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ANAESTHETIC POTENCY IN EPIDURAL ANALGESIA

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IN an extensive literature on epidural analgesia there appears to be some difference of opinion as to the intensity of nerve block produced by different drugs, perhaps partly attributable to the fact that many patients are given light narcosis in addition to nerve block, nowadays, which may conceal differences of drug action. In quoting a few of the many authorities, the following drugs are listed according to the original author's assessment of the relative strengths as regards sensory and motor block, independent of duration, although the actual concentrations used by the different observers varied.

Massey Dawkins (1949). Cinchocaine>Procaine.
Bromage (1954a). Lignocaine>Amethocaine>Cinchocaine>Procaine.
Pitkin (1946). Piperocaine>Procaine>Amethocaine.
Lull and Hingson (1945). Piperocaine>Procaine>Butethamine>Amethocaine.
Blundell et al. (1955). Hexylcaine>Lignocaine>Procaine>Amethocaine>Piperocaine.
Crawford (1953). Lignocaine>Hexylcaine>Procaine.
Abajian (1943). Diethoxin>Procaine.

Thus, amethocaine is considered more powerful than procaine by Bromage and weaker than procaine by Pitkin, Blundell et al. and Lull and Hingson. Piperocaine was found to be more powerful than procaine by Pitkin and Lull and Hingson but weaker than procaine by Blundell et al. Other differences of opinion arise over the relative potencies of lignocaine and hexylcaine.

This investigation has been made to ascertain within the limits of restricted clinical trial, the relative potencies of several local anaesthetics in epidural analgesia. Preliminary trials of sacral epidural block (47) and spinal epidural block (53) were carried out to provide experience, using procaine, piperocaine, amethocaine and lignocaine. From a study of this series two principal facts emerged. Firstly, that there appeared to be a far greater variability of response to a given dose than is the case in subarachnoid anaesthesia. For every case of epidural block, twenty intrathecal spinal anaesthetics were administered in the routine work of this department using amethocaine, amylocaine, cinchocaine or procaine. Since uniformity as regards predictability of the extent of block and intensity of relaxation is obtained in upwards of 80 per cent of subarachnoid spinal anaesthesias, the method has formed the basis for comparison with results from epidural anaesthesia. Secondly, anatomical variations accounting for failures were less in the spinal than in the caudal epidurals; it was therefore decided to adopt a standard technique of spinal epidural block and to accumulate comparative series of cases with different anaesthetic solutions. None of the preliminary trial cases have been included, except in the study of toxic reactions. Because of the practical difficulty of delays engendered by the assessment of the results of weak solutions and by the administration of supplementary anaesthesia, the investigation has taken six years to complete.

TECHNIQUE

Patients were given intravenous premedication of papaveretum 20 mg and hyoscine 0.4 mg for the 10 stone (63 kg) adult with proportional variation for body weight. Reduced dosage was applied also for senility, cachexia and for anaemia with haemoglobin below 70 per cent. The skin over the selected supraspinous ligament was infiltrated with procaine and ephedrine 30 mg and the epidural space identified by release of digital pressure applied to a syringe of normal saline. Nineteen or 20 S. W. G. Luer-Lok short bevel needles were employed. In every case blood pressure charts were made throughout operation. Details of site of spinal puncture and classification of operations appear in table I.

Twelve drugs were used and twenty anaesthetics were conducted for each of thirty-five different solutions and for two techniques with viscous amethocaine. The compositions of the solutions are listed in table II. Structural formulae of all the drugs except Carbocaine and Amplicaine, which are depicted here, were presented by Lloyd (1955). In solutions containing adrenaline the concentration varied from 1/300,000 to 1/150,000, the latter strength being used for laminectomies where an avascular extradural space was advantageous.

TABLE I

Data of operation, site of spinal puncture and age of the 740 cases.

Site of operation	No. of cases
Upper limb and chest wall	12 (mastectomy 8)
Stomach	21 (gastro-enterostomy 15)
Liver	14 (hydatid 8)
Gall bladder	12 (cholecystectomy 7)
Spleen	6 (hydatid 2)
Other upper abdominal ...	82 (epigastric and umbilical hernia 47)
Gut	53 (Appendicectomy 35)
Uterus and ovaries ...	25 (hysterectomy 7)
Testis and tunica	21 (hydrocele 11)
Ureters	49 (transplantation 25)
Bladder	77 (prostatectomy 41, cystolithotomy 24)
Other lower abdominal ...	204 (inguinal hernia 186)
Kidney	23 (nephrolithotomy 9)
Spine	60 (laminectomy 46)
Other	7
Lower extremities	42
Perineal and gynaecological	32

Site of interspinous puncture	No. of cases
T 3	2
T 4	5
T 5	2
T 6	5
T 7	2
T 8	5
T 9	11
T 10	29
T 11	15
T 12	58
L 1	131
L 2	156
L 3	228
L 4	91

Average age of patients 40.7 years.
Age range from 14 to 80 years.

ASSESSMENT OF RESULTS

Tests of the extent of motor loss, analgesia and hypalgesia were made after injection until no further block developed. Sympathetic block was not awaited prior to commencement of surgery. The extent of hypalgesia was noted at both upper and lower levels as it was found easier to identify the site of pain response in moving from hypalgesic areas to those of normal sensation. Whilst judgment of hypalgesia to pin prick with allowance for apprehensive exaggeration, stoical indifference, ignorance and lack of co-operation is largely arbitrary and personal, every attempt was made to categorize the anaesthesia before incision. Patients were finally classified in the five following groups:

A. Perfect analgesia and good relaxation, inability to raise the legs from the table or to contract the rectus abdominis. Operation conducted without supplements, comparable to subarachnoid spinal anaesthesia.

B. Satisfactory analgesia for surgery without supplements, but relaxation incomplete, tactile and pressure sensations permissible if not painful.

C. Satisfactory analgesia for surgery with intravenous pethidine 100 mg or thiopentone, administered after the incision was completed, for pain from deeper tissues. Pain due to recession of analgesia was excluded.

D. Analgesia not enough for incision without light narcosis, application of towel clips caused only discomfort.

E. Hypalgesia five segments or more, negligible to slight motor weakness and sympathetic block, towel clips painful, narcosis essential.

This classification only applied to surgery conducted within the anaesthetized zone and an extension of surgical stimuli, for example, to unanaesthetized upper abdomen or pelvis was discounted. A few cases with excellent relaxation equivalent of group A, but complaining of pain on incision, were classified as group B. Personal judgment was unavoidable in allowing for the more intense stimuli arising from visceral traction; cases classified as B for inguinal herniotomy

TABLE II
Details of solutions tested.

