Sarcoidosis, Firefighters Sarcoidosis, and World Trade Center “Sarcoid-Like” Granulomatous Pulmonary Disease

To the Editor:

Dr. Izbicki and colleagues1 are to be congratulated on their report of 26 patients with “sarcoid-like” granulomatous pulmonary disease (SLGPD) in Fire Department of New York (FDNY) World Trade Center (WTC) rescue workers. This adds to their previous descriptions of “WTC cough,” persistent airway hyperreactivity, and accelerated decline in lung function that have provided the largest share of the medical literature on WTC lung disease. All these contributions have been uniquely benefited by the serial (including pre-WTC attack) observations available to these investigators.

To the question of whether SLGPD is truly sarcoidosis may be added whether it is the same as the “sarcoidosis” reported in New York City firefighters previous to September 11, 2001.2 Figure 1 in the article by Izbicki et al1 suggests that the incidence of SLGPD is no different after September 11, 2001, with the exception of the marked increase in the year following. No pre-WTC FDNY sarcoidosis patients had airway hyperactivity by history or bronchoprovocation, in contrast to the recent group. The authors suggest that the prevalence of asthma-like symptoms, airways obstruction, and hyperreactivity distinguishes SLGPD from sarcoidosis, although investigators at Mt. Sinai in New York1–3 have reported all of these in sarcoidosis with frequencies varying with the stage of disease, ethnicity, and smoking history.

On the question of whether SLGPD is truly sarcoidosis, it should be noted that all 26 patients met the definition of sarcoidosis6 by having more than one organ system involved because all had mediastinal or hilar adenopathy. Indeed, diagnosis was established by mediastinal biopsy in the majority (16 patients). However, several observations are unusual for sarcoidosis: (1) the frequency and site of extrathoracic findings; only six cases (23%) were extra-thoracic, of which only one case (bones, joints, skin) was typical of sarcoidosis, while five cases were unlikely to have been considered as sarcoidosis previously, showing only pelvic adenopathy or splenomegaly on CT; (2) the rarity (two cases) of diffusion impairment, which is common in all stages of sarcoidosis including stage I; and (3) the absence of progression. Greater insight into this question would be provided by serum angiotensin-converting enzyme levels and most specifically by Kveim reactivity, which is uniquely seen in sarcoidosis.

This question is now of greater than clinical interest with the recent decision of the New York City Medical Examiner2 to rescind his previous finding and conclude that the sarcoidosis death of a worker fleeing a nearby building was indeed WTC related and therefore homicide. Unlike the FDNY cases in the article by Prezant et al.,3 this young woman was briefly exposed to the plume and died 5 months later. Autopsy showed evidence of longstanding cardiac sarcoidosis. Pulmonologists and pathologists will undoubtedly be subjected to our adversarial judicial system to attribute cause and allot compensation of WTC-exposed patients with sarcoidosis (as well as other lung diseases of unknown etiology).

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The author has no conflict of interest to disclose.

REFERENCES


Oxyhemoglobin Dissociation Curve Clarification

To the Editor:

I read with interest the report by Das et al1 on hemoglobin Bassett producing low pulse oximeter and co-oximeter readings. Since I practice intensive care medicine, two things struck me about this report. One is the implication that the low oximetry readings were related to a rightward shift of the oxyhemoglobin dissociation curve. From the data
the authors presented, the measured $\text{PO}_2$ at 50% saturation ($\text{pSO}_2$) was normal and the oximetry was low. This suggests the dissociation curve was not shifted and the low oximetry seen in their patient was due to the fraction of the abnormal hemoglobin not binding oxygen. Secondly, the authors state that a rightward shift in the dissociation curve results in a higher $\text{pSO}_2$. However, they incorrectly state that a rightward shift in the oxyhemoglobin dissociation curve occurs with blood transfusion, reduced levels of 2,3-diphosphoglycerate (2,3-DPG), hypophosphatemia, and hypothyroidism. Reduced levels of 2,3-DPG result in a leftward shift of the curve and increased affinity of oxygen binding to hemoglobin (lower $\text{pSO}_2$). Reduced levels of 2,3-DPG are known to occur with hypothyroidism and hypophosphatemia. As well, stored blood has reduced levels of 2,3-DPG, potentially resulting in a leftward shift of the curve after transfusion. 

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REFERENCES

Intubating ICU Patients With Ketamine

Adverse Effects That Can Occur

To the Editor:

We read with interest the article by Dr. Walz et al1 and wish to enlighten the readers to a misconception about a drug that is mentioned and used in the ICU setting. The review article is excellent but requires additional explanation about the drug ketamine.

According to the authors, ketamine, when used in the ICU for intubation, stimulates the CNS system, thereby causing an increase in BP and heart rate. This phenomenon is usually expected by anesthesiologists, surgeons, and critical care specialists alike. Intubating conditions are also improved. That may well be, but the increase in BP and heart rate are not routinely achieved.

On the contrary, critically ill patients with minimal physiologic reserve do not always manifest this condition; rather the opposite can occur: hypotension and bradycardia. Not only can it happen in the operating room but also in the ICU setting.

Ketamine is thought to stimulate cardiovascular functions by several mechanisms. First, it directly stimulates the sympathetic nervous system, resulting in the release of catecholamine. Second, ketamine increases tissue and circulating norepinephrine levels by inhibiting their neuronal and extraneuronal re-uptake. Elevated serum cortisol levels during ketamine anesthesia were demonstrated in elective (noncritically ill) patients, suggesting that the agent produced adrenocortical stimulation. Critically ill patients with minimal physiologic reserve are maximally compensating for hypovolemia, hypoxemia, fluid-electrolyte, acid-base, and nutritional problems.

