LETTERS TO THE EDITOR

RECONSTRUCTION OF LARYNGOTRACHEAL WAR INJURIES WITH THE MEDIAN LAYER OF THE DEEP CERVICAL FASCIA
From Major JDC Bennett, RAMC and Professor SC Guha

Sir, We would be grateful if you would consider this letter in response to the paper which appeared in your February edition by D Danic et al (Reconstruction of laryngotracheal war injuries with the median layer of the deep cervical fascia. J R Army Med Corps 1995; 141: 16-19).

Trauma and the Larynx
Laryngeal trauma was one of the subjects covered at a day seminar held at the Royal Army Medical College 12 July 1994, when it was affirmed, as the authors acknowledge (JR Army Med Corps 1995; 141: 16-19), that it is difficult to develop treatment protocols in injuries which vary and are rare. There were, over a 23 year period, only 120 laryngeal injury patients at a Level One Trauma Centre in Dallas which sees over 150,000 casualties a year (1). This makes expertise difficult to acquire; as yet no SAC committee has set a minimum number of cases a senior registrar must treat before being accredited! The reasons it accounts for less than 1% of the injuries seen in most major centres are rapid death from asphyxiation, anatomic protection by the mandible and sternum, and failure to recognise laryngeal trauma in a patient suffering multiple injuries.

Types Of Injury
Injuries to the larynx are mostly closed, being caused by blunt trauma such as karate kicks or deceleration injuries. Typically, a car occupant strikes the wheel or dashboard with the extended neck. “Clothesline” injuries are also seen, and were the reason for military vehicles in Northern Ireland having a post fitted in front of the command hatch. In contrast, the authors describe war injuries. This distinction must be remembered, as many non-penetrating injuries do not require open surgical intervention (2). That does not mean to say that diagnostic procedures are not necessary. Failure to recognise an oesophageal injury may lead to mediastinal sepsis and tracheoesophageal fistula. The larynx should be examined endoscopically to look for exposed of fractured cartilages, damage to vocal cords, or other signs indicating damage.

Management
There is controversy as to whether tracheostomy under local anaesthetic or orotracheal intubation is more likely to cause iatrogenic injury. In a study of 21 patients seen over three years, there were 12 who received penetrating trauma (comprising 5 gunshot and 7 stab wounds). Active airway control was needed to 18 (90%), and this was either by intubation (67%) or emergency tracheostomy (33%) (3). Surgical exploration is indicated in any neck injury where there is stridor, dysphonia, cartilage disruption, or swelling of the neck from bleeding or emphysema. This is generally by thyrotomy, consisting of an anterior incision with midline division of the thyroid cartilage and separation of the vocal cords at the anterior commissure. Fractures can be splinted over a modified attempt to reduce and hold by multiple sutures alone may result in stenosis and malformation. In the report of 8 cases mentioned, such molds or stents were placed in six. The decision was based on the severity of the tracheal ring injury or the loss of cricoideal structural integrity. The stents were fashioned from finger cots over sponges secured with transcutaneous sutures and removed endoscopically at two to six weeks. Indwelling laryngeal keels were required in four to prevent web formation when vocal cord disruption had occurred. They were formed from Silastic sheet and secured with transcutaneous wires. It is difficult to see how these methods can be satisfactorily replaced with support by a fascial layer. In order to lessen the likelihood of chondritis, exposed cartilage should be covered by resutting the mucosa that has been avulsed and remains attached to the distal airway (4). This appears much more amenable to fascial substitution.

The Nature Of The Median Layer Of Deep Cervical Fascia

Fig 1a + 1b. Diagram of pretracheal or median layer of deep cervical fascia
The terminology of the many fascial layers and spaced within the neck is complicated, and varies with time and between countries. The cylindrical visceral compartment of the neck is surrounded by a thin layer of fascia. This encloses the strap muscles and fuses in the midline over the thyroid gland. It is also known as the pretracheal fascia. Posteriorlaterally, it fuses with the carotid sheath. It blends below with the capsule of the thyroid gland and attaches above to the hyoid bone. This is the anatomical basis of the movement of the thyroid gland during swallowing. The portion of visceral fascia around the strap muscles has also been called the middle cervical fascia (5). We dissected the laryngeal area in a series of fresh autopsy specimens. It can be seen that the fascia is rather flimsy and would seem to serve better as a substitute for laryngeal mucosa, but nothing more substantial such as cartilage.

Fig 2. Dissection of median layer of deep cervical fascia/pretracheal fascia of fresh cadaver

The timing of the operative repair is important as infection, drying, overflow of secretions are likely to make the fascia less suitable. It was not used in a series of six cases of laryngeal bullet wound injuries which received delayed repair; of these, three (50%) required a laryngectomy (6).

