Regional lung function in bilateral diaphragmatic paralysis

T. C. AMIS,* G. CIOFETTA, J. M. B. HUGHES AND L. LOH
Department of Medicine, Royal Postgraduate Medical School, Hammersmith Hospital, and National Hospital for Nervous Diseases, London

(Received 31 December 1979/27 March 1980; accepted 18 July 1980)

Summary

1. The distribution of regional function in the lungs of six patients with bilateral diaphragmatic paralysis was investigated by continuous inhalation and infusion of the radioactive gases $^{81m}$Kr and $^{85m}$Kr during tidal breathing.

2. In the supine and right lateral decubitus postures the vertical distribution of ventilation per unit alveolar volume was less in the dependent zones, the reverse of that found in normal subjects. In the upright posture ventilation was slightly decreased at the lung base. Perfusion per unit alveolar volume was more uniformly distributed than normally in the upright posture, and decreased from superior to inferior in the supine posture. In the lateral decubitus posture, perfusion of the lower lung was greater than that of the upper. Ventilation/perfusion ratios were more uniformly distributed in the patients than in normal subjects, except in the right lateral decubitus posture.

3. Alterations in the distribution of ventilation may be explained in terms of the altered mechanical interaction of chest wall, mediastinal and abdominal contents, with selective use of intercostal and accessory muscles. The effects on the distribution of blood flow are probably related to the low end-expiratory lung volume.

Key words: krypton ($^{81m}$Kr, $^{85m}$Kr), lung mechanics, radioactive gases.

* Present address: Department of Physiological Sciences, School of Veterinary Medicine, University of California, Davis, California, U.S.A.

Correspondence: Dr J. M. B. Hughes, Department of Medicine, Royal Postgraduate Medical School, Hammersmith Hospital, London, W12 0HS.

Introduction

In recent years the effect of bilateral diaphragmatic paralysis on pulmonary gas exchange has been emphasized. Newsom Davis, Goldman, Loh & Casson (1976) reported significant hypercapnia and hypoxia in bilateral diaphragmatic paralysis. In a group of six patients, they found that some had raised $P_{a}CO_{2}$ and reduced $P_{a}O_{2}$ in the upright posture; when these patients were supine $P_{a}O_{2}$ fell further and $P_{a}CO_{2}$ rose. These changes in blood gases were attributed to hypoventilation associated with loss of inspiratory muscle function; these patients breathed rapidly and shallowly, but with voluntary hyperventilation normal blood gas tensions could be restored.

Nevertheless, mismatching of alveolar ventilation ($V_{A}$) and perfusion ($Q$) is a much more frequent cause of impaired gas exchange than overall hypoventilation. Since the diaphragm is the principal muscle of inspiration during quiet breathing in normal subjects it would not be surprising if its paralysis altered the mechanics of breathing and the distribution of inspired gas. Thus $V_{A}/Q$ mismatching may be contributing to reduced $P_{a}O_{2}$ recorded in such patients. Past work (Roussos, Fukuchi, Macklem & Engel, 1976; Roussos, Fixley, Genest, Cosio, Kelly, Martin & Engel, 1977b) suggests that selective use or non-use of the diaphragm can alter the distribution of inspired gas in normal subjects.

In this study, the distribution of ventilation and perfusion in the lungs of patients with bilateral diaphragmatic paralysis has been compared with distributions found in normal subjects. Since gas exchange may be very sensitive to posture in these patients, who can develop hypercapnia and hypoxaemia at night while supine (Newsom...
Davies et al., 1976), the effect of body position was also studied.

Measurements of regional lung function were made by using the short-lived gases $^{81m}$Kr (half-life 13 s) and $^{85m}$Kr (half-life 4.4 h) using the technique introduced by Amis, Ciofetta, Clark, Hughes, Jones & Pratt (1978).

