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Clinical correlates of angiographically diagnosed idiopathic pulmonary hypertension

We read with great interest the report by Dr H H Gray and his colleagues (June 1990;**45**: 442–6). Several points are raised which, we feel, merit comment and, hopefully, clarification. The most critical one is the distinction between what we prefer to call major vessel chronic thromboembolic pulmonary hypertension and small vessel idiopathic pulmonary hypertension.

This distinction has major management implications. The most vital one is that chronic thromboembolic pulmonary hypertension is potentially subject to surgical correction by thromboendarterectomy; idiopathic pulmonary hypertension is not. Furthermore, as Dr Gray and colleagues note, medical management of the latter often is not successful, with transplantation as the "final option." We have evaluated and followed more than 220 patients with major vessel, chronic thromboembolic pulmonary hypertension who have undergone pulmonary thromboendarterectomy[1]; our experiences in making this diagnosis may be germane.

One of the central problems in determining the diagnosis has been the terminological confusion created by the World Health Organisation classification of idiopathic pulmonary hypertension, a classification which has outlived its usefulness. Particularly troublesome is the uncertainty introduced by the "thromboembolic" subcategory.

We would submit that a "1990s" classification would be "small vessel pulmonary hypertension" and "large vessel thromboembolic pulmonary hypertension." In the first category are patients whose pulmonary hypertension arises from obstruction in the small, distal "resistance" vessels of the lung. Various lesions cause such obstruction, as has been amply demonstrated. Among these are so called "thrombotic" lesions. In our view patients with such lesions should no longer be described as having thrombo*embolic* pulmonary hypertension as no evidence for embolism has been offered. More likely, such lesions arise in situ from endothelial injury.

In the second category are patients whose pulmonary hypertension arises from obstruction of the large *elastic* arteries (main, lobar, segmental). These organised obstructing thrombi arise, in virtually every patient, from embolisation of venous thrombi. This distinction is not only pathogenetically more useful; it is also operationally critical. Patients in the second category can be substantially aided (even "cured") by thromboendarterectomy; patients in the former category cannot.

Other considerations follow once this distinction is made: anticoagulation in true thromboembolic ("large vessel") hypertension is, in our view, essential. The same can be said of Greenfield filter placement, to protect against further embolism in patients with substantial, large vessel thromboembolic pulmonary hypertension. As Mansour *et al* and others have noted, the morbidity and mortality of this procedure is negligible (in contrast with caval ligation or plication). In small vessel hypertension the value of anticoagulation is much less certain and filter placement is not indicated in the absence of venous thrombosis. We agree with Dr Gray and colleagues that differentiating the two conditions is difficult and frequently impossible on clinical grounds. We have, however, found a much higher frequency of a history compatible with deep venous thrombosis or pulmonary embolism (over half of all patients) in cases of major vessel obstruction (perhaps this is because one of us has taken the history in each of these cases).[2] There also is one distinctive physical finding in large vessel thromboembolic pulmonary hypertension: a flow murmur (as in congenital pulmonary artery branch stenosis), due to partial obstruction by a chronic thrombus; one or more of these murmurs can be heard *over the lung fields during breath holding* in some 30% of patients. We have not heard such murmurs in patients with small vessel pulmonary hypertension.

As Dr Gray and colleagues have suggested, the perfusion lung scans and pulmonary angiograms in these two groups differ substantially. In regard to perfusion lung scans, we have not found segmental or larger perfusion defects in patients with small vessel pulmonary hypertension. All patients with major vessel chronic thromboembolic pulmonary hypertension have had one (usually more and larger) such defect. (But commonly the perfusion scan defects *underestimate* the extent of major vessel obstruction.)

The key test is, of course, the pulmonary angiogram. It is quite distinctive in the two conditions. In small vessel pulmonary hypertension patent and normally tapering elastic arteries are seen, with "pruning" of the small, distal vessels (that is, no "capillary" blush). In large vessel chronic thromboembolic pulmonary hypertension various patterns are seen in the central arteries: frank obstruction, peculiar taperings and irregularities, webs, and bands. These many patterns reflect the variability in the way in which central (main, lobar, segmental) thrombotic occlusions organise and recanalise. Direct fibreoptic angioscopy helps diagnosis occasionally.

We concur that lung biopsy does not help to distinguish the two conditions. All the small vessel lesions "characteristic" of small vessel pulmonary hypertension can be found in major vessel thromboembolic pulmonary hypertension and in other disorders associated with pulmonary hypertension.[3] Biopsy therefore may obfuscate rather than elucidate the diagnosis. History taking, lung scanning, pulmonary angiography, and angioscopy are the most useful techniques for obtaining a diagnosis.