According to Lippmann et al,4 there was an early progressive increase in heart rate, cardiac index, arterial and venous pressure, stroke work and oxygen delivery, oxygen consumption, and oxygen extraction. In critically ill patients, however, ketamine did not produce uniform responses and was not without some adverse effects. There was a diversity of responses in mean arterial pressure, heart rate, cardiac index, oxygen consumption, oxygen extraction, and venous admixture. In some cases, ketamine may even cause maldistribution of systemic blood flow resulting in inadequate tissue oxygenation. Patients in this series were severely stressed and critically ill with depleted catecholamine and adrenocortical stores. Moreover, prolonged severe stress blunts sympathetic and/or adrenocortical stimulation by ketamine. Lippmann et al4 concluded that the variability of ketamine responses in these patients was largely attributable to the balance between a direct myocardial depressant effect and a stimulatory sympathomimetic action of ketamine. This is altered in the critically ill patient whose compensatory responses may be affected to different degrees. For example, the hypovolemic patient may respond to sympathetically stimulated tachycardia but be unable to respond with increased cardiac output. Those with limited myocardial reserves and increased demands may respond to ketamine with reduced cardiac output. They also conclude that although ketamine may be the agent often used in emergencies, whether it be in the operating room/ICU, its side effects, even in small doses, may lead to unanticipated severe adverse effects. Even though ketamine may facilitate intubation conditions, the adverse effects must be weighed carefully in critically ill patients.

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Bronchiectasis in Acute Pneumonia . . . Pseudobronchiectasis

To the Editor:

I read with interest in a recent issue of CHEST (June 2007)1 the case report “A 53-Year-Old Man With Fever, Clubbing,
pears.4 It is only after destruction of the elastic tissue and bronchial following control of the infection or inflammation the dilatation disappears. The process is reversible, because the patient has only dilated bronchi, which is a common reversible finding seen in patients with pneumonia of any cause and is referred to as pseudobronchiectasis or functional bronchiectasis.3,5

As a result of either infection or inflammation of the bronchi, dilatation of the bronchi can occur. The process is reversible, because following control of the infection or inflammation the dilatation disappears.4 It is only after destruction of the elastic tissue and bronchial musculature and replacement by scar tissue that the anatomic change so characteristic of bronchiectasis is seen.5 A study6 of 60 consecutive cases of pneumonia in previously asymptomatic adults revealed 25 patients with bronchial dilatation during the acute phase of the illness, with 20 of those patients subsequently returning to normal. As the bronchial dilatation may persist for up to 3 to 4 months after the resolution of acute pneumonia, a consequent high-resolution CT scan examination for bronchiectasis7 should be performed at least 6 months after the infection has resolved to avoid this pitfall and to confirm the diagnosis of bronchiectasis.7,8

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7 Pontius Jr, Jacobs LG. The reversal of advanced bronchiectasis. Radiology 1957; 68:204–208

“Nailing” the Evidence

To the Editor:

Dr. Gomez and colleagues’ deserve praise for a well-conducted laboratory investigation of the effects of endogenous albumin on acid-base balance and tolerance of acidosis. However, the accompanying editorial by Dr. Kaplan,2 entitled “Another Nail in Albumin’s Coffin,” is misleading and deserves comment. The title and text of the editorial imply that clinical treatment with exogenous albumin is without benefit and should be abandoned. Drawing these conclusions from the study by Gomez et al,3 which examined the effects of hypercapnia on acid-base status, nitric oxide balance, and BP in rodents with normal albumin, hypoalbuminemia, or analbuminemia is beyond the limits of this focused preclinical investigation. The assumption that these results may apply to humans requires testing, and we cannot advocate changing clinical practice based on such data.

A consensus statement4 of the clinical use of albumin and other colloid solutions in critically ill patients exists to guide the appropriate use of these agents, and neither that document nor the cited reference4 in the editorial espouse the use of any colloid for modulating acid-base balance. Having never seen albumin administered to patients solely for buffering acidosis, it seems unlikely that even additional human data, as suggested by Dr. Kaplan, would alter current prescribing practices for this drug. However, it is equally incorrect to consider that the results of the study by Gomez et al3 may inform clinical practice and should prompt clinicians to discontinue their appropriate use of albumin. In the absence of clinical evidence in favor of or against the use of albumin, we should consider whether our nails are scaling a coffin or erecting a barrier to real evidence.

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2 Kaplan LJ. Another nail in albumin’s coffin. Chest 2007; 131:1276–1277

Response

To the Editor:

I received Dr. Martin’s letter1 with great enthusiasm, as it indeed underscores the visceral reaction that often accompanies colloid therapy. I agree that one would not abandon albumin solely on the basis of Dr. Gomez’s well-done and elegant study. However, I would advocate caution when selecting colloid therapy for plasma volume expansion (PVE) or acid-base management. Albumin offers no advantage over other colloid therapies for PVE. The results from the oft-touted SAFE trial2 declared that albumin was as safe as normal saline solution for PVE in the critically ill. Recall that normal saline solution PVE often creates hyperchloremic metabolic acidosis; an undesirable consequence of PVE, and one that may be decidedly “un-SAFE” with a preexisting acidosis.

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To the Editor:

We read with interest the hypothesis-generating study by Fremont et al in a recent issue of CHEST (June 2007) in which the authors, based on the observation that patients identified as having postobstructive pulmonary edema had a lower mean edema fluid/plasma protein ratio, concluded that “postobstructive pulmonary edema is a form of hydrostatic pulmonary edema.” Even though the assumption may eventually be proven to be true, we think it is currently based on a tenuous laboratory abnormality, the low pulmonary edema fluid/plasma protein ratio. The dramatic clinical presentation of a patient with upper airway obstruction and postobstructive pulmonary edema forces the attending physicians to use, in a knee-jerk fashion, nebulized salbutamol or racemic epinephrine and IV dexamethasone in a desperate attempt to avoid reintubation. However, selected studies in animal and ex vivo human lungs have demonstrated that therapy with β-agonists can accelerate the rate of alveolar fluid clearance within hours of starting treatment via an increase in intracellular cyclic adenosine monophosphate that results in increased Na+/K+ transport across type II alveolar cells through up-regulation of the apical sodium and chloride channels and Na+/K+–ATPase. In addition, a single dexamethasone injection has been shown to modulate lung epithelial Na+ channels and Na+/K+–ATPase and to increase alveolar fluid clearance, thereby accelerating recovery from pulmonary edema. Therefore, the medications used in the management of patients with presumed postobstructive pulmonary edema may facilitate the reabsorption of edema fluid and lead to an erroneously high edema fluid/plasma protein ratio.