**Materials and methods**

Steady-state administration of $^{81m}$Kr for measurement of regional pulmonary ventilation ($V_A$) perfusion ($Q$), and ventilation/perfusion ratio ($V_A/Q$) was described by Fazio & Jones (1975) and Harf, Pratt & Hughes (1978). However, quantification of count-distribution images obtained with $^{81m}$Kr is complicated by the influence of regional lung volume and washout of the radioactive gas. Amis et al. (1978) introduced a procedure involving equilibration and clearance of the longer-lived radioactive gas $^{85m}$Kr (half-life 4.4 h) in addition to steady-state administration of $^{81m}$Kr in order to calculate regional ventilation per unit alveolar volume ($V_A/V_A$) and perfusion per unit alveolar volume ($Q/V_A$). In the steady state, the distribution of the shorter half-life radioactive gas $^{81m}$Kr is flow determined whereas that of the longer-life $^{85m}$Kr is proportional to volume. Division of a $^{81m}$Kr flow-weighted image by a $^{85m}$Kr volume-weighted image produces an index of regional ventilation or perfusion per unit alveolar volume. The principal gamma energy of $^{85m}$Kr (150 keV) is sufficiently similar to the 190 keV of $^{81m}$Kr that geometric factors may be assumed to be the same for both isotopes.

Since the inspired concentration of either gas is not measured all regional radioactivity counts are related to those of the total lung field (Mannell, Prime & Smith, 1966). By monitoring the clearance of $^{85m}$Kr after recording an equilibrium image, the specific ventilation of the total lung field may be obtained from an initial slope analysis of a semilogarithmic plot of the residue curve. This value is then used to calculate regional specific ventilation in absolute units (1 min$^{-1}$l$^{-1}$). Regional and total values for specific ventilation are used to correct the perfusion index for ventilatory washout of infused $^{81m}$Kr. The use of $^{81m}$Kr for the measurement of regional $V_A/Q$ has been described by Harf et al. (1978).

A group of six patients with bilateral diaphragmatic paralysis were studied. Their characteristics are presented in Table 1. Informed consent was obtained from each patient and the study was approved by the Hospital Ethical Committee. The diagnosis was confirmed by the failure of patients to generate a significant change in transdiaphragmatic pressure ($\Delta P_{di}$) during a maximal inspiration [normally the change in transdiaphragmatic pressure ($\Delta P_{di}$) is >25 cm water]. Transdiaphragmatic pressure was measured by using gastric and oesophageal balloons. Total lung capacity (TLC) for each patient was measured in a constant-volume body plethysmograph, and vital capacity (VC) and forced expired volume in 1 s (FEV1.0) were measured with a dry spirometer.

The clinical features of patients no. 1, 3, 4 and 5 have been described previously by Newsom-Davis et al. (1976). They all had moderate

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Total lung capacity* (%</th>
<th>Vital capacity* (%)</th>
<th>FEV1.0* (%)</th>
<th>$\Delta P_{di}$† (cm water)</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>39</td>
<td>M</td>
<td>170</td>
<td>68</td>
<td>63-7</td>
<td>57-8</td>
<td>58-1</td>
<td>0</td>
<td>Spinal poliomyelitis</td>
</tr>
<tr>
<td>2</td>
<td>55</td>
<td>F</td>
<td>149</td>
<td>63</td>
<td>58-4</td>
<td>40-9</td>
<td>42-1</td>
<td>0</td>
<td>Limb girdle dystrophy</td>
</tr>
<tr>
<td>3</td>
<td>45</td>
<td>M</td>
<td>175</td>
<td>66</td>
<td>82-1</td>
<td>30-0</td>
<td>27-8</td>
<td>0</td>
<td>Kugelberg-Welander syndrome</td>
</tr>
<tr>
<td>4</td>
<td>40</td>
<td>F</td>
<td>153</td>
<td>56</td>
<td>Unobtainable</td>
<td>17-3</td>
<td>13-0</td>
<td>0</td>
<td>Acid maltase deficiency</td>
</tr>
<tr>
<td>5</td>
<td>35</td>
<td>M</td>
<td>178</td>
<td>68</td>
<td>41-6</td>
<td>20-0</td>
<td>19-5</td>
<td>+5</td>
<td>Acid maltase deficiency</td>
</tr>
<tr>
<td>6</td>
<td>65</td>
<td>M</td>
<td>172</td>
<td>84</td>
<td>94-6</td>
<td>73-0</td>
<td>66-7</td>
<td>0</td>
<td>Polyneuritis (Guillain-Barre)</td>
</tr>
</tbody>
</table>

* Measured in the upright posture, shown as a percentage of the predicted normal value (Cotes, 1979); FEV1.0, forced expired volume in 1 s.
† $\Delta P_{di}$ is the change in transdiaphragmatic pressure generated during a maximal inspiration; in normal subjects $\Delta P_{di}$ is >25 cm water (Loh et al., 1979).
costal or accessory muscles in these patients.

