REGIONAL EPIDURAL ANAESTHESIA IN THE DOG WITH LIGNOCAINE AND BUPIVACAINE

MAXIM I. LEBEAUX

SUMMARY

Regional epidural anaesthesia was produced with lignocaine and bupivacaine in dogs with permanently implanted catheters in the lumbar epidural space. Neurological techniques were used to evaluate and measure the signs of anaesthesia. The response to both drugs in the dog paralleled the response in man, bupivacaine proving to be more potent than lignocaine, having less propensity to spread, and less of a sedative effect.

Epidural anaesthesia is a commonly employed technique of regional anaesthesia in man. Correlation of local anaesthetic activity in experimental animals with results in man has been difficult. In this regard, an experimental method used to study epidural anaesthesia in the cat was described by Duce and associates (1969). The feline, however, tends to be stoical, uncommunicative and sedentary, and effects on neural conduction caused by local anaesthetics, particularly on sensory pathways, are difficult to evaluate. There is also a large discrepancy between the size of the cat and man. Consequently a similar model has been developed using the dog because it is tractable, mobile, responds readily to noxious stimuli by vocalizing and snapping and is more comparable in size to man.

The surgical implantation of a catheter provided a preparation whereby solutions of drugs could be easily injected into the same area in the epidural space without stress and therefore the necessity to premedicate the animal. This report will describe therefore (1) the procedure developed for implantation of a catheter into the epidural space of the dog, (2) the neurological examination utilized to assess drug effects at discrete levels of the spinal cord, (3) response of the dog to epidural administration of the well known and widely used local anaesthetic lignocaine, (4) response to a newly developed agent, bupivacaine, and finally, (5) a comparison of the responses to these two agents by dog and man.

METHODS

Surgical technique.

Male beagle dogs (10.0 ± 0.5 kg) were anaesthetized with pentobarbitone sodium and prepared for surgery in the lumbar area. Aseptic surgical procedures were employed. Since the catheter was to be placed in the sixth lumbar vertebra on the right side, this vertebra was identified and an incision made through the skin on the midline from the seventh to the fifth lumbar.

The subcutaneous connective tissue and fat were dissected, exposing the spinous processes of the sixth, seventh lumbar vertebrae, and the heavy aponeurosis overlying the sacrospinalis muscle on the right side. The aponeurosis was incised immediately lateral to its attachments to the spinous processes, and the muscle was separated from the bone of the spine and dorsal laminar arch of the sixth lumbar vertebra by blunt dissection. The bone of the spine and the dorsal laminar arch cephalad to the cranial articular surface, caudal to the caudal articular surface and ventrolaterally to the origin of the transverse process was visualized. In the beagle, the area of bone exposed, exclusive of the vertebral spine, was about a square centimetre. The periosteum of the exposed bone was stripped by scraping with a scalpel blade.

A 20-in. length of polyvinyl catheter (o.d. 1.9 mm, i.d. 1.4 mm) was prepared prior to surgery by winding several loops of No. 34-gauge stainless steel wire tightly about the catheter 2.0-2.5 mm from one tip and tying securely. The wire provided an anchor for the adhesive to the catheter, which, in turn, anchored the catheter in the vertebral bone.

A laminotomy was performed with a dental burr, 2 mm lateral to the base of the sixth vertebral spine, in the centre of the area heretofore described. The hole was drilled through the laminar arch and into the epidural space. The drill was angled and directed ventrally and toward the midline during this operation so that eventual placement of the catheter orifice

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epidurally would be as near to the midline as possible, directly over spinal cord segment coccygeal 2–3. Haemorrhage was completely controlled and the exposed vertebral bone surrounding the hole kept dry so that the adhesive would adhere properly. The thickness of the bone was ascertained, the catheter tip was trimmed appropriately and inserted into the hole up to the wire winding. Care was taken so that protrusion of the catheter tip into the epidural space did not exceed 0.5 mm.

