Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Discrepancy between Measured Oxygen Tension and Saturation in Patients with Leukemia

To the Editor:

We read with great interest the article by Prakash, Divertie and Banks (Chest 75:345-350, 1979) on respiratory failure in leukemic patients. Because of the nature of our institution, we are frequently faced with decisions regarding the intensity of multiple organ support justified in patients for whom only limited further therapy exists. We are greatly encouraged by reports from other physicians who share our enthusiasm to provide maximum, although time-limited, support for the life-threatening complications of acute leukemias.

We are, however, somewhat surprised by the statement of Prakash and colleagues in which they attribute oxygen desaturation, despite high PaO₂ levels, to the high metabolism of the elevated numbers of white cells seen leukemia. For a given PaO₂ two factors influence oxygen saturation: hemoglobin affinity for oxygen, usually expressed by the P₅₀; and the presence of ligands which combine irreversibly with hemoglobin and possess a greater hemoglobin affinity than does oxygen.

Fever, hypercapnia, metabolic acidosis, low levels of 2,3DPG are all factors which displace the oxyhemoglobin dissociation curve to the right, thus lowering hemoglobin saturation for a given PaO₂. These conditions are often present in leukemic patients with acute respiratory failure. Methemoglobin, sulfhemoglobin and carboxyhemoglobin may reduce the amount of hemoglobin available to be saturated by oxygen. Saturation is thus lower than expected at a given PaO₂. The importance of abnormal hemoglobins in critically-ill patients has recently been emphasized. If leukocyte metabolism were the only factor influencing oxygenation, one would expect to find a normal arterial PaO₂ and saturation and a very low venous PaO₂ and saturation, reflecting increased oxygen consumption.

Unexplained hemoglobin desaturation in leukemic patients merits further study and, if it proves a consistent observation, would bear great clinical relevance. Investigative efforts are therefore necessary to ascertain whether this finding is due to a shift of the oxyhemoglobin dissociation curve or the presence of abnormal hemoglobin ligands.

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REFERENCE

1 Cohn JD, Engler PE: Shunt effect of carboxyhemoglobin. Crit Care Med 7:54-58, 1979

To the Editor:

Drs. Carlon and Turnbull have very properly pointed out that a twice repeated statement which appeared in our paper “Aggressive Therapy in Acute Respiratory Failure from Leukemic Pulmonary Infiltrates” needs further explanation. This referred to factors which may aggravate hypoxemia in some leukemic patients. A recent abstract (Fox MJ, Brody JS, Weintraub LR: Leukocyte larceny; A cause of spurious hypoxemia. Am Rev Respir Dis 194:113, April 1979 Supplement) also calls attention to a progressive fall in arterial oxygen tension and content occurring during laboratory testing of blood from two leukemic patients with high white blood cell counts. The authors speculate that this phenomenon might produce tissue hypoxia in vivo if the circulation time became prolonged or leukostasis occurred. As a local or regional phenomenon, this seems entirely possible.

The original observation by our colleague, Dr. W. W. Douglas, in a patient with an acute blastic crisis of chronic granulocytic leukemia was of great interest to us, but we quoted him incorrectly. There was indeed a progressive hypoxemia in vivo and a discrepancy between arterial oxygen tension and saturation, but not as we recorded. This is regrettable and it seems proper and appropriate to have this clarified. Dr. Douglas’ clarification follows.

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To the Editor:

As far as I know, Drs. Carlon and Turnbull are correct when they say that leukemia per se does not cause a shift in the oxygen-hemoglobin dissociation curve. Furthermore, I have never seen a decrease in the oxygen saturation, as expressed by the concentration of oxyhemoglobin relative to the sum of oxyhemoglobin and reduced hemoglobin, when the oxygen tension was normal.

However, we have seen a discrepancy between O₂ tension (PaO₂) and the reported O₂ saturation (SaO₂) in patients with leukemia from two causes. As noted by Carlon and Turnbull, SaO₂ may be reported to be low because of elevated carboxyhemoglobin (COHb) levels. This occurs because spectrophotometric oximeters report SaO₂ as the concentration of oxyhemoglobin relative to the sum of oxyhemoglobin, reduced hemoglobin and carboxyhemoglobin.