Name of drug	Strength	Mean dose (mg)	Other constituents
Procaine hydrochloride B.P.	2%	610	0.9% sodium chloride
	2%	609	0.9% sodium chloride adrenaline
	1%	486	0.9% sodium chloride
	1%	462	0.9% sodium chloride adrenaline
Butethamine formate N.N.R.	1%	455	0.9% sodium chloride
	1%	441	0.9% sodium chloride adrenaline
Amethocaine hydrochloride B.P.	1/300	88	0.9% sodium chloride
	1/300	88	0.9% sodium chloride adrenaline
	1/500	76	0.9% sodium chloride
	1/500	77	0.9% sodium chloride adrenaline
	Viscous (preliminary saline)	86	6% polyvinylpyrrolidone adrenaline 1/140,000
	Viscous (terminal saline)	89	6% polyvinylpyrrolidone adrenaline 1/140,000
Tutocaine (p-amino-benzoyl dimethyl-amino-methyl butanol hydrochloride)	1%	387	0.9% sodium chloride
	1%	361	0.9% sodium chloride adrenaline
Amylocaine hydrochloride B.P.C.	2%	501	0.5% sodium chloride 2% glucose
	2%	548	0.5% sodium chloride 2% glucose adrenaline
Piperocaine hydrochloride U.S.P.	2%	490	nil
	2%	514	adrenaline
	1%	349	0.45% sodium chloride
	1%	353	0.45% sodium chloride adrenaline
Hexylcaine hydrochloride N.N.R.	1%	373	nil
	1%	374	adrenaline
Diethoxin hydrochloride (beta diethylaminoethyl-p-ethoxy benzoate hydrochloride)	2%	571	nil
	2%	585	adrenaline
	1%	415	nil
	1%	405	adrenaline
Lignocaine hydrochloride B.P.C.	2%	503	nil
	2%	458	adrenaline
	1%	316	0.45% sodium chloride
	1%	314	0.45% sodium chloride adrenaline
Carbocaine (d-l-N methyl-pipecolic acid 2-6-dimethylanilide)	1%	235	adrenaline

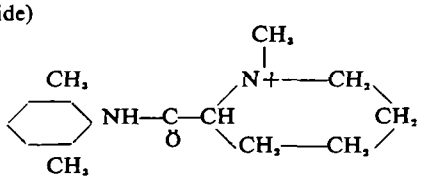
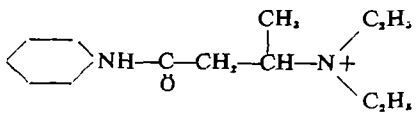


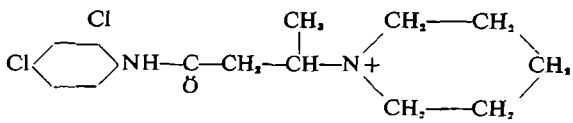
TABLE II—*continued*

Name of drug	Strength	Mean dose (mg)	Other constituents
Amplicaine	2%	710	nil
	2%	750	adrenaline

2% beta diethyl-amino-n-butyro-anilide hydrochloride



0.4% piperidino-n-butyro 2,4 dichloranilide hydrochloride



Cinchocaine hydrochloride B.P.	1/300	98	0.27% sodium chloride 3% glucose
	1/300	90	0.27% sodium chloride 3% glucose adrenaline
	1/600	58	0.45% sodium chloride
	1/600	63	0.45% sodium chloride adrenaline

or gastro-enterostomy might easily prove to be C for appendicectomy or gastrectomy respectively. Since the majority of cases were categorized on the basis of stimuli arising from the parietes, reclassification as a result of reaction to prolonged visceral traction was avoided in order to preserve uniformity of comparison as far as possible. Reclassification from B to C was avoided in cases where supplements were given only to provide the surgeon with greater relaxation as frequently occurred with Tutocaine. The distribution of cases according to this scheme is shown in table III.

A consideration of the unsupplemented cases for each solution gives an indication of relative potency (table IV). In the case of distribution which shows a sharp peak at a given intensity of block—for example, lignocaine, Tutocaine and procaine—it seems reasonable to assume that the sampling was adequate, but other solutions showing irregular distribution presumably require larger case samples in order to provide a satisfactory picture of average intensity of action. The less satisfactory samples have been marked with an asterisk in table IV and fortunately the powerful drugs have given good distribution curves.

Perhaps the most significant finding was the augmentation of the degree of nerve block produced by the addition of adrenaline. Whilst adrenaline is usually added to anaesthetic solutions to prolong anaesthesia and to limit systemic reactions, increased intensity of motor and sensory block was demonstrable with all the solutions except three. The mean intensity obtained was about the same for the solutions with and without adrenaline for 1 per cent hexylcaine, 1 per cent butethamine and 1 per cent procaine, but in spite of the absence of potentiation by adrenaline there was narrowing of the range of response and the anaesthesias became more uniform in intensity. Increasing the concentration of procaine to 2 per cent did bring about adrenaline potentiation and it is possible that different concentrations and greater numbers of cases would result in similar findings for hexylcaine and butethamine. One per cent lignocaine with adrenaline gave a better blockade than 2 per cent lignocaine plain, and similarly 1 per cent piperocaine with adrenaline was stronger than

TABLE III

Case distribution according to intensity of nerve block, categories A to E described in text.

Solution	Without adrenaline					With adrenaline				
	A	B	C	D	E	A	B	C	D	E
Procaine 2%	3	10	5	2	–	6	10	3	1	–
Procaine 1%	–	1	3	8	8	–	1	2	11	6
Butethamine 1%	2	4	4	7	3	1	5	5	8	1
Amethocaine 1/300	5	6	5	4	–	13	5	1	1	–
Amethocaine 1/500	1	4	3	11	1	6	3	4	7	–
Amethocaine viscous preliminary saline	–	–	–	–	–	9	4	5	2	–
Amethocaine viscous terminal saline	–	–	–	–	–	5	5	8	2	–
Tutocaine 1%	–	4	6	10	–	–	12	7	1	–
Amylocaine 2%	–	5	2	10	3	1	9	3	6	1
Piperocaine 2%	3	3	7	5	2	5	5	4	4	2
Piperocaine 1%	–	5	5	9	1	2	6	4	8	–
Hexylcaine 1%	2	9	3	5	1	2	7	8	2	1
Diethoxin 2%	4	5	3	6	2	6	10	2	1	1
Diethoxin 1%	–	4	6	9	1	–	9	8	3	–
Lignocaine 2%	7	9	3	1	–	12	7	1	–	–
Lignocaine 1%	1	7	5	4	2	10	5	4	1	–
Carbocaine 1%	–	–	–	–	–	3	8	5	3	1
Amplicaine 2%	–	4	5	6	5	1	6	5	6	2
Cinchocaine 1/300	–	2	4	12	2	4	3	5	6	2
Cinchocaine 1/600	–	1	4	7	8	–	2	7	7	4

2 per cent piperocaine plain. The number of unsupplemented anaesthesias was trebled by the addition of adrenaline to 1 per cent Tutocaine and to 1/300 cinchocaine, and doubled for 2 per cent amylocaine and 1 per cent diethoxin.

