

LETTERS TO THE EDITOR

Surgical treatment of carcinoma of the oesophagus and Laurence O'Shaughnessy

I read, in Mr Raymond Hurt's interesting historical review (July 1991;46:528-35), that Professor Ferdinand Sauerbruch had been given a pass to cross the British lines around Dunkirk in 1940, to try to save the life of Mr Laurence F O'Shaughnessy, who had been wounded in the chest. As I was 12 years old when my mother and I left our burning home in Dunkirk in May 1940 and as I am now a sexagenarian thoracic surgeon, everything that concerns both the battle of Dunkirk and the history of thoracic surgery is of interest to me. I wrote to Mr Hurt, who confirmed that his information was based on GG Jamieson's *Surgery of the Oesophagus*, and more precisely on a chapter written by Professor RG Elmslie, professor of surgery at Queen Elizabeth Hospital in Woodville, Australia.¹ The phrase reads: "The thin link between Sauerbruch and British oesophageal surgery started between the first and second world wars with O'Shaughnessy, who died of chest wounds on the beaches of Dunkirk before his old chief, Sauerbruch, given safe conduct through the British lines, could reach him."

I have been interested for a long time in the circumstances of the death of a brilliant young British thoracic surgeon, whose curved dissector we all use. I personally insist on its eponym as a daily mark of respect for the memory of a man who had started, in the 1930s, the surgical interest in coronary disease as well as in oesophageal cancer.

In the Imperial War Graves Commission's register of the names of those who fell in the 1939-45 war and have no known grave his name is written. He died on 27 May 1940, which is the very day when the British Expeditionary Forces were ordered to fall back on the Yser river line. I had been told years ago that Mr O'Shaughnessy was hit on

the beach while helping wounded men to embark into rescue boats and that he slumped into the water at once and disappeared. These circumstances are probably not correct, as he died on the day before the embarkation started from the beaches, either in the town or in the harbour. What is certain is that his name is engraved on Dunkirk's war memorial consecrated to soldiers whose bodies were never recovered (figure).

If his body remained lost, I doubt very much that Mr O'Shaughnessy had received medical care or that Professor Sauerbruch was called to help, even if in that tragic chaos a message could have been sent from Dunkirk to Berlin, received, answered, and transmitted to the allied troops around the town. In his memoirs,² Professor Sauerbruch does not mention the fact. Considering how he was keen on reminding the reader of his international links and deeds, being more discreet about his relations with the German Government, this is surprising. He writes about coming from Berlin to Holland, Belgium, and France later in June, to inspect military hospitals. There are proofs of links between surgeons from opposite belligerent countries during the two world wars, particularly through Switzerland; but most of them concerned professional information, especially on abdominal and brain wounds.

Perhaps Mr O'Shaughnessy's body was never found; perhaps it was unidentified and lies in one of those many graves where are written the moving words "Known unto God." In any case, may this letter say to British surgeons that their promising young colleague has not been forgotten in France and to British people that a war comradeship, twice sealed in blood, tears, and mud during a quarter of a century, is vivid in our hearts and must be remembered at a time when we are trying to build Europe together.

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- 1 Elmslie RG. Perspectives in the development of oesophageal surgery. In: Jamieson GG, ed. *Surgery of the oesophagus*. London: Churchill-Livingstone, 1988:3-8.
- 2 Sauerbruch F. *Mes souvenirs de chirurgien* (translated by Forsletter M). Paris: Denoël, 1952:257-60.

