The carbon monoxide bound to hemoglobin also causes a leftward shift for oxygen, carbon monoxide displaces oxygen from hemoglobin, and volumes percent oxygen of 17. Even taking into account hypoxia. As the affinity of hemoglobin for oxygen, carbon monoxide displaces oxygen from hemoglobin, reducing the amount of oxygen available for delivery to the tissues. The carbon monoxide bound to hemoglobin also causes a leftward shift of the oxyhemoglobin dissociation curve. Tighter binding of oxygen to hemoglobin in the presence of carboxyhemoglobin further decreases oxygen delivery to the tissues.

Other alternative mechanisms of carbon monoxide toxicity include carbon monoxide binding to hemoproteins such as myoglobin, cytochrome oxidase, tryptophan deoxygenase, and tryptophan catalase. Regardless of the mechanism of carbon monoxide toxicity, carboxyhemoglobin levels provide the best marker currently available for carbon monoxide toxicity.

Numerous studies, including references 1-12 cited in our article, have demonstrated that exposure to carbon monoxide from smoking or heavy atmospheric carbon monoxide pollution causes adverse health effects in susceptible patients. The US Environmental Protection Agency has stated that ambient carbon monoxide levels exceeding 9 ppm for an eight-hour average exceed air quality standards. According to the Couburn equation, a constant 9 ppm carbon monoxide exposure for eight hours in the presence of light or moderate physical activity would produce a carboxyhemoglobin level of 1.4 percent.

On the basis of currently available data, as stated in our article in Chest, “It would appear to be inadvisable to transfuse susceptible patients with blood containing high levels of carboxyhemoglobin, particularly when multiple transfusions are required.”

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Limitations to Quality Control Materials for CO-oximeters

To the Editor:

Spruzem et al recently discussed the problem of interference with values obtained from CO-oximetry by lipemia (Chest 1984;86:84-6). Rather than the center of the problem, however, the results they reported are merely a symptom of a more pervasive problem. That problem is the lack of adequate quality control materials for these spectrophotometric devices.

The technique of CO-oximetry involves the measurement of absorption spectra for different hemoglobin species. The IL 282 measures absorption at two different bandwidths for each of four species of hemoglobin. From these absorption data, the instrument's microprocessor calculates a value for total hemoglobin, and then converts the absorption data for each species into a percentage of the total. It also then calculates the oxygen content of the bound oxygen only, expressing the result as “volumes percent oxygen.” Because of this methodology, it stands to reason that an error in measurement of any of the directly measured parameters will result in errors in the calculated values. The data presented by Spruzem et al serve as only one example of interference with the absorption spectrophotometer. Interference with absorption data for methemoglobin and carboxyhemoglobin have been reported.

Although there is no easy method for verifying the values obtained as measured parameters (short of running parallel samples for comparison), the value obtained from calculation of volumes percent oxygen can easily be double-checked. This check alone should have revealed the presence of an analytic error. The data reported show a total hemoglobin value of 17 g/100 ml, oxyhemoglobin of 77 percent, and volumes percent oxygen of 17. Even taking into account rounding error, 17/(77 x 17) = 1.299. This value represents the factor for combining capacity of hemoglobin with oxygen. This factor is a constant 1.39. Utilizing a similar calculation to check the second set of data reported, 18/(14 x .90) = 1.333.

The only commercially available quality control product for CO-oximetry devices consists of lysed bovine erythrocytes tonemometered with 100 percent carbon monoxide. The resulting product demonstrates high values for carboxyhemoglobin, and, as a result, low values for volumes percent oxygen. The high carboxyhemoglobin value masks the performance of the other absorption measurements. It also masks errors made by the microprocessor in calculation of the value for volumes percent oxygen. The accepted reference standard for spectrophotometers, cyanmethemoglobin, is useful only for calibrating the total hemoglobin channel of the instruments.

Until a product is marketed which allows consistent check of performance of all channels of the spectrophotometer on a routine basis, errors such as those described will continue to occur. Only the careful surveillance of data reported by clinicians will prevent misinterpretation and misuse of the data.

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To the Editor:

We wish to thank Mr. Van Driel for his comments concerning our recent report. He raises the following points concerning clinical oximetry using the model 282 CO-oximeter: 1) spectral interference may produce erroneous measured and calculated results, 2) clinicians must carefully interpret the results, and 3) there is no convenient way to determine the accuracy of results. We tried to emphasize the first two points in our report. We agree with the third point, but feel it is a separate issue since, as shown in Table 2 of our report, a hyperlipemic blood sample may produce erroneous measurements in a well-calibrated, accurate CO-oximeter.

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The one-point calibration of the CO-oximeter is very simple to perform using the commercially available control solution. If needed, the hemoglobin channel alone may be calibrated quickly using a commercially available reference (CalDye) which is traceable to the cyanmethemoglobin standard.

