The authors state that the pleural effusion in their case was not a parapneumonic effusion because there were no parenchymal infiltrates on chest roentgenograms. However, the reliability of chest roentgenograms in ruling out parenchymal infiltrates in the presence of a pleural effusion (massive initially, and requiring decortication within two weeks) can be questioned. In this case, the history of a low-grade fever and findings of leucocytosis and left shift on admission suggests an infection. The background of seizure disorder, anticonvulsive medications, broken or missing teeth and most importantly, recovery of multiple anaerobic organisms from the pleural fluid suggests an aspirational (gravitational) route of infection, from the mouth to the lung. The characteristics of the pleural fluid-exudate, low glucose level, negative gram stain and neutrophilic predominance (increasing in number from 2400/cu mm to 4000/cu mm) are all very consistent with a parapneumonic effusion evolving into an empyema. Determination of pH of the pleural fluid is helpful in differentiating complicated (empyemic or loculated) from benign parapneumonic effusions. Measurement of pH of pleural fluid from the initial thoracocentesis and serially in this case may have resulted in a decision to institute chest tube earlier.

Pleural effusions in yellow nail syndrome are usually, bilateral or unilateral, at times massive, and tend to be chronic. In the few cases where information is available regarding the pleural fluid cell count, the predominant cells have been lymphocytes. Hiller et al. reported normal concentration of glucose in the pleural fluid. To add low glucose level as another possible characteristic of the pleural effusion in yellow nail syndrome, on the basis of present information is not tenable and may be misleading.

To the Editor:

We do not disagree with the general comments relative to a low glucose level in pleural fluid, as presented in the first paragraph of the communication by Sahn and Good, although the purpose of our case report was not to review all aspects of glucose in pleural effusion. We disagree with the opinion of Sahn and Good that the initial cause of the pleural effusion in our case was due to empyema. The fluid obtained by thoracocentesis at the time of admission did not demonstrate a reduced pH, purulence, or bacterial growth from properly obtained anaerobic cultures. Under no known medical criteria could this be considered an empyema or parapneumonic effusion. Surely one would have difficulty explaining the reduced level of glucose found on the initial thoracocentesis on the basis of "phagocytosing leukocytes and multiplying bacteria."

Sahn and Good point out that the thoracocenteses performed 60 and 90 minutes after glucose loading may have allowed insufficient time for the glucose to appear in the

O'Donohue the pleural manifestation occurred by accident, not associated with the common etiologic relationship of the yellow nail syndrome, namely, the primarily defective lymphatic circulation. The history data on their patient as having profound mental retardation and being under continuous use of anticonvulsants and sedatives, suggests a propensity toward the development of aspiration episodes from which pneumonitis or lung abscess with empyema might concur.

A febrile course coupled with pleural fluid examinations showing an initial count of 2400 cells/cu mm, predominantly polymorphonuclear leukocytes, which ultimately became frank pus, was consistent with an infective process. It is here remarked that previous cases of the yellow nail syndrome exhibited lymphocytic effusions. Subsequent isolation of anaerobic pathogenic organisms in the case reported by Angelillo and O'Donohue, Jr., provided further support to the preliminary diagnosis of pneumonia by aspiration with parapneumonic effusion. Additional information that "no parenchymal infiltrates were observed on chest roentgenograms," did not rule out concurrent lung inflammation, but rather reflected the interpretation of follow-up films after effective treatment.

The mechanisms for low glucose levels in the pleural exudate are an excessive utilization by inflammatory cells and the impairment of diffusion across the thickened inflammatory membrane lining the pleural cavity. Results of thoracocenteses performed 60 and 90 minutes after loading with glucose and showing no rise in glucose levels supported the idea of an undue delay in glucose transportation from the blood to the pleural fluid.

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REFERENCES

To the Editor:

The concluding sentence of the case report by Angelillo and O'Donohue states that the yellow nail syndrome "may also be included in the list of diseases that are associated with a glucose level in the pleural fluid of less than 10 mg/100 ml." This statement is on shaky ground since there is an additional diagnosis—empyema—which could explain the low glucose levels. The low glucose levels were all found in pleural fluid aspirate, seven days or less from the date when the empyema was diagnosed.

