HUMAN OXYGENATION BY AIR DURING ANAESTHESIA: THE RELATION OF VENTILATORY VOLUME AND ARTERIAL OXYGEN SATURATION

BY

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SUMMARY

Ventilation and arterial oxygen saturation have been measured in 18 patients during anaesthesia with ether or halothane carried in air. All patients were ventilated artificially with a Jefferson ventilator at a frequency of 16 breaths per minute and variable tidal volume. Ventilation has been expressed as a percentage of Radford's standard, Nunn's standard and the patient's pre-operative resting ventilation. 110 per cent of both Radford's and Nunn's standard ventilation and 98 per cent of the patient's own resting ventilation were found to yield a mean arterial oxygen saturation of 95 per cent. Below this the mean saturation fell sharply with decreasing ventilation. Cyanosis was never visible even with a saturation as low as 70 per cent, although desaturation was evident in the colour of the arterial blood when sampled.

The use of air as a vehicle gas for volatile anaesthetic agents during inhalation anaesthesia has long been considered a safe practice, particularly in the case of the open drop technique with ether. On the other hand, anaesthetists have long been in the habit of administering oxygen-enriched gas mixtures for the sole purpose of preventing hypoxaemia. The availability of an oxygen-rich atmosphere, however, does not guarantee the adequacy of arterial blood oxygenation or carbon dioxide elimination, unless the maintenance of proper pulmonary ventilation is ensured.

In recent years, some anaesthetists have investigated the validity of oxygenation by air during anaesthesia, having been influenced by the introduction of calibrated inhalers for volatile anaesthetics and using self-inflating reservoirs for artificial ventilation. They consistently emphasize that provided adequate pulmonary ventilation be maintained it is unnecessary to administer a higher concentration of oxygen than is present in ordinary air, except in certain circumstances.

Since January 1960, more than 4,000 patients have been anaesthetized with volatile agents vaporized in room air in this Department. The apparatus consisted of a calibrated vaporizer for ether or halothane, an Oxford Inflating Bellows or a Jefferson Ventilator with Wakai T-valve, and a Ruben valve connected to the endotracheal tube. This open non-rebreathing system permits ventilation of the lungs of the patients with air containing a predetermined concentration of a volatile agent.

In the first series of twelve patients inhaling ether and air from the EMO inhaler, the average of arterial oxygen saturation levels was 91.8 per cent during spontaneous respiration and 95.2 per cent during controlled respiration. It has been subsequently revealed, while measuring arterial oxygen saturation of the patients under various conditions of artificial respiration that there is some correlation between arterial oxygen saturation levels and ventilatory volumes.

This paper concerns the oxygenation curves obtained by plotting the levels of arterial oxygen saturation against ventilatory volumes which are expressed as percentages of modified Radford's standard ventilation, Nunn's alternative standard ventilation during anaesthesia, and the patients' resting minute volumes.

METHOD

Eighteen adult patients (11 males) were used for this study. These patients underwent various types
of operations in the supine or lateral position, with the exception of intrathoracic procedures. They had no apparent cardiopulmonary disorders and had haemoglobin indices of more than 70 per cent Sahli.

Pre-anaesthetic medication consisted of pentobarbital sodium 100 mg and atropine sulphate 0.5 mg, 2 hours and 1 hour respectively prior to the induction of anaesthesia. Anaesthesia was induced with thiamyl sodium in doses ranging from 200 to 300 mg, followed by endotracheal intubation facilitated by 40 mg suxamethonium chloride. Artificial ventilation by air containing 3 per cent ether or 1 per cent halothane was started immediately after administering suxamethonium, and the patient was kept under hyperventilation for about 1 minute. In most cases ventilatory interruption due to endotracheal intubation did not exceed 30 seconds. At the first sign of the receding effect of suxamethonium, 21 mg of d-tubocurarine chloride was injected intravenously to maintain apnoea. Intermittent positive pressure ventilation was continued, using the unit consisting of a calibrated vaporizer, a Wakai T-valve (Wakai, in preparation), a Jefferson Ventilator, and a Ruben valve (fig. 1). By manipulating the pressure-regulating valves and the rate adjuster, it was possible to ventilate the patient’s lungs with definite tidal volumes or minute volumes of air containing the weak anaesthetic vapour. Ventilation was measured with a Dräger volumeter connected to the expiratory side of the Ruben valve. The value was converted to BTPS. The Dräger volumeter operates with around +7.5 per cent errors of flow rates in sinusoidal air flow (Byles, 1960).

