References

1. SMITH NT, WESSELING KH, DE WIT B. Evaluation of two prototype devices producing noninvasive, pulsatile, calibrated blood pressure measurement from a finger. *Journal of Clinical Monitoring* 1985; **1:** 17–29.
2. WESSELING KH, SETTELS JJ, DE WIT B. The measurement of continuous finger arterial blood pressure noninvasively in stationary subjects. In: SCHMIDT TH, DEMBROSKI TM, BLÜMCHEN G, eds. *Biological and psychological factors in cardiovascular disease*. Berlin, Heidelberg: Springer-Verlag, 1986.
3. GRAVENSTEIN JS, PAULUS DA, FELDMAN J, MCLAUGHLIN G. Tissue hypoxia distal to a Peñaz finger blood pressure cuff. *Journal of Clinical Monitoring* 1985; **1:** 120–5.

Fluid flow through dural puncture sites

We read the paper by Cruickshank and Hopkinson[1] with interest. We too have performed a similar study of flow through cadaveric dura which we would like to present for comparison, because it differed in some aspects.

Samples of lumbar dura were obtained from the pathology department of the Freeman Hospital in Newcastle. A segment of the dura was mounted across the cut body of a 2 ml Becton Dickinson syringe, and a waterproof seal obtained using cyanoacrylate glue and adapted small cable hose clips. One sample of dura could yield up to 10 segments. The dura was kept moist at all times. The chamber of the syringe was then filled with oxygenated physiological saline and the preparation immersed in a water bath that also contained physiological saline at 35–37°C. The syringe was connected to a reservoir of saline via a Travenol blood-giving set at a pressure of 3.0 kPa measured using a manometer between the reservoir and specimen. The relatively high pressure was used in order to simulate the loss of fluid in the upright position, when headache is usually worse.

The dura was then punctured using either a 22- or 25-gauge Quincke-type spinal needle and the flow measured by counting the number of drops per unit time in the chamber of the giving set. A total of 10 samples were tested, five for each size of needle, with half the specimens from each sample being used for each bevel orientation. The flow was measured immediately after puncture, at 20 minutes, 24 hours and 48 hours after puncture in order to try and observe the loss of fluid over the time period at which headache usually occurs.

The results for puncture sites remaining patent at testing are shown in Table 1. Our results after initial puncture and 20 minutes later were similar to those found by Cruickshank and Hopkinson, and confirm the importance of needle size and the relative unimportance of needle bevel orientation in the initial period. There was also little difference in flows between the two orientations after 24 and 48 hours, although slightly fewer punctures were patent after puncture with the bevel parallel to the fibres. However, this was not found to be statistically significant.

Table 1.

Needle gauge and orientation	Percentage of punctures still patent			
22 parallel	100	97	15	9
22 horizontal	100	95	24	12
25 parallel	100	86	10	2
25 horizontal	100	90	15	5
	0	20 minutes	24 hours	48 hours

The lack of difference between the orientations is surprising in view of the strong clinical evidence in the difference in incidence of headache. Mihic[2] showed the effect of altering the needle bevel orientation to be of greater importance than needle size, with a reduction in the incidence of headache from 17.24% to 0.71% with 22-gauge needles, and from 15.15% to 0% with 25-gauge needles. We can only conclude that either loss of CSF is not the prime cause of post lumbar puncture headache, or that this experimental model does not reflect the *in vivo* state at all.

Newcastle General Hospital, Newcastle upon Tyne NE4 6BE

E.F. JANES
S. PENNEFATHER
K. WILKINSON

References

1. CRUICKSHANK RH, HOPKINSON JM. Fluid flow through dural puncture sites. An *in vitro* comparison of needle point types. *Anaesthesia* 1989; **44:** 415–8.
2. MIHIC DN. Postspinal headaches needle surfaces and longitudinal orientation of the dural fibers. Results of a survey. *Regional Anesthesia* 1986; **9:** 54–6.

Weight-determined dosage of atracurium besylate

Drs Harrison and Gunn (*Anaesthesia* 1989; **44:** 692) have suggested that doses of muscle relaxants should be determined by the fat-free mass rather than gross body weight from their experience with vecuronium bromide.

We found that the volume of distribution at steady state (VdSS) and clearance of atracurium were less in morbidly obese subjects.[1] All patients received atracurium on a mg/kg basis as determined by the gross body weight. The onset of neuromuscular blockade was faster, but its duration was not significantly longer in the obese individuals. The plasma concentrations corresponding to 50% recovery (Cp50) were 52% greater in the obese patients.

Neuromuscular relaxants such as atracurium are ionised compounds whose volume of distribution approximates to extracellular fluid volume (ECF). The smaller ECF volume per kg body weight in obese patients probably accounts for their smaller volume of distribution. However, obese individuals in our study required a higher plasma concentration of atracurium to attain the same degree of neuromuscular blockade as in lean individuals. This effect would counteract the alteration produced by changes in volume of distribution. We therefore concluded that obese individuals would require comparable doses of atracurium, on a mg/kg basis as subjects with normal body weight. It

may hold true for atracurium in obese subjects, but it remains to be shown for the other relaxants, such as vecuronium bromide.

