 0.125% and ropivacaine at 0.1% appear to be the most widely used agents (Zaric *et al.*, 1996).

A number of local anaesthetic agents, of different concentrations and doses have been used to produce epidural anaesthesia in the dog and cat and have been discussed by Skarda (1996). The selection depends mainly on the weight of the animal, the extent of anaesthesia required and the onset time and duration of effect. A dose of 1 mL of 2% lidocaine per 4.5 kg is recommended to produce anaesthesia of the body caudal to the first lumbar vertebra and will be effective some 10–15 min after injection. Bupivacaine at a concentration of 0.75% has a latent period of 20–30 min (Heath *et al.*, 1985). Anaesthesia for abdominal and orthopaedic surgery caudal to the diaphragm is normally obtained with 1 mL/5 kg of 2% lidocaine with 1 in 200 000 epinephrine or 0.5% bupivacaine.

A reduced volume of 2% lidocaine at 1 mL/6 kg is normally effective for caesarean section. There are various reasons suggested for this apparent sensitivity in pregnancy (Butterworth *et al.*, 1990). Different agents appear to have a different duration of action. Two per cent solutions of lidocaine, procaine and carbocaine have produced satisfactory anaesthesia for 60–120 min, whereas bupivacaine and etidocaine have a duration of 4–6 h. In an experimental comparison of bupivacaine and ropivacaine at a variety of concentrations, with or without epinephrine, a duration ranging from 103 min (0.75% ropivacaine) to 163 min (0.75% bupivacaine) was observed (Feldman & Covino, 1988).

The position of the animal following injection is important. If a unilateral effect is required, the animal should remain on that side until anaesthesia is effective. For bilateral effects, the animal should be placed in dorsal recumbency. In the conscious dog, loss of anal sphincter tone will indicate the onset of

anaesthesia.

#### *Opioids*

The use of epidural opioids to produce analgesia has been reviewed by Cousins and Mather (1984) and Morgan (1989). In order to produce an effect following epidural injection, opioids must diffuse through the *dura* and pass into the dorsal horn. They are thought to work on pre-synaptic sites by preventing the release of substance P and on post-synaptic receptors to hyperpolarize the cells. Hence, they obtund nociception without any significant effect on motor function. The potency of the different opioids, when they are administered intrathecally, is not directly related to systemic potency but is a function of lipid solubility.

#### *Morphine*

This is the most useful of the opioid class of drugs, when administered epidurally, due to its high potency and long duration of action. In dogs, epidural morphine at a dose of 0.1 mg/kg has an onset time of 20–60 min and a duration of action of 16–24 h (Bonath & Saleh, 1985). A dose of 0.1 mg/kg of the preservative-free material is recommended in both species. It would appear to have similar effects in the cat (Tung & Yaksh, 1982). A recent study by Yaksh *et al.* (1999) has demonstrated that a sustained release encapsulated form of morphine had a duration of action of 62 h when administered to dogs by the epidural route. This would appear to have considerable potential for the provision of long-term analgesia. Only about 0.3% of epidural morphine is thought to cross the meninges (Durant & Yaksh, 1986).

Epidural morphine has been demonstrated to provide a significant reduction in the amount of halothane needed to produce and maintain general anaesthesia in the dog (i.e. a reduction in minimum alveolar concentration) (Valverde *et al.*, 1989).

#### *Pethidine*

This agent has local anaesthetic properties in addition to its opioid activity. In cats, it has been shown that it has a rapid onset of action and a dose related duration of action from 1 h to 4 h (Tung & Yaksh, 1982). Its epidural potency is considered to be 1/35th that of morphine.

#### *Methadone*

In cats, a dose of 0.7–1 mg/kg has a rapid onset of

action but a duration of only around 4 h. Information on its use in the dog is sparse.

#### *Oxymorphone*

This agent has been used more extensively in North America but information on its epidural use is relatively sparse. A dose of 0.1 mg/kg has a duration of 10 h in the dog (Popilskis *et al.*, 1991).