Arterial blood samples were drawn during the early period of operation after ventilation was kept constant for 5 minutes. More than two blood samples were obtained from each patient under various ventilatory minute volumes. The positive pressure induced in the airway varied from 8 to 28 cm H₂O and the ventilatory rate was fixed at 16 per minute except for four occasions when marked hyperventilation was induced. Each blood sample was analyzed for oxygen content and

![Fig. 1](image-url)

**Fig. 1**
Anaesthetic system used in this study.
The Wakai T-valve and the Jefferson ventilator are placed between the EMO ether vaporizer or Fluotec vaporizer and the Ruben valve.
### TABLE OF RESULTS

Minute volumes of intermittent positive pressure ventilation, expressed in ml and as percentages of the three ventilation standards, and the resultant arterial oxygen saturations during ether-air and halothane-air anaesthesia in 18 subjects. Arterial blood samples were drawn after ventilation was kept constant for 5 minutes.

<table>
<thead>
<tr>
<th>Subject, operation performed and anaesthetic agents</th>
<th>Induced minute volume (ml) BTPS*</th>
<th>Radford's standard minute volume (ml) BTPS and ratio of induced ventilation (%)</th>
<th>Nunn's standard minute volume (ml) BTPS and ratio of induced ventilation (%)</th>
<th>Resting minute volume (ml) BTPS and ratio of induced ventilation (%)</th>
<th>Arterial oxygen saturation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Y.M., 19, M, 158 cm, 50 kg Tendinoplasty, left hand (halothane-air)</td>
<td>7700 165 4790</td>
<td>157 4900 160.5</td>
<td>86 4800 88</td>
<td>53.5 45.4 70.5</td>
<td>96.8</td>
</tr>
<tr>
<td>(2) T.H., 43, F, 148 cm, 61 kg Right nephropexy, cholecystectomy (ether-air)</td>
<td>7480 133.5 5440</td>
<td>141.5 5300 110</td>
<td>99.5 6800 80</td>
<td>88.5 98.5</td>
<td>91.0</td>
</tr>
<tr>
<td>(3) H.K., 22, F, 152 cm, 45 kg Craniotomy (ether-air)</td>
<td>3344 83.5 4220</td>
<td>88 3800 109</td>
<td>67 3200 83.5</td>
<td>70.5 93.5</td>
<td>87.5</td>
</tr>
<tr>
<td>(4) K. H., 42, M, 162 cm, 50 kg Right lumbar sympathectomy (halothane-air)</td>
<td>11660 122 4560</td>
<td>270 4300 203.5</td>
<td>129 5720 97</td>
<td>133 92.2</td>
<td>95.4</td>
</tr>
<tr>
<td>(5) M. H., 19, F, 154 cm, 47 kg Craniotomy (halothane-air)</td>
<td>14795 356 4160</td>
<td>361 4100 228</td>
<td>103 6500 66</td>
<td>98.9</td>
<td>84.6</td>
</tr>
<tr>
<td>(6) Y.K., 23, M, 159 cm, 60 kg Open reduction right hip joint (ether-air)</td>
<td>5620 135 5150</td>
<td>148.5 5100 116</td>
<td>95 6590 81</td>
<td>92.3</td>
<td>83.0</td>
</tr>
<tr>
<td>(7) H.I., 35, M, 167 cm, 66 kg Left inguinal colostomy (ether-air)</td>
<td>7645 133 5730</td>
<td>141 5400 134</td>
<td>153 5700 154</td>
<td>97.2</td>
<td>98.8</td>
</tr>
<tr>
<td>(8) A.Y., 15, F, 158 cm, 59.5 kg Curettage, left humerus (ether-air)</td>
<td>6732 122 5520</td>
<td>118 5700 107</td>
<td>70 6300 61.5</td>
<td>89.3</td>
<td>71.0</td>
</tr>
<tr>
<td>(9) M.I., 36, F, 152 cm, 50 kg Breast amputation (ether-air)</td>
<td>5280 137.5 3840</td>
<td>135 3900 105.5</td>
<td>91 5000 69</td>
<td>94.4</td>
<td>84.8</td>
</tr>
</tbody>
</table>
Table of Results (continued)