Medical School,
Beech Hill Road,
Sheffield S10 2RY

S.S. GILL

Reference

1. DUCHARME J, DONATI F, GILL SS, VARIN F, BEVAN DR. Pharmacokinetics and pharmacodynamics of atracurium in morbidly obese subjects. *Canadian Journal of Anaesthesia* 1989; **36: 3** (part 2): S149.

Propofol and atracurium in familial periodic paralysis

This is a report of the successful use of propofol and atracurium for two operations on a patient with familial periodic paralysis. The 26-year-old woman was admitted in the first instance for bilateral antral lavage. She had suffered from many episodes of periodic paralysis since infancy, a few of which were very severe. Five other members of her family and one of her daughters also suffered from the condition. One of her sisters had once required controlled ventilation of the lungs after an operation. The serum potassium levels of this woman and the other members of her family were normal during attacks. She was perfectly well at the time of admission and the laboratory investigations were normal. She received diazepam 15 mg, orally as a premedicant. Propofol 2.5 mg/kg, atracurium besylate 0.5 mg/kg and morphine 3 mg were given intravenously. The trachea was intubated and the lungs ventilated; anaesthesia was maintained with nitrous oxide, oxygen and isoflurane. Muscle relaxation was reversed at the end of the operation with neostigmine, 2.5 mg and glycopyrronium 0.5 mg. She was transferred to the intensive therapy unit for observation. Recovery was uneventful.

Eight months later the woman was readmitted for septal reconstruction, turbinate reduction and bilateral radical antrostomies. She received the same anaesthetics and again recovery was uneventful.

Familial periodic paralysis is an hereditary condition thought to be transmitted by an autosomal dominant gene. It is characterised by recurrent attacks of muscle weakness or flaccid paralysis occasionally associated with abnormality of the serum potassium. The skeletal muscles are affected and the bulbar muscles spared. Attacks may be precipitated by emotional excitement, high carbohydrate intake, severe exertion, cold, infectious diseases and accidental or surgical trauma. Thiopentone and muscle relaxants are also implicated.

South Infirmary,
Cork,
Ireland

J.F. WALSH
M. SIDDIQ

Cardiac arrest during laparotomy

We read with great interest the letter written by Drs Doyle and Mark (*Anaesthesia* 1989; **44:** 448). We encountered a similar occurrence recently during a total abdominal hysterectomy performed on a generally healthy woman of 46 years. She showed signs of extreme anxiety before anaesthesia despite diazepam 10 mg orally. She received 100 μg fentanyl and 0.25 mg vecuronium for pre-induction priming. Tracheal intubation was performed without any difficulties after injection of 250 mg sodium thiopentone and 8 mg vecuronium. Maintenance was continued with 70% N_2O in oxygen. Monitoring was by means of ECG, noninvasive automatic blood pressure, capnometer, pulse oximeter, and nerve stimulator to the ulnar nerve. Tears were observed in the patient's eyes at the first incision, and therefore a further dose of 100μg fentanyl was given. Shortly afterwards, when the uterus was being clamped by the surgeon there was a sudden decrease in pulse rate, from 80 to 50 beats/minute. Atropine 1 mg was immediately injected, but the bradycardia persisted until asystole was observed. The pulse oximeter ceased to demonstrate activity at the same time. The operation was immediately suspended, and another 1 mg atropine was injected. There was no immediate improvement so closed cardiac massage was started together with manual artificial ventilation of the lungs with 100% oxygen. Simultaneously 1 mg adrenaline was administered intravenously. The first ECG signs appeared shortly afterwards, which rapidly developed into tachycardia of 120 beats/minute; the blood pressure was now 130/80 mmHg.

The patient stabilised so it was decided to continue with the operation and, until the end of the procedure, anaesthesia was maintained with 0.6% halothane, 50% N_2O in oxygen, with the addition of fentanyl and vecuronium as necessary. There were no further problems during the remainder of the surgery or in the postoperative period. No signs were found of brain or cardiac damage, and the patient made no mention of awareness during the anaesthesia.

There is a similarity in our case to that of Doyle and Mark. Our patient also underwent an abdominal surgical procedure, did not receive anticholinergic drugs before the vecuronium, and responded with cardiac arrest during strong, painful stimulation under light anaesthesia. Contrary to the case cited, we continued with the anaesthesia after the resuscitation.

The absence of further complications emphasises the benign nature of vagally mediated cardiac arrest.[1] The bradycardia appeared shortly after administration of fentanyl and probably indicates a pharmacological interaction between this drug and vecuronium.[2]

Despite all the innovations and improvements in pharmacology, and in the use of sophisticated monitoring devices, our conclusion from this case is that it is essential to continue to observe the basic principles of anaesthesia.

Rambam Medical Centre,
Technion Institute,
Haifa, Israel

M. RUS
B. ROSENBERG
H.J. BIRKHAHN

References

1. POLLOK AJP. Cardiac arrest immediately after vecuronium. *British Journal of Anaesthesia* 1986; **58:** 936–7.
2. INOUE K, EL-BANAYOSY A, STOLARSKI L, REICHELT W. Vecuronium induced bradycardia following induction of anaesthesia with etomidate or thiopentone, with or without fentanyl. *British Journal of Anaesthesia* 1988; **60:** 10–7.