The Alveolar Air Equation Again!

To the Editor:

I enjoyed the article by Gross and Israel (Chest 1981; 79:311-15) until I got to the appendix. I do not know what "idealized" means when applied to the alveolar air equation, but if it means wrong, then I agree with the authors. The fact that they are quoting other authors is no excuse. The error in alveolar oxygen partial pressure is 0.05 times the alveolar partial pressure of carbon dioxide while breathing air, and 0.25 times the alveolar partial pressure of carbon dioxide while breathing 100 percent oxygen. The values used for alveolar oxygen partial pressure are, therefore, not correct and thus the scale for arterial carbon dioxide tension and fraction of oxygen are not properly aligned.

I have been told by allied health workers that if an equation appeared in Chest it must be right. Please let me present one of the correct equations for alveolar air, each of which shows that the exchange ratio effect is proportional to the amount of inert gas in equilibrium with the subject or patient, and, therefore, is maximal when the patient is breathing air and completely absent when breathing only oxygen. One equation which shows this is:

\[ \text{PAO}_2 = (\text{PA}-47) \text{FIO}_2 - \text{PAO}_2 (\text{FIO}_2 + \frac{1-\text{FIO}_2}{R}) \]

In the article referred to above there is no error when arterial carbon dioxide is assumed 0 (though no one as yet has attained this value). The error is a maximum at an arterial carbon dioxide tension of 120 and an inspired oxygen fraction of 1. The latter error is 30 mm Hg.

H. F. Helmholz, Jr., M.D., F.C.C.P.
Rochester, Minnesota

To the Editor:

We are aware of Dr. Helmholz’s observation that the "idealized" alveolar air equation is an abbreviated form of the alveolar air equation, the complete form of which states:

\[ \text{PAO}_2 = (\text{PA}-47)\text{FIO}_2 - \frac{\text{PACO}_2}{R} + \left[ \frac{\text{PACO}_2 \cdot \text{FIO}_2 (1-R)}{R} \right] \]

where \( \text{PAO}_2 \) = alveolar oxygen tension
\( \text{Ps} \) = barometric pressure
\( \text{FIO}_2 \) = inspired oxygen concentration

The "idealized" equation results when the bracketed portion of the full equation is deleted. This results in such an extremely small change that West states "the term in square brackets is a correcting factor of small magnitude."

We do not share Dr. Helmholz’s distress over the small error introduced by utilizing the abbreviated form for the purposes of our study. In fact, even under the conditions cited by Dr. Helmholz to contrive the maximum possible "error" of 30 mm Hg in the alveolar oxygen tension (\( \text{PAO}_2 = 120 \text{ mm}, \text{FIO}_2 = 1 \)), the overall percentage of error is approximately 5.5 percent. Furthermore, at this portion of our nomogram, the difference would be unlikely to change the a/A ratio, as visually estimated, by more than a factor of 0.02. Finally, the results of our clinical trials did not reveal a notable effect of the alleged "error" in our ability to estimate the appropriate parameter.

We fully recognize the lack of precision of the a/A ratio nomogram when compared with theoretic and/or mathematical models. However, we do not view this as an important disadvantage based on our data. A far greater disadvantage might occur if clinicians relied simply on "experience" for managing oxygen therapy, simply because the "right" approach as perceived by Dr. Helmholz proved too cumbersome or detailed for routine practical use. We believe the simplicity of use and acceptable results of our clinical trial far override the insightful but perhaps exaggerated objections of Dr. Helmholz.

Richard Gross, M.D.,
Robert H. Israel, M.D., F.C.C.P.
Rochester, New York

REFERENCES

2 Gross B, Israel RH. A graphic approach for prediction of arterial oxygen tension at different concentrations of inspired oxygen. Chest 1981; 79:311-15

Angiographic Relief of Coronary Artery Spasm with Sublingual Nifedipine

To the Editor:

Coronary artery spasm is an important cause of angina pectoris. It may be superimposed upon coronary atheroma or induced by mechanical irritation and relieved by oral or intracoronary nitroglycerine or isosorbide dinitrate. The calcium channel blocking agent nifedipine is widely used in clinical practice for the relief of coronary artery spasm, but direct angiographic proof of its efficacy has not been reported or documented. We describe a case of angiographically-documented, mechanically-induced coronary artery spasm relieved by sublingual nifedipine.