From our point of view, the edema fluid/plasma protein ratio may be confounded by the medical treatment that is instituted and potentially can lead to the misclassification of the etiologies of pulmonary edema in selected cases. A retrospective analysis of the authors’ extensive data bank of patients with pulmonary edema would provide an excellent opportunity to refute or validate this statement.

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The authors have not reported to the ACCP any conflicts of interest.

REFERENCES

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Response

To the Editor:

We thank Dr. Kopterides and colleagues for their comments on our study of edema fluid-to-plasma protein ratios in patients with postobstructive pulmonary edema. The authors question whether the use of medications known to increase the rate of alveolar fluid clearance (β-agonists and corticosteroids) has led to misclassification of the etiology of postobstructive pulmonary edema in our study. We agree that alveolar epithelial fluid transport can increase the edema fluid-to-plasma protein ratio through the more rapid clearance of fluid and solute compared to protein. Misclassification would be most likely to occur if substantial time had elapsed between the onset of acute pulmonary edema and sampling of the edema fluid and plasma. In our study, the median time to fluid collection was very short, 1.5 h (interquartile range, 0.5 to 5 h). Furthermore, rapid alveolar fluid clearance could only lead to misclassification of patients with underlying hydrostatic pulmonary edema, who might be misclassified as having increased permeability edema because of an elevated edema fluid-to-plasma protein ratio; patients with increased permeability pulmonary edema would not be misclassified. In our study, 7 of 10 patients had edema fluid-to-plasma protein ratios in the hydrostatic range (< 0.65). Two patients had levels that were slightly above this cutoff point at 0.66 and 0.69, still suggesting a predominant hydrostatic mechanism. One patient had an initial ratio of 0.80, suggesting either a nonhydrostatic mechanism or the possibility that sampling of the edema fluid took place after alveolar epithelial fluid transport had begun. Thus, among the 10 patients studied, only 1 patient had the potential to be misclassified. Furthermore, only a minority of patients received β-agonists or corticosteroids during the study period: one patient received albuterol, two patients received epinephrine, and four patients received corticosteroids. In summary, the available evidence including the low edema fluid-to-plasma protein ratio in the majority of the patients despite intact alveolar fluid clearance strongly supports a hydrostatic mechanism of edema fluid formation in postobstructive pulmonary edema.

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REFERENCES


Recurrence of Severe Pulmonary Hypertension Following the Removal of a Lung Allograft

To the Editor:

Deb et al (June 2006) previously reported a patient with pulmonary arterial hypertension (PAH) in whom lung transplantation appeared to have acted as a “bridge to recovery.” Idiopathic PAH was diagnosed in 1992 in the patient, who eventually received a single-lung transplant in 1994. The posttransplant course was complicated by the development of chronic renal failure and chronic rejection. Due to ongoing infection in the allograft, and after determining that the allograft was essentially nonfunctional, the allograft was removed in August 2004. The mean pulmonary arterial pressure was 33 mm Hg prior to explant, and 6 months later a repeat right heart catheterization documented a mean pulmonary arterial pressure of 35 mm Hg. This was the first case showing significant improvement of PAH following lung transplant and subsequent removal of the transplanted lung.

Following a change in the patient’s geographic location, our center assumed care of the patient in August 2006. At that time, the patient was on multiple therapies for recurrent PAH. These included sildenafil (20 mg tid), bosentan (125 mg bid), and IV treprostinil (60 ng/kg/min). Despite this aggressive treatment, the patient’s functional status was class IV, and echocardiograms continued to show signs of right ventricular dilatation and impaired function with elevated pulmonary arterial systolic pressure (70 to 80 mm Hg). Unfortunately, recurrent line infections and difficult central vein access (due to her history of recurrent central access for hemodialysis) precluded the ongoing use of IV treprostinil. The patient was unable to tolerate therapy with subcutaneous remodulin or inhaled iloprost. Compassionate use of imatinib was started at a dosage of 200 mg daily. She was successfully tapered off treprostinil, and the central line was discontinued. The patient has continued to respond well to imatinib therapy with regression to functional class III, improvement in 6-min walk distance from <100 to 340 m, and stability of estimated pulmonary arterial systolic pressure and right ventricular function seen on echocardiograms. While imatinib has several molecular targets, its ability to block the receptor for platelet-derived growth factor, a powerful mitogen of the pulmonary circulation, could be beneficial in patients with PAH. Following promising case reports, ongoing clinical trials are investigating the use of imatinib in patients with PAH.

The eventual recurrence of PAH in this patient is important to note. The “multiple-hit hypothesis” of PAH assumes that a genetic predisposition, combined with environmental factors, leads to the development of idiopathic PAH. Severe pulmonary hypertension developed in our patient, followed by significant improvement in PAH symptoms and hemodynamics following transplant, followed by recurrence of severe PAH following removal of the transplanted lung. While the use of immunosuppressive medication to prevent allograft rejection may have played a role in the patient’s improved PAH, we believe it unlikely, as these medications have not been effective in the treatment of PAH. Rather, with insertion of the allograft in 1994, an enormous shift in the pulmonary circulation (97% of perfusion to the allograft by ventilation-perfusion scan) permitted a great reduction in the endothelial stress to the vascular bed of the native lung. Following years of reduced endothelial stress after receiving the transplant, the patient’s PAH

4 Matthay MA, Folkesson HG, Clerici C. Lung epithelial fluid transport and the resolution of pulmonary edema. Physiol Rev 2002; 82:569–600
improved significantly. However, as her allograft eventually failed and was then removed, her native load took on a progressively greater proportion of the pulmonary blood flow. This increase in volume and endothelial stress likely permitted recurrent injury to the vascular endothelium, which, combined with a genetic predisposition, led to the return of severe PAH.

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REFERENCES


Response

To the Editor:

On behalf of my co-authors, I would like to thank our colleagues for assuming the care of this complicated patient and providing important follow-up information. Their report adds to the growing body of literature attesting to the potential utility of imatinib and possibly other forms of antiproliferative therapy as we move beyond the era of vasodilator therapy in our quest for a cure for this devastating condition.

