has been encouraged by our housestaff for the evaluation of hypoxemia in patients breathing room air.

The following altitude-baseline table can be used for convenience in place of equation (4).

<table>
<thead>
<tr>
<th>Altitude (Feet)</th>
<th>Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>142</td>
</tr>
<tr>
<td>500</td>
<td>139</td>
</tr>
<tr>
<td>1000</td>
<td>136</td>
</tr>
<tr>
<td>1500</td>
<td>134</td>
</tr>
<tr>
<td>2000</td>
<td>131</td>
</tr>
<tr>
<td>2500</td>
<td>129</td>
</tr>
<tr>
<td>3000</td>
<td>126</td>
</tr>
<tr>
<td>3500</td>
<td>123</td>
</tr>
<tr>
<td>4000</td>
<td>121</td>
</tr>
<tr>
<td>4500</td>
<td>119</td>
</tr>
<tr>
<td>5000</td>
<td>116</td>
</tr>
<tr>
<td>5500</td>
<td>114</td>
</tr>
<tr>
<td>6000</td>
<td>111</td>
</tr>
<tr>
<td>6500</td>
<td>109</td>
</tr>
<tr>
<td>7000</td>
<td>107</td>
</tr>
<tr>
<td>7500</td>
<td>105</td>
</tr>
<tr>
<td>8000</td>
<td>102</td>
</tr>
<tr>
<td>8500</td>
<td>100</td>
</tr>
<tr>
<td>9000</td>
<td>98</td>
</tr>
<tr>
<td>9500</td>
<td>96</td>
</tr>
<tr>
<td>10000</td>
<td>94</td>
</tr>
</tbody>
</table>

Michael E. Perry, LTC, MC;  
Robert J. Browning, B.S.; and Neal B. Kindig, Ph.D.,  
Pulmonary Disease/Clinical Investigation Service,  
Fitzsimons Army Medical Center,  
Aurora, Colorado

Reprint requests: Dr. Perry, Pulmonary Disease Service,  
Fitzsimons Army Medical Center, Aurora, Colorado 80045

To the Editor:

The difference between ideal alveolar \( P_{O_2} \) and arterial \( P_{O_2} \) \( P(A-a)O_2 \) is a measure of the severity of the pathophysiological mechanisms which cause hypoxemia. I am pleased that Perry et al agree that the mental arithmetic simple expedient for calculating \( P(A-a)O_2 \) by summing arterial \( P_{CO_2} \) and \( P_{O_2} \) and subtracting from 142 (at sea level; lower value for higher altitude) described in reference 3 is "correct enough" and practical for clinical use. The difference between the exact and the expedient calculation is small. We would rather have the housestaff approximate the difference by the summing technique rather than not make any estimation in their patients because of the complexity of the "exact" calculation. The recognition of pulmonary insufficiency without an increase in \( P(A-a)O_2 \) can quickly alert the house officer to a defect in respiratory control (example: primary or drug induced) or respiratory muscle failure (example: myasthenia gravis or Guillain-Barré syndrome) from primary disorders of the lungs which characteristically give an increased \( P(A-a)O_2 \).

Karl E. Wasserman, M.D., F.C.C.P.
UCLA School of Medicine, Harbor-UCLA  
Medical Center, Torrance, California

The Dolovich Reaction

To the Editor:

We have recently undertaken a study on the prevalence of allergic reactions in people exposed to animal allergens by virtue of their employment either as full-time or part-time animal handlers or as animal experimentalists. We found asthma, rhinitis and contact allergy as the main allergic symptoms, with a high degree of correlation between animal induced asthma and a positive immediate skin prick test to the animal species inducing the asthmatic reaction.

Of the 17 subjects with animal asthma and positive immediate skin prick test reactions, three developed late skin reactions around the test site. These late reactions were maximal at about six hours and were large, red, warm, tender edematous areas involving about half the forearm and resolving by the following day.

The serum of these individuals did not contain specific IgG antibody to the eliciting animal materials (serum and male urine) as determined by both the enzyme-linked immunosorbent assay (ELISA) and by double gel diffusion.

On the basis of the time course of this reaction there is an undoubted parallel with the Arthus (type III) reaction which does involve specific IgG antibody to the antigen; however, in view of the absence of detectable specific IgG and the minute amount of allergen introduced by the skin
prick test, it seems that another mechanism is operating.