Pathological assessment of mediastinal lymph nodes in lung cancer: implications for non-invasive mediastinal staging

We read the article by Dr K Kerr and his colleagues (May 1992;47:337-41) with interest. We would like to reinforce the serious reservations raised about this paper by Mr David Kaplan in his editorial (p 332). The essential thesis of the paper appears to be that the size of mediastinal lymph nodes does not aid prediction of metastases from carcinoma of the lung. We would like to make three points: (1) The selection criteria for those patients studied has already excluded most patients with malignant invasion of the mediastinum and presumably larger lymph nodes and there is also a noticeable absence of smaller normal sized nodes in their sample. It is not surprising that the remaining patients do not show a correlation between size and malignancy. It has also long been known that 10-15% of normal sized lymph nodes contain metastases and are not detected by mediastinoscopy or computed tomography. (2) Although the authors are at pains to point out which node stations were sampled, they have not related this to the expected chain of spread of each tumour. (3) Most importantly, they have measured the wrong lymph node diameter. Computed tomography images in the axial plane cut nearly all lymph nodes through their short axes. Assessment of lymph node size is made from the *smallest* diameter. This is sensible when the normal elongated ovoid shape of a lymph node is considered. The width will increase in malignancy before the length and may be measured by computed tomography. The authors' use of the *maximum* diameter renders extrapolation of their results to preoperative staging erroneous.

Overall, we believe that this paper is misleading and that the authors have no grounds for their comments on mediastinal computed tomography. Better studies¹ have consistently shown the value of preoperative imaging.

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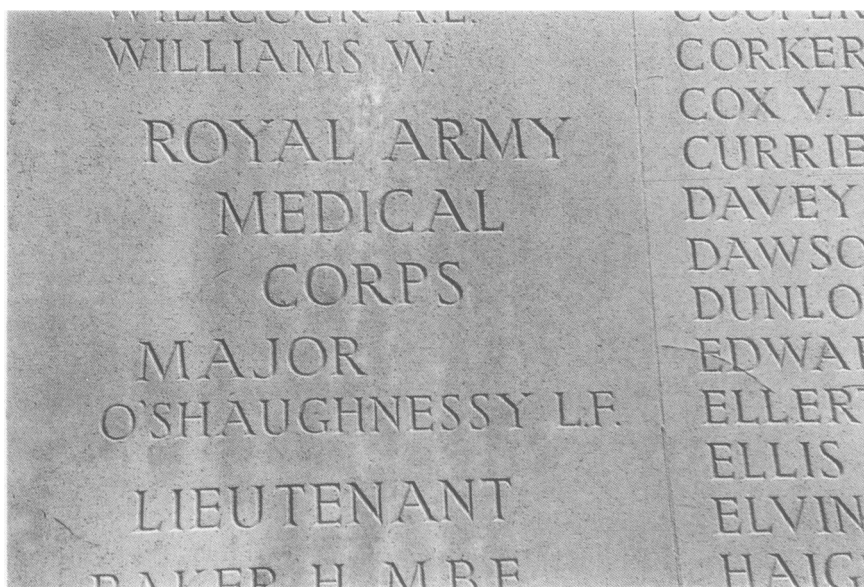
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- 1 Buy JN, Ghossein MA, Poirson F, Bazot M, Meay E, Malbec L, et al. Computed tomography of mediastinal lymph nodes in non-small cell lung cancer. *Journal of Computer Assisted Tomography* 1988;12:545-52.

AUTHOR'S REPLY Concern has been expressed about the selection of patients in our study. The statement by Drs Weston and Goddard that we "excluded most with malignant invasion of the mediastinum and presumably larger lymph nodes" is categorically untrue. Only patients with unequivocal mediastinal adenopathy on plain chest radiograph were excluded by the referring physicians and thus were not entered into the study. As the authors will be well aware, such patients represent a small fraction of individuals with mediastinal adenopathy and would not be expected to skew our data significantly for mediastinal nodes in the range 5-45 mm. Furthermore, as we emphasised in the paper, it is in our very group of "potentially operable" patients that lymph node histology is relevant and we do not accept that this biases our results.

The number of lymph nodes obtained per patient is perhaps low, but this is the number



Mr O'Shaughnessy's name on the Dunkirk memorial.

that we could identify at surgery. For this to have biased our results one would have to assume either that we selectively sampled small lymph nodes that were malignant or that even smaller nodes would have been more likely to be benign. The former is not true and the latter unproved and certainly not supported by the remainder of the data in our study.

