APNEA, SUCTION, AND HYPERVENTILATION: EFFECT ON ARTERIAL OXYGEN SATURATION

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MANY investigators have observed arterial oxygen desaturation during apnea and tracheal aspiration in anesthetized patients.[1-8] A few studies[9-12] in man have correlated the duration of apnea with the degree of arterial hypoxemia. However, none of these studies have evaluated the effectiveness of hyperventilation with oxygen for a brief, specific interval prior to apnea in preventing arterial oxygen desaturation.

We have attempted to determine the amount of arterial desaturation incurred by patients anesthetized with nitrous oxide and oxygen when subjected to one minute periods of apnea both with and without prior hyperventilation with oxygen. In addition, we investigated the effect of endotracheal suction during apnea on arterial oxygen saturation.

METHOD

Eleven adolescent and adult patients were studied during pulmonary resection (wedge resection, segmental resection, or lobectomy) in the lateral position. Although all but one patient had pulmonary tuberculosis, disturbances in ventilation were minimal as determined by vital capacity, maximum breathing capacity, and exercise tolerance. Anesthesia was induced with intravenous thiopental and maintained with endotracheal nitrous oxide and oxygen in a semi-closed circle absorption system (6:2 liters per minute). Supplemental thiopental, meperidine, and a succinylcholine infusion were also employed. The patient's lungs were continuously hyperventilated either manually or with a Stephenson ventilator, and in eight patients minute ventilation was monitored with a Roswell Park Ventimeter. Mean end-tidal alveolar gas samples obtained in four patients during routine ventilation had P_{CO_2} levels ranging from 10 mm. of mercury to 28 mm. of mercury by infrared analysis.* The arterial oxygen saturation (Sa_{O_2})[13] was continuously monitored with a Wood's single scale direct reading ear oximeter. The instrument was calibrated in the usual manner,[14] including comparison with Van Slyke Sa_{O_2} analyses, and standardization was performed at least every ten minutes during the experimental period to minimize local circulatory changes in the pinna affecting the readings. The pretrial Sa_{O_2} varied from 95 to 101 per cent with a mean of 99 per cent.

Two series of trials were performed on each patient. In one series, apnea was produced for one minute both with and without endotracheal suction.† In a second series the patient's lungs were hyperventilated with oxygen for 15 seconds ‡ followed by one and two minute periods of apnea. Also, in this latter series a trial of one minute of apnea with endotracheal suction was performed.

Each patient was subjected to all the trials but the sequence was varied. If breathing or bucking occurred, the results were disregarded. Trials were performed in 10 patients with the pleura intact, repeated in four with the pleura open, and in one case performed only with the pleura open. Following each trial routine ventilation with nitrous oxide and oxygen was resumed and at least four minutes allowed to elapse before the next trial was begun.

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* Analyzer designed by A. C. Young, University of Washington.

† Suction was performed for 20 seconds during apnea with a catheter passed through the side arm of a Rovenstine connector; a negative flow of 13 liters per minute was used.

‡ Hyperventilation consisted of 10 ventilatory cycles with oxygen at a tidal volume of 500-600 cc. in the 15 seconds prior to apnea. Preceding this the flow of nitrous oxide was discontinued, the rebreathing bag emptied, and the system flushed with oxygen prior to each ventilatory cycle.

TABLE 1

INDIVIDUAL AND MEAN CHANGES IN ARTERIAL OXYGEN SATURATION (PERCENTAGE Sa_{O_2})

Patient	Pleura Closed					Pleura Open			
	No Prior Hyperventilation		Hyperventilation O_2 15 Sec.			No Prior Hyperventilation		Hyperventilation O_2 15 Sec.	
	Apnea 1 Min.	Apnea 1 Min. + Suction	Apnea 1 Min.	Apnea 2 Min.	Apnea 1 Min. + Suction	Apnea 1 Min.	Apnea 1 Min. + Suction	Apnea 1 Min.	Apnea 1 Min. + Suction
1	−12	−12	+2	−6	+3				
2	− 1	0	+1	0	+2	− 5	− 8	+1	+2
3	−11	−13	+2	−1	+1	−15	−15	+1	0
4	− 8	−22	+4	+1	+1				
5	− 6	− 5	+2	−1	0				
6	− 5	− 6	+2	0	+1				
7	−13	−11	+2	0	−4				
8	− 2	− 6	+2	0	+1	−10	− 2	+1	+1
9	− 4	− 3	+1	+1	+2				
10	− 9	−13	+1	+2	+2	−28	−31	+3	+1
11						−10	−31	+3	−1
Sa_{O_2} Mean Change	− 8	− 9	+2	0	+1	−14	−17	+2	0
Standard Error	±1.3	±1.9	±0.25	±0.66	±0.40	±3.5	±5.4	±0.45	±0.54
"*P*" of Mean*	<0.01	<0.01	<0.01		<0.05	<0.01	<0.05	<0.01	