Electromyographic (EMG) data on the intercostal or accessory muscles must have been affected to various degrees on the basis of the range of vital capacity (17–73% of the normal predicted value) but without the complete (or virtually complete) paresis shown by the diaphragm.

For each study, patients were placed in front of a large field gamma-camera (Toshiba-Jumbo GCA, 202) linked to a digital computer (Nova 1220, Data General Co.) which displayed information from the camera as a colour-coded image on a 64 × 64 matrix. Information from the camera was automatically recorded on magnetic discs (CDC model 848-12) for later replay and processing. Data could be collected as an accumulated count-distribution image (static mode) or as a series of frames under dynamic conditions (dynamic mode).

Patients were studied in the upright and supine postures (patients no. 1–5) or upright and right lateral decubitus (patient no. 6). They breathed through a mouthpiece attached to a large-bore three-way tap, one arm of which was attached to a T-piece connected by wide-bore tubing to an extractor fan which pumped gas to waste outside of the laboratory. The other arm of the T-piece was open to room air. The third arm of the three-way tap was attached to a non-rebreathing valve which was connected in closed circuit with a spirometer and a soda lime carbon dioxide absorber. The spirometer was surrounded by lead shielding (0.5 cm thick).

Inhalation of $^{81m}$Kr was commenced and once a steady state was achieved (after six to ten breaths) a static distribution image was obtained containing around 250 000 counts; this took 1–2 min depending on the activity of the generator. The $^{81m}$Kr inhalation was then stopped. After decay of radioactivity in the lung, the intravenous infusion of $^{81m}$Kr in 5% glucose solution was started. After waiting again for a steady state, a distribution image of approx. 250 000 counts was obtained in 1–2 min. After stopping the intravenous infusion, the patient was switched into the closed-circuit spirometer system at functional residual capacity (FRC). A number of deep breaths were taken in order to equilibrate with the $^{81m}$Kr in the spirometer circuit before the patient returned to normal breathing; after sufficient counts had been accumulated, the clearance of $^{81m}$Kr from the lung was monitored until counts over the lung were less than 10% of the initial count rate. The time taken for the rebreathing and washout manoeuvres was usually 6–7 min. The total radiation dose was estimated at 160 mrad to the lungs, 30 mrad to the right side of the heart, 20 mrad to adipose tissue and 2 mrad to other tissues.

In the upright posture, strips for analysis were selected on the count-distribution images centrally down each lung. In the supine and lateral decubitus postures, one vertical strip was situated cranial to the heart, the other just cranial to the diaphragm. Cranial–caudal (i.e. horizontal) strips were chosen down the centre of the upper and lower lung in the lateral decubitus posture, and superior and inferior horizontal strips were chosen in the supine posture. When the sitting of strips for analysis were chosen the perfusion image was consulted for the position of the artifact of activity produced by the pool of $^{81m}$Kr in the subclavian veins, superior vena cava, right heart and pulmonary arteries. These areas were avoided as sites for analysis of perfusion studies. The computer divided these strips into 5–15 subregions, for each of which values for $V/V_A$, $Q/V_A$ and $V_A/Q$ were calculated.

**Results**

**Ventilation**

Findings in patients are shown in relation to results in normal subjects in Figs. 1 and 2. The range of values for normal subjects has been taken from studies by using the same technique as Amis (1979).

Results are presented for the left lung only; results in the right lung were similar. Regional $V/V_A$ increased from $0.84 \pm 0.15$ (1 SD) superiorly to a maximum of $1.09 \pm 0.08$ and then decreased to $0.88 \pm 0.17$ at the lung base (Fig. 1a). Horizontal analysis showed no systematic differences across the lungs.

Three patients were studied supine. In the vertical direction, $V/V_A$ decreased from $1.06 \pm 0.06$ superiorly to $0.75 \pm 0.19$ inferiorly although over the superior two-thirds of the lung $V/V_A$ was relatively uniform. This is a reversal of the gradient of $V/V_A$, found in normal subjects (Fig. 1b). Horizontal distributions of $V/V_A$ measured for the inferior strip demonstrated little change from cranial to caudal.