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REFERENCE


Extranodal Extension in Metastatic Non-small Cell Lung Cancer

To the Editor:

We read with much interest the study of Lee et al (April 2007) on the prognostic significance of extranodal extension (ENE) of metastatic non-small cell lung cancer. This is a much needed area of research, and there is strong agreement with the authors’ goal of improving existing pathologic staging standards. However, we feel that the authors’ conclusions could be strengthened by addressing the following concerns:

1. A reproducible definition of ENE is not provided. Generally considered synonymous with the term extracapsular extension, this term implies the presence of proliferating malignant cells outside the capsule of an involved lymph node. As an example, Fleischmann et al in 2005 provided the following definition for ENE: “...perforation of the capsule by tumor tissue with extracapsular growth. Histopathologically, extranodal extension must be differentiated from tumor deposits in the pericapsular lymphatics.” The definition of terminology and the application of criteria for assessing ENE should be explicitly listed in the “Materials and Methods” section; the failure to do this results in the inability to reproduce the study results and could lead to erroneous conclusions based on the current data.

2. The photomicrograph proffered to depict ENE (Fig 1 in the article) does not, in our opinion, appear to demonstrate ENE. We interpret the image to depict metastatic tumor within capsule-confined lymph node parenchyma and pericapsular lymphatics, and believe it does not demonstrate unequivocal evidence of ENE.

3. While we do not dispute that the identification of ENE may be of significant prognostic value, it is our experience that variations in surgical technique may limit the assessment of ENE in some practices. For example, in our academic medical center, specimens from thoracic lymph node biopsies and excisions are often received as partially cauterized and fragmented specimens with variable amounts of attached perinodal soft tissue. Lymph node handling and sectioning methods will need to be expanded to account for these potential limitations if the documentation of ENE is to become the standard of practice.

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To the Editor:

The major purpose of our published research1 was to find out the significance of extranodal extension (ENE) of regional lymph nodes (LNs) in surgically resected non-small cell lung cancer. We would like to respond to the comments from Bell et al, as follows:

1. Our response to the concern about a reproducible definition of ENE is that the terminology and application of criteria for assessing ENE had been explicitly addressed in the “Materials and Methods” section,1 as “... (2) extranodal extension, in which cancer cells invaded beyond the capsule of the LN.” Pericapsular lymphatic tumor deposit, whether present or not, was not included in the definition of ENE. The term ENE was used in various cancers and in the literature for many years, and different subclassifications of ENE have been proposed.2-5

2. Our response to the concern about the previously shown photomicrograph depicting ENE is that the photomicrograph we proffered in our article1 demonstrated that the metastatic tumor was not only invading through the vanishing LN capsule but was also accompanied by vascular invasion, a frequent phenomenon that was significantly correlated with ENE (p < 0.001) [Table 1 in our article].1 Our specimens of LNs with ENE frequently showed an extensive extranodal tumor area, even replacing LN architecture. We would like to share with the readers more photomicrographs of ENE demonstrating various severities of ENE (Fig 1).

3. Our response to the concern about “variations in surgical technique may limit assessment of ENE in some practices” is that all patients cared for by our team had undergone systemic dissection of LNs. By using surgical clips and scissors during LN dissection, the problem of partially cauterized and fragmented specimens could be avoided. However, if in that case, thoroughly examining fragmented lymph nodes by sectioning at 1- to 2-mm intervals, which was described in the last paragraph of the “Materials and Methods” section,1 carefully processing different cut surfaces,6 and integrating microscopic observations from every field would facilitate ENE evaluation.

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Figure 1. Top: Extranodal tumor extension causing fibrosis in the perinodal adipose tissue outside the vanished LN capsule (hematoxylin-eosin, original × 13.2). Center: Extensive extranodal soft-tissue cancer involvement with desmoplasia was demonstrated on the left side (hematoxylin-eosin, original × 13.2). Bottom: LN architecture was almost effaced by tumor cell nests with extranodal fatty tissue involvement (hematoxylin-eosin, original × 33).
Timing of Antibiotics and Community-Acquired Pneumonia

To the Editor:

We read with interest the recent article in CHEST (June 2007) by Kanwar et al1 about the misdiagnosis of community-acquired pneumonia (CAP) after the implementation of the “4-h antibiotic administration rule.” We agree that the topic is of great relevance; however, we have the following comments. Although a retrospective cohort study evaluating different points in time (ie, before and after the implementation of the rule) was appropriate, the fact that it was at a single center makes their observations dependant on singularities of a particular center such as, for example, changes in staffing, equipment, temporary policies other than the 4-h rule. This makes the external validity of the study questionable. Obtaining data from other centers and at different calendar times for comparison would achieve stronger conclusions. Also, the table of results (Table 2 in the article) illustrated only hard outcomes such as mortality and length of hospital stay, which the study was not powered to examine.

Finally, the authors concluded that “compliance with the 4-h antibiotic-administration rule led to an increase in the misdiagnosis of CAP, and subsequently to greater utilization of inappropriate antibiotics.” Based on their data, we have tabulated the rate of misdiagnosis of CAP in Table 1. We see a trend toward significant bias. The final diagnosis of pneumonia was based on subjective criteria, and when the authors used more stringent definitions of pneumonia (definitions A and B) the difference between the two groups was no longer significant ($p = 0.06$ and $0.17$, respectively). Along the same line, the mean time to the administration of antibiotics was not significantly different between the groups. Moreover, though the 2005 cohort received a higher proportion of antibiotics, 34.2% of cases including 35.6% with a final diagnosis of CAP did not receive antibiotics within the prescribed 4-h period, so final conclusions based on this could lead to misinterpretations.

The authors have reported to the ACCP that no significant conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

To the Editor:

We appreciate the interest of Srivaths and Corrales-Medina in our study.1 Their conclusions are based on the assumption that our final diagnosis was biased.2 We disagree that using hospital discharge diagnosis would lead to a bias. This diagnosis was based on the assessment of the attending and consultant physicians, and none of them were involved in the study.

We agree that we cannot generalize our study to every single hospital; this was not our intent. However, similar concerns about the 4-h rule have led the Infectious Diseases Society of America/American Thoracic Society 2007 guidelines to recommend the administration of antibiotics in the emergency department rather than adhering to a “specific time window.”3 The guidelines also caution that “improvements in one area may be offset by worsening in a related area.”