* Values obtained from the standard "t" test of the mean and Fisher's *Statistical Tables*.[21]

RESULTS

The individual and mean changes in Sa_{O_2} are presented in the table along with the standard errors and "P" values for differences of the mean from zero. With the pleura closed, one minute of apnea during nitrous oxide-oxygen anesthesia resulted in a mean decrease of 8 per cent Sa_{O_2}, whereas during one minute of apnea following hyperventilation with oxygen for 15 seconds, there was a mean rise of 2 per cent Sa_{O_2} (fig. 1). Apnea for one minute with the pleura open caused a mean fall of 15 per cent Sa_{O_2} and again hyperventilation with oxygen for 15 seconds resulted in a rise of 2 per cent Sa_{O_2} during subsequent apnea. Endotracheal suction during apnea did not significantly alter changes in Sa_{O_2} ($P > 0.5$). The differences in mean values between the nonhyperventilated and hyperventilated groups of trials were all statistically significant ($P < 0.01$ with the intact pleura, $P < 0.05$ with the open pleura). The parametric metod of Link and Wallace[11] was used to analyze these differences in mean values.

Arterial oxygen desaturation to or below 93 per cent Sa_{O_2} was observed in 25 of 30 trials in which one minute of apnea was not preceded by hyperventilation with oxygen. On the other hand, in every trial with prior hyperventilation, the Sa_{O_2} was maintained above 95 per cent. Although patients differed in their responses, the individual patient showed consistent changes in Sa_{O_2} with the various procedures. A typical pattern of the desaturation curve during apnea obtained by recording oximeter readings at five-second intervals is illustrated in figure 2.

DISCUSSION

Among the studies correlating duration of apnea with arterial oxygen desaturation, that of Boba and co-workers[10] is most closely related to our experiments. Boba studied 15

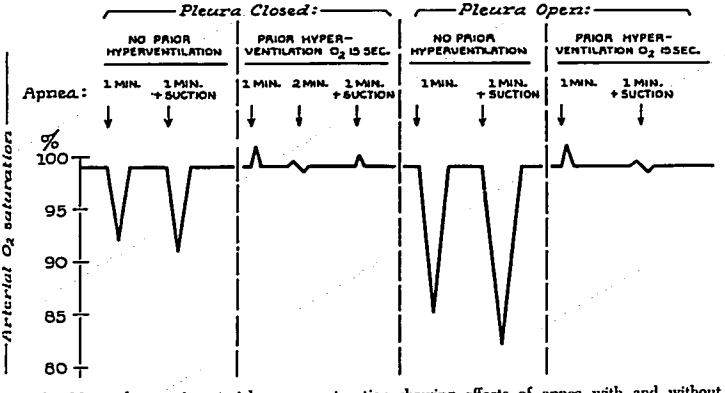

FIG. 1. Mean changes in arterial oxygen saturation showing effects of apnea with and without prior hyperventilation with oxygen. Influence of endotracheal suction during apnea and effect of opening the pleura are also indicated.

normal patients adequately ventilated with nitrous oxide-oxygen (6:3 liters per minute) who were subjected to one minute periods of apnea with and without endotracheal suction. There resulted an average fall of 15 per cent Sa_{O_2} as determined with intermittent arterial Van Slyke analyses. Although the effect of prior hyperventilation with oxygen was not studied, when these patients were exposed to insufflation of oxygen (4 liters per minute) through a catheter during apnea, the average decrease was less than 5 per cent Sa_{O_2}. Our study confirms their observation that endotracheal suction during apnea does not influence the degree of arterial desaturation.

The significance of acute arterial desaturation below the range of 90 to 93 per cent can be appreciated from an examination of the oxyhemoglobin dissociation curve. A 35 mm. of mercury decrease in arterial P_{O_2} (from 110 to 75 mm.) is reflected in a decrease of Sa_{O_2} from 99 to 94 per cent, yet tissue oxygenation apparently remains normal.[16] Below an Sa_{O_2} of 93 per cent the dissociation curve steepens, and a further reduction of 35 mm. of mercury arterial P_{O_2} results in an Sa_{O_2} of about 75 per cent with resultant tissue hypoxia[17] and profound cardiovascular changes.[18-21] The first cardiovascular response to hypoxemia, consisting of small but significant increases in heart rate, is seen in conscious normal men when the Sa_{O_2} rapidly decreases to 93 per cent.[22, 23] Thus, we as well as others[10] consider an Sa_{O_2} of 90 to 93 per cent to represent a critical range below which severe arterial hypoxemia may rapidly occur.

