laboratories and from rigorously monitored clinical trials into which patients are entered after they give informed consent. I appreciate and applaud Dr. Chambers' enthusiasm and search for improvement.

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REFERENCE

1 Holmes EC. Preoperative neoadjuvant therapy in NSCLC: open season. J Natl Cancer Inst 1991; 83:228

An Unusual Cause of Electrical Alternans

To the Editor:

This electrocardiogram (ECG) (Fig 1) was recorded during an acute exacerbation in a patient with bronchial asthma. At that time his heart rate (92 beats per minute) was two times his respiratory rate (46 breaths per minute). As a result, the variation in the amplitude of QRS complexes that sometimes occurs with excessive respiratory movement occurred with alternate heart beats, giving an appearance of electrical alternans. This was most marked in lead AVL. The "electrical alternans" was abolished when the ECG was recorded in some leads (Fig 1, bottom row) with the patient holding his breath for the duration of two or three heartbeats.

The patient made an uneventful recovery with usual therapy for bronchial asthma. Clinical features, electrocardiography, plain radiographs of the chest, and echocardiography did not reveal any evidence of cardiac or pericardial disease.

Electrocardiographic electrical alternans is associated with pericardial effusion or myocardial disease. In the latter, it indicates an adverse prognosis. The mechanism causing an appearance resembling electrical alternans in this patient is probably uncommon; it has not been previously reported. Nevertheless, if electrical alternans is encountered in a patient whose heart rate is exactly twice his respiratory rate, the simple bedside maneuver described above may prevent diagnostic and prognostic confusion. Recently, the value of clinical information in the interpretation of ECGs in patients with suspected myocardial infarction has been questioned. This case shows how clinical correlation, as well as the presence of the interpreter when the ECG was being recorded, dramatically altered the interpretation of an ECG abnormality.

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REFERENCES

2 Fisch C. Electrocardiography and vectorcardiography. In: Braun-
Communications to the Editor

Reactive Airway Disease after Chlorine Gas Exposure

To the Editor:

We read with great interest the case report by Moore and Sherman. Chlorine gas exposure has multiple toxic effects on the respiratory system, including immediate airflow obstruction, ARDS, and death. The long-term effects can include abnormalities in gas transfer, restriction, and airflow obstruction. These generally resolve with time to a variable degree. Additionally, the severity and resolution of these effects appear to be related to the degree of exposure, premorbid pulmonary status, and the degree of hypoxemia on initial presentation.

In 1990, we reported two cases of reactive airway dysfunction syndrome (RADS) following chlorine exposure to chlorine gas. Both patients had an asthmatic diathesis extending over time. One individual has had persistent asthmatic symptoms for the past six years; the symptoms have ameliorated with time, although she continues to be symptomatic. She is receiving minimal medication at the present time, compared with her immediate post-exposure needs. Before exposure, she was neither a smoker nor an asthmatic. The other individual was also exposed to chlorine fumes. There was no history of pre-exposure asthma, and he had discontinued smoking several years prior to his exposure. Not only did this individual have asthmatic symptoms, which responded to bronchodilators, but he also had persistent hypoxemia, which has resolved over the years. His exercise stress test initially accentuated his hypoxemia, and his primary symptoms were those of dyspnea on exertion. His condition has ameliorated with time.

Thus, chlorine exposure can present with asthmatic symptoms and RADS, as described by Brooks et al. Our experience has been that the amount of asthma (diagnosed either by symptoms or by the medication needs) ameliorates with time. Hypoxemia with poor cardiopulmonary reserve was observed in one individual, and this also resolved with time.

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REFERENCES

To the Editor:

We welcome the comments of Drs Demeter and Cordasco. We agree that our patient probably does have RADS, although he does not fulfill all of the criteria set forth by Brooks et al. Clearly the cases reported by Demeter and Cordasco represent persistent hyperreactive airways disease after chlorine gas exposure. Our patient differs from theirs in that his symptoms and level of disability have not ameliorated with time. On the contrary, six years after the exposure this patient still requires home oxygen therapy, oral corticosteroids at high doses, frequent use of beta-agonist inhalers, and frequent injections of subcutaneous epinephrine. In a recent telephone conversation, he informed us that his symptoms have actually worsened since our report was submitted. He is barely able to perform normal activities of daily living and reported that his physicians referred him to a transplant center for possible heart-lung transplantation.

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Pulmonary Lymphangioleiomyomatosis

To the Editor:

I read the very interesting case report by Huml et al., which appeared in the December 1991 issue of Chest. I was curious to know whether the patient described so well by the authors had sarcoidosis or a localized sarcoidal reaction. It would be helpful in making this distinction if the authors were to state whether noncaseating granulomata were observed outside the thorax in the organs examined at autopsy. It would also be of interest to know something of the patient's ethnic background.

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REFERENCE

To the Editor:

In our case report, the absence of multigain involvement with noncaseating granulomas speaks against systemic sarcoidosis. No evidence of noncaseating granulomas was found outside the thorax. This raises the very interesting possibility that an immunologic mechanism may play a role in the etiology or that the presence of noncaseating granulomas may represent a localized immunologic reaction to lymphangioleiomyomatosis. Further study and clinical correlation are necessary to prove this hypothesis.

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