THE CHANGES IN BLOOD GASES ASSOCIATED WITH VARIOUS METHODS OF INDUCTION FOR ENDOTRACHEAL ANESTHESIA

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WITHIN the past few years many new drugs and techniques have been added to the armamentarium of the anesthesiologist. Among those that have become increasingly popular are the rapid acting barbiturates and the curariform drugs. The combination of these drugs has permitted the development of a technique of so-called rapid induction and endotracheal intubation.

The use of this technique has been especially desirous in a busy operating room. The time required to prepare a patient for operation under endotracheal anesthesia can be reduced from about thirty minutes to five or ten minutes. It has been thought, however, that although rapid induction and intubation may be expedient, it may also sufficiently disrupt normal physiological processes so as to make it an undesirable technique.

Some anesthesiologists and surgeons have been reluctant to employ the rapid intravenous administration of drugs to facilitate induction and endotracheal intubation. This hesitancy has been justified because even though numerous clinical reports of the successful use of this technique have appeared, very few objective observations have been made.

The purpose of this investigation has been to collect objective information concerning changes in arterial blood gases during the use of a variety of types of induction before endotracheal intubation. Thus, it has been possible to determine which technique is least disturbing to the physiology of respiration, circulation and the transportation of gases.

It has also been observed that the inhalation of 100 per cent oxygen while hyperventilating before induction will effectively minimize any subsequent disturbances to the respiratory gases and hydrogen ion concentration.

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MATERIAL

The subjects in this study were 50 patients undergoing various operations which required endotracheal anesthesia. No special effort was made to avoid patients who had disease entities other than those for which operation was performed. An attempt was made, however, to avoid patients who from the anatomical structure of the head and neck, would present difficult subjects on whom to perform orotracheal intubation. In patients who had short, thick necks and limited extension, prominent upper teeth, receding mandible, edema of the floor of the mouth and tongue, or who had diseases of the larynx, laryngeal exposure and intubation would be an extremely difficult procedure.

The ages of the patients ranged from 21 to 82 years.

METHODS

All of these patients received preanesthetic medication appropriate to their age group and the anticipated procedure.

On arrival of the patient in the operating room, an infusion of 5 per cent glucose in water was begun. A blood pressure cuff and stethoscope were applied to the patient's other arm and an initial pulse rate and blood pressure recorded. Readings of the blood pressure were obtained for this study from the time of induction of anesthesia until ten minutes after the completion of intubation, and were taken as often as was feasible.

An arterial puncture was performed on each patient, using a 20 gauge stilet needle in the brachial artery at the antecubital space. Five arterial blood samples were drawn: (1) after sedation and before the administration of oxygen, (2) at the end of a three minute period of inhalation of 100 per cent oxygen, (3) at the time of intubation, (4) two minutes after intubation and (5) ten minutes after intubation. In one group of 19 patients, control bloods were obtained the day before, or several days after, operation.

Three techniques of induction and endotracheal intubation were used: (1) The rapid administration of pentothal® sodium and a curariform drug before endotracheal intubation was used on a total of 41 patients. All of these patients inhaled 100 per cent oxygen for a three minute period before the induction, and 22 patients were asked to hyperventilate during the inhalation of 100 per cent oxygen. (2) The second method was the slow induction using cyclopropane and ether until the patient was sufficiently relaxed to permit an atraumatic intubation. There were 6 patients in this group. (3) A topical anesthetic agent was applied to the pharynx, larynx and trachea as the only anesthesia before intubation. There were 3 patients in this group, 2 of whom inhaled 100 per cent oxygen before intubation.

Each sample of arterial blood was analyzed for oxygen content and capacity and carbon dioxide content using the Van Slyke technique.

The hydrogen ion concentration of each sample of blood was determined by the glass electrode technique. The arterial oxygen saturation was calculated and plasma carbon dioxide determined according to Van Slyke (1).