The authors misquoted our findings. When looking at all those with the admitting diagnosis of pneumonia, mean time to antibiotic was significantly lower for 2005 (187 min) compared to 2003 (230 min). Moreover, 65.8% of patients in 2005 received antibiotics within 4 h compared to 53.3% in 2003 ($p = 0.007$). However, looking at patients with the final diagnosis of pneumonia, the 4-h rule did not improve the timing of antibiotics. Our results clearly show that the 4-h rule resulted in lowering the threshold for starting antibiotics without a significant benefit in patients who were confirmed to have pneumonia.

We support antibiotic administration promptly when patients are admitted with pneumonia and when adequate evaluation is done so the correct diagnosis is achieved. We believe that the removal of the 4-h rule will improve the accuracy of the admitting diagnosis and decrease unnecessary antibiotic usage.

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Table 1—Misdiagnosis of CAP and Mean Time to Administration of Antibiotics

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<th>Variables</th>
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<td></td>
<td>n = 199</td>
<td>n = 319</td>
<td></td>
</tr>
<tr>
<td>Misdiagnosis of CAP</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Using definition A</td>
<td>24.1</td>
<td>41.1</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Using definition B</td>
<td>55.3</td>
<td>64</td>
<td>0.06‡</td>
</tr>
<tr>
<td>Time to administration of</td>
<td>251 ± 165</td>
<td>250 ± 204</td>
<td>0.95¶</td>
</tr>
<tr>
<td>antibiotics, min</td>
<td></td>
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</tr>
</tbody>
</table>

*Values are given as % or mean ± SD, unless otherwise indicated. Misdiagnosis of CAP = No. of patients with a final diagnosis of pneumonia/No. of patients admitted to the hospital with a diagnosis of pneumonia × 100; final diagnosis of pneumonia = diagnosis of pneumonia during hospital stay (as documented in the progress notes) or on hospital discharge (per the hospital discharge summary) by the attending physician, infectious disease specialist, or a pulmonologist; definition A = chest radiograph showing an infiltrate or consolidation, and one or more among shortness of breath, cough, sputum production, and a temperature of > 37.8°C; definition B = an infiltrate seen on a chest radiograph and two or more of the symptoms and signs for definition A.

†By Fisher exact test.
‡By t test.
Real-time Sonography With Central Venous Access

The Role of Self-Training

To the Editor:

The recent article in CHEST (July 2007)1 on internal jugular access with sonography establishes obvious benefits for the procedure despite the reluctance of present-day colleagues to recognize the decrease in multiple needle passes, cervical hematoma, and pain to the conscious patient. Whether it is comfort or pride in the “blind” technique among attendings, residents, and fellows, these traits beckon an opportune change for the patient’s sake.

Real-time sonography performed with a one-operator technique using either hand for needle insertion and probe support should be encouraged to develop ambidextrous skill for sonography of the right or left internal jugular and subclavian veins. In my routine, the probe is held but not set down, while the needle is inserted. The exceptions are obese patients with multifolded skin; short necks; and tracheostomies, severe heart failure, and orthopenia.2

Adjunctive measures have been suggested to promote safety and comfort with the procedure, namely: (1) routine use of a non-Trendelenburg position; (2) minimal anesthetic infiltration to offset iatrogenic vein compression; (3) use of a micropuncture set to reduce venous delenburg position; and (4) use of the transverse view to probe the most central diameter of the vessel. These points were obtained from over 15 years of self-training in sonography for thoracoabdominal and peripheral evaluation, for difficult arterial insertion, and for venous entry into the internal and external jugular veins, subclavian veins, and innominate veins. The result of this experience became evident in the endomyocardial biopsy of cardiac transplants.2

Self-training in real-time sonography should be included as a benefit of recognition and certification, as given by the proctoring system of the American College of Emergency Physicians, the American College of Surgeons, and the American College of Chest Physicians. Though these entities don the recognized authority to initiate competency for quality assurance and to promote an undefined legal standing to perform the procedure, self-training, once recognized as a self-willed earnest effort to achieve excellence, is implicitly regarded as an egocentric, deceiving, deprecatory method of learning. Bravado without medical and legal appreciation!

In any specialty, self-training is the product of different concepts, the harbinger of new management, and a precursor of organized evaluation and acceptance. What role should it play in the formal education of a physician for an organized entity such as a hospital, the American Colleges, or other medical associations? Just as the proctoring system is recognized as a form of training for the physician by the accepted medical organizations, why shouldn’t self-training be afforded an equal stance in the eyes of the same medical organizations since: (1) both methods aim to achieve the same results: safety and competency; and (2) self-training in a new approach or technique when accepted by a medical organization allows for the development of a proctoring system to promote that approach or technique.

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The author has reported to the ACCP that no significant conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

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References


Response

To the Editor:

I thank Dr. Olivier for his comments regarding my article.1 Although I appreciate Dr. Olivier’s recommendations to develop ambidextrous skill as well as his “adjunctive measures,” the purpose of my article was to propose a standardized approach for ultrasound-guided internal jugular access as opposed to offering “pearls” that develop with experience. As Dr. Olivier states, his “points were obtained over 15 years.” Clearly, self-training is integral to proficiency in all aspects of medicine. The astute physician constantly reviews their technique and style with an attention toward self-improvement. Self-training should, however, be used in conjunction with, and not instead of formal didactics and instruction from experts/mentors. In an article by Mey and colleagues,2 when performed with the two-operator technique, the experience of the physician controlling the needle did not influence procedural success or complication rate, whereas both were significantly reduced when the physician manipulating the sonographic probe was experienced. This illustrates the fact that there is a learning curve associated with sonography, and that guidance based on misinformation can harm our patients. The responsible way to develop skills in any procedure is to understand the concepts of the procedure, learn the psychomotor skills, and integrate them with clinical judgment and experience. Medical simulation combines deliberate practice and feedback with the goal of achieving mastery.3 By combining didactics, simulation, and mentorship, the learning curve may be significantly reduced. It is the responsibility of our collective societies to develop formal training guidelines before they are imposed on us from third parties. Until these guidelines become available, it is my hope that the recommendations I proposed will lay some of the groundwork to improve training and education in ultrasound-guided central venous catheterization.