The one patient who was studied in the right lateral decubitus posture demonstrated a marked
FIG. 1. Vertical distribution of $\bar{V}/V_A$ (arbitrary units) in the lungs of patients with bilateral diaphragmatic paralysis in the (a) upright, (b) supine and (c, d) right lateral decubitus postures in comparison with results obtained in normal human subjects. (Shaded areas within lung diagrams indicate lung regions analysed, $n =$ no. of subjects.) ▲, Patient no. 1; ■, no. 3; ●, no. 4; □, no. 5; ◆, no. 6.

FIG. 2. Ventilation and volume count distribution images obtained during continuous inhalation of $^{81}$Kr ($V$) and $^{85}$Kr ($V_A$) in a normal subject (left) and patient no. 6 with bilateral diaphragmatic paralysis (right) in the right lateral decubitus posture. (Posterior views, cranial to right.) Relative size of each lung indicates approximately its share of ventilation ($\bar{V}$) or end-expiratory volume ($V_A$). Note the similar distribution of lung volume in the patient and normal subject but greater ventilation in the upper lung of the patient, the reverse of normal.
regional lung function in diaphragm paralysis

489

reversal of the normal distribution of specific ventilation. Fig. 2 shows steady-state count-distribution images reflecting the distribution of ventilation (81mKr) and volume (85mKr) in a normal subject and in the patient with bilateral diaphragm paralysis in the right lateral decubitus posture. In the vertical direction \( V/V_A \) decreased from 1.36 superiorly in the upper lung to 0.54 inferiorly (Fig. 1c), and in the lower lung \( V/V_A \) decreased from 1.62 superiorly to 0.48 inferiorly (Fig. 1d). The upper lung/lower lung ratio was 1.26 compared with 0.39 in the normal subject (Fig. 2). From cranial to caudal along an isogravity line, \( V/V_A \) was relatively uniformly distributed in both upper and lower lung.

**Perfusion**

In the vertical direction, \( Q/V_A \) increased from 0.69 ± 0.11 superiorly to a maximum of 1.16 ± 0.13 before decreasing over the base of the lung to 1.05 ± 0.11. Results in the right lung were similar. The vertical gradient of \( Q/V_A \) is less in the patients than in the normal subjects (Fig. 3). Perfusion was evenly distributed across the lungs.

In the supine posture, \( Q/V_A \) decreased in the vertical direction from 1.14 ± 0.09 superiorly to 0.71 ± 0.09 inferiorly. This contrasts with the more even distribution of perfusion down the lung in normal subjects (Fig. 3). A similar result was found when a cranial vertical strip was analysed. The horizontal distribution of pulmonary perfusion was assessed for both inferior and superior lung strips, with identical findings. \( Q/V_A \) was uniformly distributed, being 0.9 ± 0.15 cranially and 0.97 ± 0.1 caudally.

In the one patient examined in the right lateral decubitus posture, \( Q/V_A \) was greater in the lower lung (area weighted mean relative perfusion per unit alveolar volume 0.84) than in the upper (0.58), i.e. an upper/lower lung ratio of 0.69. The difference between the two lungs is not as great as that observed in normal subjects. The vertical distribution of \( Q/V_A \) was studied for the caudal vertical strip only. In the upper lung, perfusion decreased from 1.16 over the superior third of the lung to 0.74 over the inferior third; in the lower lung, \( Q/V_A \) decreased from 1.48 over the superior quarter to 0.64 over the inferior quarter (Fig. 3). In the horizontal direction, in both upper and lower lungs, \( Q/V_A \) was uniformly distributed.

**Ventilation/perfusion ratio**

In the vertical direction, \( V_A/Q \) decreased from 1.20 ± 0.17 superiorly to 0.85 ± 0.08 inferiorly, most of this decrease occurring over the superior fifth of the lung. Results in the right lung were similar. The vertical gradient of \( V_A/Q \) was less in patients than in normal subjects (Fig. 4). For a horizontal strip, across the base of the left lung, \( V_A/Q \) was slightly lower medially (0.07 ± 0.13) than laterally (1.09 ± 0.09). Results were similar in the right lung.