OBSERVATIONS AND RESULTS

Rapid Induction with Oxygen Inhalation

This group of patients was asked to breathe 100 per cent oxygen for three minutes before they were put to sleep. In this series of patients 5 received tri-(diethylaminoethoxy) benzene triethyliodide (flaxedil®), 9 received succinylcholine chloride (anectine®),|| 5 received decamethonium bromide (syncurine®) || as a muscle relaxant. Enough sodium pentothal was administered to produce sleep and then a curarizing dose of muscle relaxant was given. When the desired effect was obtained, up to 500 mg. of pentothal was given rapidly. As soon as apnea was present the endotracheal intubation was accomplished and the endotracheal tube was connected directly to a circle filter gas machine. Nitrous oxide and oxygen were administered in a semi-closed system with carbon dioxide absorption. Respirations were supported or controlled as necessary for a period of time which depended upon the curariform drug used.

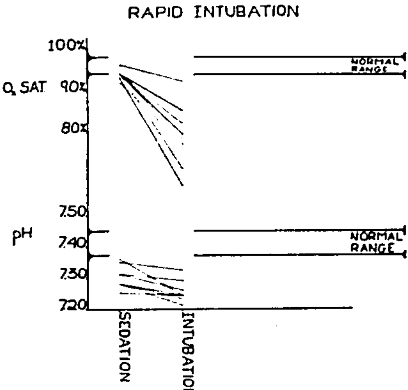

FIG. 1.

It was in this series of patients that control bloods were taken before sedation. In a considerable proportion of the patients the arterial oxygen saturation fell below normal after sedation (table 1, fig. 2).

|| The curariform drugs used in this study were supplied by Burroughs Wellcome & Co. (U.S.A.) Inc., Tuckahoe 7, N. Y.

TABLE 1

CHEMICAL CHANGES IN ARTERIAL BLOOD WITH INDUCTION OF ANESTHESIA

Rapid Induction with Oxygen

Time of Sampling	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH
	Case 1				Case 2				Case 3				Case 4				Case 5			
Sedation	88	40	50	7.38	97	53	47	7.42	90	50	37	7.50	96	52	59	7.32	94	52	45	7.43
After O_2	100	50	50	7.38	100	50	45	7.42	98	50	37	7.48	100	52	58	7.32				7.42
Intubation	100	54	56	7.30	95	52	51	7.39	100	53	46	7.41	100	56	66	7.28				
2 min after	100	55	60	7.33				7.40	100	50	45	7.42					100	56	58	7.35
10 min. after	100	53	52	7.38	100	54	50	7.40				7.46					100	54	53	7.38
Control	94	49	43	7.43	97	49	37	7.48	90	47	36	7.48	92	49	42	7.44				
	Case 6				Case 7				Case 8				Case 9				Case 10			
Sedation	92	45	45	7.39	90	48	53	7.35	93	49	45	7.42	93	41	39	7.43	93	44	41	7.42
After O_2	95	42	41	7.40	100	48	52	7.35	98	49	47	7.41	94	41	39	7.43	100	42	40	7.42
Intubation	100	45	49	7.35	100	52	60	7.30	95	52	51	7.39	100	45	45	7.38	94	47	50	7.36
2 min. after	100	46	53	7.33	100	45	45	7.38	95	54	63	7.31	100	40	38	7.42	90	49	57	7.32
10 min. after	100	49	54	7.34	100	45	48	7.38	92	55	64	7.31	100	41	38	7.42				
Control	93	46	39	7.47	93	49	41	7.48	94	49	44	7.45					90	46		
	Case 11				Case 12				Case 13				Case 14				Case 15			
Sedation					88	42	46	7.38	88	42	45	7.35	94	57	50	7.46	93	46	45	7.39
After O_2	98	44	42	7.42	100	46	42	7.38	100	43	50	7.32	100	58	51	7.46				7.37
Intubation	96	46	46	7.39	98	49	58	7.31	100	48	53	7.30	100	61	60	7.42	95	49	53	7.35
2 min. after	100	44	43	7.40	100	49	57	7.32	94	49	57	7.32	100	61	59	7.41				7.38
10 min. after	100	45	45	7.40	99	45	39	7.45	96	47	57	7.29	100	59	56	7.42	100	45	44	7.39
Control	93	46	42	7.44	97	42	39	7.42	94	42	38	7.43	94	48	42	7.45	94	49	48	7.40
	Case 16				Case 17				Case 18				Case 19							
Sedation	87	47	45	7.42	89	44	41	7.43	87	49	49	7.39	89	50	42	7.48				
After O_2	99	49	47	7.41	96	45	45	7.38	98	51	57	7.33	98	49	42	7.47				
Intubation	87	51	50	7.40	69	51	59	7.31	100	54	65	7.28	100	50	50	7.40				
2 min. after	93	53	60	7.34				7.29	98	53	64	7.29	100	52	48	7.42				
10 min. after	98	52	54	7.36				7.35	99	54	65	7.29	100	52	49	7.42				
Control	95	50	45	7.45					92	45	43	7.42								