A few drops of the adhesive* were applied to both bone and catheter at their vertebral junction. Setting of the adhesive occurred rapidly, fixing the catheter in situ and sealing the hole. Sterile saline injected through the catheter before closing the incision revealed leaks or malfunction. The catheter was emplaced subcutaneously and exposed at the nape of the neck by using a 12-gauge, 18-in. needle. The protruding tip of the catheter was sealed by clamping and protected with a stockinette collar taped securely around the neck of the dog. Recovery was uneventful and the dog used the day following surgery.

**Design and conduct of experiment.**

Five male beagle dogs were prepared with epidural catheters and treated for four successive days with 2.0% lignocaine with adrenaline 1:100,000 (pH 6.5–6.8). Heparinized blood samples were obtained immediately upon completion of the injection and at 2, 5, 10, 15, 20, 30, 45, 60, 120 and 240-minute intervals thereafter. Treatment of the animal was performed with the dog standing on a table and restrained by an assistant. The sealed tip of the catheter was trimmed, and with a needle of appropriate size introduced into the lumen, a volume of 5.0 ml of solution was administered over a 2-minute interval.

In another series of experiments, twelve male beagles were treated with 0.5% bupivacaine with adrenaline 1:100,000 (pH 5.0) in a like manner to those treated with lignocaine. These dogs, however, were prepared and used as controls for studies over a 5-month period involving other local anaesthetic agents. In all cases, however, the dogs were used the day following surgery and were used only once.

The start of the injection was noted as the start of the experiment and was the time from which onset was calculated. The term onset of block was defined in these experiments as describing the time at which anaesthesia was established (sign of block appeared). Duration of block was the time between onset and recovery of function (sign of block disappeared). No attempt was made to measure regression of anaesthesia in two spinal segments as is often done in humans. Though there are means in the canine model to assess regression as will be indicated hereafter, this phenomenon cannot be followed as closely as in man. Each leg was evaluated independently for the signs of weight support, flexor reflex and digital pain (one dog, two rear legs, etc.).

Ataxia, foot drop, tail paralysis, loss of weight support by the rear limbs and loss of sensation in various dermatomes occur during epidural anaesthesia. Loss of weight support, loss of flexor reflex, loss of digital pain and loss of scrotal pain were chosen as the primary experimental endpoints for measuring the spread and duration of epidural anaesthesia because they exhibit clear endpoints, possess delineated nerve pathways and exemplify as nearly as possible either motor or sensory function (table I). Other signs, called overt, also appear subsequent to epidural blockade. Horner’s syndrome, abdominal breathing, and front limb paresis were used as a means of assessing exaggerated segmental spread. Sedation, tremor (preconvulsive sign), and convulsions were interpreted as indications of systemic toxicity. The onset and duration of these overt signs were not measured but their frequency of appearance was recorded.

Gross necropsies were performed on the five dogs used in the lignocaine studies 3 weeks after catheter implantation. Confirmation of the location of the tip of the catheter in the epidural space and appraisal of pathology in surrounding structures were made.

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*Aron Alpha-(Alpha Cyanoacrylate) Toagosei Chemical Industry Co., Ltd, Tokyo, Japan.*
RESULTS
Table II summarizes the responses of five dogs to treatment with lignocaine on four successive days. The sequence of events after administration was similar on each day. Initially, as epidural blockade developed, there was loss of weight support by the pelvic limbs, followed by loss of response to stimulation of the scrotum and digits of the rear legs. Lastly, there was loss of the flexor reflex. After a period of about 90 minutes, recovery began in the reverse order to the sequence observed during onset. First, the flexor reflex returned, then responses to scrotal and digital stimulation (pain), and finally recovery of weight support occurred.

With respect to the reproducibility of the responses to repeated doses of lignocaine, statistical analysis of the data obtained on the four successive days of treatment revealed that insofar as duration was concerned, day one differed from days three and four but not always from day two. There were no differences in onset except for weight support on day four.

Peak blood levels (fig. 1) of the dogs treated epidurally on four successive days. Each curve represents mean values of five dogs.

Table II. Response of the dog to the epidural administration of 100 mg of lignocaine hydrochloride* on four successive days.