<table>
<thead>
<tr>
<th>Subject, operation performed and anaesthetic agents</th>
<th>Induced minute volume (ml) BTPS</th>
<th>Radford's standard minute volume (ml) BTPS and ratio of induced ventilation (%)</th>
<th>Nunn's standard minute volume (ml) BTPS and ratio of induced ventilation (%)</th>
<th>Resting minute volume (ml) BTPS and ratio of induced ventilation (%)</th>
<th>Arterial oxygen saturation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(10) M.N., 20, M, 162 cm, 45.5 kg, Right nephrectomy (halothane-air)</td>
<td>5610 4760</td>
<td>118 130</td>
<td>110</td>
<td>95.5</td>
<td></td>
</tr>
<tr>
<td>(11) O.M., 25, M, 164 cm, 48.5 kg, Craniotomy (ether-air)</td>
<td>4480 6050</td>
<td>134 132</td>
<td>112</td>
<td>98.8</td>
<td></td>
</tr>
<tr>
<td>(12) Y.A., 52, M, 163 cm, 59 kg, Radical resection left maxilla (ether-air)</td>
<td>5620 8140</td>
<td>144.5 170.5</td>
<td>110</td>
<td>96.6</td>
<td></td>
</tr>
<tr>
<td>(13) Y.O., 22, M, 160 cm, 54 kg, Left nephrectomy (halothane-air)</td>
<td>4390 5445</td>
<td>124 109</td>
<td>93.5</td>
<td>96.0</td>
<td></td>
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<tr>
<td>(14) Z.I., 25, M, 162 cm, 55 kg, Gastrectomy (ether-air)</td>
<td>5300 6182</td>
<td>117 123</td>
<td>123</td>
<td>98.0</td>
<td></td>
</tr>
<tr>
<td>(15) E.Y., 32, F, Curettage, right tibia (halothane-air)</td>
<td>4640 11880</td>
<td>256 237.5</td>
<td>229</td>
<td>98.0</td>
<td></td>
</tr>
<tr>
<td>(16) K.N., 28, M, 164 cm, 53 kg, Craniotomy (halothane-air)</td>
<td>4920 8030</td>
<td>163.5 164</td>
<td>114</td>
<td>96.8</td>
<td></td>
</tr>
<tr>
<td>(17) T.T., 36, F, 148 cm, 40 kg, Craniotomy (halothane-air)</td>
<td>3570 16830</td>
<td>472 466.5</td>
<td>194</td>
<td>97.8</td>
<td></td>
</tr>
<tr>
<td>(18) K.I., 22, M, 169 cm, 45 kg, Gastroenterostomy (halothane-air)</td>
<td>4170 10780</td>
<td>256 229</td>
<td>185</td>
<td>96.8</td>
<td></td>
</tr>
</tbody>
</table>

* Induced minute volume measured by the method shown in figure 1 was converted into BTPS.
† Radford's standard minute volume was converted into BTPS multiplying by 1.080. This figure was then reduced by 960 ml assuming the average upper airway deadspace eliminated by intubation in this study to be 60 ml.
‡ Resting minute volume was measured by applying volumeter directly to mouth-piece, which partly eliminated upper airway dead space. Though the volumeter was heated by the expired air, the figures are still slightly smaller than BTPS.
saturation by Van Slyke's manometric method as modified by Goldstein, Gibbon, Albritten and Stayman (1950). The oxygen saturation levels were plotted against the ventilatory volumes expressed as percentages of the three ventilation standards.