CHEST, 77: 2, FEBRUARY, 1980

COMMUNICATIONS TO THE EDITOR 243
pleural fluid. They reference an article by Russakoff et al. as providing evidence that there may be a lag of from two to four hours before glucose appears in the pleural fluid after glucose loading. In that article, seven cases were presented. Four of the patients had tuberculosis, and two had carcinoma with pleural metastases. One would expect, as Russakoff et al. point out, that the curve for glucose in the pleural fluid would be flat when extensive thickening of the pleural membrane is encountered. The study also demonstrated that in every case, except one, an increase in the glucose level of the pleural fluid was observed in the first 90 minutes, even though the curves remained relatively flat as compared to the graphs of the glucose level in the blood. It should also be added that four of the seven curves of glucose tolerance are clearly abnormal, with either excessive elevations of the glucose level in the blood or prolongation of the increased glucose levels in the blood beyond three hours. Russakoff et al. commented that no broad sweeping conclusions are to be drawn from these data.

The fact that the glucose level in the pleural fluid was still 10 mg/100 ml after antibiotic therapy and drainage via a chest tube for five days, in addition to the presence of clear pleural fluid that was free of bacteria and white blood cells, is somewhat different from the observation by Sahn and Good that "several days are required for the glucose concentration in the pleural fluid to rise above 60 mg/100 ml, despite appropriate antibiotic therapy and drainage." Specifically, one of the reasons for submitting our case report was that the very low glucose levels in the pleural fluid could not be explained by empyema alone or by other pathologic mechanisms known to be associated with a reduced glucose level in the pleural fluid.

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Nitroglycerin-Induced Bradycardia and Hypotension in Acute Myocardial Infarction

To the Editor:

Come and Pitt. have described their experience regarding the rare occurrence of bradycardia and hypotension associated with the use of nitroglycerin. We would like to describe our recent experience with this unusual phenomenon.

CASE REPORT

A 67-year-old white man was admitted for treatment of unstable angina. Propranolol 20 mg and 2 percent nitroglycerin ointment were immediately started. Then, almost simultaneously with the administration of these medications, he started complaining of chest pain again. Nitroglycerin 0.3 mg was immediately given sublingually. After one to two minutes, he developed blurring of vision and became very diaphoretic. The blood pressure and heart rate before nitroglycerin was 180/100 mm Hg and 90/min respectively. After nitroglycerin, the former dropped to 60 mm Hg palpatory and the latter to 60/

min. Then the patient started gagging and the blood pressure and heart rate increased spontaneously to 120/80 mm Hg and 90/min respectively before atropine and dopamine could be administered. His sensorium improved. The serial ECG and cardiac enzymes revealed changes of acute anteroseptal myocardial infarction. Propranolol and nitroglycerin ointment in increasing dosages were continued without further adverse effects.

This case illustrates the unusual occurrence of bradycardia with hypotension secondary to nitroglycerin. The possibility of carotid sinus hypersensitivity cannot be ruled out. However, the circumstances of this case point strongly to a nitroglycerin-induced phenomenon. The mechanism of bradycardia cannot be explained at this time.

The clinical importance of this patient is to emphasize that the first dose of nitroglycerin in suspected myocardial infarction should be carefully supervised.

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REFERENCES

Tracheal Obstruction in Chronic Lymphocytic Leukemia

To the Editor:

Chronic lymphatic leukemia is a disease in which old lymphocytes accumulate in various organs of the body producing signs and symptoms of the disease. Obstructive symptoms, produced by enlarged lymphoid masses, are extremely uncommon unless there is associated generalized lymphadenopathy and hepatosplenomegaly. We wish to present a patient who posed an extremely difficult diagnostic problem with compression of the trachea by enlarged nodes, but without signs of generalized bulky disease.

CASE REPORT

A 91-year-old active caucasian woman, treated successfully for ten years with chlorambucil, presented with symptoms of upper airway obstruction and syncope. Physical examination confirmed stridor, scattered adenopathy in cervical triangles, clear lung fields and no hepatosplenomegaly or generalized adenopathy. Laboratory data confirmed chronic lymphatic leukemia in partial remission. Chest x-ray film revealed mild cardiomegaly and tomography of the larynx and trachea revealed narrowing of the membranous portion of the trachea by extrinsic pressure (Fig 1). The patient underwent bronchoscopic examination which revealed no intraluminal pathology and biopsy of mediastinal lymph nodes measuring 2-3 cm in longest diameter which were compressing the trachea. Histology of these lymph nodes was compatible with malignant lymphoma, lymphocytic, well-differentiated, diffuse type. She was referred for irradiation.

DISCUSSION

Intrinsic upper airway obstruction has been reported rarely in benign and malignant neoplasm of the oral cavity, but

244 COMMUNICATIONS TO THE EDITOR

CHEST, 77: 2, FEBRUARY, 1980