<table>
<thead>
<tr>
<th>Day</th>
<th>Onset (min)</th>
<th>Weight support</th>
<th>Flexor reflex</th>
<th>Digital pain</th>
<th>Scrotal pain</th>
<th>Duration (min)</th>
<th>Full recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>X</td>
<td>3.0 ± 0.0</td>
<td>8.2 ± 0.0</td>
<td>8.2 ± 0.0</td>
<td>5.2 ± 0.0</td>
<td>141.2 ± 1.6</td>
<td>96.6 ± 4.2</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>±0.7 ± 0.0</td>
<td>±1.6 ± 0.6</td>
<td>±1.6 ± 0.6</td>
<td>±1.6 ± 0.6</td>
<td>±3.6 ± 0.4</td>
<td>±9.4 ± 0.3</td>
</tr>
<tr>
<td>2</td>
<td>X</td>
<td>2.6 ± 0.5</td>
<td>6.8 ± 0.6</td>
<td>6.2 ± 0.4</td>
<td>4.4 ± 0.5</td>
<td>116.6 ± 1.3</td>
<td>86.4 ± 4.2</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>±0.6 ± 0.4</td>
<td>±1.3 ± 0.4</td>
<td>±1.1 ± 0.5</td>
<td>±0.5 ± 0.5</td>
<td>±4.2 ± 0.3</td>
<td>±7.3 ± 0.6</td>
</tr>
<tr>
<td>3</td>
<td>X</td>
<td>2.2 ± 0.4</td>
<td>9.2 ± 0.6</td>
<td>7.8 ± 0.4</td>
<td>6.6 ± 0.6</td>
<td>111.6 ± 1.1</td>
<td>55.2 ± 4.2</td>
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<tr>
<td></td>
<td>SD</td>
<td>±0.1 ± 0.3</td>
<td>±1.7 ± 0.3</td>
<td>±1.1 ± 0.5</td>
<td>±0.5 ± 0.5</td>
<td>±4.2 ± 0.3</td>
<td>±7.3 ± 0.6</td>
</tr>
<tr>
<td>4</td>
<td>X</td>
<td>4.2 ± 0.7</td>
<td>7.8 ± 0.8</td>
<td>7.2 ± 0.6</td>
<td>5.8 ± 0.6</td>
<td>103.0 ± 1.9</td>
<td>67.6 ± 4.2</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>±0.8 ± 0.6</td>
<td>±1.3 ± 0.6</td>
<td>±1.3 ± 0.6</td>
<td>±0.6 ± 0.6</td>
<td>±11.0 ± 1.9</td>
<td>±6.3 ± 0.6</td>
</tr>
<tr>
<td></td>
<td>SE</td>
<td>±0.0 ± 0.0</td>
<td>±0.0 ± 0.0</td>
<td>±0.0 ± 0.0</td>
<td>±0.0 ± 0.0</td>
<td>±0.5 ± 0.1</td>
<td>±0.5 ± 0.1</td>
</tr>
</tbody>
</table>

Each value represents five animals.

Table II. Response of the dog to the epidural administration of 25 mg of bupivacaine† on four successive days.

<table>
<thead>
<tr>
<th>Day</th>
<th>Onset (min)</th>
<th>Weight support</th>
<th>Flexor reflex</th>
<th>Digital pain</th>
<th>Scrotal pain</th>
<th>Duration (min)</th>
<th>Full recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>X</td>
<td>3.75 ± 1.76</td>
<td>8.17 ± 4.0</td>
<td>5.13 ± 4.96</td>
<td>6.64 ± 6.41</td>
<td>331.5 ± 5.35</td>
<td>161.2 ± 36.2</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>±0.2 ± 2.0</td>
<td>±1.2 ± 1.2</td>
<td>±0.8 ± 0.8</td>
<td>±0.6 ± 0.6</td>
<td>±16.2 ± 10.4</td>
<td>±24.9 ± 19.1</td>
</tr>
<tr>
<td>2</td>
<td>X</td>
<td>4.2 ± 0.5</td>
<td>7.8 ± 0.8</td>
<td>7.2 ± 1.4</td>
<td>5.8 ± 1.9</td>
<td>103.0 ± 1.9</td>
<td>67.6 ± 4.2</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>±0.7 ± 0.6</td>
<td>±1.3 ± 1.3</td>
<td>±1.3 ± 1.3</td>
<td>±0.6 ± 0.6</td>
<td>±11.0 ± 1.9</td>
<td>±6.3 ± 0.6</td>
</tr>
<tr>
<td></td>
<td>SE</td>
<td>±0.0 ± 0.0</td>
<td>±0.0 ± 0.0</td>
<td>±0.0 ± 0.0</td>
<td>±0.0 ± 0.0</td>
<td>±0.5 ± 0.1</td>
<td>±0.5 ± 0.1</td>
</tr>
</tbody>
</table>