Ventilation standards.

A suitable indication of ventilation was required to express the various ventilatory minute volumes in the individual subject which produce certain arterial oxygenation levels. The Radford nomogram (Radford, Ferris and Kriete, 1954; Radford, 1955) has been suggested for use during anaesthesia (Scurr, 1956; Nunn, 1960a). In order to standardize the conditions of ventilatory measurement in this study, the figures obtained from the Radford nomogram were converted to BTPS and then reduced by 960 ml assuming the average upper airway deadspace eliminated by endotracheal intubation to be 60 ml. Nunn (1960b) has suggested an alternative method of predicting arterial carbon dioxide tension during anaesthesia from the ventilation. The present author adopted this method for standardizing the minute volume of artificial ventilation in order to maintain the arterial carbon dioxide tension at 40 mm Hg during anaesthesia, which has been suggested as a suitable index for the maintenance of ventilation by Woolmer (1960). As another ventilation standard, the patients' resting minute volumes were used.

RESULTS

The induced minute volumes of artificial ventilation in each subject expressed as percentages of the three ventilation standards, together with the resultant arterial oxygenation levels are set out in the table. There were nine patients in whom anaesthesia was maintained by the constant inhalation of 1 per cent halothane in air. The hypotension associated with the combined use of d-tubocurarine and halothane, to which attention has been drawn by previous investigations (Johnstone, 1956; Bryce-Smith and O'Brien, 1956; Marrett, 1957; Hansen, Davies and Hardy, 1959), was less significant, even under positive pressure ventilation, when d-tubocurarine was injected slowly in diluted solution than in patients in whom anaesthesia was maintained by

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**FIG. 2**

The relation of arterial oxygen saturation and ventilatory minute volume expressed as a percentage of Radford’s standard
the constant inhalation of 3 per cent ether in air while d-tubocurarine was injected in non-diluted form (Oda et al., 1961).

The arterial oxygen saturation levels plotted against Radford's ventilation standard (fig. 2) formed a hyperbolic distribution, although with a wide range of distribution, which resembled the oxygen dissociation curve of haemoglobin. The dotted line is a smooth curve closely approximating to the mean figures obtained from all saturation and ventilation values lying within each 20 per cent interval on the abscissa. On this curve arterial oxygen saturation of 95 per cent or more is likely to be produced by ventilation of more than 110 per cent of the standard, but arterial oxygen saturation drops steeply as ventilation decreases below 110 per cent of the standard.

The arterial oxygen saturation levels plotted against Nunn's alternative ventilation standard during anaesthesia show an almost similar distribution to that noted when these were plotted against the modified Radford's ventilation standard, and also with a wide range of variation (fig. 3). On the estimated ventilation-oxygenation curve on Nunn's standard, it appears that 110 per cent ventilation is likely to be necessary for 95 per cent saturation of arterial blood. It is also shown that oxygen saturation drops steeply as ventilation decreases below 110 per cent of Nunn's standard.

Figure 4 shows the distribution of arterial oxygen saturations plotted on the standard of the patients' resting minute volumes. The arterial oxygen saturations formed a hyperbolic curve with a surprisingly narrow range of distribution compared with the former two plottings. On the estimated ventilation-oxygenation curve, arterial oxygen saturation of 95 per cent or more is likely to be secured by the ventilation of more than 98 per cent of the patients' resting minute volumes, and arterial oxygen saturation also drops steeply as ventilation decreases below 98 per cent of the resting minute volume.

Observation of each ventilation-oxygenation curve reveals that ventilation of more than 110 per cent of the standard increases the arterial oxygen saturation only to the extent of 5 per cent, while 70 per cent ventilation of Radford's standard causes desaturation of around 70 per cent, 70 per cent of Nunn's standard causes 74 per cent desaturation, and 70 per cent of the patient's resting minute volume causes 77 per cent desaturation of oxygen.