Because of the major management implications of making the correct diagnosis, we hope that the confusion evoked by "mixing" large vessel thromboembolic pulmonary hypertension with small vessel pulmonary hypertension (in which thrombotic lesions may occur) can be dissipated; patients with the former, potentially remediable, condition can then be recognised and managed appropriately.

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AUTHOR'S REPLY We welcome the comments of Professor Moser and his colleagues, whose experience in the field of idiopathic pulmonary hypertension is well known. Our paper was a retrospective review of patients presenting with unexplained pulmonary hypertension and as such suffers from certain weaknesses. Our finding of a lower incidence of prior deep venous thrombosis or pulmonary embolism in the group with asymmetrical pulmonary arteriopathy than that in Professor Moser's group, and the absence of pulmonary flow murmurs in the case records, may represent one of these weaknesses as the incidence of these findings would almost certainly be higher in a prospective study when someone with a particular interest in the subject makes the clinical assessment.

We agree entirely with their comments concerning the distinction between patients with idiopathic pulmonary hypertension. The histological differentiation into the three WHO categories (primary plexogenic, thromboembolic, and pulmonary veno-occlusive disease) may be difficult and indeed makes the assumption that there are in fact three separate disease entities, whereas it may be that a range of diseases exists. Such a differentiation is often clinically unhelpful and, as clinicians, we agree that until the aetiologies of idiopathic pulmonary hypertension are more clearly defined it may be more helpful to make distinction between patient groups based on therapeutic options. The distinction that Professor Moser and his colleagues use in dividing patients into those with small and large vessel pulmonary obstruction has a lot to recommend it from a therapeutic point of view and has the additional advantage that patients are not given a diagnostic label that is based on speculation about the causes of their pulmonary hypertension. If the causes of idiopathic pulmonary hypertension become clearer and if an imaging or pathological technique becomes available that reliably separates patients into these aetiological groups, the patient can be given an accurate diagnosis. Until then a distinction based on therapeutic groupings would have more practical benefit.

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Antemortem recognition of brain metastases in malignant mesothelioma

Drs M Huncharek and J Muscat report that antemortem diagnosis of central nervous system metastases from pleural mesothelioma is rare, with only three reports of antemortem diagnosis (July 1990;**45**:571). I suspect that this is due to underreporting rather than to the rarity of the condition. Indeed, on the day I read the article I received a necropsy report

for a 52 year old plumber with extensive malignant pleural mesothelioma, who had been admitted with impaired consciousness and left sided long tract signs and whose computed tomogram showed at least four cerebral metastases, two in the brainstem and two in the right partietal region. Both of these tumours were subsequently confirmed at necropsy. In addition, there was extensive tumour affecting the pericardium, epicardium, and myocardium and extending extensively through the peritoneum.

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I agree with Dr Lewis that the small number of published cases of antemortem diagnosis of cerebral metastases in pleural mesothelioma may be due to underreporting. Unfortunately, I do not have data to support or refute this contention. I reviewed about 200 cases of pleural mesothelioma in 1986–87 and was surprised to find that many physicians were unaware that the tumour often metastasised.

Over the last few years I have developed a strong interest in research examining how scientific data are used in the courts and in public policy decision making.[1] Through my work in the asbestos area I became aware that injured workers seeking compensation through the courts often faced defence attorneys who vigorously questioned a diagnosis of pleural mesothelioma if metastatic disease was present. This prompted me to review a large number of cases coming to necropsy, and document the occurrence of metastatic spread, which was quite common.[2] This report also cited earlier studies further supporting this fact.

With the incidence of pleural mesothelioma increasing, cerebral metastases will undoubtedly be found more often. I hope that information such as that detailed by Dr Lewis will find its way into medical publications. In this way confusion concerning the natural history of this disease will be avoided.

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Local anaesthesia for fibreoptic bronchoscopy

We read with interest the report by Dr A R Webb and colleagues (June 1990;45:474–7) in which they compared transcricoid injection of lignocaine with the traditional "spray as you go" technique. They found the transcricoid technique to be more effective.

On reading the "Methods" section of the paper, one finds that both groups of patients received topical anaesthesia with lignocaine, but that the location and timing of administration differed. Patients who received transcricoid anaesthesia had lignocaine delivered to the subglottic space in an anteroom several minutes before the passage of the bronchoscope, whereas the other group of patients received a topical application of lignocaine to the upper surface of the vocal cords about 1½ minutes before the bronchoscope was passed between the cords. There were further applications to the bronchi during the procedure.