#### *Fentanyl*

There is considerable debate as to the role of epidurally administered fentanyl. Its high lipid solubility reduces its meningeal permeability and its CSF potency is low. It would appear that administration of fentanyl by the intravenous route produces a similar effect (Loper *et al.*, 1990). Its only use appears to be as an adjunct to other epidurally administered drugs due to its rapid onset of action (Fischer *et al.*, 1988).

#### *Butorphanol*

Information from published studies on this agent suggests that there is no advantage to be gained by administering it epidurally compared with the intravenous route (Camann *et al.*, 1992). In dogs, an epidural dose of 0.25 mg/kg has been shown to reduce the minimum alveolar concentration (MAC) of isoflurane by 31% and to have a duration of action of about 3 h (Troncy *et al.*, 1996).

#### *Buprenorphine*

This has a slow onset of action of about 60 min. The analgesic effects appear to be similar to those of morphine with a potency ratio of 8:1 which gives a dose of 12.5 µg/kg equivalent to 0.1 mg/kg of morphine (Chrubasik *et al.*, 1987).

#### *Alpha 2 adrenoreceptor agonists*

These drugs interact with the adrenergic system in the spinal cord to inhibit central transmission on nociceptive information and this effect does not appear to be related to the vasoconstrictive effect of the drugs. It is usual to observe sedative effects after epidural administration due to the systemic uptake of the drugs.

#### *Xylazine*

This agent has been used in a variety of species and can produce profound analgesia. The majority of information is available from large animals and the onset appears to occur within 30 min

and have a duration of up to 3 h. No published information appears to be available for the dog and cat.

#### *Medetomidine*

This is the most potent of the currently available alpha 2 agonists. In experimental dogs, the ED50 for epidural medetomidine to a heat stimulus was 10 Hz/kg (Sabbe *et al.*, 1994). A dose of 15 µg/kg produces postoperative analgesia for 4–8 h (Vesal *et al.*, 1966). In cats, a dose of 10 µg/kg raised the hind limb pain threshold for a period of 20–245 min after injection. Most of the cats vomited after the medetomidine and all appeared to be mildly sedated, which was due to the central effects of the drug following absorption.

#### *NMDA antagonists*

There appear to be conflicting results on the use of epidurally administered ketamine. Since its mode of action is due mainly to an effect on 'wind-up' or central hyperalgesia, it is likely that it will be most useful as part of a regime rather than as a sole agent. Whilst it has been suggested that ketamine may be neurotoxic, it may be due to the preservative. A study using preservative free ketamine failed to demonstrate toxicity (Borgbjerg *et al.*, 1994). It has recently been reported that a dose of 0.4 mg/kg of ketamine administered by the epidural route is an effective analgesic in the dog for a period of up to 90 min (Rao *et al.*, 1999).

#### *Combination of drugs*

It is logical to suggest that combinations of the various drugs will enhance analgesia. Synergism between a variety of compounds has been demonstrated. Synergism between local anaesthetics and opioids has been demonstrated in the dog (Wang *et al.*, 1993) and between alpha 2 agonists and opioids in the dog (Branson *et al.*, 1993). Various other combinations have been shown to be effective in other species.

#### *Preservatives*

There are very few drugs, apart from morphine which are marketed specifically for epidural use. The neurotoxicity of these preservatives has not been extensively tested. Sodium sulphide, benzethonium chloride, chlorbutanol and disodium EDTA have caused concern (Olek & Edwards, 1980; Ford & Raj, 1987; Wang *et al.*, 1992; Yaksh, 1996).

### *Contra-indications*

There are a number of contra-indications to the production of epidural anaesthesia/analgesia in the dog and cat. Infectious skin disease, particularly that which involves sepsis, in the region of the lumbosacral area, is an absolute contra-indication. Uncorrected hypovolaemia, in such situations that follow road traffic accidents or any other cause of haemorrhage, is also an absolute contra-indication. Bleeding disorders, which may be either therapeutic or physiological, form another class of contra-indication as does degenerative central or peripheral axonal disease. Anatomical abnormalities which may be congenital or which arise as a result of trauma and make access to the lumbosacral space either extremely difficult or impossible are also absolute contra-indications. Relative contra-indications include such conditions as bacteraemia, some neurological disorders and low dose heparin therapy.