In table V are incidental findings which may be of interest although they are only approximations. The A/B ratio gives an indication of motor block. In addition, the category of muscle relaxation was noted in all cases as good, moderate, slight or nil and the ratio $\frac{\text{Good} + \text{Moderate}}{\text{Slight} + \text{Nil}}$ has been obtained for all the weaker solutions where the majority of cases were in categories C to E. From the two ratios drugs have been grouped in order of relative intensity of motor blocking activity in table V. Dilatation of veins of legs, warming of toes and congestion of the penis to an obvious degree was evidence of sympathetic blockade and drugs have been similarly arranged in order of activity. Although lignocaine gave a 50 per cent development of anaesthesia much sooner than other drugs, the time for full onset or about 90 per cent spread did not differ markedly. Drugs have been arranged in order of speed of full onset of anaesthesia in table V and details are given in table VI. Evaluation of duration of anaesthesia can only be made accurately when the operation outlasts the paralysis and analgesia and when the patient is conscious. The recession of analgesia and of paresis was noted every time it occurred during operation in the unsupplemented anaesthesias and the time of recession is taken from full onset (table VI). Motor recession preceded pain by some 10 to 30 minutes with lignocaine, carbocaine and amplicaine, and general anaesthesia had to be undertaken to provide relaxation in quite a number of cases. The longest time at which painless skin closure took place when greater than the mean recession time has also been noted; naturally the number of cases depended on an uncontrollable variable, the duration of surgery. The estimated probable average duration is based on the figures in the two preceding columns of table VI and upon postoperative observations of skin analgesia, admittedly a less satisfactory method. Lignocaine, diethoxin and hexylcaine appear to be of about equal duration. The fall in blood pressure was looked for in every case with the object of comparing the pressor blocking activity of different drugs and it was found that the number of cases with severe falls of blood pressure corresponded fairly closely to the number of high

TABLE IV

Relative potency of test solutions (for sensory block).

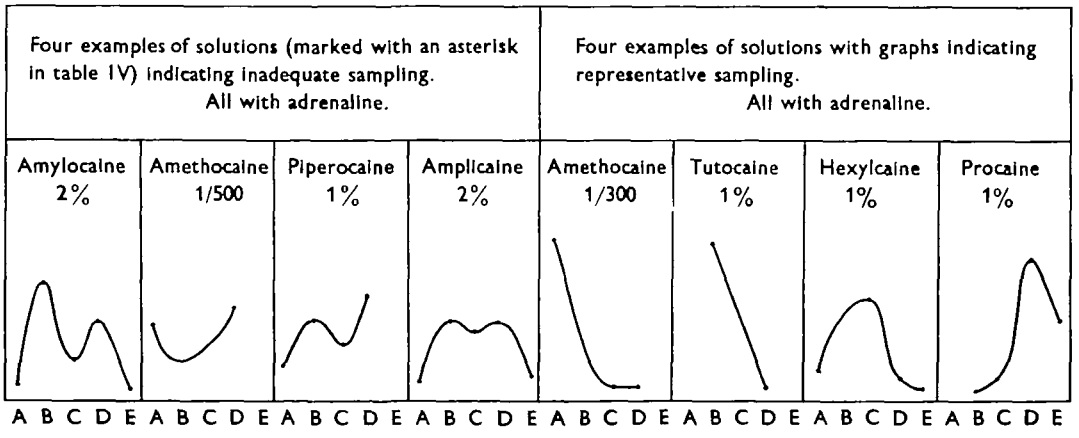
Solution in order of apparent potency	Number of unsupplemented anaesthesias
Lignocaine 2% adrenaline	19
Amethocaine 1/300 adrenaline	18
Lignocaine 2%	16
Procaine 2% adrenaline	16
Diethoxin 2% adrenaline	16
Lignocaine 1% adrenaline	15
Procaine 2%	13
Tutocaine 1% adrenaline	12
Amethocaine 1/300	11
Carbocaine 1% adrenaline	11
Hexylcaine 1%	11*
Piperocaine 2% adrenaline	10*
Amylocaine 2% adrenaline	10*
Amethocaine 1/500 adrenaline	9*
Diethoxin 2%	9*
Hexylcaine 1% adrenaline	9
Diethoxin 1% adrenaline	9
Piperocaine 1% adrenaline	8*
Lignocaine 1%	8
Cinchocaine 1/300 adrenaline	7*
Amplicaine 2% adrenaline	7*
Piperocaine 2%	6*
Butethamine 1% adrenaline	6
Butethamine 1%	6
Amethocaine 1/500	5*
Piperocaine 1%	5*
Amylocaine 2%	5*
Tutocaine 1%	4
Diethoxin 1%	4
Amplicaine 2%	4
Cinchocaine 1/600 adrenaline	2
Cinchocaine 1/300	2
Cinchocaine 1/600	1
Procaine 1% adrenaline	1
Procaine 1%	1

TABLE V

Drugs in approximate order of intensity of action of motor and sympathetic block, and onset and duration of anaesthesia.

Relative intensity of motor to sensory block	Intensity of sympathetic block
Amethocaine	Lignocaine, excellent
Lignocaine, Cinchocaine, Diethoxin — similar	Amethocaine, Cinchocaine, Tutocaine, Butethamine — good
Piperocaine	Amylocaine, moderate
Carbocaine, Hexylcaine, Procaine — similar	Piperocaine, variable early (10)
Butethamine, Amylocaine, Amplicaine — similar	Procaine, variable late (20)
Tutocaine	Others variable

Speed of onset	min	Duration of anaesthesia	min
Tutocaine	14	Cinchocaine	210
Diethoxin	16	Amethocaine	170
Lignocaine	16	Lignocaine	100
Procaine	16	Diethoxin	100
Amylocaine	17	Hexylcaine	90
Piperocaine	17	Tutocaine	80
Butethamine	17	Carbocaine	80
Amplicaine	18	Butethamine	80
Hexylcaine	20	Amylocaine	80
Carbocaine	21	Amplicaine	70
Amethocaine	22	Piperocaine	70
Cinchocaine	24	Procaine	55



blocks in the series, as might be expected. Although procaine appeared to give more cases of severe hypotension than the others, very large numbers of strictly comparable cases would be required to assess any difference in vasomotor blockade.

TOXICITY

After intensity of action the most important consideration is toxicity and quite a few of the formidable number of toxic effects listed by Moore (1956) were encountered in these patients. They have been presented in detail and include twelve cases from the trial series mentioned in the introduction and one case of convulsions. Broadly the reactions appear to be of two types, those occurring during or in the first few minutes after injection and those appearing after 10 minutes. Early reactions comprised sweating, vomiting, transient fall of blood pressure, shivering, rigidity of limbs, vertigo, grunting, shouting, delirium, jactitation, transient loss of consciousness, and convulsions. The case of convulsions was a thin patient aged 60, with 18 months history of prostatic obstruction, Hb 70 per cent, blood urea 45 mg per cent, and blood pressure 125/80 mm Hg. Twenty ml of 2 per cent diethoxin were injected in the third lumbar space with

TABLE VI
Data on onset and duration of anaesthesia in minutes from which the figures in table V have been obtained.