Each value represents twelve animals.

Response of twelve dogs to the epidural administration of 25 mg of bupivacaine†

<table>
<thead>
<tr>
<th>Day</th>
<th>Frequency of overt signs**</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8/12</td>
</tr>
<tr>
<td>2</td>
<td>7/12</td>
</tr>
<tr>
<td>3</td>
<td>4/12</td>
</tr>
<tr>
<td>4</td>
<td>2/12</td>
</tr>
<tr>
<td>5</td>
<td>0/12</td>
</tr>
</tbody>
</table>

* 5.0 ml 2.0% lignocaine hydrochloride.
** 1 Horner's syndrome. 2 Abdominal breathing. 3 Sedation. 4 Paresis of front limbs. 5 Convulsions or preconvulsive signs.
† 5.0 ml 0.5% bupivacaine.
‡ Only seven dogs measured to full recovery—others recovered within 24 hours.
minutes shows no statistical difference,* these data may suggest greater absorption with repeated treat-
ment and may account for the shorter duration on
de these days. Gross examintion at necropsy 3 weeks
after surgery revealed that the catheters were still
firmly in place. Externally, at the site of catheter
implantation, no marked changes had occurred in
the vertebral bone or surrounding tissues. Within
the vertebral canal, however, some inflammatory
responses had occurred in all dogs about the orifice
of the catheter in the epidural space. In some cases
adhesions between dura and vertebra had occurred
and in one instance overgrowth of granulation tissue
ocluded the opening of the catheter. These changes,
it appeared, were chronic, progressive and accumu-
lative and undoubtedly minimal in early stages of
the experiment.

The responses to bupivacaine are also summarized
in table II. In general, the sequence of effects
occurring after bupivacaine was the same as after
lignocaine. However, the duration of action of bupi-
vacaine was consistently about double that of ligno-
caine, as reflected in the longer duration of block of
all parameters.

In most animals, both with lignocaine and bupi-
vacaine, there developed, in addition to the measured
anaesthetic parameters, overt signs of exaggerated
blockade and systemic toxicity. The frequency of
these signs is summarized in table II. Statistical
analysis† reveals that there is a significant difference
between the two drugs for the signs of sedation
and front limb paresis. These data suggest, therefore,
a greater spreading propensity and central sedative
effect for lignocaine.

It should be reported that during the 4 days of
 treatment with lignocaine, there were four instances
or respiratory embarrassment severe enough to cause
cyanosis in the visible mucous membranes, one dog
requiring supplementary oxygen. One dog exhibited
slight nausea (lip licking and retching) and one dog responded with what appeared to be the
Schiff-Sherrington phenomenon (increased reflex
excitability of the front limbs caused by block of
spinal impulses to hind part of body). There
were no overt signs or side effects in the dogs treated
with bupivacaine other than those noted in table
II. No preconvulsive or convulsive signs occurred
with either drug.

*Two-way analysis of variance of related samples.
†Fisher Exact Probability Test.

Discussion

Anaesthetists who perform epidural anesthesia in
humans are concerned with drug responses such as:
(1) onset; (2) duration; (3) spread; (4) effects on
motor and sensory pathways; (5) systemic toxicity.
Bromage (1965) has described these effects and
devised methods for assessing and quantifying them
in man. The canine model reflects responses similar
to those observed in man, but evaluation, unfortu-
nately, cannot be as sophisticated since man can
be subjective (describe sensation, move specific
muscles), whereas in the dog one must rely on
observation of the manner in which the animal
ambulates and/or responds to stimuli. Interpretation
of the neurological response to epidural anaesthesia
in animals depends, therefore, upon correlation of
the signs of dysfunction with the spinal nerve com-
ponents affected.