### *Complications and side-effects*

A variety of complications may arise from the use of epidural analgesia in the dog and cat. The first one to be considered is technical failure. It may not always be possible to locate the lumbo-sacral space and/or insert a needle into the epidural space. This appears to be more common in fat animals where the location of the landmarks may be more difficult. In a series of some 636 dogs, it was reported that analgesia was absent in some 12% of animals in which the technique was attempted (Heath *et al.*, 1989). It is, however, important to ensure that sedation is adequate and its lack is not mistaken for inadequate anaesthesia. In this published report, no reference was made to the experience of the individual carrying out the procedure.

If CSF is obtained when the needle is inserted into the spinal canal, then the needle should be withdrawn and the procedure repeated. It is suggested that a subarachnoid injection can be made but only 50% of the calculated dose of local anaesthetic solution is used (Skarda, 1996). If blood is obtained following insertion of the needle, then the needle should be discarded, another one selected and a further attempt made to insert it into the epidural space. Intramuscular injection of local anaesthetic solution may produce signs of toxicity, such as convulsions and/or cardiopulmonary collapse. A regional block will not be induced.

It has been reported that large defects in the *dura*, produced by the insertion of a spinal needle, may result in a headache in humans, possibly due to

an increased leakage of CSF. A Whitacre needle is likely to produce a smaller defect for a given size of needle and a bevel orientation parallel to the *dura* fibres produces a smaller defect than when it is perpendicular. Cardiovascular function respiratory rate and blood gases are not affected by epidural anaesthesia which entered as far forward as the cranial thoracic dermatomes (Nolte *et al.*, 1983).

Unsedated healthy dogs compensate for reduced spinal sympathetic outflow by increased vasopressor secretion (Stanek *et al.*, 1980). This mechanism may be suppressed in aged and sick dogs. Hypotension should be prevented by pre-loading with a crystalloid solution but treatment may also be necessary with a crystalloid and/or a vasopressor drug (Butterworth *et al.*, 1986).

Neurological complications which may occur after the induction of epidural anaesthesia include Horner's syndrome, Shiff Sherrington-like reflexes and signs associated with local anaesthetic toxicity, such as muscle twitch, coma and convulsions.

Urinary retention has been described after epidural anaesthesia and, if large volumes of fluid are administered, this may require intervention, either by manually squeezing the bladder through the abdominal wall or by catheterization. Animals with full bladders are likely to show considerable discomfort, hence, it is essential that careful attention is paid to the state of the bladder.

Relatively few side-effects have been observed following the administration of epidural morphine. Pruritus was reported in four animals in a series of 250 (Valverde *et al.*, 1989). There is a lot of anecdotal evidence which suggests that hair growth is slow over the site of lumbo-sacral injection when the technique is used in dogs. However, extensive experience would suggest that there is no real scientific evidence to support this.

## REFERENCES

- BERNARD, C. M. & HILL, H. F. (1992). Physical and chemical properties of drug molecules governing their diffusion through spinal meninges. *Anesthesiology* **77**, 750-756.
- BIER, A. (1899). Versuche uber cocainisierung des ruckenmarkes. *Deutsche Zeitschrift fur Chirurgie* **51**, 361-369.
- BONATH, K. H. & SALEH, A. S. (1985). Long term pain treatment in the dog by peridural morphines. *Proceedings of the 2nd International Congress of Veterinary Anesthesia*. pp. 7-10, October, Sacramento, California.

