Correspondence

Arterial Oxygen Tensions

To the Editor,—Recent correspondence in your columns by Dr. Markello (Anesthesiology 27: 334, 1966) and Drs. Laver and Slater (Anesthesiology 27: 335, 1966) draws attention to the paradoxical decrease in arterial oxygenation which occurs during hyperventilation. The causes of this decline in arterial \( P_{O_2} \) are complex, and there appears to be some confusion about the interrelation of the several factors involved.

It is commonly assumed that the increase in the alveolar/arterial \( P_{O_2} \) tension gradient which occurs during anesthesia must be due to an increase in the pulmonary venous admixture caused by the passage of blood through atelectatic or relatively underventilated areas of lung. Such areas of atelectasis have seldom been demonstrated. On the other hand it does not appear to be sufficiently well recognized that falls of cardiac output can themselves decrease the arterial \( P_{O_2} \) by lowering the oxygen content of the blood comprising any given degree of venous admixture. Many investigators in the past, based their calculations of venous admixture on the assumption that there is normal arterial/mixed-venous oxygen content difference during anesthesia. This is understandable in view of the natural reluctance to the sampling of mixed venous blood. However, recent studies in this Department \(^1\) have shown that changes of arterial \( P_{CO_2} \) have a marked effect on the cardiac output during anesthesia, and outputs as low as 2 liters/minute have been recorded at low \( P_{CO_2} \). Studies of mixed venous oxygen content (from right ventricle) in a few patients confirm these low outputs.

In addition to these factors, the alveolar/arterial \( P_{O_2} \) gradient is influenced by the actual level of the alveolar \( P_{O_2} \), the \( pH \) of the blood, the temperature of the patient, and the hemoglobin concentration. The interplay of various factors is perhaps most easily demonstrated by an example (table 1).

Our table illustrates the hyperventilation of a hypothetical but typical anesthetized patient with the following variables maintained constant: temperature: 37\(^\circ\) C., Hb: 14 g./100 ml., inspired oxygen: 30 per cent (\( P_{O_2} \): 214 mm. of mercury), \( R \): 0.8, \( V_{O_2} \): 200 ml./minute. During hyperventilation the arterial \( P_{CO_2} \) drops from 40 to 20 mm. of mercury. The assumed change in cardiac output represents the mean of our current series of measurements of the cardiovascular effects of hyperventilation. Assuming that the venous admixture is unchanged at 6 per cent of the cardiac output, it is instructive to infer the effect of this degree of hyperventilation on the arterial \( P_{O_2} \) and percentage oxyhemoglobin saturation. We are aware that “venous admixture” does not necessarily consist solely of mixed venous blood, and regard it as no more than a convenient index of pulmonary maldistribution.

It is startling to find that, under the conditions postulated, hyperventilation does not increase the arterial \( P_{O_2} \). We should have been hesitant to write this letter had we not ample experimental verification of the hypothesis that hyperventilation can lower the arterial \( P_{O_2} \). Even so we remain slightly diffident about incorporating this iconoclastic doctrine into the instruction which we give our residents! It should, however, be remembered that under different conditions, particularly at a lower alveolar \( P_{O_2} \), hyperventilation may raise the arterial \( P_{O_2} \). \(^2\)

The real lesson is that many factors influence the arterial \( P_{O_2} \) and it is not profitable to discuss the causation of changes in arterial oxygenation without considering all the relevant factors.

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References


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Table 1

| Variable Factor          | Patient Normally Ventilated 40 min. Hg | Hyperventilated 20 mm. Hg | \( \text{A-a) } P_{O_2} \) gradient
|--------------------------|----------------------------------------|---------------------------|-------------------------------
| Alveolar \( P_{O_2} \)   | 166 mm. Hg                             | 190 mm. Hg                | \( \text{A-a) } P_{O_2} \) gradient increases (A—a) gradient. |
| Arterial/mixed venous oxygen content difference | -4 L./m. 5 vols. % | 2.8 L./m. 7 vols. % | \( \text{A-a) } P_{O_2} \) gradient. |
| Cardiac output           | 7.4                                    | 7.6                       | \( \text{A-a) } P_{O_2} \) gradient. |
| Arterial pH              | 48 mm. Hg                             | 84 mm. Hg                | \( \text{A-a) } P_{O_2} \) gradient. |
| Arterial \( P_{O_2} \)   | 118 mm. Hg                             | 101 mm. Hg               | \( \text{A-a) } P_{O_2} \) gradient. |
| Arterial Saturation      | 98.2%                                  | 98.4%                     | \( \text{A-a) } P_{O_2} \) gradient. |

Myasthenic Syndrome

To the Editor:—The recent report by Drs. Sanger and Kinyon (Sensitivity to curare by a patient with undiagnosed myasthenia gravis syndrome, Anesthesiology 27: 325, 1966) describes a further case of a myasthenic syndrome associated with bronchogenic carcinoma. The first such case was described in 1953, and since then a number of reports have confirmed the clinical and electromyographic features of this condition. The need for prolonged positive pressure ventilation after operation when muscle relaxants have been employed has also been reported.

The principal clinical features have been fatigueability and weakness of the proximal muscles, especially in the pelvic girdles and thighs, reduced or absent tendon reflexes, poor response to neostigmine and, as in the present case, an abnormal sensitivity to tubocurarine. Patients with this syndrome have also shown an increased sensitivity to depolarizing relaxants. In patients with myasthenia gravis, ocular and bulbar symptoms predominate and the weak muscles are easily fatigued. The muscles of patients with the myasthenic syndrome are less easily fatigued and indeed with sustained or repeated contractions a gradual initial increase in strength may be observed before fatigue develops. A very high proportion of the reported cases had, or subsequently developed, a small cell bronchogenic carcinoma, although some patients with this syndrome have been observed as long as nine years without a carcinoma becoming manifest.

In addition to the clinical features which differentiate myasthenia gravis from the myasthenic syndrome associated with bronchogenic carcinoma, electromyography has proved the most conclusive method of identifying this condition. In the rested muscle, the action potential evoked by a single maximal nerve stimulus is very much reduced in amplitude compared with a normal response, whereas at fast rates of stimulation (10–200 seconds) a progressive and striking increase in the response is observed. The action potentials reach from 2 to over 20 times the initial amplitude and accounts for the fact that the power of a voluntary contraction may be normal despite the weak response to a single twitch in the rested muscle. Post-tetanic facilitation persists for a period of 20 to 30 seconds after a period of repetitive nerve stimulation and provides the basis for a simple screening test for this syndrome. The amplification of the action potential evoked in the hypothenar muscles by a single maximal stimu-