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Natural History of Stage I Lung Cancer

This estimate by Raz et al (July 2007)\(^1\) of surgical benefit and overdiagnosis magnitude in patients with stage I non-small cell lung cancer (LC) should be viewed with caution.

1. The comparison of survival in operated vs unoperated persons lacks balance because the former are surgically-pathologically staged, and the latter are clinically staged. As is well known, surgery frequently upstages clinical evaluation.
2. If the cohort declining surgery were skewed by a higher frequency of comorbidities, it would account, in part, for their diminished 5-year survival rate vs the surgically treated cohort.
3. As the authors\(^1\) note, the systematic understaging of unoperated persons conveys an unfavorable estimate of the natural history of untreated stage I non-small cell LC (ie, reverse Will Rogers Effect).
4. The authors\(^2\) incorrectly identify pseudodisease (ie, “false diagnosis,” “iatrogenic pseudodisease,” “lanthanide disease,” and “clinically irrelevant cancer”)\(^3\) with the estimated 5-year survival rate of 11% in persons with clinical stage I disease who declined surgery. Overdiagnosed cases are represented by an undetermined proportion of the 11% of 5-year survivors who will later succumb to non-LC plus those dying of non-LC (100 – 78 = 22%) within 5 years. This figure is similar to the proportion of excess cases (attributed to overdiagnosis) in the intervention cohorts of the Mayo Clinic screening trial\(^2\) (22%) and Czech screening trial\(^3\) (24%).
5. The assignment of cause of death is problematic, particularly in persons with LC who frequently have competing, lethal comorbidities. Death certificates are inaccurate sources for this information.\(^4\) Some states assign precedent cancer as the default diagnosis when the cause is uncertain. The Mayo Clinic trial\(^2\) and the Czech trial\(^3\) circumvented this difficulty by assigning a panel to review the medical records of the deceased.
6. Because overdiagnosed persons are destined to die of other causes, their inclusion in a treated cohort generates a spurious benefit as measured by LC survival.\(^5\)
7. Survival is an invalid metric of efficacy.\(^6,7\) Due to the surgical mortality rate (2%) and the long-term harm of lobectomy (which foreshortens the course of older smokers’ characteristic lethal comorbidities) in understaged persons (30%) and overdiagnosed persons (22+%), a reduction in LC mortality in the remaining 40% that is sufficient to more than offset this increase in non-LC mortality must be attained to achieve a net benefit. This reduction can be achieved solely by the surgical interdiction of advanced LC, which was not achieved in the intervention cohorts of the Mayo Clinic trial\(^2\) and the Czech trial.\(^3\) A preliminary assessment of the CT scan trials has, similarly, shown no reduction.\(^8\)

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The authors have reported to the ACCP that no significant conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

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REFERENCES


Response

To the Editor:

We agree with Drs. Reich and Asaph that our study has limitations by nature of utilizing retrospective cancer registry data. Since it would be unethical to randomize stage I non-small cell lung cancer (NSCLC) patients to treatment or no treatment, population-based observational studies are the next-best data source to describe the natural history of stage I NSCLC. As mentioned in our article,\(^1\) the lack of information on staging methods likely results in underestimation of survival in patients with untreated stage I disease. For clarification, a lung cancer-specific 5-year survival of 22% and overall survival of 11% means that for a cohort of patients with stage I NSCLC with complete follow-up, 78% will have died of lung cancer, 11% will have died of other causes, and 11% will have survived for 5 years. Assuming that 5 years is sufficient time to estimate long-term survival from lung cancer, the percentage of patients with pseudodisease can be estimated by adding the 11% of survivors to a proportion of the 11% of patients who died of other diseases who would not have died of lung cancer had they survived. While Reich and Asaph argue that overall survival is not an adequate measure of efficacy for surgical resection in stage I NSCLC, it is hard to argue with data showing the excellent overall survival of patients with surgically resected stage I NSCLC, especially small tumors, compared with the survival of patients who refuse surgical resec-
tion. These survival estimates include perioperative deaths and deaths from comorbid conditions.

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**Response**

To the Editor:

We thank Dr. Laohaburanakit for his comment on the potential impact of concomitant use of systemic corticosteroids during acute exacerbations of chronic bronchitis (AECB) on the main findings of our metaanalysis that compared second-line to first-line antimicrobials for the treatment of patients with AECB. We agree with our colleague that use of corticosteroids should be standardized because they modulate local and systemic inflammatory response and, thereby, may act as a potential confounding factor. Indeed, a Cochrane review revealed that treatment with corticosteroids, compared to placebo, was associated with fewer treatment failures in patients with AECB (odds ratio, 0.48; 95% confidence interval, 0.34 to 0.68). Acknowledging this evidence in another metaanalysis, we commented on the potential confounding role of the administration of corticosteroids when evaluating the comparative effectiveness of several second-line antimicrobials for the treatment of AECB.

Unfortunately, 8 of 12 randomized controlled trials (RCTs) included in the present metaanalysis did not provide data on the concurrent administration of systemic corticosteroids for the management of AECB. As concerns the remaining four RCTs, in the trial by De Vlieger et al., “concomitant methylprednisolone consumption of > 8 mg was an exclusion criterion,” while in the three remaining RCTs the compared groups of patients were similar regarding use of systemic corticosteroids during AECB (44% vs 39%, 100% vs 80%, and 19% vs 19%, in the RCTs by Ulmer et al., Mertens et al., and Bachand et al., respectively). Specific information on the treatment success of corticosteroid recipients was not given.

We performed a subgroup analysis by including only the three latter RCTs that mentioned concomitant use of corticosteroids. Our main finding was the same, namely that first-line antimicrobials were associated with lower treatment success compared to second-line antimicrobials (odds ratio, 0.42; 95% confidence interval, 0.22 to 0.79), a result that should be in the context of several other factors that influence clinical decision making and that are discussed in our article.