- BORGBJERG, F. M., BJORN, A. S., SVENSSON, A., FRIGAST, C. & GORDH, T. (1994). Histopathology after repeated intrathecal injection of preservative-free ketamine in the rabbit: a light and electron microscopic examination. *Anesthesia and Analgesia* **79**, 105–111.
- BRANSON, K. R., KO., J. C., TRANQUILLI, W. J., BENSON, J. & THURMAN, J. C. (1993). Duration of analgesia induced by epidurally administered morphine and medetomidine in dogs. *Journal of Veterinary Pharmacology and Therapeutics* **16**, 369–372.
- BROMAGE, P. R. (1967). Physiology and pharmacology of epidural analgesia. *Anesthesiology* **28**, 592–622.
- BROOK, G. B. (1935). Spinal (epidural) anaesthesia in the domestic animals. *Veterinary Record* **15**, 659–667.
- BUTTERWORTH, J. F., PICCIONE, W., BERRIZBEITIA, L. D., DANCE, G., SHEMIN, R. J. & COHN, L. H. (1986). Augmentation of venous return by adrenergic agonists during spinal anaesthesia. *Anesthesia and Analgesia* **65**, 612–616.
- BUTTERWORTH, J. F., WALKER, F. O. & LYSAK, S. Z. (1990). Pregnancy increases median nerve sensitivity to lidocaine. *Anesthesiology* **72**, 962–965.
- CAMANN, W. R., LOFERSKI, B. L., FANCIULLO, G. J., STONE, M. L. & DATTA, S. (1992). Does epidural administration of butorphanol offer any clinical advantage over the intravenous route? *Anesthesiology* **76**, 216–220.
- CHRUBASIK, J., VOGEL, W., TROTSCHLER, H. & FARTHMAN, E. H. (1987). Continuous plus on demand epidural infusion of buprenorphine versus morphine in postoperative treatment of pain. Postoperative epidural infusion of buprenorphine. *Arzreimittelforschung* **37**, 361–363.
- CORNING, J. L. (1885). Spinal anaesthesia and local medication of the spinal cord. *New York Medical Journal* October 31st, 483–485.
- COUSINS, M. J. & MATHER, L. E. (1984). Intrathecal and epidural administration of opioids. *Anesthesiology* **61**, 276–310.
- DUCE, B. R., ZELECHOWSKI, K., CAMOUGIS, G. & SMITH, E. R. (1969). Experimental epidural anaesthesia in the cat with lignocaine and amethocaine. *British Journal of Anaesthesia* **41**, 579–587.
- DURANT, P. A. C. & YAKSH, T. L. (1986). Distribution in cerebrospinal fluid, blood and lymph of epidurally injected morphine and insulin in dogs. *Anesthesia and Analgesia* **65**, 583–592.
- FELDMAN, H. S. & COVINO, B. (1988). Comparative motor blocking effects of bupivacaine and ropivacaine, a new amino amide local anaesthetic in the rat and dog. *Anesthesia and Analgesia* **67**, 1047–1052.
- FELDMAN, H. S., DVOSKIN, S., ARTHUR, G. R. & DOUCETTE, A.M. (1996). Antinociceptive and motor-blocking efficacy of ropivacaine and bupivacaine after epidural administration in the dog. *Regional Anaesthesia* **21**, 318–326.
- FISCHER, R., LUBENOW, T. R., LECEAGA, A., MCCARTHY, R. J. & IVANOVICH, A. D. (1988). Comparison of continuous epidural infusion of fentanyl-bupivacaine in management of postoperative pain. *Anesthesia and Analgesia* **67**, 559–563.
- FORD, D. & RAJ, P. (1987). Peripheral neurotoxicity of 2-chloroprocaine and bisulfite in the cat. *Anesthesia and Analgesia* **66**, 719–722.
- HALL, L. W. & CLARKE, K. W. (1991). *Veterinary Anaesthesia* 9th edn ch. 10, pp. 183–187. London: Bailliere Tindall.
- HALLWORTH, D., JELLCOE, J. A. & WILKES, R. G. (1982). Hypotension during epidural anaesthesia for Caesarean section. *Anaesthesia* **37**, 53–56.
- HEATH, R. B., BROADSTONE, R. V., WRIGHT, M. & GRANDY, J. L. (1985). Bupivacaine and mepivacaine lumbosacral analgesia in dogs. *Proceedings of the 2nd International Congress of Veterinary Anaesthesia*. 7–10 October, Sacramento, California, pp. 162–163.
- HEATH, R. B., BROADSTONE, R. V., WRIGHT, M. & GRANDY, J. L. (1989). Using bupivacaine hydrochloride for lumbosacral epidural analgesia. *Compendium of Continuing Education for the Practising Veterinarian* **11**, 50–55.
- JOSHUA, J. O. (1956). Epidural anaesthesia. *Veterinary Record* **68**, 801–803.
- LEBEAUX, M. I. (1973). Experimental epidural anaesthesia in the dog with lignocaine and bupivacaine. *British Journal of Anaesthesia* **45**, 549–555.
- LEWIS, M., THOMAS, P. & WILKES, R. G. (1983). Hypotension during epidural analgesia for Caesarean section. *Anaesthesia*, **38**, 250–253.
- LOPER, K. A., READY, L. B., DOWNEY, M., SANDLER, A. N., NESSLY, M., RAPP, S. & BADNER, N. (1990). Epidural and intravenous fentanyl infusion are clinically equivalent after knee surgery. *Anesthesia and Analgesia* **70**, 72–75.
- MILLER, M. E., CHRISTENSEN, G. C. & EVANS, H. E. (1964). *Anatomy of the dog*, pp. 539–542. London: W. B. Saunders Company Ltd.
- MORGAN, M. (1989). The rational use of intrathecal and extradural opioids. *British Journal of Anaesthesia* **63**, 165–168.
- NOLTE, J. G., WATNEY, G. C. C. & HALL, L. W. (1983). Cardiovascular effects of epidural blocks in dogs. *Journal of Small Animal Practice* **24**, 17–21.
- OLEK, A. & EDWARDS, C. (1980). Effects of anesthetic treatment on motor neuron death in xenopus. *Brain Research* **191**, 483–488.
- PAPILSKIS, S., KOHN, D., SANCHEZ, J. A. & GORMAN, P. (1991). Epidural vs intramuscular oxymorphone analgesia after thoracotomy in dogs. *Veterinary Surgery* **20**, 462–467.
- PASCOE, P. J. (1997). Drugs in the epidural space. *Proceedings of the 6th International Congress of Veterinary Anaesthesiology*. September, Halkdiki, Greece, pp. 53–61.
- RAO, K. N. M., RAO, K. V., MAKKERA, S. & NAIDU, K. S. (1999). Ketamine as epidural anaesthetic in dogs. *Indian Veterinary Journal* **76**, 61–62.
- SABBE, M. B., PENNING, J. P., OZAKI, G. T. & YAKSH, T. L. (1994). Spinal and systemic action of the alpha 2 receptor agonist dexmedetomidine in dogs. Antinociception and carbon dioxide response. *Anesthesiology* **80**, 1057–1072.
- SKARDA, R. (1996). Local and regional anesthetic and analgesic techniques: dogs. *Lumb and Jones' Veterinary Anaesthesia*, 3rd edn. Thurman, J. C., Tranquilli, W. J. & Benson, G. J., ch. 10A, pp. 434–447. Baltimore: Williams and Wilkins.



