

by the immediate insertion of chest tube drains, the establishment of either a subclavian venous access (by casualty personnel) or an internal jugular line by the anaesthesia department. Tamponade to us is the greatest risk, and we delay the induction of anaesthesia until the patient and surgeon are ready for instant chest opening.

The singular inattention to seat-belts, and the high intake of alcohol amongst the general population, leads to many blunt chest injuries, and not a few penetrating injuries like the one illustrated in the enclosed photograph. Here, the young man was impaled by a fence rail of dimensions 6 inches by 3 inches. It penetrated to the right of the sternum, passed completely through the thoracic cage, emerged from the rear, passed through the car seat, and caused a splenic rupture in the rear seat passenger. In the photograph, the stake may be seen both anteriorly and posteriorly, Miraculously, no major vessel damage occurred, the patient made a speedy recovery, and has no external evidence of the horrific presenting picture save a few skin scars.

Should Glasgow be short of cases, we would be delighted to forward a few for their care!

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Mr I Reece and Mr K G Davidson in their paper 'Emergency surgery for stab wounds to the heart' (*Annals*, September 1983, vol 65, p 304) point out the value of immediate anterolateral thoracotomy for the relief of tamponade in their group 3 patients ('apparently dead', unconscious, absent pulses, dilated pupils, no recordable blood pressure).

Immediate thoracotomy is also of value in the hypovolaemic traumatised patient with cardiorespiratory arrest. External cardiac massage is ineffectual in the hypovolaemic 'empty' heart (1). Internal massage enables maintenance of cerebral circulation and therefore brain function and in addition this can be enhanced by distal thoracic aortic occlusion. This dramatic procedure, especially when undertaken in the resuscitation room in casualty can yield extremely rewarding results.

Baker *et al.* record the results and value of emergency room thoracotomy for cardiorespiratory arrest in 175 traumatised patients. Correlation between admission status and outcome of thoracotomy after cardiorespiratory arrest revealed the following survival rates. No vital signs 6.6%, Agonal 20%, Profound shock 34.1%, Mild shock with subsequent deterioration 56.3%. Overall, the survival rate was 19.6%. If patients with irreversible head injuries are eliminated, the survival rate was 24%.

The best results were achieved where the major site of injury was the lung 57.1%, heart 27.7%, hilar vessels 25%. However, 5 of the 33 survivors had their major injuries in the abdomen.

Undoubtedly emergency thoracotomy in casualty can be lifesaving for the patient in extremis. Mr Reece and Mr Davidson point out the value of this procedure in cardiac wounds. However, its use should be extended to other traumatised patients and its value and technique taught to surgeons in training.

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Reference

- 1 Baker C, Thorn A, Trunkey D. The role of emergency room thoracotomy in trauma. *J Trauma* 20:848-54.

Unoperated abdominal aortic aneurysms: presentation and natural history

I must admit that I still feel a great excitement regarding this subject even though I am no longer personally involved in either research or in the clinical situation of aortic aneurysms. I thoroughly enjoyed the recent paper on the subject (*Annals*, September 1983, vol 65, p 311) and am pleased that the findings and conclusions are similar to my own (1). As I commented in a previous letter (2) there is also a need to have some non-invasive method of identifying and assessing aneurysms before rupture. Clinical examination and ordinary X-ray examination for calcification are by no means diagnostic or accurate in every case. Following clinical, pathological and ultrasonic studies, I put forward a suggestion that ultrasonic measurement of the transverse diameter of an abdominal aneurysm (rather than the antero-posterior diameter) offered a most useful clinical method of monitoring size and likelihood of

rupture (3). Whether anyone else can confirm this or offer an alternative thesis would be interesting and could perhaps be of significant practical importance.

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References

- 1 McGregor JC. Unoperated ruptured abdominal aortic aneurysm. *Br J Surg* 1976;63:113-16.
- 2 McGregor JC. Letter. *Ann R Coll Surg Engl* 1980;62:485.
- 3 McGregor JC. Abdominal aortic aneurysms. Ultrasonic measurement of the transverse diameter and its prognostic significance in the light of pathological observations. *Postgrad Med J* 1977;53:737-40.

Anaesthesia for laparoscopy: alfentanil and fentanyl compared

I would like to make a few comments on the above article by B Kay *et al.* (*Annals*, September 1983, vol 65, p 316). A comparison was drawn between these two drugs regarding, among other things, the time for return of spontaneous respiration following reversal of neuromuscular blockade. The time for alfentanil was 16.3 ± 4.7 s and that for fentanyl was 69.4 ± 15.2 s. A cursory glance might lead one to expect a four fold difference in the times to recovery of spontaneous respiration using the regimens as stated. The reference points are taken as from the start of the tachycardia following neostigmine and atropine to the point where respiration commences. The tachycardia is in fact due to the unopposed action of atropine, the effect of neostigmine coming on somewhat later, certainly more than 16.3 s later (1,2). Thus the onset of spontaneous ventilation cannot be assumed to be as a result of antagonism of neuromuscular blockade. The 10 mg alcuronium would probably be wearing off by this time. It would therefore be as logical to take the time of injection of reversal agents as the first reference point and thus the figures would then look somewhat different.

The authors also state that ventilation was reduced towards the end of the operation to allow pCO_2 to rise. Was there always an identical interval between this event and reversal? A useful addition to the method would have been a recording of end-tidal CO_2 . In this case reversal could have been given at a set level of CO_2 . Without this addition how do the authors know that the levels of CO_2 at the time of reversal was comparable? Additional information would have been gained about the relative shifts in the CO_2 response curves as a result of the residual blood-opiate concentrations.

Without these data, I feel that the results as presented are a little misleading although there is obviously a significant difference between these two regimens.

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References

- 1 Mirakhor RK, Dundee JW, Jones CJ, Coppel DL, Clarke RSJ. Reversal of neuromuscular blockade: dose determination studies with atropine and glycopyrrolate given before or in a mixture with neostigmine. *Anesth Analg (Cleve)* 1981;60:557.
- 2 Hammond J, Wright D, Sale J. Pattern of change of bronchomotor tone following reversal of neuromuscular blockade: a comparison between atropine and glycopyrrolate. *Br J Anaesth* (in press).

B Kay, A T Cohen, F Shaw and T E J Healy reply as follows:

We agree that the interpretation of measurements made from the time of onset of tachycardia may lack precision, but the onset of tachycardia is a patient event that can be identified exactly. It was considered that the use of this to establish a baseline time measurement would reduce the effect of variations in the duration of the period between injection and onset of action of the reversal drugs. Several patients did breathe spontaneously at this time, but this time marker has at least equal validity with the time when the reversal agents were injected for those patients incapable of breathing spontaneously until neuromuscular block was reversed.

Ventilation was reduced towards the end of the operation to allow a rise in PCO_2 to provide a stimulus for patients to breathe. No attempt was made to standardise $F_{ET}CO_2$. The manoeuvre is more difficult and time consuming than it might appear on first