Solution	Mean time of full onset	Range of onset	Cases of recession, time from full onset m=motor; s=sensory	Longest time for painless closure	Probable duration
Procaine 2%	13	10–16	s. 30, 35, 40	70	60
Procaine 2% adrenaline ...	15	10–18	s. 65, 70		75
Procaine 1%	16	10–20			45
Procaine 1% adrenaline ...	19	15–22	s. 50, 80		65
Butethamine 1%	18	13–30	s. 55, 80	75	70
Butethamine 1% adrenaline	17	5–25	s. 60, 75, 90	85	90
Amethocaine 1/300	22	10–30		180	>180
Amethocaine 1/300 adrenaline	26	20–30		110	>200
Amethocaine 1/500	17	10–25			120
Amethocaine 1/500 adrenaline	24	17–37	s. 105, 135	140	130
Amethocaine viscous adrenaline	26	15–40		180	>200
Tutocaine 1%	13	10–18	s. 50	75	70
Tutocaine 1% adrenaline ...	16	12–25	s. 80, 95	85	90
Amylocaine 2%	16	10–25	s. 50, 68	75	70
Amylocaine 2% adrenaline	19	10–30	s. 60, 65	100	90
Piperocaine 2%	15	10–20	s. 50, 50, 60	60	60
Piperocaine 2% adrenaline ...	18	10–25	s. 70	70	85
Piperocaine 1%	17	14–22	s. 55		70
Piperocaine 1% adrenaline ...	17	12–27	s. 50, 55, 75, 80, 80		75
Hexylcaine 1%	20	15–30	s. 40, 95	80	70
Hexylcaine 1% adrenaline ...	20	15–35	s. 50, 50, 55, 70	160	115
Diethoxin 2%	17	12–30	s. 80		100
Diethoxin 2% adrenaline ...	14	8–25	s. 110	125	130
Diethoxin 1%	16	10–20	s. 60, 70, 70	85	80
Diethoxin 1% adrenaline ..	18	10–24	s. 65, 70, 100	95	95
Lignocaine 2%	16	5–25	s. 65, 75, 75, 100; m. 50, 60, 60, 65		95
Lignocaine 2% adrenaline ...	20	12–28	s. 80, 100, 110. m. 60	110	110
Lignocaine 1%	14	8–20	s 80, 90; m. 40, 85.		80
Lignocaine 1% adrenaline ...	16	10–25		90	110
Carbocaine 1% adrenaline ...	21	12–25	s. 65, 70, 85; m. 40, 60		80
Amplicaine 2%	18	12–25	s. 60, 65; m. 45		65
Amplicaine 2% adrenaline ...	19	12–25	s. 55, 60; m. 75	80	80
Cinchocaine 1/300	22	15–30		155	>200
Cinchocaine 1/300 adrenaline	27	15–50			>250
Cinchocaine 1/600	23	15–35	s. 160		180
Cinchocaine 1/600 adrenaline	22	15–30		205	220

moderate difficulty, aspiration tests being negative. During the customary pause following the first 10 ml of solution the patient began shouting and shivering. This subsided and the second 20 ml were injected. Immediately following this he became confused, recommenced shouting incoherently and within one minute he started to convulse. The first fit lasted 8 seconds, followed by relaxation and coma with deep respirations. Fits recurred at about 10-second intervals and he had six clonic convulsions before control was obtained with 250 mg of 2½ per cent thiopentone. Respiration was present throughout. Consciousness had nearly returned 20 minutes after the onset and no analgesia was demonstrable. General anaesthesia was given for the prostatectomy and recovery was uneventful. The case quoted by Bromage (1954b) was similar and he considers rapid absorption from the extradural space was responsible. Absorption giving rise to subconvulsive symptoms detailed in table VII indicates that this phenomenon appears often, and although anatomical differences may account for rapid absorption it seems that certain drugs are more dangerous than others. Whilst sensitivity to premedicating drugs cannot be excluded, experience with intrathecal spinal anaesthesia in similar patients indicates that it is uncommon and mainly confined to vomiting. Signs of early toxicity were most marked with diethoxin, amylocaine, Amplicaine, piperocaine and procaine; hypotension occurred with procaine, Amplicaine, amylocaine and piperocaine.

Lignocaine figures prominently in delayed reactions attributable to late absorption. It is impossible to say how close the blood level for coma approaches the convulsive level but this late absorption cannot be regarded as devoid of danger. Nevertheless since it is slower in onset, adequate precautions may be taken and generalized analgesia is not unhelpful in surgery. The control of toxic symptoms from procaine has been well established in intravenous therapeutic analgesia and the usual emergency facilities; 2½ per cent thiopentone, oxygen, apparatus for intubation, suction and 1/200,000 noradrenaline should be immediately available. The lowest incidences of all types of toxic reaction were found with amethocaine, Carbocaine and cinchocaine. Adrenaline tended to protect against toxic reactions but the total incidence of such reactions was not reduced by halving the concentration of lignocaine, diethoxin and procaine.

OTHER FINDINGS

In table VIII it has been shown that with increasing age a greater intensity of nerve block results from all the solutions used in these cases, considered collectively.

Bromage (1954c) noted that there was a strong association between age and dosage, less of an anaesthetic solution being required for an equivalent degree of block as age progresses, this being attributed to anatomical changes. It would be difficult to say to what extent the apparently greater spread is due to enhanced nerve penetration or nerve susceptibility, since, as the block becomes more intense so the spread appears greater, that is, hypalgesic zones become analgesic. In fact this may be the explanation for the difference noticed by Bromage (1954d) in the spread of equal quantities of 1/600 cinchocaine and 1.2 per cent lignocaine, the latter giving the more extensive anaesthesia, as it is much more powerful.

Whilst the negative pressure of the extradural space is known to vary, so also did the injection characteristics of these patients. The same needles were used in the 740 cases and ease or difficulty of injection was recorded at the time. Twelve per cent were difficult, considerable force being required and 7 per cent were easy, hardly any pressure being needed up to the end of the injection. Neither ease nor difficulty of injection was particularly associated with good or poor anaesthesia with flow back or with toxic symptoms. In 9 per cent of cases there was marked flow back into the syringe, suggestive of thecal puncture. Drip back was usual in most cases but the pressure and rate fell off rapidly. Marked drip back was usual in cases of reflux on aspiration. In one extreme case the whole of the anaesthetic solution (40 ml) could be aspirated into the syringe, as is occasionally possible when the needle point is incorrectly placed in ligament, but proof was obtained of correct entry. Great resistance with pain on injection was sometimes encountered in cases of prolapsed disc, presumably from adhesions. Because of the

TABLE VII

Systemic toxic reactions.

Numbers .....Time of onset from injection in minutes.
Numbers in brackets ..... Duration of symptom in minutes.
Coma .....Not awakened by painful stimuli.
Comatose .....Transient awakening with pain.
Dizzy .....Distressing vertigo.