- SPREULL, J. S. A. (1958). Accidents associated with anaesthesia. *Veterinary Record* **70**, 981.
- STANEK, B., SCHWARTZ, M., ZIMPFER, M. & RABERGER, G. Plasma concentrations of noradrenaline and adrenaline and plasma renin activity during extradural blockade in dogs. *British Journal of Anaesthesia* **52**, 305–311.
- TRONCY, E., CUVELLIEZ, S. & BLAIS, D. (1996). Evaluation of analgesia and cardiorespiratory effects of epidurally administered butorphanol in isoflurane anesthetized dogs. *American Journal of Veterinary Research* **57**, 1478–1482.
- TUNG, A. S. & YAKSH, T. L. (1982). The antinociceptive effects of epidural opiates in the cat: studies on the pharmacology and the effects of lipophilicity in spinal analgesia. *Pain* **12**, 343–356.
- VALVERDE, A., DYSON, D. H., McDONELL, W. N. & PASCOE, P. J. (1989). Use of epidural morphine for pain relief in the dog. *Veterinary Comparative Orthopaedics and Traumatology* **2**, 55–58.
- VESAL, N., CRIBB, P. H. & FRKETIC, M. (1996). Postoperative analgesic and cardiopulmonary effects in dogs of oxymorphone administered epidurally and intramuscularly and medetomidine administered epidurally: a comparative clinical study. *Veterinary Surgery* **25**, 361–369.
- WANG, B. C., LI, D., HILLER, J. M., SIMON, E. J., BUDZILOVICH, G. & HILLMAN, D. E. (1992). Lumbar subarachnoid ethylenediamine-tetraacetate induces hindlimb tetanic contractions in rats: prevention by CaCl<sub>2</sub> pretreatment: observations of spinal root nerve degeneration. *Anesthesia and Analgesia* **75**, 895–899.
- WANG, C., CHAKRABARTI, M. K. & WHITWAM, J. G. (1993). Specific enhancement by fentanyl of the effects of intrathecal bupivacaine on nociceptive efferent but not sympathetic efferent pathways in dogs. *Anesthesiology* **79**, 766–773.
- YAKSH, T. (1996). Epidural ketamine: a useful mechanically novel adjuvant for epidural morphine. *Regional Anesthesia* **21**, 508–513.
- YAKSH, T. L., PROVENCHER, J. C., RATHBURN, F. R. & KOHN, F. R. (1999). Pharmacokinetics and efficacy of epidurally delivered sustained-release encapsulated morphine in dogs. *Anesthesiology* **90**, 1402–1412.
- ZARIC, D., NYDAHL, P. A., PHILLIPSON, L., SAMUELSSON, L., HEIERSON, A. & AXELSSON, K. (1996). The effect of continuous lumbar epidural infusion of ropivacaine (0.1%, 0.2% and 0.3%) and 0.25% bupivacaine on sensory and motor block in volunteers: a double blind study. *Regional Anaesthesia* **21**, 14–25.  
(Accepted for publication 18 August 2000)