There was no significant difference in blood oxygenation between the groups anaesthetised with 3 per cent ether and 1 per cent halothane.

In all the subjects studied, cyanosis was under no circumstances observed. This was true even during hypoventilation sufficient to cause an arterial oxygen saturation of 70.5 per cent in a 19-year-old male (Subject 1), although the colour of the blood had become warningly dark at the time of sampling.

**DISCUSSION**

The potential dangers of using air as the only means of blood oxygenation during inhalation anaesthesia have been suggested by many authors. Weitzner, King and Ikezono (1959) studied the rate of arterial oxygen desaturation during apnoea after hyperventilation with air and with 100 per cent oxygen. In the former circumstances arterial oxygen saturations dropped to dangerous levels within 1½ minutes, while in the latter oxygen saturation was well maintained during the first 2 minutes. They admitted that the use of 100 per cent oxygen prior to apnoea would have an advantage over ordinary air in maintaining oxygen saturation longer at an acceptable level. The polarographic study of the same problem by Heller and Watson (1961) also suggested that it is advantageous to use 100 per cent oxygen instead of 21 per cent oxygen (room air) prior to induced apnoea. Fujimori and Virtue (1960), in their electroencephalographic study, also stressed the greater safety obtained by the use of 100 per cent oxygen for ventilating the subject's lungs before intubation using the apnoeic technique.

The results of these investigators manifestly demonstrated more rapid desaturation after breathing air than after breathing oxygen-enriched mixtures prior to the onset of apnoea. The fact has often been emphasized that the body cannot store a significant amount of oxygen. The question arises: "What would be an adequate ventilation to maintain the normal levels of arterial oxygen saturation, using gas mixtures of different oxygen concentration or tension?" The possibility of air being inadequate for man's breathing, in relation to its density or altitude, has long been known.
Fig. 3
The relation of arterial oxygen saturation and ventilatory minute volume expressed as a percentage of Nunn’s standard.

Fig. 4
The relation of arterial oxygen saturation and ventilatory minute volume expressed as a percentage of resting minute volume.
Human respiratory and circulatory responses in themselves work to combat the hypoxic condition due to exposure to low tension of oxygen, and in normal man inhalation of 10 per cent oxygen at sea level (76 mm Hg, equivalent in tension to the altitude of 5180 m) provokes marked respiratory stimulation leading to increased minute volume. The circulatory response appears as the progressive increase of the pulse rate and cardiac output with decrease of oxygen tension below normal. These physiological responses, however, fail to maintain the normal arterial oxygen saturations, and the inhalation of 18 per cent oxygen results in 94 per cent saturation and of 16 per cent oxygen results in 91 per cent saturation (Dripps and Comroe, 1947). Having a relatively high affinity for oxygen, the physical nature of haemoglobin affords an oxyhaemoglobin saturation of approximately 84 per cent at an altitude of 4,000 m (Lowry, 1961).

To a certain extent the oxygen saturation may be increased under anoxic conditions by hyperventilation (Houston, 1946). The question of ventilation by air during anaesthesia should be discussed with these limitations in mind. Faulconer and Lattrell (1949) reported saturations as low as 70 per cent during ether-air anaesthesia, while they noted an oxygen tension of 128 mm Hg in the air under the mask before adding ether, instead of 152 mm Hg at the altitude of 305 m. This oxygen tension under the mask is equivalent to 16.8 per cent oxygen at sea level. Here arises the problem of suitable breathing systems when using air as the principal diluent of volatile agents. Modern efficient automatic non-rebreathing valves with minimal deadspace are available and the author would like to stress the value of a non-rebreathing system while using air, because it enables pulmonary ventilation to be accurately measured.