Node station data were included to give the reader some impression of where in the mediastinum the lymph nodes originated. The lack of data relating lymph node station to site of primary tumour in no way detracts from any of the conclusions in our study.

We do not accept that we have measured the wrong lymph node diameter. Computed tomography measurements are made in the shortest diameter as this value is the one least likely to be artificially inflated by oblique cuts on the scan. Mediastinal lymph nodes are haphazardly orientated and also vary widely in shape. In addition, the relationship between increasing lymph node width and length put forward by Drs Weston and Goddard is a gross oversimplification. Lymph nodes do not enlarge symmetrically and their asymmetrical expansion is not constant in pattern. Important variables include the location of the malignant deposit within the node, which part of the lymph node (if any) is enlarged by the malignant deposit, and how much of the node is replaced by tumour. Computed tomography measurements of the shortest diameter represent a simplified score of lymph node size, specifically designed to eliminate artefact and minimise differences between the angle of section of lymph nodes. In contrast, we had access to the whole lymph node and thus were able to make actual measurements that were representative and repeatable and—most importantly—allowed comparison between the benign and the malignant group. Measuring maximum diameter in no way invalidates our conclusions, and we stated quite clearly in our paper that direct comparison between published computed tomography measurements of nodes and our data were not intended and that that this would be erroneous.

Drs Weston and Goddard quote selectively a study that has been performed directly to investigate the value of mediastinal computed tomography. Several recent studies,¹⁻⁴ including one of our own,³ have indicated that preoperative computed tomography is a far from perfect approach to staging the mediastinum in lung cancer.

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- Whittlesey D. Prospective computed tomographic scanning in the staging of bronchogenic cancer. *J Thorac Cardiovasc Surg* 1988;95:876-82.
- McKenna RJ, Libshitz HI, Mountain CE, McMurtrey MJ. Roentgenographic evaluation of mediastinal nodes for preoperative assessment in lung cancer. *Chest* 1985;88:206-10.
- Martini N, Heelan R, Wescott J, Bains MS, McCormack P, Caravelli J, et al. Comparative merits of conventional computed tomographic and magnetic resonance imaging. *J Thorac Cardiovasc Surg* 1985;90:639-68.
- Dales RE, Stark R, Sankaranarayanan R. Computed tomography to stage lung cancer: approaching a controversy using meta-analysis. *Am Rev Respir Dis* 1990;141:1096-101.
- Wathen CG, Kerr KM, Reid W, Wightman AJA, Best JKK, Millar AM, et al. A compar-

ison of cobalt [⁵⁷Co] bleomycin scanning and contrast-enhanced CT scanning for assessment of the mediastinum in lung cancer. *Chest* 1990;97:1148-51.

Barcelona's asthma epidemics

I found Dr C Picado's article on the clinical characteristics of the Barcelona asthma epidemics (March 1992;47:197) a very clear-cut and stimulating summary. His assertion that these findings would support a search for other potential point sources of aeroallergens or air pollutants as a cause of a high incidence of asthma in particular areas was suggested in a leader on soya bean asthma in the *Lancet*¹ and this undoubtedly needs reinforcing. The difficulty lies where so called outbreaks are less dramatic, when it is important to try to find a definition of an epidemic day. The simplest method is to take any day when numbers of exacerbations of asthma are outside two standard deviations from the mean for a period of time. This may be somewhat harsh but has the benefit of simplicity. In order to define these "high spots" one may need to look at areas of a country where asthma is thought to be more common by looking at standardised hospitalisation ratios rather than deaths. There are wide variations in the UK for both hospitalisation rates and mortality and this would seem a suitable starting point.