CASE REPORT

A 37-year-old man who had sustained a large anterior myocardial infarction ten months prior to investigation underwent coronary angiography because of residual mild angina pectoris on exertion. Results of general examination...
were normal and the blood pressure was 120/70 mm Hg. The apex beat was dyskinetic and he had an apical 4th heart sound. The ECG showed anterior myocardial infarction. He had a graded exercise test using the Bruce protocol; he ran for eight minutes and reached his target heart rate (180 beats/minute) without chest pain or ST segment change.

Coronary angiography was performed by the Judkins technique. The left ventricular end-diastolic pressure was 22 mm Hg. The left ventriculogram showed akinesis of the anterior wall and apex and hypokinesis of the septum. The left anterior descending coronary artery was obstructed at its origin and a ghost of the distal artery filled from the circumflex and right coronary arteries. The circumflex artery was normal.

The first injection into the right coronary artery showed a normal proximal vessel. The catheter tip slid down the artery and the pressure wave form became damped. The catheter tip was removed and reinserted. The patient complained of chest pain without ECG change and during the subsequent injection, the vessel appeared to have a fresh narrowing. After a second injection, the narrowing became more severe (Fig 1a). The catheter tip was withdrawn, nifedipine (10 mg) was given sublingually, and after four minutes, the pain subsided. There was no change in arterial blood pressure. A further series of right coronary arteriograms in multiple views showed no evidence of narrowing or irregularity of the arterial lumen; the spasm had disappeared (Fig 1b).

DISCUSSION

This case presents direct evidence of the abolition of coronary artery spasm and the relief of angina pectoris by sublingual nifedipine.

T. Schwartz, M.D.; D. Halon, M.B., Ch.B.; Y. Hasin, M.D.; and M.S. Gotsman, M.D.,
Department of Cardiology, Hadassah University Hospital, Jerusalem, Israel

Unanticipated Response to Alpha-Adrenergic Blockade in Pulmonary Hypertension

To the Editor:

We wish to report an unanticipated response to parenteral alpha-adrenergic blockade with tolazoline hydrochloride in a patient with primary pulmonary hypertension.

CASE REPORT

A 22-year-old black woman presented with a six-month history of progressive fatigue and dyspnea on exertion. Physical examination revealed a well developed female in sinus rhythm with clinical evidence of right ventricular hypertrophy and pulmonary hypertension. Diagnostic cardiac catheterization revealed the following pressures (mm Hg): right atrial, a=17, v=20 (mean=17); right ventricle=100/8; pulmonary artery=100/55 (mean 65); pulmonary wedge=10; left atrium=8; left ventricle=150/8. Three days later, in order to ascertain the degree of pulmonary vascular reactivity, the patient's hemodynamic response to tolazoline was ascertained. Tolazoline, 1 mg/kg, was administered into a peripheral IV line over one minute with continuous monitoring of the hemodynamic response. Within two minutes, the patient became profusely flushed and complained of back discomfort and palpitations. Heart rate increased from 100 to 150 beats per minute. After three minutes, pulmonary artery pressure increased from 100/55 to 140/75 mm Hg (mean=95) and cardiac output increased from 3.5 to 5.75 L/min with systemic blood pressure rising from 130/106 to 140/110 mm Hg. Calculated pulmonary vascular resistance fell by 11 percent and systemic vascular resistance fell by 38 percent. The patient's discomfort resolved within ten minutes while hemodynamic parameters returned to baseline within 45 minutes.

Dresdale and colleagues first reported the value of tolazoline hydrochloride infusion in assessing pulmonary vascular activity in patients with pulmonary hypertension. They gave 25 mg "parenterally." Subsequently, Grover and colleagues reported their experience with eight children with elevated pulmonary vascular resistance greater than twice...