Solution		Symptoms	Dose (mg)
Procaine 2%	5	Semiconscious, pulseless, pale	500
	10	Comatose	680
	12	Generalized analgesia	600
	23	Drowsy	
Procaine 2% adrenaline	40	Comatose	640
Procaine 1%	0	Shivering	400
	3	Dizzy, vomiting, hypotension	500
	7	Pale, vomiting, pulseless	500
Procaine 1% adrenaline	1	Shivering, dizzy	370
	2	Dizzy	500
	2	Dizzy, confused	440
	3	Confused, semiconscious, 15 coma (180)	400
	5	Dizzy	500
	20	Drowsy, generalized analgesia	500
Butethamine 1%	3	Drowsy (25)	500
	5	Dizzy (5) Generalized analgesia, drowsy	500
	5	Dizzy (10)	450
	7	Drowsy	500
	7	Comatose, cyanotic (20) generalized analgesia	450
	8	Comatose	350
	10	Drowsy, 20, comatose	500
	15	Drowsy	500
Butethamine 1% adrenaline	3	Dizzy, 10 drowsy	460
	3	Vomiting, 22 cyanotic drowsy (60)	450
	4	Nausea, pallor, drowsiness, 10 comatose cyanotic (20)	400
	10	Cyanotic, drowsy	400
	14	Drowsy	300
	15	Drowsy	460
	20	Drowsy	450
Amethocaine 1/300		Nil	
Amethocaine 1/300 adrenaline		Nil	
Amethocaine 1/500	15	Shivering	80
Amethocaine 1/500 adrenaline		Nil	
Amethocaine viscous	5	Dizzy	90
	25	Confused, delirious	90
Tutocaine 1%	5	Dizzy, sweating	400
	6	Dizzy, nausea	400
Tutocaine 1% adrenaline	5	Shivering, jactitation of legs	300
Amylocaine 2%	1	Sweating, transient unconsciousness, jactitation, hypotension, pallor	600
	2	Confused, restless, vomiting, sweating, hypotension	500
	15	Drowsy, pale, generalized analgesia	440

TABLE VII *continued*

Solution		Symptoms	Dose (mg)
Amylocaine 2% adrenaline	1	Vomiting	400
	2	Shivering	600
	20	Generalized analgesia	540
	20	Confused, comatose	600
Piperocaine 2%	4	Sweating, 20 generalized analgesia	500
	17	Nausea, drowsy	400
	20	Generalized analgesia	520
	20	Comatose	500
Piperocaine 2% adrenaline		Nil	
Piperocaine 1%	7	Confused, rigid, pale, pulseless, transient unconsciousness	400
Piperocaine 1% adrenaline	3	Shivering	340
	6	Sweating	400
Hexylcaine 1%		Nil	
Hexylcaine 1% adrenaline	0	Twitching of limbs, drowsy	370
	0	Shivering	400
	1	Dizzy, hypotension, 15 generalized analgesia drowsy	370
	3	Dysarthric, comatose, 10 shivering	390
Diethoxin 2%	0	Confusion, shouting, delirium, convulsion	400
	13	Transient unconsciousness, rigid, cyanotic (3), confused	720
Diethoxin 2% adrenaline	12	Nausea, 27 pallor, coma (75)	580
Diethoxin 1%	0	Shivering	500
	0	Dizzy, comatose	400
	1	Shivering	400
	2	Dizzy	450
Diethoxin 1% adrenaline	0	Shivering	400
	1	Shivering	450
	2	Shivering, dizzy, tachycardia, 10 comatose, delirious, dysarthric, cyanotic, shallow respiration	350
	4	Vomiting	400
	4	Drowsy	350
	7	Dizzy	400
Lignocaine 2%	8	Drowsy	500
	10	Drowsy, dysarthric, 25 coma (60)	480
	12	Generalized analgesia, 30 coma (40)	800
	20	Generalized analgesia, comatose	600
	35	Drowsy	540
	40	Coma (30)	500
Lignocaine 2% adrenaline	30	Drowsy	500
	40	Coma (120)	580
	40	Shallow respiration, cyanotic, 55 comatose	400
	45	Drowsy, shallow respiration, cyanotic	320
Lignocaine 1%	8	Drowsy	370
	10	Generalized analgesia, coma (80), respiration 14/min.	310
	14	Comatose, confused, twitching fingers	300
	16	Generalized analgesia	400
	20	Comatose	330
	100	Drowsy, twitching of fingers	320

TABLE VII—*continued*

Solution		Symptoms	Dose (mg)
Lignocaine 1% adrenaline	15	Drowsy, twitching of hand	360
	15	Pallor, coma (60)	400
	20	Drowsy	380
	30	Drowsy	400
Carbocaine 1% adrenaline	4	Dizzy	200
Amplicaine 2%	3	Dizzy	800
	5	Drowsy	800
	5	Dizzy	800
	6	Jactitation, grunting, transient loss of consciousness, pallor, hypotension, 20 confusion, dysarthria, tremor	800
	7	Dizzy, 15 drowsy	700
	10	Twitching of limbs	800
	10	Confusion, dysarthria, drowsiness, hypotension, vertigo, nystagmus, twitching (20)	800
	20	Dizzy (30)	500
Amplicaine 2% adrenaline	0	Shivering, twitching, dizzy	600
	6	Nausea, retching	800
	15	Drowsy (100)	800
	15	Sweating, vomiting, hypotension	800
	20	Tremor, drowsiness	700
	25	Comatose	600
Cinchocaine 1/300		Nil	
Cinchocaine 1/300 adrenaline		Nil	
Cinchocaine 1/600	0	Transient confusion, grunting, twitching	33
Cinchocaine 1/600 adrenaline	0	Transient unconsciousness (4), twitching, rigidity	67

possibility of rapid systemic absorption with toxic manifestations being due to damage to small vessels, the incidence of bloodstained reflux was of interest. Nine cases (1.2 per cent) showed reflux of small quantities of bloodstained fluid, none of them was associated with toxic symptoms. In 13 cases, thecal tap was performed accidentally and the anaesthetic solution was subsequently injected either at the same or at an adjacent space without clinical evidence of entry of the drug into the theca. Once subarachnoid anaesthesia did occur.

TABLE VIII

Age group	Number of cases	Per cent unsupplemented anaesthesias (A+B)
Up to 20	81	32
21 to 30	171	33
31 to 40	174	43
41 to 50	135	49
51 to 60	121	50
61 +	58	54

The patient, aged 40, was a well nourished fellah with bilateral ureterolithiasis secondary to bilharziasis. One per cent procaine in saline was injected in the third lumbar space, he complained of feeling dizzy, and unco-operative struggling occurred after 20 ml and a fluid jet under great pressure came from the needle. The needle was withdrawn and the patient placed supine. No analgesia developed in 10 minutes and a further 30 ml of procaine were introduced in the same space without drip back or flow back. In 15 minutes there was complete paralysis and anaesthesia to the second thoracic segment which lasted two hours. Since 1 per cent procaine does not give such a complete anaesthesia extradurally, partial entry into the theca is presumed.

Most authorities, for example Dogliotti (1939) and Pitkin (1946), rely on choice of interspace for distribution of the anaesthetic solution, but Bromage (1954e) mentions the use of a head down tilt for cephalic spread and a sitting posi-

tion to limit the upward spread. It was decided to investigate the precise extent of gravitational spread to the lumbosacral region. For this purpose cases were classified into two groups as regards site of injection and into five groups as regards downward spread. These data were abstracted from 663 cases as the other postural techniques were too few to warrant inclusion and cases of saline filling were excluded. The results are shown in table IX. The comparative intensity of block was more difficult to determine as the critical test of tolerance to intrapelvic or perineal surgery was only applicable in a limited number of conscious cases. However, it is noted that a maximum of 14 per cent were classified "S5 good". Although it would be desirable to know what is the exact expectation of sacral analgesia, equally intense as that of the lower thoracic region, obtained by optimal gravitational movement of solution, this can only be estimated as certainly not greater than 30 per cent. From "% S5" the overall influence of gravity is definite and the maintenance of the sitting position for at least 5 minutes after lumbar injection gave approximately a two-thirds chance of obtaining complete sacral hypalgesia.