The idea that the particle size is important is crucial. Does Dr Picado have any idea of the genuine particle size of the soya dust that was inhaled on epidemic days as opposed to that of the dust used in wet antigen challenge? A theory was put forward many years ago that very small particles would be exhaled immediately after inhalation because of their small size and dynamics. If the soya particles are submicronic in size Dr Picado's hypothesis would tend to oppose such a theory unless only limited lung retention of what is clearly a highly allergenic substance was able to cause an acute response.

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- Anonymous. Asthma and the bean. *Lancet* 1989;ii:538-40.

AUTHOR'S REPLY Although I tried to explain the most striking peculiarities of Barcelona's outbreaks, one of the questions raised in Dr Ayre's letter made me realise that in the article I missed one aspect that may be crucial for detecting an "unusual day" so far as the number of patients suffering asthma exacerbations is concerned.

Apparently, as he suggests, the simplest method of determining whether a day is unusual is to count the number of patients suffering exacerbations and then to take any day with numbers outside two standard deviations from the mean for a period of time. The experience of Barcelona suggests, however, that, for defining an unusual day, recording the names of the patients is probably better than counting the number of asthmatic patients attending the emergency room. This suggestion is based on the fact that in Barcelona's asthma epidemics several patients suffered repeated episodes of severe acute asthma, which in the more extreme cases required admission to the intensive care unit for artificial ventilation. The history of

the patient most severely affected offers some interesting insights.

The patient, a 56 year old woman (Miss MB), had been suffering from asthma since childhood. For many years the disease followed a chronic course with occasional mild or moderate exacerbations that never required hospital admission. She was treated with bronchodilators, cromoglycate, and finally beclomethasone. She also received short courses of oral corticosteroids during exacerbations, some of which were precipitated by exposure to house dust and pollen. A prick test showed positive reactions to *Dermatophagoides pteromyssinus*. In February 1981 she suffered a sudden and severe asthma attack requiring mechanical ventilation for the first time. Within the next two years she was admitted to hospital 18 times and she underwent artificial ventilation on 10 of those occasions. When the first episodes of asthma outbreaks were detected during 1981 and 1982 it became evident that Miss MB was always affected in these episodes. In fact, on epidemic days she was one of the earliest to attend the emergency department. The arrival of Miss MB triggered the "red alert" for an asthma outbreak. Although not all emergency attendances of Miss MB were on epidemic days many of the occasions could be considered as "unusual days," with more cases than expected of severe asthma admitted to hospital. She continued to suffer frequent exacerbations and finally died in June 1984 as a consequence of hypoxaemic encephalopathy and pneumonia. Combined treatment with high doses of different bronchodilators (beta mimetics, ipratropium bromide, and theophylline), beclomethasone, and oral corticosteroids proved to be totally ineffective for preventing severe asthma attacks.

Other patients in Barcelona suffered recurrent asthma attacks and often several of these "repeaters" coincided once or more with Miss MB in the emergency room. Some of these days might not be considered unusual days according to a statistical criterion (number of patients) but were clearly unusual when we looked at the names of the patients admitted to the hospital. Interestingly, the history of Miss MB was initially published as an example of recurrent idiopathic sudden severe bronchospasm.¹ Many reports have described patients who suffer recurrent attacks precipitated by unknown causes. These patients, like patients affected in Barcelona's outbreaks, often have asthma not responding to treatment. Perhaps the erratic oscillation of symptoms is a reflection of changes in the concentrations of environmental allergens. As only the most severe cases are usually seen in the emergency room, counting the number of patients admitted to hospital would not reveal the existence of an atmospheric factor causing less severe exacerbations of asthma in many more patients who do not usually require hospital admission. If two or more asthmatic patients are repeatedly attending the emergency room the same day the possibility that an environmental factor is causing the asthma attack should be suspected.

Unfortunately, to my knowledge there are no studies on the particle size of the soya dust. The size of the particles concerned in epidemic asthma do not necessarily need to be submicronic and might be similar in size to mould spores. Dr Ayres knows that fungal spores have been implicated in episodes of epidemic asthma.² In addition, O'Hollaren *et al*³ have recently reported that exposure to the