Simply excision of the damaged area is sometimes a possibility. If the cricoid is so badly injured that it is beyond repair, the uppermost tracheal ring may be anastomosed to the thyroid cartilage (7). Up to 6cm of cervical trachea can be resected and repaired by end to end anastomosis. Tension is removed from the suture line by division of the thyroid muscles (8). It can be seen that, owing to a combination of the relative rarity of penetrating laryngeal trauma causing lack of experience and hence expertise, innovation has not been hitherto forthcoming. The authors describe excellent results, based on a technique which, on theoretical grounds, has little to recommend it. This may well be one of those situations where medical advances are made from war.

HEAT ILLNESS AND CYSTIC FIBROSIS
Major MCM Bricknell, RAMC

Sir, Two recent papers (1,2) have suggested that the cystic fibrosis gene might contribute to the pathogenesis of heat illness. The prevalence of cystic fibrosis is approximately 1:2500 with the heterozygote state occurring in about 1:25 Caucasians. Should the Services screen for the heterozygote or the homozygote state or cystic fibrosis to reduce heat casualties?

The presentation of cystic fibrosis as recurrent heat exhaustion (1) has to be extremely rare compared to the familiar respiratory, endocrine and gastrointestinal manifestations. Thus testing for the homozygous state in healthy service personnel would have an extremely low true positive detection rate and would not be effective.

It is suggested that heterozygotes for the cystic fibrosis gene could have saltier sweat compared to non-heterozygotes and thus these individuals may be at risk of salt-deficiency heat exhaustion in hot climates (2). Screening for heterozygotes is now technically feasible (3).
Could and association between the heterozygote state and heat illness be demonstrated?

A case-control study might be conducted to compare the prevalence of the heterozygote state in soldiers who develop heat illness with the prevalence in a matched military population. It is unlikely that known confounders eg. alcohol, concurrent illness, inadequate acclimatisation, inappropriate clothing could be avoided.

A positive case-control study would demonstrate an association between the heterozygote state and heat illness but it would not be possible to determine the magnitude of the increased risk of heat illness. This could only be achieved by a longitudinal study. The low absolute incidence of heat illness in the Army (73 per 100,000 (4) makes this study impossible to undertake.

Even if heat illness is diagnosed should an individual be tested? The low incidence of heat illness in servicemen combined with the high prevalence of the heterozygote carriers state suggests that the heterozygote state is not a significant factor in the development of heat illness. Thus being a heterozygote carrier would have very little positive predictive value for the risk of developing heat illness in the future even if the case-control study showed an association. Therefore there would be no justification to restrict the employability of heterozygote servicemen and thus the test is of no value.

Finally there are significant ethical problems associated with genetic testing. The issues have already been identified for screening the healthy population for heterozygote status in relation to pre-pregnancy and antenatal care (3). The ethical implications of such testing in relation to employment also need to be very carefully examined.

I suggest that testing servicemen for the cystic fibrosis gene as a means to reduce heat casualties is unlikely to be worthwhile.

I am etc

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2. HOWORTH PJN. The biochemistry of heat illness. JR Army Med Corps 1995; 141: 40-41.
4. DICKINSON JG. Heat illness in the services. JR Army Med Corps 1994; 140: 7-12.

EXERCISE ECG’S
From Dr I Perry, MFOM, DAvMED, FRAeS

Sirs, I read with interest the results of the first 10 years of Maximal Exercise Testing as part of the extended Pulheems examination. JR Army Med Corps 1995;141: 71-74.

This particular test has now been dropped as part of the protocol for routine follow-up in all professional pilots covered by the Joint Aviation Authority of Europe (1) due to the extremely high rate (10%) of false positives.

Thallium scanning and in some cases Angiography have had to be used to prove that the exercise test was false. The only criteria when exercise testing will be carried out is when there is an abnormal resting ECG, where there may be symptoms suggestive of Angina Pectoris, or at the discretion of a specialist in Aviation Medicine.

I recently have been obliged to carry out maximal exercise testing for the Japanese Aviation Authorities who were recruiting Europeans as Pilots. It will be some time before I can publish a full analysis of all the results, but I can say that of 188 with normal resting ECG subjects of a mean age of 45.8 years the false positive rate was 12%. (The Extended Pulheems rate was 25%)

Twenty of my survey went on to Thallium Scanning, whom eighteen were normal. Two went on to Angiography and as expected gave normal results. The other two dropped out, as I found that they were taking anti-hypertensives. A drug screen was carried out on a urine sample. Both denied taking anything, but as the Authorities would not accept their positive exercise tests the selection procedure stopped.

It seems we now have a difference in philosophy. In the JAA, exercise testing will only be done when clinically indicated. Other agencies still seem to regard maximal exercise testing as a screening tool regardless of any symptomaticology, costs and the high false positive rate.

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REFERENCES