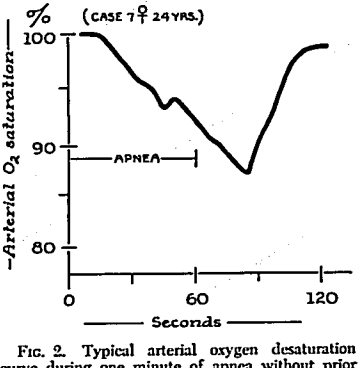

FIG. 2. Typical arterial oxygen desaturation curve during one minute of apnea without prior hyperventilation with oxygen.

Summary and Conclusions

Data obtained by ear oximetry in 11 patients anesthetized with nitrous oxide and oxygen (25 per cent oxygen) show that one minute of apnea resulted in arterial oxygen desaturation to or below 93 per cent in three-fourths of the trials. Hyperventilation with oxygen for 15 seconds prior to apnea caused arterial oxygen saturation to remain above 95 per cent in every patient during two minutes of apnea with the pleura intact, and one minute of apnea with an open pleura.

Endotracheal suction during apnea did not significantly affect changes in arterial oxygen saturation. Desaturation was greater when the patient was subjected to apnea with the pleura open.

Because of the slope of the oxyhemoglobin dissociation curve, severe arterial hypoxemia can rapidly occur when the arterial oxygen saturation decreases below the range of 90 to 93 per cent. To prevent hypoxemia, the data suggest that during nitrous oxide-oxygen anesthesia, a 15 second interval of hyperventilation with oxygen should precede all periods of deliberate apnea.

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FRACTURE REDUCTION A mixture of meperidine and levallorphan in a ratio of 80 to 1 given intravenously was used in the reduction of fractures and dislocations in 39 cases with only 3 failures. The dose for adults is 100 mg. of meperidine plus 1.25 mg. of levallorphan and one-half to three-quarters of this in children. Ten minutes after slow intravenous administration the patient is ready for manipulation. Patients are told they will not be asleep, but will not feel any pain. The effects wear off by the time the post reduction film is ready. The need for general anesthesia is much reduced. The drugs come already mixed as "Pethilorfan." (*Pearson, J. R.: Reduction of Fractures and Dislocations Without a General Anesthetic, Brit. Med. J. 1: 706 (Mar. 5) 1960.*)

FAMILIAL ANESTHETIC DEATHS A man aged 21 was given meperidine 100 mg. and atropine 0.6 mg. prior to anesthesia consisting of thiopental, nitrous oxide, oxygen and halothane. Within 20 minutes he was pale, cyanosed with dry skin, hypotensive, and with tachycardia. Anesthesia was stopped, the operation concluded within 30 minutes. The patient remained deeply unconscious, his skin hot and sweaty. He was transfused with cold blood. Recovery ensued over the next 1½ hours, and his subsequent course was uneventful. Inquiry revealed that of the 24 relatives of the patient given general anesthesia, 10 had died. In only one case was the condition necessitating the operation likely to have caused death on its own accord. The above patient is the only one who has been affected and survived. Study of the family tree suggests that the factor causing the reaction is inherited as a dominant gene. The same pattern appears to have been followed in all patients—hyperpyrexia occurring posoperatively followed by convulsions and death. (*Denborough, M. A., and Lovell, R. R. H.: Letter to the Editor: Anaesthetic Deaths in a Family, Lancet 2: 45 (July 2) 1960.*)

SUCCINYLCHOLINE PAIN A higher incidence of muscle pain after succinylcholine occurred in patients undergoing minor procedures and in out-patients and a much lower incidence in patients older than 60 years of age. The use of fresh solutions of succinylcholine bromide and the use of atropine and neostigmine after the administration of succinylcholine did not reduce the incidence of muscle pain. The prior administration of gallamine in doses of 20 mgm. lowered the incidence and severity of muscle pains, but tended to raise the dosage of succinylcholine necessary to produce relaxation; and the resumption of adequate ventilation after such short procedures as bronchoscopy was apt to be delayed, sometimes making the use of edrophonium or neostigmine necessary. (*Foster, C. A.: Muscle Pains that Follow Administration of Suxamethonium, Brit. Med. J. 2: 25 (July 2) 1960.*)