Loss of weight support. Supportive weight by
pelvic limbs is largely dependant upon proper func-
tion of the extensor muscles of the knee. These
muscles, principally the quadriceps femoris, are
supplied by motor fibres couring in the femoral
nerve which rises at spinal roots L4-5.* Loss of
weight support occurs when anaesthesia in this
segment blocks conduction and inability to extend
and lock the knee joint ensues. Albeit that sensory
proprioceptive feedback is an important component
of this function, the activity of weight support can
be considered principally motor activity. Since the
dog is self motivated to stand, the endpoints of
onset and recovery of this function are readily
discernable. Consequently, as long as this function
is impaired, anaesthesia must involve at least the
L4-5 segment.

Flexor reflex. The flexor reflex has both a sensory
and motor element but that portion of the arc which
supplies nerve fibres to the flexor muscles of the leg
and foot (which operate to withdraw the limb after
painful stimulus) is motor. These pathways are con-
tained in the sciatic nerve and its divisions, whose
roots are primarily L6-7 and S1-2. Withdrawal or
nonwithdrawal of the limb after digital stimulus
denotes whether or not conduction has been blocked
in these spinal roots.

Pain. Vocalization and snapping by the dog is inter-
preted as a subjective response to a painful stimuli
and was used as a measure for presence or absence
of sensory activity in the appropriate segment. In
this manner, therefore, the extent of spread of epi-
dural anaesthesia can be evaluated.

*The iliopsoas and sartorius muscles, principal flexors
of the hip joint, are also innervated by the femoral nerve.
**Digital pain in hind limb.** The skin of the digits and of the web between them is very sensitive. Digits three, four and five in the rear paw are supplied by cutaneous sensory fibres carried in the peroneal and tibial nerves whose spinal roots as extensions of the sciatic are L6–7, S1–2. The dorsal and medial aspects of digits one and two, however, are supplied by fibres carried in the saphenous nerve, whose roots, as a division of the femoral, are L4–5. This nice distinction in dermatomal innervation of two closely related anatomical parts supplied by separated spinal roots can be used to assess the degree and duration of anaesthetic spread. Obviously, if all digital dermatomes are blocked, anaesthesia has extended to include at least L4–5; return of sensation to digits one and two (but not three, four and five) implies that regression has occurred to at least spinal segment L5.

**Scrotal pain.** The caudal surface of the scrotum is supplied by sensory fibres carried in the pudendal and caudal scrotal nerves whose roots are located in S1–2–3. The cephalic surface of the scrotum, on the other hand, is supplied by sensory fibres contained in the genital nerve whose roots are located at L3–4. The absence of pain in the caudal scrotal skin is indicative of anaesthesia in S3; recovery of pain sensation in this part means regression of anaesthesia to at least S2. Similarly, absence of pain in the anterior surface of the scrotal skin and sheath indicates block of spinal roots as high as L3; return of sensation to this area implies regression to at least L4.

**Other parameters.** Other postural and spinal reflexes were used for adjunct evaluation of the extent of epidural anaesthesia, but not measured. The patellar reflex, afferent and efferent components of which are entirely contained in the femoral nerve, is a test for function in roots L4–5. Tail paralysis as a sign of block in the coccigeal nerve trunks (spinal roots S3, coccygeal 1–2) and cutaneous dermatomal responses to stimuli are also aids in determining the segmental spread of anaesthesia.

**Overt signs of exaggerated spread.** Horner's syndrome as seen in the dog is characterized by prolapso of the membrana nictitans, ptosis of the upper lid and miosis. The response may appear unilateral or bilateral depending upon the manner of spread of the drug. Innervation of structures associated with this syndrome is via preganglionic sympathetic fibres which exit from the spinal canal at T1–2–3 and arrive at their destination by way of the sympathetic trunk and the cranial cervical ganglion. Therefore, this sign indicates spread to thoracic segments T1–2–3.

