A Misnomer

To the Editor:

I believe the designation "posterior mediastinal sarcoidosis" in the article by Russel et al (Chest 1986; 90:402-64) is anatomically incorrect as per the illustrative material in the paper.

The two CT scans both show the lesions in question to be mainly in the "middle" mediastinum (right paratracheal region). This is the most easily accessible area of the mediastinum for mediastinoscopy and biopsy. In both cases the "posterior" mediastinum is not involved.

In Figure 1, A and B are transposed and the CT scan is displayed upside-down (with the spine above and anterior chest below), contrary to the usual method of presentation.

In Figure 2 A, the legend refers to "widening of superior mediastinum and aortopulmonary window involvement", neither of which are in the posterior mediastinum. In Figure 2 B, the CT scan clearly shows extensive paratracheal nodal involvement, especially on the right, plus nodular densities anterior to the superior vena cava and aortic arch in the anterior mediastinum.

In both cases, mediastinoscopy by the Carlens technique would have been positive and would have obviated thoracotomy.

In the references furnished by the authors, none of the papers referring to posterior mediastinal involvement utilized CT scans. CT scans are essential for a true anatomic localization of disease and surpass previous imaging techniques such as conventional tomography.

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Purulent Rhinosinusitis is Also a Cause of Sepsis in Critically Ill Patients

To the Editor:

In a recent review, Norwood and Givetia evaluated current sepsis in critically ill patients, but forget sinusitis, an important source of infection in nasotracheally-intubated patients.

We prospectively studied all patients in an intensive care unit between October, 1986 and August, 1987 who required nasotracheal intubation and mechanical ventilation for more than six days. All patients underwent CT scan of the frontal, ethmoidal, sphenoidal and maxillary sinuses. Sinusitis was defined by sinus opacification and air-fluid level. Maxillary sinusitis on CT scan was confirmed by a transantral maxillary puncture and aspiration of fluid, which allowed us to isolate bacteria.

Of 30 patients studied, CT scan sinusitis was noted in 28 cases (93 percent). Purulent fluid aspirate from maxillary sinus puncture revealed bacteria in 25 cases (83 percent). Gram-negative bacteria in 85 percent, Gram-positive in 15 percent. Candida species in 5 percent, and polymicrobial infection in 40 percent of the cases. The same bacteria were found in blood cultures in eight cases and in pleural effusion in four cases. Treatment of such sinusitis is controversial. A study comparing removing of the nasotracheal tube (including tracheostomy), antibiotic therapy, and surgical drainage of the sinus has been proposed.

In nasotracheally-intubated patients, sinusitis is a frequent complication, representing five percent of the nosocomial infections.2 Underestimate is frequent because of a paucity of clinical signs.4 Sinusitis is found in about 25 percent of nasotracheally intubated patients,12 but these studies were not prospective and sinus puncture was not always obtained. In our prospective study, purulent sinusitis was found in 83 percent of patients intubated for six to nine days. Thirty-three percent of these patients had little purulent drainage around the nasotracheal tube, and all patients had fever.

In critically ill patients, clinical features appear too late to make a rapid diagnosis of purulent sinusitis. These data emphasized the need for a CT scan for sinusitis in patients with a nasotracheal tube.


REFERENCES


To the Editor:

The comments of Dr. Guerin and colleagues are greatly appreciated. As cited in their letter, sinusitis is becoming a more frequently identified cause of infection in patients with nasotracheal intubation. This should have been mentioned in our review.

It has been our experience, however, that sinusitis is an infrequent cause primarily because we almost never use nasotracheal tubes. My concern is that nasogastric tubes may also cause similar problems, and we are presently utilizing small bore gastrostomy tubes much more frequently because of this concern.

The data presented by Dr. Guerin shows an alarmingly high rate...
of sinusitis (83 percent) in patients with nasotracheal tube in place greater than six days. However, it is not clear from his data whether, in fact, the sinusitis was the source of the patient’s fever. How many patients’ fever resolved following antibiotic therapy for sinusitis? Were some of the cases not treated?

We do not routinely use nasotracheal intubation in our Emergency Department, even if cervical spine injury is suspected. Most patients can be safely nasotracheally intubated either directly or with a bronchoscope while maintaining the neck in neutral position. If nasotracheal intubation is not possible, I prefer surgical cricothyrotomy over nasotracheal intubation.