![Fig. 3. Vertical distribution of \( Q/V_A \) (arbitrary units) in the lungs of patients with bilateral diaphragmatic paralysis in the (a) upright, (b) supine and (c, d) right lateral decubitus postures in comparison with results in normal subjects. For symbols see Fig. 1.](image-url)
In supine patients, \( V_A/Q \) was slightly lower superiorly (0.87 ± 0.08) than inferiorly (1.06 ± 0.11). Fig. 4 shows that \( V_A/Q \) is more evenly distributed in the patients than in the normal subjects. \( V_A/Q \) was uniformly distributed in the horizontal direction.

In the one patient studied in right lateral decubitus, \( V_A/Q \) was greater in the upper lung (area weighted mean 1.58) than in the lower lung (0.68), i.e. an upper/lower lung ratio of 2.32 compared with 1.14 in normal subjects. Within the upper lung, \( V_A/Q \) decreased from 1.08 over the superior third of the lung to 0.75 at its inferior aspect. Within the lower lung, \( V_A/Q \) continued to decrease from 1.1 superiorly to 0.78 over the inferior quarter (Fig. 4). Note that for the within-lung analyses, regional counts for \( V \) and \( Q \) are expressed as a percentage of the total for that lung only. In the horizontal direction, \( V_A/Q \) was evenly distributed in the upper lung, but in the lower lung \( V_A/Q \) decreased from 1.04 over the cranial fifth of the lung to 0.76 over the caudal fifth.

Discussion

The effect of paralysis of the diaphragm on regional lung function has been studied previously only in patients with unilateral paresis (Arborelius, Lilja & Senyk 1975a,b; Ridyard & Stewart, 1976; Weitzenblum, Moyses, Hirth, Kuissu & Methlin, 1977). In general, reduction in ventilation, alveolar volume and perfusion was found at the base of the lung overlying the paralyzed diaphragm. In the present study, the distribution of regional specific ventilation in the upright posture was slightly more uniform than in normal subjects. There was a difference near the lung bases where decreased function was found. This result agrees with the findings of previous authors. The major finding was a reversal of the normal vertical distribution of specific ventilation in horizontal postures. A similar reversal was shown by Roussos et al. (1977b) when normal subjects breathed voluntarily without using their diaphragm. They suggest that the redistribution of inspired gas achieved by voluntary use of different muscle groups implies regional pleural pressure swings. The pattern of respiratory muscle action, which patients with bilateral diaphragm paralysis are forced to adopt, may also result in unequal pleural pressure swings over the lung.

Consider the right lateral decubitus posture (Fig. 5). With paralysis of the diaphragm, there is no active tension available to overcome the abdominal hydrostatic pressure gradient and the patient breathes by enlarging his chest cavity using intercostal and accessory muscles or the passive recoil of the chest wall. The dependent lung is sandwiched between (a) the mediastinal contents, which cannot be effectively raised as no tension can be developed by the diaphragm, (b) the diaphragm itself, with the hydrostatic pressure gradient in the abdomen forcing it cranially into the thoracic cavity, and (c) the lower chest.
Regional lung function in diaphragm paralysis

Fig. 5. Sagittal section of thorax and abdomen of a normal human subject (a) and a diaphragmatic paralysis patient (b) in the right lateral decubitus posture. At FRC (continuous lines) in the normal subject the hydrostatic gradient of abdominal pressure (Pab.) may be opposed by a pressure (Pdi.) developed by active contraction of the diaphragm. In the patient abdominal pressure forces the diaphragm cranially and FRC is lower than in the normal subject. Expansion of the lower lung on inspiration (broken lines) is restricted by abdominal pressure, mediastinal weight and the surface on which the patient lies. V/Vₐ is greater in the upper lung; the gradient of ventilation from upper to lower lung is the reverse of normal. Absolute values of V/Vₐ are greater because of the reduction of FRC.

wall, movement of which is restricted by the surface on which the patient lies. Thus there is little room for this lung to expand. Superiorly, the upper hemithorax is unrestricted and the abdominal pressure is lower. Thus intercostal and accessory muscles expand the thorax in such a way that the pleural pressure changes by a greater amount over the upper lung than the lower. An unequal distribution of swings in pleural pressure during tidal breathing develops in supine dogs after bilateral phrenic nerve section (D'Angelo, Sant'Ambrogio & Agostoni, 1974). Tidal changes in pleural pressure were greater over the third intercostal space than over the sixth intercostal space and changes there were greater than on the diaphragmatic surface.

