THE RATE OF ARTERIAL OXYGEN DESATURATION DURING APNEA IN HUMANS

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THE DESIRABILITY of a simple portable anesthetic apparatus requiring no tanks of gas or carbon dioxide absorber for use during wartime or civilian catastrophe is self evident. Because we believe that the EMO Ether Vaporizer,[1] which can use atmospheric air as the vehicle for ether vapor, is the best apparatus available for this purpose, we have used it frequently for clinical anesthesia. During a course of study [2] on the arterial oxygen saturation during ether-air anesthesia using this apparatus, it became apparent that little was known of the rate of oxygen desaturation of arterial blood during apnea, especially the apnea associated with tracheal intubation. As a corollary, it became desirable to determine whether varying the tension of oxygen in the atmosphere inhaled prior to the onset of apnea would affect the rate of desaturation. Such knowledge would answer many questions that arise during clinical anesthesia: How much time may be spent in exposing the larynx of the apneic patient? Should patients' lungs be ventilated with oxygen before attempting tracheal intubation? How long may a patient remain apneic after cessation of controlled respiration while waiting for resumption of spontaneous respiration? Others have investigated factors related to endotracheal anesthesia which may effect arterial desaturation: tracheal intubation,[3, 4] extubation,[5] and endotracheal suction.[5] However, the influence of duration of apnea does not appear to have been investigated.

MATERIAL AND METHODS

Fourteen unselected patients ranging in age from 25 to 78 years who were to undergo elective general surgical procedures were used for this study. An arterial puncture using a 20 gauge, 2 inch styletted spinal needle was performed at the anticubital fossa. The needle was left *in situ* so that arterial blood samples could be obtained at will during the study.

Thiopental 2½ per cent was then given intravenously until the patient's eyelash reflex was gone. This required an average dose of 320 mg. with a range of 200 mg. to 400 mg. Sixty to 100 mg. of succinylcholine were injected intravenously as a single dose and a 0.2 per cent succinylcholine drip was started and maintained at a flow rate sufficient to keep the patient apneic throughout the study.

Approximately one minute after injection of the thiopental, and after the insertion of an oropharyngeal airway, a face mask was applied and the subject's lungs were hyperventilated thirty times a minute by means of an Oxford Inflating Bellows [6] using air in a nonbreathing technique. Such ventilation assured a minute volume between 15 and 20 liters. After two minutes of such hyperventilation, the patient was permitted to remain apneic for at least 90 seconds with the mask removed. In one instance the patient's lungs remained unventilated for 2 minutes. The mask was then reapplied, and the patient's lungs were similarly hyperventilated with oxygen for two minutes. The mask was then removed and the patient remained apneic for 2 to 4 minutes, after which the patient's lungs were reventilated and the anesthesia continued as it would have been normally.

Two milliliter heparinized blood samples were obtained in the following sequence during the study: before the start of anesthesia; at the end of the 2 minute period of air hyperventilation; after 1 minute of apnea following air hyperventilation; after 1½ minutes of apnea; after the 2 minute period of oxygen hyperventilation; and 4 samples at the end of consecutive one-minute intervals during the four minute period of apnea following oxygen hyperventilation. The time intervals were varied slightly in some patients either for technical reasons or because of differences in the subject's physical status. Pulse rate was noted and blood pressure was measured by