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power indicates that this was more than likely an ideologically motivated, personal choice rather than a “career move.” In addition, being a leader in the SA was not just a political statement; the SA was Hitler’s private, paramilitary, terrorist, militia organization that perpetrated numerous acts of violence throughout Europe, primarily targeting Jews and political opponents. One of the most horrific examples includes the now infamous Kristallnacht (night of broken glass) in 1938 in which >30,000 Jews were arrested and deported to concentration camps in a single night. The slogans of the SA included “terror must be broken by terror” and “all opposition must be stamped into the ground.”23 At that time, Dr. Wegener was a Lt. Colonel in this organization.3

THE ACCP MASTER CLINICIAN AWARD

The arguments used to not take away the award are somewhat specious; this decision should not have been controversial. Up until Dr. Rosen’s article in September 2007, the ACCP had made no mistakes regarding Dr. Wegener; the information was lacking, and no apologies are necessary. However, with the current information (gained at great personal effort by Dr. Woywodt3,4), the ACCP must withdraw the award. In this present era of terrorism—specifically with recent acts of terrorism being perpetrated by physicians in London—national medical societies must remain as far removed as possible from any potential acts of war or terror: past, present, or future. Dr. Rosen’s arguments for Dr. Wegener keeping his award make little sense. Whether or not he has been legally convicted of “war crimes” is irrelevant; his being a high-ranking officer in the brownshirts is more than reason enough to rescind his award. Finally, the statements regarding the date that the Master Clinician Award was presented (1989) and date of Dr. Wegener’s involvement in the Nazi party (from 1932 to 1945) as factors in determining whether or not the award should be retracted depend on these awards from national medical societies, the leadership of national medical societies, and specifically the ACCP.

DR. WEGENER’S NAZI CAREER

Dr. Wegener’s voluntary enlistment and rapid rise to leadership in the Nazi storm troopers (SA) is not synonymous with stating that he was merely a member of the Nazi party; it has significant implications, both ideological and practical. The fact that Dr. Wegener apparently joined the SA prior to Adolph Hitler’s official rise to

Letters Re Dr. Friedrich Wegener

Editor’s Note: Because of space constraints, we are publishing four of the letters received. They contain interesting comments and reflect divergent points of view.

Richard Irwin, MD, FCCP
Editor in Chief, CHEST

Time Does Not Heal All Wounds

Medical Luminaries, National Socialism, and the American College of Chest Physicians

It was with great interest that we read Dr. Rosen’s recent article1 (September 2007) on the wartime activities of Dr. Friedrich Wegener. Dr. Rosen is to be praised for his delicate and articulate handling of a complex and difficult situation. We were, however, surprised, disappointed, and confused with the ultimate conclusions and actions—or lack thereof—by the American College of Chest Physicians (ACCP). Our letter has two major points: (1) to describe why the ACCP Master Clinician Award should in fact be posthumously taken away; and (2) to enunciate why the eponymous use of the term Wegener granulomatosis should be terminated.

WEGENER GRANULOMATOSIS: WHAT MUST BE THE END OF AN ERA

Having a disease named after someone is a tremendous and rare honor in the field of medicine. If such a person is found to have taken part in criminal activities, physician leaders are honor bound to do all they can to rectify the mistake and remove the accolade.5 It is inappropriate and irrelevant to state that subsequent “good” actions and statements may absolve one from having been involved in such organized criminal activities. If this disease were known as Himmler’s granulomatosis, there would not be national debate as to the continued use of this eponym. The Vasculitis Foundation of North America has stated that “as patients and family members, we would prefer a different name for our disease.”6

The ACCP and Dr. Rosen have been given an opportunity to bring historical accuracy to the memory of a man who participated in the evil Nazi regime, which visited death and destruction to so much of the world from 1933 to 1945. This opportunity cannot, should not, and must not be missed.

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References

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Friedrich Wegener
The Past and Present

To the Editor:

Dr. Mark Rosen has elucidated the deliberations of the American College of Chest Physicians (ACCP) in reviewing its decision in 1989 to bestow an award on Dr. Friedrich Wegener.1 The 1989 decision took place without the ACCP having the benefit of Dr. Wegener’s complete curriculum vitae. We applaud the frankness and good will of the ACCP in engaging in this discussion. While we understand the dangers of the “retrospectoscope,” we believe that the ACCP, in spite of its carefully considered judgment, should move to correct a misjudgment of the past and retract the award to Dr. Wegener. What has changed is not the values or mores of the ACCP, as strongly held in 1989 as today, but biographical facts are evident today that if known in 1989 would have disqualified Dr. Wegener as an ACCP awardee then, as they do now.

This discussion has been brought about by biographical information concerning Dr. Wegener’s professional life brought to light by one of the authors2 and conveyed to the ACCP by the other author. Dr. Rosen’s column details Dr. Wegener’s National Socialist Party (Nazi) membership, his ascension to rank of “physician Lt Colonel” in the storm troopers, and his professional services in Lodz, Poland. It is true that there is no currently available evidence of Dr. Wegener as a direct perpetrator in the heinous crimes of the National Socialist regime in the Warthegau or elsewhere in Nazi occupied Europe. Nevertheless we believe Dr. Wegener is morally accountable for choosing to join the Nazi organizations liable for expounding a philosophy of violence inevitably producing mass murder; and for remaining publicly silent about events until his death in 1990. By such silence, he chose to remain a bystander rather than bear witness to the genocidal crimes he observed in the Lodz ghetto. Bearing witness may have been all the Jewish and Romany people, incarcerated, brutally mistreated in the ghetto, and inexorably annihilated, could have hoped for.

Dr. Wegener’s motivation for joining the Nazi party and its storm troopers went to the grave with him. But to varying degrees, it may be attributed to the following: (1) careerism: Dr. Wegener’s supervisor and academic mentor Martin Staemmler was one of the major proponents of the regime; (2) family ties: his brother was a devoted party member who rose to the rank of “general” in the Schutzstaffel (SS) and Gauleiter; and (3) temporal appeal: the Nazi biological agenda had great allure for physicians, anthropologists, and biologists. This agenda, and careerism, attracted almost 50% of German physicians to join the Nazi party.

Regardless of motive, Dr. Wegener chose to join organizations dedicated to the racial agenda that led to the industrialized genocide carried out by the Third Reich. He then chose silence.