An alternative explanation for the reversal of the vertical gradient of specific ventilation may be related to the effect of lung volume on lung and chest wall compliance. There is little information available on the effect of diaphragm paralysis on FRC. McCredie et al. (1962) reported normal values for FRC in three patients in both upright and supine postures although Huettemann & Huckauf (1970) reported FRC to be 57% of the predicted normal value in the upright posture falling to 50% of the predicted normal value in the supine posture. In addition, a low end-expiratory lung volume may cause secondary changes in the lung such as stiffening and collapse from altered surfactant properties (Gibson, Pride, Newsom Davis & Loh, 1977). The distribution of regional volume in patients with bilateral diaphragmatic paralysis has not been reported. The work of Roussos, Martin & Engel (1977a) on diaphragmatic relaxation and Rehder, Sessler & Rodarte (1977) on the effects of anaesthesia and muscle paralysis in normal subjects in decubitus postures suggest that patients with diaphragmatic paralysis may have a low end-expiratory lung volume and the vertical gradient of regional lung volume may increase, particularly in horizontal postures. Under these circumstances, the expansion of dependent lung regions may be so reduced that airway closure and gas trapping occurs. Because of relatively high airway opening pressures, ventilation to those regions at that lung volume may be reduced (Milic-Emili, Henderson, Dolovich, Trop & Kaneko, 1966).

In unilateral diaphragmatic paralysis, redistribution of perfusion from the base towards the apex of the lung on the affected side has been recorded (Arborelius et al., 1975a,b; Ridyard & Stewart, 1976; Weitzenblum et al., 1977). In contrast to the situation in normal subjects, Q/Vₐ decreased from superior to inferior in patients with bilateral diaphragmatic paralysis in the supine posture (Fig. 3). Although the vertical distribution of regional lung volume was not measured in the patients it is probable that there is a regional volume gradient as great or greater than that in normal subjects and a lower end-tidal-expiratory lung volume. Therefore, the effect of low lung volumes on extra-alveolar vessel resistance (Hughes, Glazier, Maloney & West, 1968) may be responsible for the decrease in Q/Vₐ down the lung in these patients. In addition, local hypoxic vasoconstriction in the dependent zones may have contributed to the decrease in Q in order to preserve a normal Vₐ/Q gradient.

The present studies indicate that Vₐ/Q is topographically more uniformly distributed in
patients with bilateral diaphragmatic paralysis than in normal subjects in the supine and upright postures. Nevertheless, intraregional inhomogeneity of $V_a/Q$ is almost certainly present and the real dispersion of $V_a/Q$ ratios in the lung must be much larger than demonstrated by the topographical measurements; indeed, the ideal alveolar–arterial oxygen difference increases when these patients go from the upright to the supine posture (Löh, Hughes & Newsom Davis, 1979).

In the right lateral decubitus posture, there is gross topographical mismatching of ventilation and perfusion. Interestingly, this posture is of considerable importance to patients with bilateral diaphragmatic paralysis since it allows them to achieve a horizontal posture where gravity aids in the descent of the diaphragm. Thus they achieve some mechanical advantage in expanding their lungs and often use this posture for sleeping, but at the expense of topographical mismatching of ventilation and perfusion.

In anaesthetised, paralysed and mechanically ventilated normal subjects, the dispersion of $V_a/Q$ is greater during mechanical ventilation than was the case during awake spontaneous breathing. This is associated with increased ventilation of superior regions with persistence of the normal vertical gradient of blood flow (Landmark, Knopp, Rehder & Sessler, 1977; Chevrolet, Martin, Flood, Martin & Engel, 1978). These results support the findings reported here in spontaneously breathing patients with bilateral diaphragm paralysis.

Acknowledgments

We are grateful to Dr J. P. Lavender for permission to use facilities in the Diagnostic Radiology Department Scanning Unit at Hammersmith Hospital and to Mr J. Clark and Mr I. Watson of the MRC Cyclotron Unit for the supply of radioactive gases. The technical assistance of Mrs Hazel Jones and Mr T. Pratt is gratefully acknowledged. Dr T. P. Amis was supported by a scholarship from the University of Queensland, Australia and material in this paper formed part of a thesis accepted for the Ph.D degree by the University of London (Amis, 1979).

References