The reason for the increased COHb readings in some patients with leukemia is not known; it may represent an artifact or, if COHb levels are truly increased, may reflect an increased rate of heme degradation. Accelerated leukocyte metabolism may result in a decreased venous PaO₂; if this is so, higher levels of COHb would be expected for any given blood carbon monoxide tension because of the competition between O₂ and carbon monoxide for reduced hemoglobin.

In other patients, SaO₂ may be reported to be different than the PaO₂ if a significant time interval elapses between measurement of the two values; this difference appears to be related to the high rate of O₂ consumption within the blood sample by the increased population of neutrophils. Both causes were present in the following illustrative case.
When leukocyte counts are very high, a lapse of several minutes between measurement of Po₂ and So₂ will result in a discrepancy between the two values, suggesting abnormal oxygen-hemoglobin dissociation as well as spurious diagnosis of hypoxemia. In patients with markedly elevated neutrophil counts, delays in analysis must be avoided. In some situations the patient should be taken to the electrodes to expedite analysis of the sample.

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REFERENCES

Massive Subcutaneous Emphysema following Bronchography

To the Editor:

A case of massive subcutaneous emphysema, an unusual complication of bronchographic studies done via the nasal route, is reported. Possible causes are described.

Case Report

A 45-year-old man was admitted for bronchographic studies. The bronchographic procedure was performed after proper tests of sensitivity and premedication. The propylene-dye (Dionosil) oily dye was introduced via a nasal endotracheal catheter under fluoroscopic control. A diagnosis of bronchietasis of the right middle lobe was made.

The patient was well for 12 hours. After that, he noticed tightness of the chest and slight difficulty in deglutition. A diagnosis of subcutaneous emphysema of the neck was made. There was a gradual increase in subcutaneous emphysema. By the next morning the subcutaneous emphysema extended to the whole of the trunk, both arms, the neck, and the face (Fig 1). After intensive care, the emphysema regressed slowly; and in ten days, it had resolved completely.

Discussion

Despite the widespread use of bronchographic studies, I could not find any recorded instance of subcutaneous emphysema as a complication following use of the nasal route, although surgical emphysema of the neck due to leakage of air through the site of puncture in the cricothyroid membrane is a common complication in bronchographic studies using the cricothyroid route. 1 Won et al 2 reported a case of severe subcutaneous and mediastinal emphysema complicated by pneumothorax following transcricothyroid bronchographic studies.

Possible causes for the development of subcutaneous emphysema in our case may be (1) cough, leading to increased intra-alveolar pressure; (2) local chemical irritation due to retained dye; or (3) exaggerated mechanical obstruction of the airway due to retained dye, which led to an increase in alveolar pressure. It is difficult to say whether these three factors acted singly or in combination in the formation of subcutaneous emphysema in our case, but the route of air dissection in this case was possibly due to increased pressure leading to alveolar rupture, with air travelling to the hilum, neck, head, and wall of the chest by the interstitial route along peribronchial and perivascular sheaths.

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REFERENCES

Effect of Fracture of Pacing Lead on R-Wave Sensing

To the Editor:

I would like to express my appreciation of the article entitled “Fracture of Pacing Electrode Mimicking Failure of Pulse Generator” by Salem et al (Chest 74:673-674, 1978). I note that these authors used a pacing system analyzer with a known output of voltage to evaluate the electrode; the problem may not have been as recognizable if a device (such as an external pacemaker) with an unknown output of voltage had been used.

As an engineer, I cannot resist correcting the following minor error in the discussion by Salem et al: “At the same time, the increased resistance of the electrode with an intact pathway should enhance sensing of the endocardial signal” (p 674). The statement is confusing, since a broken lead does not have an intact pathway. Actually, while a slightly greater voltage would exist at the tip of the electrode, the voltage would be noticeably reduced at the pacemaker by the resistance at the break. At the same time, it is likely that sufficient voltage would remain to properly inhibit the pacemaker.

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Figure 1. Chest x-ray film taken 20 hours after bronchographic procedure, demonstrating advanced subcutaneous emphysema in soft tissues of neck and wall of chest (arrows) and presence of dye in bronchopulmonary segments.