By the use of the lateral position predominantly unilateral analgesia is obtained in a large proportion of cases, but quite often it was found that more intense analgesia resulted on the upper side. It is assumed that this paradoxical block is due to puncture of the extradural space to the wrong side of the midline. Since completing this investigation the author has used the Tuohy needle to provide a directional as well as a gravitational movement of anaesthetic solutions and the impression has been gained that, for maximal lumbosacral or thoracocervical distribution of the anaesthetic, the injection should be made at L4 interspace, bevel down in the sitting position or at T12 or L1 interspace bevel up (cephalic), in a steep head down lateral position, respectively. In each case the position must be maintained for at least 5 minutes.

TABLE IX

Lumbosacral penetration in relation to postural technique.

Above L3 .....Hypalgesia not reaching the front of the knee.
L3 .....Hypalgesia reaching front of knee.
L5 .....Hypalgesia to front of leg.
S2 .....Hypalgesia to back of knee.
S5 .....Hypalgesia to anal margin.
S5 poor .....Intrapelvic surgery painful.
S5 patchy .....Some roots escaped analgesia.
S5 good .....Intrapelvic surgery painless.
% S5 .....% of one postural technique with hypalgesia to anal margin.

Position	Case distribution for hypalgesia or analgesia							
	Above L3	L3	L5	S2	S5	S5 poor and S5 patchy	S5 good	%S5
Injection sitting 5 minutes sitting Puncture L1 to L4	7	13	34	28	148	38 (26%)	21 (14%)	64
Injection sitting 5 minutes lateral or supine Puncture L1 to L4	10	9	20	16	77	16 (21%)	5 (6%)	58
Injection lateral 5 minutes lateral or supine Puncture L1 to L4	10	30	42	39	87	—	—	42
Injection lateral 5 minutes lateral or supine Puncture T12 or higher	30	10	29	7	17	—	—	18

TABLE X
Volume-segment distribution of 660 cases.

Volume (ml)	Segments of hypalgesia					Mean segmental block
	5–9	10–14	15–19	20–24	25–30	
45 and greater	1	6	39	4	–	16
40–44	3	46	83	20	2	17
35–39	2	30	41	18	3	16
30–34	20	46	79	14	–	14
25–29	18	47	44	7	3	14
24 and less	16	32	30	5	1	14

TABLE XI
Volume-segment relationship of 200 cases of "B" intensity of nerve block.

Volume (ml)	Range of segmental block	Mean segmental block	No. of cases
45 & greater	16–23	19.8	13
40–44	12–23	17.6	47
35–39	10–26	15.1	26
30–34	6–24	15.4	51
25–29	6–20	13.3	38
24 & less	8–20	14.4	25

Since the descent of anaesthetic solution is variable, it is suggested that studies of the relationship between volume and segmental block might incorporate data of lumbosacral penetration as well as the extent of cephalic spread. In table X this relationship only shows a dubious general tendency for a larger volume to give a more extensive block. The fact that potency influences apparent spread has already been suggested and owing to the great range of nerve blocking activity of the different solutions, any simple relationship between volume and extent of spread might be concealed. If that were the case the potency factor could be eliminated by examining the volume-segment relationship in one category of nerve block. Category "B" was probably the most accurately delineated type of block and in table XI it is obvious that the relationship has become only a little more definite. The enormous variation makes it virtually impossible to predict the total area of blockade produced by a given volume of solution.

Employing the principle of the injection of a viscous solution to limit the extent of block as practised by German workers, Bromage (1954f), using lignocaine 1.4 per cent with adrenaline and 7.5 per cent polyvinyl pyrrolidone, found that the ultimate spread did not appear to be limited in extent. The technique of Buchholz and Lesse (1950) consists of preliminary saline filling of the extradural space until resistance to injection occurs or pressure symptoms arise, then injection of 10 ml of solution containing 6 per cent polyvinyl pyrrolidone, adrenaline 1/150,000 and amethocaine hydrochloride in a dose ranging from 30 to 120 mg according to age and type of operation. Site of puncture, from the interspace of T2 to L3 also depends on the surgical requirements. The preliminary filling with saline is intended to open up the extradural space into the intervertebral foramina and to supply a hydrostatic cushion limiting the vertical spread of the anaesthetic solution. If it is true that limitation of spread is obtained in this way, then reversing the procedure by injecting the saline after the amethocaine should cause a very much wider spread. Accordingly two series of cases were conducted. In the first, the technique of Buchholz and Lesse was followed, except that the dosage of viscous solution was kept within a narrow range; in the second, the saline filling was performed last.

The simple 1-300 amethocaine hydrochloride in normal saline with 1/200,000 adrenaline forms a control series both for segmental block and for intensity of resultant anaesthesia. As all these amethocaine solutions gave good anaesthesia, segmental block was easy to determine in the manner already described. There was no case of recession and the longest time for painless skin closure was 180, 90 and 110 minutes for preliminary saline, terminal saline and 1/300 solution, respectively. Relaxation was generally excellent for all three. The results in table XII show surprisingly little difference in the distri-

TABLE XII

Two techniques with viscous solutions and 1/300 amethocaine as control.

Solution	Mean time of full onset (min)	Mean number of segments and range	Number of unsupplemented anaesthesias	Mean dose (mg) and range	Mean volume of saline and range
Viscous amethocaine preliminary saline	28 20–40	15 9–23	13	86 60–90	78 20–120
Viscous amethocaine terminal saline	24 15–35	17 11–26	10	89 75–105	90 52–180
1/300 amethocaine with adrenaline	26 20–30	17 8–28	18	88 67–100	—

bution and degree of segmental block produced by the three techniques. There is some limitation of spread by the viscous solution preceded by saline filling and the range is less, but it is disappointing in degree. The 1/300 solution in equivalent dosage gave better results as regards intensity of block, but it would be improper to assume that this finding would be upheld by an extensive series of cases, particularly if conducted by an operator thoroughly experienced in the rather complicated German technique. Nevertheless the 1/300 solution is unlikely to give a less efficient block than the viscous solution and the extent of spread is only a little greater. It should be mentioned that the saline filling was rather a prolonged and generally unpleasant experience for the patient. In conclusion the author has the impression that the success of the German workers may not be so much due to their technique as to the fact that amethocaine is one of the two most powerful drugs available, has fewer toxic manifestations than lignocaine in equipotent doses, and gives longer anaesthesia than all the other local anaesthetics with the exception of cinchocaine. The use of doses which can be limited to 100 mg with adrenaline gives excellent and prolonged epidural anaesthesia. It is unfortunate that the drug has gained an unfavourable reputation because of absorption of toxic doses from mucous membranes in topical anaesthesia (Hewer, 1953).