Dr. Wegener’s Nazi party membership and storm trooper rank distinguishes him from drafted German citizens serving in the armed forces during World War II. During his 6-year tenure in Poland as an army and health office pathologist, Dr. Wegener’s office was three blocks from the Lodz ghetto. The ghetto included Jews from Lodz as well as Jews and Romany deported from the Reich. They were incarcerated, starved, and utilized as slave labor before their deportation to the Chelmno death camp. There they died in mobile gas vans, and their ashes were scattered in the woods. It is inconceivable that Dr. Wegener did not see nor know what awaited those human queues in Lodz, boarding the open trains to take them on their last journey.

After 1945, Dr. Wegener resumed his career at the first opportunity, continued his work, and avoided ever publicly commenting on what he had surely seen and knew of. We comprehend silence during the Nazi regime as there is no moral requirement for heroism. However, it would have been far less courageous and even morally required to speak out at some time and place after the war. There was still risk, however. The German medical establishment even to this day favors silence, encouraging “not soiling one’s own nest” (nestrerschmutzen). Some in the medical community who spoke out were in various ways ostracized, punished, or driven from their chosen professional paths and not only in Germany.5 As Dr. Wegener attained international acclaim, his testimony may have helped the German medical establishment finally free itself of its shameful past of the National Socialist era. In addition, he could have provided additional eyewitness evidence to further refute the revisionists and holocaust deniers. But Dr. Wegener chose silence.

For this, he must be held morally responsible and thus ineligible for honors from the ACCP or other organizations expressing humanitarian ideals. We urge the ACCP to retract the Master Clinician Award from Friedrich Wegener. We also encourage the replacement of the disease eponym Wegener syndrome with that of ANCA-positive vasculitis in all publications of the ACCP. Not only do Dr. Wegener’s life choices more than justify this, but like so many eponyms it is either an inaccurate reflection of the contribution of the eponymee or an inapt expedient.6

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Comments on Dr. Wegener Editorial

To the Editor:

“The only record of this occurrence is in the Convocation Program printed that year.” In fact, there is a recording of Dr. Wegener’s presentation.

I attended the American College of Chest Physicians (ACCP) meeting in Boston in 1989 (I was 33 at the time), and I remember very well the presentation Dr. Wegener made at the meeting. As I was unable to attend his presentation, I managed to attend the award ceremony just to see the man (a tall, thin, apparently proud man) and, above all, I bought a tape recording of the presentation. I remember listening to the tape the following days in my car on my way to my hospital back in France. He was speaking in German (the first words were Es ist eine grosse Ehre . . . or something very close to that), and each sentence was immediately translated in English (my friend Dr. Sergio Salmeron who attended the presentation believes Dr. U. Speckes was the translator). It was very moving to hear this man describing not only “his” disease and the way he discovered it, but also where he came from, and there was apparently (I did not see them, unfortunately) at least one slide with his family and one slide from Löbeck. As far as I remember, there was no allusion to his past in the World War II years. Maybe this recording could provide evidence as to whether Dr. Wegener made any false or misleading statement regarding his past?

Shortly after the meeting (or a couple of years later), somebody told me about Dr. Wegener’s “unclear” past. I thought the ACCP was aware of that past and had considered there was insufficient evidence to deny or withdraw the award.

Regarding what to do now, I support your current decision: if the award was given in good faith, as it was “scientifically” deserved, as there seems to be so far no evidence of Dr. Wegener participating directly in war crimes, and if there is no evidence of false or misleading statements, I believe he can be left with the benefit of doubt (benefit of doubt).

Regarding the tape, I have been unable to locate it, but other attendees must have this tape, as well as, maybe, the ACCP and/or the company that made the recording. I certainly appreciate ACCP’s transparency on all this . . .

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On Wegener and the ACCP

To The Editor:

I read your editorial “Dr. Friedrich Wegener, the ACCP, and History” with great interest. In addition to the specific issues and concerns you addressed—namely the special recognition given by the American College of Chest Physicians to Dr. Wegener—the eponymous association of Wegener with the syndrome he described has also been called into question. Should we continue to call this disease Wegener granulomatosis? Several other, rather notorious, Nazis have eponymous disease associations. They include Hans Reiter, who was directly and personally implicated in multiple war crimes, including typhus experiments carried out on concentration camp victims. In 2003, an international group of rheumatology journal editors decided to eliminate usage of the term Reiter syndrome, and this eponym no longer appears in many journals nor in recent editions of several internal medicine textbooks (largely replaced with the term reactive arthritis). An analogous decision was made regarding Hallervorden-Spatz disease when it became clear that Julius Hallervorden’s wartime reputation was remarkably enhanced by his dissections of “wonderful material”: 500 brains obtained from euthanized “feeble-minded individuals.” Dr. Wegener was never convicted of any war crime. His war-time records have largely “disappeared.” He also never apologized for, or even publically acknowledged, his very early membership in the Sturm Abteilung (SA) Brownshirts and then the Nazi party. I have chosen not to use the term Wegener granulomatosis in my professional and educational activities and instead use the term granulomatous vasculitis. When my lack of eponymous usage is questioned, it provides an opportunity for historical education.

I also would like to point out a most interesting coincidence. The term Wegener’s granulomatosis was introduced into the English medical literature and promoted by the pathologists Jacob Churg and Gabriel Goldmann in 1954. Dr. Churg was born in 1910 in the eastern European Jewish shtetl of Dollinow. Following graduation from the medical school in Wilno in 1936, he immigrated to New York City and joined his uncle, Louis Chargin, Chief of Dermatology at Mt. Sinai. He later became a renal pathologist of great renown and has a disease named after him: Churg-Strauss syndrome. Is it not ironic that Dr. Wegener’s fame is largely attributable to an eastern European Jew, who, had he not escaped to the United States, would certainly have been incarcerated in a ghetto, perhaps even the notorious Lodz Ghetto, where Dr. Wegener was dissecting victims just 3 years later in 1939?

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Dr. Emmett is Chairman of Internal Medicine, Baylor University Medical Center. I have no conflict of interest to declare except that I am the child of Holocaust survivors, was born in a displaced persons’ camp in Austria, and my oldest sister together with many uncles, aunts, and cousins were murdered by the Nazis.

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