In 1974, Dolovich et al. described late skin reactions that were considered to be mediated by IgE and where histology of the skin test site revealed edema, mast cell degranulation and mainly eosinophilic perivascular infiltration. There were few neutrophils and no evidence of intravascular thrombi; neither did immunofluorescence reveal immunoglobulin or complement deposition. All these observed features are not characteristic of an Arthus reaction.

We believe we have observed the same reaction to animal allergens and suggest that a late skin reaction mediated by IgE be termed a Dolovich reaction.

J. H. Edwards, M.D.,
and A. E. Cockcroft, M.D.
MRC Pneumoconiosis Unit,
Llandough Hospital,
Penarth, S Glamorgan, Wales, UK

Reprint requests: Dr. Edwards, c/o J. Mouat, Esq, Medical Research Council, Llandough Hospital Near Penarth, Glamorgan CF6 1XW, Wales

REFERENCES


Volume-Adjustment of MMF: New Term Needed

To the Editor:

I fully agree with the views recently expressed in this journal by Cockcroft and Berscheid (1980; 78:595) concerning the measurement of forced expiratory flow over the middle half of vital capacity after the application of a bronchomotor drug. Their results show that adjustment to total lung capacity (when forced vital capacity changes), as previously proposed,1 does not suffice when TLC changes: in this case, the adjustment is to be done to absolute lung volume. This approach is the same as that advocated for the adjustment of flow-volume curves after bronchostenosis by Bouhuys et al.2 Although aware of the influence of lung volume on forced expiratory flow, Leuallen and Fowler3 did not discuss the possible consequences of an acute change in volume on the MMF; their original report2 was limited to the comparison of MMF to other ventilatory function tests.

It seems to me that the adjustment proposed raises a problem of terminology: how could we best call this forced expiratory flow measured over the middle half of the pre-bronchomotor FVC, which is obviously (Fig 2 of Cockcroft and Berscheid) no longer a maximal mid-expiratory flow? The full wording, "forced expiratory flow over 25-75% of baseline FVC" seems rather cumbersome.

Dan B. Teculescu, M.D.
Vandoeuvre/Nancy, France

REFERENCES

3 Leuallen EC, Fowler WS. Maximal mid-expiratory flow Am Rev Tuberc 1955; 72:783

Anti-inflammatory Action of Terbutaline in Man

To the Editor:

It has been observed that beta-adrenoceptor agonists exert an anti-inflammatory action in skin reactions of experimental animals.1 In man, the response to intradermal injection of histamine provides a convenient model for study of altered vascular permeability and has been used to investigate the anti-inflammatory potential of a beta-adrenoceptor agonist in normal volunteers.

Histamine (0.5 μg), histamine and terbutaline (100 μg) or histamine, terbutaline and propranolol (20 μg) were injected (0.1 ml) in three sites in volar forearm skin. After 15 minutes, areas of skin swelling (mm²) were (mean ± SEM):

128.8 ± 5.0 for histamine (n = 24)
96.3 ± 7.1 for histamine + terbutaline (n=15) (P<0.0005)
114.0 ± 8.0 for histamine + terbutaline + propranolol (n=15, ns).

These results demonstrate that when a beta-adrenoceptor agonist is applied locally, there is significant reduction of the histamine-induced skin lesion. This effect of terbutaline may be attributed to beta-adrenoceptor stimulation, since it is blocked by the beta-adrenoceptor antagonist propranolol. Our findings suggest an anti-inflammatory action of locally administered beta-adrenoceptor agonists by opposition of the permeability component of plasma protein extravasation.3 This property of terbutaline could contribute to the therapeutic effect of inhaled terbutaline and other beta-adrenoceptor agonists in obstructive lung disease.

Andreas Hoffmann, M.D.,
Dieter Conen, M.D.,
Medizinische Universitätspoliklinik,
Departement f Innere Medizin,
Basel, Switzerland;
and John Morley, Ph.D.
Department of Clinical Pharmacology,
Cardiothoracic Institute,
London, England

Reprint requests: Dr. Hoffmann, Department of Innere Medizin, Kantonspital, CH-4031 Basel, Switzerland

REFERENCES


Response of Myelomatous Pleural Effusion to Chemotherapy

To the Editor:

Pleural effusion due to plasma cell infiltration of the pleura is an uncommon manifestation of multiple myeloma. We describe a patient with IgG myeloma and bilateral