In these circumstances it is not surprising that the group given transcricoid anaesthesia should cough less. It is likely that the lignocaine which was introduced into the trachea would spread widely in the bronchial tree during the coughing bout which followed the injection. It is also likely that the lignocaine would anaesthetise the inferior and medial surfaces of the vocal cords as it was removed from the bronchial tree by coughing and ciliary action. The bronchoscope is in contact with these surfaces of the vocal cords during bronchoscopy, but the "spray as your go" method would have anaesthetised only the upper surface of the cords. In the absence of any subglottic anaesthesia, one would expect the patient to cough and experience discomfort as the bronchoscope was passed into the unanaesthetised trachea and bronchial tree.

We have evolved a non-invasive technique for the instillation of local anaesthesia into the subglottic space and trachea. It is our practice to anaesthetise the nose and the upper surface of the vocal cords by the techniques described by Dr Webb and colleagues. We then position the bronchoscope directly above the vocal cords and we introduce 2-4 ml of 4% lignocaine as a bolus during inspiration. This enters the trachea and main bronchi and produces a cough similar to that occurring after transcricoid injection of lignocaine. We then wait two to three minutes before proceeding with the bronchoscopy and we find the operating conditions to be as good as those described after transcricoid lignocaine.

Our technique avoids a transcricoid injection but it has two potential disadvantages. Firstly, it may add one to two minutes to the duration of the bronchoscopy; we find this to be an acceptable delay. Secondly, our technique requires an experienced bronchoscopist to guarantee subglottic placement of the lignocaine. In difficult cases where both hands are required to optimise the position of the bronchoscope, we get an assistant to introduce the subglottic bolus of lignocaine while we observe the vocal cords through a lecturescope or on a video monitor.

We would therefore agree that transcricoid injection of lignocaine may have certain advantages, especially as it will achieve good anaesthesia of the glottis and trachea for trainee bronchoscopists. We suggest that experienced bronchoscopists might prefer to administer anaesthesia by the above non-invasive technique at the cost of a slight increase in operating time.

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We read with interest the article by Dr A R Webb and others (June 1990;45:474–7). Transtracheal injection of topical anaesthetic has been widely used for bronchoscopy, bronchography, and endotracheal intubation since this method was first suggested by Caynut[1] in 1920. Hitherto, a few complications, such as cellulitis, breakage of the needle, and pretracheal abscess, have been reported.[2-4]

Previous reports recommended inserting the needle perpendicularly to the skin through the cricothyroid membrane. This may cause laceration of the trachea as a result of coughing while the local anaesthetic is injected, and we witnessed a case of bleeding due to tracheal laceration many years ago. We therefore devised this technique to prevent this potential complication. A 23 gauge

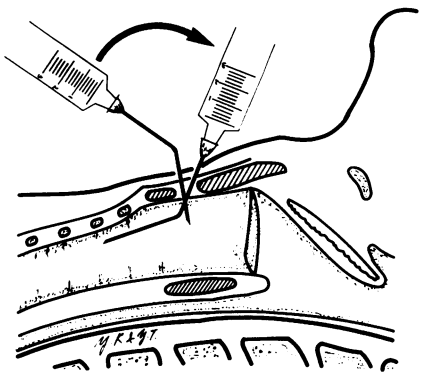

Diagram illustrating the technique of transtracheal injection with a bent needle.

hypodermic needle is bent aseptically about 45° at a point 1 cm from the tip with a cap of the needle. A bent needle, attached to a 5 ml syringe containing 3 ml of anaesthetic, is inserted perpendicularly into the trachea through the cricothyroid membrane. Easy aspiration of air confirms that the needle is within the tracheal lumen. Then the syringe is directed cephalad at an angle of about 90° while air is aspirated and the needle is advanced about 0·5 cm. This procedure makes the bent part of the needle parallel to the anterior wall of the trachea (figure). Thus there is no possibility of traumatising the trachea or dislocating the needle while the anaesthetic is instilled even if the patient coughs persistently.

We have used this technique for more than 20 years before endotracheal intubation and also for instillation of saline or mucolytic agents for treating and preventing postoperative pulmonary complications together with diazepam[5] and midazolam. No troublesome complications have been noted after more than several thousand injections, confirming the safety of this technique.

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CORRECTION

The hyperimmunoglobulinaemia E and recurrent infections syndrome in an adult

In the paper by J-P L'Huillier *et al* (September 1990;45:707–8), the 4th line from the end of p 707 should read "(0·54 × 10^9/l). . . ."