Artificial ventilation by air during anaesthesia has been shown to maintain arterial oxygen saturations. Campbell, Nunn and Peckett (1958) obtained average arterial oxygen saturations of 96.3 per cent and 95 per cent in their six subjects under spontaneous and artificial respiration respectively, when room air was the only inspired gas during thiopentone anaesthesia. Parkhouse and Simpson (1959) in their "Restatement of Anaesthetic Principles" suggested that air as a vehicle for anaesthetic vapours is ideal and should be used extensively. Ikezono, Harmel and King (1959) produced satisfactory blood oxygen levels during controlled ventilation with ether-air mixtures. They also noted saturation levels of more than 95 per cent during spontaneous respiration but observed that the lowest saturation occurred during manually assisted respiration. This was probably due to unsteady or irregular ventilatory volumes. Nandrup (1959) reported on the use of ether-air mixture with controlled ventilation. He found arterial oxygen levels to be above or equal to normal on all occasions. Poppelbaum (1960) described oximetry during ether-air and halothane-air anaesthesia for thoracic operations. His continuous measurement showed adequate oxygenation in the great majority of cases. Papantony and Landmesser (1960) reported on their own anaesthetic device for halothane-air anaesthesia. In their series the average of arterial oxygen saturations in the group in which ventilation was controlled was 94.3 per cent but only 90.7 per cent in the group in whom respiration was spontaneous. A similar unit for halothane-air anaesthesia was described by Macartney (1961), and in 102 consecutive cases the arterial blood appeared satisfactorily pink as long as ventilation was maintained in accordance with Radford's nomogram. More precisely, Cole and Parkhouse (1961) showed in sixty-two cases in which ventilation was controlled using volatile agents in air, that oxygenation levels within 2 per cent of resting levels were maintained in 92 per cent. Merrifield (1961) reported ventilatory measurements in eighteen subjects breathing spontaneously the halothane-ether azeotrope in air, with concomitant measurement of arterial oxygen saturations. The mean saturation was 95.4 per cent at a mean ventilation of 97.4 per cent of predicted ventilations. He suggested that the changes in saturation levels did not always correspond with changes in ventilation but that a fairly close relationship would be found between the two.

The results of the present study show that there is a good relationship between ventilatory volume and arterial oxygen saturation during artificial ventilation of the anaesthetized patient with air. Although the patients from whom blood samples were drawn were in light planes of anaesthesia, and the oxygen concentration in air had been reduced by ether and halothane vapour by 0.63 per cent and 0.2 per cent respectively, the relation between ventilatory volume and oxygenation levels was...
similar to that in the subject ventilated with room air.

On the three ventilation-oxygenation curves here obtained, the somewhat wide range in the distribution of arterial oxygen saturations may be attributed to many factors such as variations in alveolar ventilation due to altered physiological dead-space or altered distribution of pulmonary circulation which could be caused by the change in airway pressure, or may be attributable to variations in metabolic rate. There might also have been examples of lowered arterial oxygen levels in pre-operative resting patients as described by Lambersten et al. (1952) and Cole and Parkhouse (1961). In normal individuals breathing the same oxygen concentration a wide range in arterial oxygen levels occurs, as repeatedly shown by previous reporters (Barach et al., 1941; Pruitt, Burchell and Barnes, 1945; Dripps and Comroe, 1947).

The least random variation of oxygen saturations is on the ventilation-oxygenation curve based on the patient’s resting respiration, which suggests that the resting minute volume of individuals is the best guide for artificial ventilation with air. The wide difference between the resting minute volumes and the standard ventilations calculated from Radford’s nomogram is probably due to the individual variation in metabolic rate, diffusion capacity of the pulmonary membrane, and other factors which do not apply to the physiological nomogram based on the basal carbon dioxide production and respiratory deadspace estimated from body weight. The same can be said of Nunn’s alternative method of prediction of ventilation during anaesthesia according to carbon dioxide homeostasis. These two ventilation standards are invaluable when it is necessary to institute artificial respiration in the apnoeic or anaesthetized patient whose basal minute volume is not known, but as long as information is available about the patient’s resting minute volume prior to anaesthesia, it is the most suitable guide for blood oxygenation.