Based on the data presented by Dr. Guerin and others cited in his letter, it would seem reasonable to convert all nasotracheal tubes to orotracheal tubes as soon as possible if prolonged mechanical ventilation is needed. This should alleviate the problem altogether and prevent the often cumbersome and dangerous transfer of a patient from the intensive care unit to the radiology department for CT scanning.

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Primary Pulmonary Hypertension

To the Editor:

While I would defend Dr. Robins right to express his opinion concerning therapeutic interventions in patients with primary pulmonary hypertension (Chest 1987; 92:330-34), I take issue with his selective, and at times inaccurate, use of the published literature in this area.

Robins states that PPH is not a disease but a wastebasket, yet he contributes material to this wastebasket by lumping together all patients with this diagnosis and suggesting that the prognosis is highly variable. Although long-term survival has been documented in several cases, most studies have suggested that this is usually not the case. Furthermore, several studies have demonstrated that patients with right heart failure have a particularly poor prognosis.

I also take issue with several of the statements made by Robin concerning the use of vasodilator therapy. While he is correct in his statement that the early study evaluating hydralazine in the treatment of PPH did not show improvement in mortality or improved quality of life, it is appropriate to mention that this was a preliminary, short-term study evaluating solely the hemodynamic effects. He is incorrect, however, in stating that hydralazine therapy resulted in a significant reduction in mean pulmonary artery pressure; it was unchanged, although pulmonary vascular resistance fell. The level of PVR has been correlated with survival in PPH. In regards to his use of reference 15, the conclusions from that article were stated clearly: despite hemodynamic changes, several of these severely compromised patients died. We did not consider the therapy "successful," but rather suggested that further studies evaluating the long-term effects are necessary before a decision concerning its utility could be made. He may be interested to know that several such studies are underway or in development.

I applaud Robin's rigorous honesty about his procedure when talking to potential candidates for heart-lung transplantation, and he may be assured that others in the medical community who deal with PPH patients approach the issue of medical therapy in a similar manner.

It is unfortunate that Robin has chosen to both interpret and vilify the data from the PPH Registry prior to its complete analysis and publication, particularly since there are several inaccuracies in his statements. Several publications will be forthcoming from the Registry which will deal with these data more accurately and authoritatively.

The experience over the last decade with therapeutic interventions for PPH, including heart-lung transplantation, has generated many questions and provided few answers. Yet, it is this process that will yield more pieces to the puzzle until the mystery of PPH is ultimately solved. It is both incorrect and unfortunate for Robin to suggest that the results to date with any approach are more definitive than they actually are.

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REFERENCES

5 Hughes JD, Rubin LJ. Primary pulmonary hypertension. An analysis of 28 cases and a review of the literature. Medicine 1986; 65:36-72

To the Editor:

I thank Dr. Rubin for his vigorous response. He has been one of the prime movers in the use of pulmonary vasodilator therapy in pulmonary hypertension, so that comments by him are particularly revealing.

We have used the same diagnostic wastebaskets as have been used by essentially all students of the disease. These are clearly identified in the data paper published by us.1 No matter which references you cite, there are now 90 patients with PPH recorded in the literature with mean survival after onset of symptoms of almost six years and mean survival after diagnosis of three and one-half years. Long-term survivors are not merely statistical outliers. Thirty-seven percent lived for five years and 15 percent lived for ten years following diagnosis. Are you suggesting that somehow we combed the wide world to find the "few" patients who are long-term survivors? The paper of Rozkovec et al and an editorial in Lancet4 make the same point. The data from the PPH study likewise show a substantial cohort of patients living relatively long lives. Approximately 30 of 187 patients survived five years or longer between symptoms and first diagnosis. 4 The fact that PPH is consistent with relatively prolonged survival is now a fully confirmed, established and incontrovertible fact. What issues remain?

How representative are the data in the literature on PPH and our data with the natural life history of PPH as it occurs in real life? The answer is: no one knows. Series tend to be culled from academic centers. They may thus reflect the bias resulting from the study of a cohort with far advanced disease. Patients from academic centers tend to be treated aggressively, both with diagnostic and management modalities. They thus tend to be subjected to premature death.

As a consequence, you should restructure your thinking. You have previously consigned your patients to the kingdom of the near dead.