It is perhaps unusual to consider the economic factor in therapeutics, but there is a tangible price difference in these drugs and prices have been calculated net per patient for purposes of comparison in table XIII. Only official names have been used except for the two preparations not yet listed, namely Tutocaine and Amplicaine.

TABLE XIII

Comparative prices of drugs.

Drug	Sterling cost per patient	Cheapest source
	s. *d.*	
Procaine	2 0	British
Butethamine	2 5	American
Tutocaine	2 6	German
Diethoxin	2 7	American
Piperocaine	2 8	British
Amylocaine	2 8	British
Amethocaine	2 9	British
Amplicaine	4 3	Swiss
Cinchocaine	5 0	British
Lignocaine	5 2	French
Hexylcaine	7 8	American

MORBIDITY

Of fourteen postoperative deaths there was one which may be attributable to the anaesthetic, there were also one reactionary haemorrhage and one case of sepsis. Brief case notes are supplied.

(1) Thin poor specimen aged 60; operation one stage retropubic prostatectomy. Forty ml 1/500 amethocaine hydrochloride with adrenaline 1/200,000 at L4 space. Hypalgesia from C6 to S5, moderate but adequate relaxation of abdomen, sympathetic block and absent sweating over whole body. At 25 minutes the B.P. fell from 110/80 to 60/50 mm Hg, poor response to methylamphetamine, nearly avascular operating field. End of operation B.P. 70/40 mm Hg. Postoperatively 5 hours 110/80 mm Hg. Eight hours after operation hyperpyrexia 105°F, absent sweating, but urine output 50 ml. On the second day the patient developed anuria; epidural block (16 ml 1/300 amethocaine) resulted in 100 ml urine. On the 7th day anuria recurred and was unrelieved. Death on the 10th postoperative day, postmortem refused. It would

appear that hypotension caused an anoxaemic nephrosis in already damaged kidneys.

(2) Tall fit man aged 50 with 6 months history of sciatica. Operation laminectomy and removal of prolapsed disc. Forty ml of 1 per cent piperocaine with adrenaline 1/150,000 epidural. Hypalgesia T3 to L3, satisfactory for operation until sacral roots stimulated and supplements given. In 30 minutes B.P. fell from 110/90 to 70/50 mm Hg. Operating field nearly bloodless from combined local vasoconstriction and hypotension. Good progress until secondary haemorrhage (250 ml) from the wound on the 2nd day. Uneventful recovery.

(3) Well nourished man aged 20 with recurrent inguinal hernia. Twenty-five ml diethoxin 2 per cent with adrenaline at T12 interspace, 1¼ inch (3 cm) deep, injection under considerable pressure and painful, no flow back. Hypalgesia T9 to L4, slight paresis right leg, operating conditions satisfactory, no supplements. Patient complained of pain in the back from time of operation but discharged on 7th day. Tenderness, swelling and redness over T12 to L2 spines was treated by private doctor with antibiotics. Readmitted to hospital in 5th week with hard red induration extending 3 inches either side of spine; aspiration gave a little pus which was sterile. Laseague negative and no apparent bony involvement.

Radiography gave a picture of marked osteoarthritis with osteophyte formation. Blood leucocytes 12,000. Induration persisted for another two months. Condition resolved by the 4th month after operation. The organism could not be identified and therefore the source of the cellulitis is a matter of speculation.

DISCUSSION

Whilst the arrangement of drugs in order of potency in table IV cannot be taken as accurate in detail, the investigation does warrant an arrangement of drugs in broad order of potency of sensory blockade on the lines mentioned in the introduction. Roughly equivalent drugs are grouped together and in brackets are two which could not be tested in 2 per cent concentration. If both sensory and motor blockade were considered, the arrangement would be much the same except that Tutocaine would come after piperocaine.

$$\text{Lignocaine} > \text{Amethocaine} > \begin{matrix}\text{Diethoxin}\\ \text{Procaine}\end{matrix} > \text{Tutocaine} > \begin{matrix}\left\{\begin{matrix}\text{Carbocaine}\\ \text{Hexylcaine}\end{matrix}\right\}\\ \begin{matrix}\text{Amylocaine}\\ \text{Piperocaine}\end{matrix}\end{matrix} > \begin{matrix}\text{Butethamine}\\ \text{Amplicaine}\\ \text{Cinchocaine}\end{matrix}$$

As adrenaline enhances nerve block, all the solutions mentioned below are assumed to contain a concentration of 1/300,000 unless stated to the contrary.

Two per cent lignocaine and 1/300 amethocaine were the only solutions which produced an epidural anaesthesia comparable in intensity to intrathecal spinal anaesthesia. The duration of anaesthesia is just under 2 hours for lignocaine from the time of full onset. For intrathecal lignocaine Adams (1956) estimates the duration to be 2 hours, but the author (Stout, 1958) found the average expectation to be about 70 to 80 minutes from studies of recession time in 100 cases of light and heavy lignocaine. The incidence of signs of absorption with 2 per cent lignocaine extradurally confirms the general preference for a 1 to 1½ per cent solution owing to its lower dosage and presumed greater safety. One per cent lignocaine was found to be similar to procaine 2 per cent but inferior to 1/300 amethocaine, the respective durations being approximately 110, 60 and >180 minutes. Whilst this investigation is far from conclusive as regards toxicity, the impression was gained that 1 per cent lignocaine was less dangerous than 2 per cent procaine in respect of hypotension; it gave much better motor block and of the two solutions there is little doubt that lignocaine provides better operating conditions. The favourable points of 1/300 amethocaine have already been referred to, particularly a greater duration and equivalent, if not better relaxation than lignocaine. Its superiority to procaine mentioned by Bromage (1954a) was fully confirmed.

Diethoxin was found to be a powerful drug next to lignocaine and amethocaine in potency but the incidence of toxic stimulation of the central nervous system was high for such a small series of cases (80).

Piperocaine, butethamine, amylocaine and Amplicaine seemed to offer no particular advantages over lignocaine, amethocaine and procaine.

Tutocaine, a drug which is sometimes classed with tropacocaine and amylocaine as a toxic, out-moded spinal anaesthetic, gave a most interesting differential block. Sparing of motor fibres occurred to a degree not exhibited by any other drug, the sympathetic block was good and toxicity of the 1 per cent solution appeared low. In eight cases when the patient was comfortable

the surgeon complained of absent relaxation during surgery.

One per cent Carbocaine was compared with 1 per cent lignocaine and it was weaker, gave less motor paresis, and appeared to last a shorter time. However, the mean dose of Carbocaine was only three-quarters that for lignocaine and, further, there was practically no toxic effect in the twenty test cases. Ekenstam et al. (1956) in a series of 135 caudal blocks with Carbocaine free of adrenaline found a longer duration than for lignocaine, otherwise the two drugs were similar. It may be that adrenaline gives a greater potentiation with lignocaine than Carbocaine.

The finding of Blundell et al. (1955) that hexylcaine is more powerful than lignocaine was substantiated as far as the 1 per cent solutions without adrenaline were concerned, but with adrenaline there was no question of the superiority of lignocaine.