## Book Review

*A Practical Guide to Feline Dermatology.*

Guaguere, E. and Prelaud, P. London, Merial, 2000. 200pp (approx.) (no price given) (hard)

This up-to-date book is attractively presented so that it is both a pleasure to handle and to read. The accessibility of the book is enhanced by its Contents section which contains a summary of each chapter; this makes the book particularly useful for the general practitioner who needs to access information quickly which is both concise and useful.

The chapters are ordered logically, starting with a review of the physiology of skin and moving on to the general diagnostic approach. Major groupings of skin diseases are then addressed, starting with the most common 'Ectoparasitic disease' and moving on by stages to less common disorders, finishing with 'Skin conditions associated with behavioural disorders'. The next group of chapters provides an alternative approach by taking the reader through 'Diagnostic approaches' to a number of dermatological presenting syndromes, e.g. 'pruritic dermatoses' and 'erosive and ulcerative

dermatoses'. These are relatively brief chapters which present flow charts for considering the problem and diagnostic tests which should be used, in order of their applicability.

The photographs are of a very high quality and hold the reader's attention, making learning easy and interesting. There is sufficient detail to provide background coverage even for RCVS Certificate of Dermatology candidates.

It is difficult to find much to criticise in the book; the principal comment is that as the book is French, some of the therapeutic agents listed as being licensed products do not have a UK licence for the treatment of cats. Secondly, some commonly used disease names (e.g. rodent ulcer) are not mentioned, making it a bit harder for the user who knows some diseases only by such (outdated?) terms.

In conclusion, the book provides a highly accessible coverage of an important part of general practice; the presentation and up-to-date coverage makes the book useful to anyone with an active interest in feline dermatology.

S.K. SIVAM