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TABLE 1

ARTERIAL OXYGEN SATURATION DURING APNEA FOLLOWING VENTILATION WITH AIR AND OXYGEN

Patient	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Hemoglobin	8.0	11	13		15	15	13	9.6	12	13	12		12	13
Sex	F	F	M	M	M	M	F	F	F	F	F	M	M	F
Age	38	30	65	52	53	51	56	57	26	30	25	78	42	34
Control (arterial oxygen saturation)	93.5	93.6	94.8	97.1	87.0	91.8	96.0	95.4	96.3	93.0	97.4	93.1	91.7	92.7
Arterial Oxygen Saturation Following Ventilation with Air														
Zero time	95.8	98.0	98.5	96.6	96.8	96.0	89.8	92.5	98.1	96.3	97.3	94.6	94.9	96.0
1 minute	64.6	74.1	83.9	87.2	65.2	77.6	84.6	66.8	82.1	64.5	67.1	87.1	87.7	
1½ minutes	52.6	55.0	81.9	77.6	53.9	66.9	73.0	44.2	65.6	47.7	45.7	80.7	81.4	43.9
Arterial Oxygen Saturation Following Ventilation with Oxygen														
Zero time	94.5	98.4	99.3	98.5	99.7	98.2	98.6	99.2	99.7	98.2	98.0	98.4	97.0	99.2
1 minute		98.7	99.1	98.5	98.4		99.1	97.8	99.3	97.6		99.9	97.5	96.0
1½ minutes			98.9			98.4					97.2	98.5		
2 minutes		98.8		98.8	90.0	96.1	96.6	81.9	99.7	90.5	97.8		97.4	87.1
2½ minutes				97.8	91.0		94.9	69.0	99.9	75.4				
3 minutes		98.1									96.6		98.2	
3½ minutes									97.7					
4 minutes		95.7							97.1	48.8			97.3	

sphygomanometer each time a blood sample was obtained.

All arterial blood samples were analyzed for per cent oxygen saturation of hemoglobin by means of a Beckman DU Spectrophotometer.[7] This technique compares optical density of a hemolyzed blood sample at wave lengths of 650 mu and 805 mu. Because only the ratios of optical density are measured, the patient's hemoglobin or the amount of oxygen in solution in the plasma need not be known. This method has been compared with the Van Slyke-Neill technique for oxygen saturation analysis and has been found to have comparable accuracy over the whole range of oxygen saturations.[7]

RESULTS

Inspection of table 1 indicates that if a patient's lungs are ventilated with air, a one minute period of apnea consistently lowers arterial oxygen saturation (below 88 per cent). On the other hand, it is apparent that during a similar period of apnea following pulmonary ventilation with 100 per cent oxygen, arterial oxygen saturation is well maintained (fig. 1).

The effect of preanesthetic medication upon arterial oxygen saturation was manifested by relatively low control values in some patients. These low saturations were readily restored to normal by two minutes of hyperventilation with air.

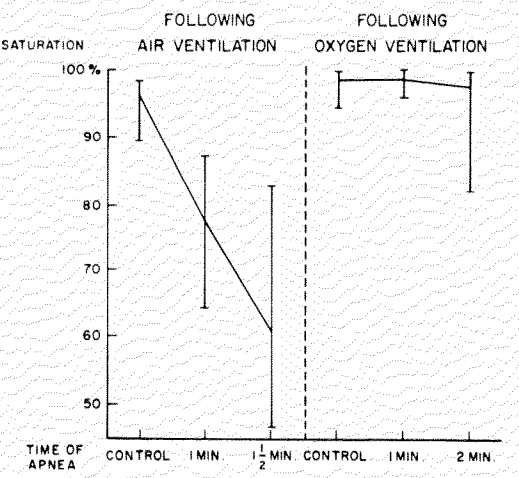

FIG. 1. Arterial oxygen saturation during apnea following ventilation with air or oxygen. The vertical lines represent the range of values obtained at the times indicated; the diagonal line connects the averages of these values.

Even though low oxygen saturations were measured in many of the subjects, (50 per cent of whom were colored) cyanosis (minimal) was evident in only one (number 10) and then only at the end of both periods of apnea (47.7 per cent and 48.8 per cent saturation); this, despite the fact that there were at least three observers at each study looking for cyanosis. This confirms the conclusions reached by Comroe and Botelho,[8] that the presence and degree of arterial oxygen saturation is difficult to assess clinically.

There was no correlation at any time between blood pressure and pulse and the state of arterial oxygen saturation during this study.