It was confirmed, of course, that cinchocaine is the longest acting of the drugs tested, but its blocking action was much feebler than 2 per cent procaine and only slightly more powerful than 1 per cent procaine, the weakest of all the solutions used. As the cinchocaine used here might have deteriorated with storage in the heat, the 1/600 solution was diluted with distilled water and used as a 1/1500 intrathecal anaesthetic by the method of Howard Jones (the saline was ¼N instead of ½N). Anaesthesia identical with the well established response to "Light Nupercaine" was obtained. The 1/300 solution, the strongest used, would probably not be regarded as safe by most authorities on local anaesthesia, but even then only 7/20 unsupplemented anaesthesias could be obtained. However, its use in conjunction with light general anaesthesia for prolonged hypalgesia would be unsurpassed.

SUMMARY

The criterion of toleration of surgery without supplemental anaesthesia was used in an attempt to evaluate relative potency of twelve local anaesthetics in epidural analgesia by samples of twenty cases with each of thirty-five solutions. The two most powerful drugs were lignocaine and amethocaine. Signs of systemic absorption such as coma and generalized analgesia were prominent with lignocaine. 1/300 amethocaine gave a better block than 1 per cent lignocaine. There was a most marked difference in potency between 2 per cent and 1 per cent procaine and hypotension seemed to occur more often with procaine than with other drugs. Two per cent procaine gave a better sensory block than 1 per cent lignocaine, but a very much weaker motor block. Carbocaine appeared slightly weaker than lignocaine but less toxic. Cinchocaine gave a weak block but of longest duration. Tutocaine showed a selective block of sensory fibres. The addition of adrenaline greatly increased the potency of nine of the twelve drugs and reduced the variation in spread of the other three.

There was a tangible increase of intensity of nerve block with age. Illustrating the effect of gravity on lumbosacral penetration, the maintenance of the sitting position for 5 minutes after lumbar injection gave a 60 per cent chance of sacral hypalgesia and a 20 per cent chance of analgesia equivalent to the lower thoracic zone. The use of a viscous solution with preliminary saline plombage gave only slight limitation of spread compared with a nonviscous preparation.

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REFERENCES

Abajian, J. (1943). Peridural segmental anesthesia with Intracaine. *Anesthesiology*, **4**, 372.

Adams, B. W. (1956). Lignocaine spinal analgesia. *Anaesthesia*, **11**, 297.

Blundell, A. E., Bodall, B., Andorko, J. E., Sweeney, J. C., and Ansbro, F. P. (1955). Clinical evaluation of drugs used in obtaining lumbar epidural anesthesia. *Anesthesiology*, **16**, 386.

Bromage, P. R. (1954). *Spinal Epidural Analgesia*, 1st ed., (a) pp. 65–68; (b) p. 116; (c) p. 80; (d) p. 82; (e) p. 75; (f) p. 71. Edinburgh: Livingstone.

Buchholz, H. W., and Lesse, K. T. (1950). Die extradurale Spinalanästhesie. *Chirurg.*, **21**, 202.

Crawford, O. B. (1953). The technic of continuous peridural anesthesia for thoracic surgery. *Anesthesiology*. **14**, 278.

Dogliotti, A. M. (1939). *Anesthesia*, 1st ed., p. 537. Chicago: Debour.

Ekenstam, B. af., Egnér, B., Ulfendahl, L. R., Dhunér, K. G., and Öljelund, O. (1956). Trials with Carbocaine. *Brit. J. Anaesth.*, **28**, 503.

Hewer, C. L. (1953). *Recent Advances in Anaesthesia and Analgesia*, 7th ed., p. 193. London: Churchill.

Hingson, R. A., and Southworth, J. L. (1946). *Conduction Anaesthesia*, 1st ed., p. 681. Philadelphia: Lippincott.

Lloyd, J. B. (1955). The chemical structure and nomenclature of the local anaesthetics. *Brit. J. Anaesth.*, **27**, 286.

Lull, C. B., and Hingson, R. A. (1945). *Control of Pain in Childbirth*. 2nd ed., p. 198. Philadelphia: Lippincott.

Massey Dawkins, C. J. (1949). *Modern Practice in Anaesthesia*, 1st ed., p. 238. London: Butterworth.

Moore, D. C. (1956). *Complications of Regional Anaesthesia*. 1st ed., p. 17. Oxford: Blackwell.

Pitkin, G. P. (1946). *Conduction anaesthesia: Clinical Studies*. Philadelphia: Lippincott.

Stout, R. J. (1958). Further trials of lignocaine in spinal anaesthesia. (In preparation.)

BOOK REVIEWS

Selected Scientific Papers and Addresses (1st edition). By Ralph Waters, M.D., Published by Richard Forreger and Forreger Co. Inc.

To read through these papers of Dr. Waters is to live one's own anaesthetic life over again. With the exception of cyclopropane, which Waters introduced into practical anaesthetics himself, there is no matter in these papers which was not a subject of serious consideration among British anaesthetists. The open airway endotracheal anaesthesia, oxygen want, acapnia and other problems connected with CO_2, spinal anaesthesia, artificial respiration—these are the subjects with which Waters deals and with which our daily life was intimately concerned.

These various addresses give me the impression that Waters got more co-operation from the surgeons with whom he worked than we did in this country. Once when I took a sphygmomanometer into the gynaecological theatre the gynaecologist asked what it was, and when I told him, he said, "We want none of your —— science here." Though that was a somewhat forcible way of expressing it, characteristic of the man, it did represent the attitude of surgeons to anaesthetists during the period covered by these addresses of Dr. Waters. In my own experience there was one great exception to this sweeping condemnation, Arthur Henry Burgess. He was interested, enthusiastic, patient, and helpful.

Contemporaries of Waters already familiar with the scientific problems with which he deals will find still further interest in the human side. Should nurses give anaesthetics? Should anaesthetists collect their fees or should surgeons do it for them? How to organize an efficient anaesthetic service? These questions have a very familiar sound. Largely due to the establishment of the Association of Anaesthetists they have been satisfactorily answered in this country. The formation of this association was the work of British contemporaries of Waters and the enhanced prestige and professional and economic position of the present-day anaesthetist is largely the result of their labours, an insight into which Waters gives so excellent an account. As this is only a first edition we may expect to have more of them. This is well, because apart from an address on Snow there is no reference to chloroform, even though Waters wrote a book on the subject.

Fluid Balance Without Tears, or The child's guide to electrolytes By G. L. Bunton, M.Ch., F.R.C.S. Price 4*s.* 6*d.*

This small pamphlet deals with the body's balance sheet with regard to water and certain salts, mainly sodium and potassium chloride, and the changes that frequently ensue after surgical operations and pathological conditions involving abnormal losses of these essential constituents. Mr. Bunton says in his preface that it is "for the guidance of students and housemen"! It is certain that there will be fewer occasions for tears if these students and housemen follow the advice given than if they fail to do so. It is perhaps too much to expect that there will be no tears. The second part of the title, *The child's guide to electrolytes*, suggests that the dose of precocity normal to each rising generation as it comes along has been somewhat unexpectedly enlarged. It is for these that the text is illustrated, presumably.

E. Falkner Hill