Paralysis of the internal and external intercostal muscles of respiration results in breathing becoming entirely diaphragmatic and can be visualized as an exaggerated rise and fall of the now flaccid abdominal wall, indicating anaesthesia in spinal segments T2 through T12. Motor and/or sensory dysfunction of either front limb denotes anaesthesia in spinal components C6–7–8, T1–2, the roots of origin of the brachial plexus. Involvement of segments C4–5 blocks the roots of the phrenic nerve with concomitant cessation of breathing and death.

**Overt signs of central effect.** Systemic toxicity is assumed to be related to excessive blood levels following overdosage or rapid absorption. This condition is exemplified by preconvulsive or convulsive states characterized by muscle tremor, reflex irritability, spasticity (local or generalized) in the former, and frank convulsions in the latter. Nausea, vomiting and sedation are other signs which indicate central involvement. All of these manifestations of systemic toxicity appear in the dog.

The results obtained in these experiments indicate that the epidural dog preparation has met many criteria for monitoring responses to drugs injected into the epidural space. These responses were characterized by typical block and recovery in neural conduction manifested by signs of failure and recovery of muscular motor and cutaneous sensory function. The surgical implantation of a catheter provided a preparation whereby solutions of drugs could be easily injected into the same area in the epidural space without stress to the animal. The catheter was maintained firmly in place and postoperative healing occurred rapidly and without complications. Unfortunately, long term use of the model was precluded, since necropsy revealed that local tissue reaction occurred about the catheter opening and involved structures in and contiguous to the epidural space. These changes ultimately led to malfunction and, in any event, cast doubt on the results obtained in successive treatments in the same animal.

Nevertheless, these experiments showed that if the preparation is used soon after surgery the results are consistent. This conclusion is supported by the data wherein very small standard deviations and standard errors occurred in the responses obtained in the five dogs treated with lignocaine on day one. Whether the trend toward shortening duration of block on days two, three and four can be related to the suggested greater absorption, or whether this is an example of tachyphylaxis cannot be resolved since the pathology observed at necropsy confuses the issue. The larger standard deviations obtained in the
dogs treated with bupivacaine can be explained, in part, by the fact that these data were collected from dogs prepared and used over a 5-month period and in part, by the fact that such wide deviations are not altogether unexpected in evaluating long acting local anaesthetics where endpoints of recovery are more masked.

The data in table II indicate that both lignocaine and bupivacaine: (1) block digital pain longer than scrotal pain; (2) block scrotal and digital pain longer than the flexor reflex and (3) block weight support sooner and longer than any of the other parameters (except full recovery).

The recovery of scrotal pain before digital pain is a good example of segmental regression since the nerve roots mediating posterior scrotal pain (S1–2–3) are farther removed from the site of injection than those mediating digital pain (L6–7, S1) and would therefore be expected to recover more quickly.

The recovery of the flexor reflex before digital pain is more difficult to rationalize, particularly since both responses depend upon conduction in the same afferent sensory pathways. One explanation for this seeming paradox would be, if, as suggested by Bromage (1967), the anaesthetic gains access to the subarachnoid space via the dural root sleeves and blocks unsheathed fibres in the corticospinal tracts. In this event pain conducting fibres to higher centres could be affected at segmental levels which would not interfere with elicitation of the flexor reflex and yet there would be no evidence of pain, even though both signs depend upon the same noxious stimulus.

The rapid onset and long duration of loss of weight support present vexing questions that cannot be entirely resolved. The rapid loss and lengthy inability to support weight even after sensation and muscular tone has returned to a marked degree must reflect dysfunction in fibres mediating proprioceptive mechanisms. By and large, proprioception is served by the large myelinated Aα and Aβ fibres which require twice the minimum concentration of drug to become blocked as do the smaller Aγ, Aδ, B and C fibres (de Jong, 1970). However, deep tendon reflexes are dependent upon the tone in muscle spindles that are mediated by Aγ fibres which are of small diameter, and are blocked sooner and longer than larger fibres (de Jong, 1970). If this is indeed the case, then the rapid onset and long duration of dysfunction in the antigravity extensor muscles of the hip and stifle joints can be understood.