Discussion

We wish to stress that all of these patients' lungs were easy to ventilate artificially, and were deliberately ventilated under optimal conditions. Such conditions are met whether air or oxygen is used when (1) a nonrebreathing valve (which permits the most rapid equilibrium of gases in the lung with the delivered gas mixture) is used, (2) where hyperventilation is employed, and (3) where the period of hyperventilation is prolonged. Obviously the use of oxygen is more advantageous, than the use of air. Hyperventilation for prolonged periods, using oxygen with a nonrebreathing valve creates the largest possible lung oxygen reservoir because it results in the most complete washout of nitrogen from the lung.[9] On the basis of these results, the value of pulmonary ventilation with oxygen, so as to create an oxygen reservoir in the lungs, is clearly indicated before periods of anticipated hypoventilation, such as may occur during tracheal intubation, extubation, and endotracheal aspiration. We are now determining whether ventilation with mixtures containing a gas such as nitrous oxide will effectively reduce the period of apnea which may be safely permitted. Fink's work [10] on diffusion anoxia has suggested that this might occur.

Although there were no clinical manifestations or sequelae of the low saturations recorded during the time intervals used in our study, it must be recognized that we were working close to the boundaries of safety. These boundaries may be narrowed under less optimal conditions. A patient should never be left apneic for prolonged periods of time in order to achieve tracheal intubation and his lungs should always be hyperventilated with oxygen before such an attempt. It is probably good practice for anyone employing apneic techniques during tracheal intubation to occasionally have an observer time the procedure. In our opinion the *total* period of apnea (*i.e.*, from the time the mask is removed to the time ventilation is resumed via the endotracheal tube) even after hyperventilation with oxygen should not exceed 45 seconds.

It must be pointed out that if the mask is left on the patients face, during apnea, with oxygen flowing, the conditions of 'Diffusion Respiration' will be simulated, and the rate of desaturation will be slowed. This situation does not occur in clinical anesthesia.

Summary

Arterial oxygen saturation during varying periods of apnea following pulmonary hyperventilation with air or oxygen was measured. Oxygen saturation fell to undesirable levels in the first minute of apnea following air ventilation and dropped to dangerous levels in another 30 seconds. Oxygen saturation was well maintained, after pulmonary ventilation with oxygen, during the first two minutes of apnea. The implications of these results are discussed in relation to clinical anesthesia.

The technical assistance of Mr. George McCoy is acknowledged.

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ARTERIAL OCCLUSION In a patient who has arterial occlusion, whether thrombotic, embolic or traumatic, sympathetic blockade may be employed either as a diagnostic procedure to evaluate sympathectomy or as a therapeutic procedure to try to get rid of vasospasm. Once anticoagulants are started, further blocks generally are not hazarded because if hemorrhage occurs it may not be recognized for some time or if recognized it may be difficult to control. However, in one study 2,116 blocks have been performed in 544 patients on anticoagulant therapy without serious hemorrhage. There is no evidence that interruption of the sympathetic nervous system improves blood flow to muscle, and vasodilatation is probably almost entirely limited to superficial vessels. One may perhaps achieve better muscular blood flow by use of vasodilators such as priscoline and histamine which, in addition to having an effect on the sympathetic nervous system, have a direct effect on blood vessels. Their disadvantage is that they dilate all blood vessels. For this reason intra-arterial injection just proximal to the affected limb may have some use. If the drug is injected slowly enough, the quantity which reaches the venous side is insufficient to produce systemic effects. (*Wright, I. S., and others: Management of Peripheral Vascular Disease. Transcription of a Panel Meeting on Therapeutics, Bull. New York Acad. Med. 35: 241 (April) 1959.*)

CHRONIC PAIN A method is being investigated of blocking nerves by means of diathermy. An 80 mm. needle, completely insulated except for the point, is placed against the nerve for ten seconds with the diathermy machine set at 30. Applying heat to a nerve by diathermy is aimed at permanent destruction, and with perfection of this method it may become possible to avoid the neuritis that is sometimes associated with alcohol block and the occasional untoward results from the use of phenol and water. (*Lundy, J. S.: Control of Chronic Pain, Proc. Staff Meet. Mayo Clin. 34: 200 (April 15) 1959.*)