Unfortunately, as noted by Dr. Van Driel, there is no convenient, simple, direct way to evaluate CO-oximeter performance (this is a problem shared by other automated analyzers). There are indirect methods of performance evaluation, but they require a well-calibrated, accurate blood gas analyzer and a calculator. One may “spot check” performance when analyzing blood from nonsmokers by comparing the measured oxygen saturation with that predicted from the oxygen tension (PaO2) using the Severinghaus equations.1 With a tonometer, one may evaluate oximeter performance more rigorously by comparing measured and predicted saturations at several different levels of saturation using blood from normal nonsmoking subjects.

A quality control program should consider methodology, equipment (including calibration/performance testing), and technician performance. We wanted to alert clinicians and technicians to a limitation of methodology: spectral interference from hyperlipemia. After the experience provided by this patient, we revised our laboratory procedures for handling blood samples, which would fall under the methodologic and technician aspects of quality control; if a laboratory refuses to perform these procedures for handling blood samples, which would fall under the methodologic and technician aspects of quality control, if a blood sample is lipemic, the observation is noted on the laboratory slip and the oximetry results are not reported.

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REFERENCE

Bronchoalveolar Lavage and Gold Salt-Induced Pneumonitis

To the Editor:

We were very interested in the article by Ettensohn et al (Chest 1984; 85:569) which gave results from bronchoalveolar lavage (BAL) in the case of gold salt-induced interstitial pneumonitis. We report similar and complementary results which confirm the value of BAL in this pathology.

CASE REPORT
A 68-year-old nonsmoker was diagnosed as having rheumatoid arthritis in July, 1983 and immediately treated with gold sodium thiopropansulfonate (Allochrysine) alone. When prescribed injections were finished at the beginning of August (total dose 1,200 mg), dyspnea developed and became quite severe. It was accompanied by asthenia, evening fever and weight loss. The patient was subsequently hospitalized. Upon admission, the patient was feverish and presented dyspnea at rest, a dry cough, light cyanosis of the extremities, and diffuse crepitant rales. There was no sign of cardiac failure. The thoracic film revealed a blurred, cloudy, poorly defined bilateral pneumonitis with predominance in the lower lobes and peri-hilar regions. Hypoxia was marked at 6.55 Kpa with hypocapnia at 4.25 Kpa and respiratory alkalosis.

All viral and bacterial sero-diagnostic tests were negative, as were tests for detection of specific precipitins. The BAL was performed in the usual manner using a total of 150 ml of sterile solution (47 percent recovery). The total cell count was found to be 2.3 x 10^6 cells of which 57 percent were lymphocytes, 17 percent macrophages, 23 percent eosinophils and 3 percent neutrophils (the normal absolute total cell count varies between 5 x 10^6 and 1 x 10^7 cells).

The pathologic examination of the surgical pulmonary biopsy revealed marked interstitial fibrosis developing around capillaries and accompanied in spots by lymphoplasmacyte infiltrates. Results of the immunofluorescence test of the pulmonary fragments was negative, as were all viral, bacteriologic and mycosal tests. The lymphoblast transformation test was positive.

Prednisone therapy (Cortancyl, 40 mg daily) was initiated. In less than one month, there was total disappearance of functional signs and almost total disappearance of radiologic signs.

The control BAL carried out one month later recovered 15 x 10^6 cells of which 45 percent were lymphocytes and 55 percent were macrophages without eosinophils.

DISCUSSION

The present results confirm the value of studying the alveolar lymphocyte count in cases of hypersensitivity pneumonitis, particularly for those drug induced.1

The primary difficulty in gold salt-induced pneumonitis is the elimination of a “rheumatoid lung.” The following criteria are normally retained in favor of gold: chronicologic relation, acute character, corticossensitivity and positive results in lymphoblast transformation test for gold salts.

The results of BAL can guide our diagnosis when a high lymphocyte count contrasts with the admitted predominance of polymorphonuclear neutrophils in the case of rheumatoid lung.1

Finally, BAL is important in controlling the evolution of the pathology. We find a continuing high lymphocyte count in control BAL, whereas clinical and radiologic signs have disappeared. Should the persistence of a high rate of lymphocytosis after clinical and radiologic recovery be taken into account in the criteria for stopping corticotherapy?

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Upper Airway Obstruction in Guillain-Barré Syndrome

To the Editor:

Rodrigues, York and Nair propose that bilateral vocal cord paralysis in their patient was directly caused by the Guillain-Barré syndrome (Chest 1984; 86:147). However, there is an alternative explanation. Unilateral vocal cord paralysis is a well documented complication of endotracheal intubation, and has been reported in at least 24 patients.1 The proposed mechanism is damage to the anterior branch of the recurrent laryngeal nerve within the larynx by an over-inflated endotracheal tube cuff. I recently saw a patient with bilateral vocal cord paralysis who had been dyspneic for several years. Her symptoms began after she was intubated for two days for treatment of drug overdose. No other explanation was available for her bilateral vocal cord paralysis, which required permanent tracheostomy for treatment.

The patient with Guillain-Barré syndrome reported was intubated...