Another explanation for the long duration of loss of weight support and of the attendant ataxia which accompanies the return to complete recovery might lie in the effect produced on spinocerebellar tracts in the spinal cord if the anaesthetic enters the subarachnoid space via the dural root cuffs. In this event the fine motor control exerted by the cerebellum would be disrupted, and for a relatively long time, since small desheathed fibres are involved.

Comparison of the responses in the dog to lignocaine and to bupivacaine (table II) reveal that though onset of block was similar, duration of block produced by these drugs was very different. Striking was the difference in sensory block where digital pain was blocked 2 hours longer and scrotal pain 1 hour longer for bupivacaine than for lignocaine. Motor block, as reflected by loss of weight support and loss of the flexor reflex, was also much longer for bupivacaine. Noteworthy was the fact that bupivacaine has less spread and systemic effect than lignocaine as reflected by the lower incidence of front limb paresis and sedation for bupivacaine.

Bromage and Gertel (1970) have indicated that the time for initial onset of sensory anaesthesia in humans for lignocaine (2.0%, 1 : 200,000 adrenaline) was 5.5 minutes and for bupivacaine (0.5%, 1 : 200,000 adrenaline) was 6.5 minutes. The response of the canine model for onset was much the same as man, 5.2 minutes for lignocaine and 6.6 minutes for bupivacaine for block of scrotal pain. These authors in the same study indicate that duration of sensory block produced by lignocaine was 100 ± 20 minutes and by bupivacaine 200 ± 31 minutes in man. Block of scrotal pain in the dog by lignocaine was 110.8 ± 6.6 minutes and by bupivacaine 189.6 ± 44.9 minutes. Though these are admittedly crude comparisons, the responses observed in the epidural dog were strikingly similar to those observed in man for these two drugs. Milligram for milligram, bupivacaine is more potent in the dog than lignocaine and the same is true for man.

The canine model, for many reasons, but particularly because it allows neurological assessment of epidural anaesthesia and seems to correlate with the response in man, promises to be a valuable tool for the investigation of potential local anaesthetic agents.

ACKNOWLEDGEMENTS

The author is indebted to Miss Georgette Kilpatrick and Mr John Holup for their technical assistance; to Dr Jack Adams for professional advice; and to Dr Murray Blair without whose support and insight this work would not have been possible.

REFERENCES

EXPERIMENTAL EPIDURAL ANAESTHESIA IN THE DOG


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ANESTHESIE EPIDURALE EXPERIMENTALE AVEC LA LIGNOCAINE ET LA BUPIVACAINE CHEZ LE CHIEN

SOMMAIRE

L'anesthésie epidurale régionale est produite avec la lignocaine et la bupivacaine chez le chien par un cathéter implanté en permanence dans l'espace epidural lombaire. On utilise les techniques neurologiques pour évaluer et mesurer les signes de l'anesthésie. Chez le chien la réponse aux deux substances est parallèle à celle de l'homme, la bupivacaine s'est révélée plus puissante que la lignocaine avec moins de tendance à s'étendre et avec un plus faible effet sédatif.

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EXPERIMENTELLE EPIDURALE ANAESTHESIE AN HUNDEN MIT LIGNOCAINE UND BUPIVACAINE

ZUSAMMENFASSUNG


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ANESTESIA EPIDURAL EXPERIMENTAL EN EL PERRO CON LIGNOCAINA Y BUPIVACAINA

RESUMEN

Se probó la anestesia epidural regional con lignocaina y bupivacaina en perros, con unos catéteres permanentes fijados en el espacio epidural lumbar. Técnicas neurologicas fueron usadas para evaluar y medir los signos de la anestesia. Las respuestas a las drogas en el perro fueron similares a las reacciones en el hombre; la bupivacaina, demostró ser más potente que la lignocaina siendo menos propensa a difundirse y con menos efecto sedativo.