The resemblance of the ventilation-oxygenation curve using a gas of fixed oxygen concentration to the oxygen dissociation curve of haemoglobin is of interest. It is suspected that the complete form of the ventilation-oxygenation curve will be an S-shaped hyperbola, though not perfected in this study to avoid the risk of exposing the subjects to too severely impaired ventilation. The addition of oxygen in the inhaled air will shift the curve to the left and the reduction of oxygen from the air will shift the curve to the right, as does the change in pH on the oxygen dissociation curve of haemoglobin. This assumption suggests that the availability of additional oxygen to the inhaled air should also be supported by an adequate volume of ventilation in any event. The reduction in oxygen tension resulting from adding the vapour of any volatile agent, at present available for the maintenance of light levels of anaesthesia, is by no means so great in extent that ventilation fails to keep oxygen saturations at the normal level, unless circulatory derangement is caused by anaesthetics or surgical manoeuvres.

The versatility of the EMO ether Inhaler (Epstein and Macintosh, 1956) and its physical principles have led to many reports of experiences or recommendations about the use of similar units vaporizing volatile agents in air in a non-rebreathing system (Parkhouse and Simpson, 1959; Ikezono et al., 1959; Soper, 1959, 1961; Papantony and Landmesser, 1960; Hingson, 1961; Cole and Parkhouse, 1961; Macartney, 1961; Merrifield, 1961; Safar and Gedang, 1961; Pearson and Safar, 1961). The limitation in the oxygen tensions in the inhaled air when using these techniques raises questions concerning the degree of ventilation required to maintain arterial oxygen saturations at normal levels. The ventilation-oxygenation curves obtained in this study may be of value as a guide to ventilation.

ACKNOWLEDGMENTS

For their continuous encouragement, my thanks are due to the Nuffield Department of Anaesthetics, and in particular to Prof. Sir Robert Macintosh, the stimulus of whose visit to Japan cannot be overestimated. I am indebted to Prof. Y. Hashimoto who gave me the opportunity to carry out this work. My thanks are also due to my colleagues for facilitating and assisting in the preparation of this paper.

REFERENCES

HUMAN OXYGENATION BY AIR DURING ANAESTHESIA


VENTILATION D'OXYGÈNE PAR L'AIR PENDANT L'ANESTHESIE: RAPPORT ENTRE VOLUME DE VENTILATION ET SATURATION ARTÉRIELLE EN OXYGÈNE

SOMMAIRE
L'auteur mesura la ventilation et la saturation artérielle d'oxygène chez 18 patients au cours d'anesthésies à l'éther ou au halothane sur "substrat-porteur" d'air. Tous ses patients furent ventilés artificiellement par un ventilateur Jefferson marchant à la fréquence de seize inspirations par minute et avec un volume d'air d'amplitude variable. La ventilation est exprimée dans la présente étude comme pourcentage du standard de Radford, du standard de Nunn et de la respiration préopératoire du patient au repos. L'auteur constata que 110% des ventilations-standard de Radford et de Nunn et 98% de la ventilation normale propre du patient au repos donnèrent une saturation artérielle moyenne en oxygène de 95%. En dessous de ces chiffres la saturation moyenne s'abaissa assez brutalement à mesure de la réduction de la ventilation. Aucune cyanose ne devint visible, même avec une saturation aussi basse que 70% — alors que l'état de "désaturation" était rendu évident par la couleur des échantillons de sang artériel.

NOTICE
The reader's attention is drawn to the fact that the articles:

"Continuous cerebrospinal fluid drainage by indwelling spinal catheter" by G. Vourc'h
(Brit. J. Anaesth., 35, 118)

and

"Problems raised by anaesthesia for thalamic and cortical exploration in neurosurgery" by G. Vourc'h, J. Hardy and M. Denavit (Brit. J. Anaesth., 35, 208)

were based upon two papers read at the meeting of the Section of Anaesthetics of the Royal Society of Medicine April 6, 1962.