With the administration of 100 per cent oxygen for three minutes the arterial oxygen saturation rose and remained at or near 100 per cent for the remainder of the procedure (fig. 2). The one exception was in a patient in whom the intubation was difficult, the arterial oxygen saturation fell to 69 per cent (indicated by *a* in fig. 2).

The carbon dioxide combining power in volumes per cent and the plasma carbon dioxide rose significantly above the control level after sedation had been produced (table 1). Sometimes both values were elevated further even during the inhalation of 100 per cent oxygen. The greatest rise occurred at the time of intubation. The carbon dioxide increased in one patient to the extent of 7.3 volumes per cent

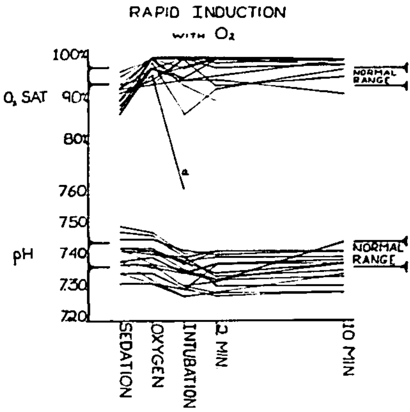

FIG. 2.

but the majority of patients in this group showed a rise of no more than 3 to 5 volumes per cent (table 1).

The hydrogen ion concentration in this series of patients had a definite pattern; it decreased steadily during the procedure, reaching its lowest value at the time of, or two minutes after, intubation and gradually returned to normal by the end of ten minutes (fig. 2).

Rapid Induction with Oxygen Inhalations and Hyperventilation

In this series of patients, the technique of induction and intubation was the same as that previously described but, in addition, this group of patients was requested to breathe deeply during the inhalation of 100 per cent oxygen. As in the previous group, the oxygen saturation of the arterial blood rose to 100 per cent and remained there for ten minutes following intubation except for 3 patients in whom intubation was exceptionally difficult (fig. 3).

The carbon dioxide content and plasma carbon dioxide had either remained the same or fallen slightly in 15 patients of this series, after three minutes of oxygen inhalation. At the time of intubation and during the period of apnea, the carbon dioxide levels and plasma carbon dioxide rose above the preinduction levels. After intubation the carbon dioxide content showed a tendency to level off at the preinduction value (table 2).

In the patients in this group the pattern of the hydrogen ion concentration was noticeably different from that of the previous series.

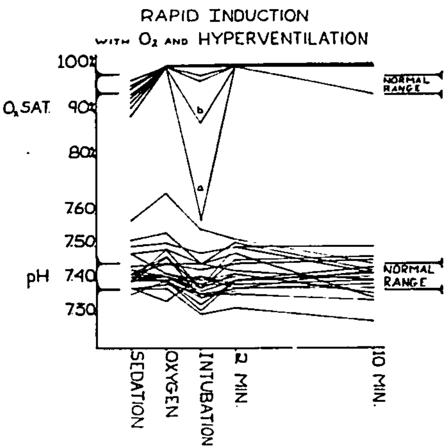

Fig. 3.

It will be observed from figure 3 that the hydrogen ion concentration showed a rise following the hyperventilation with 100 per cent oxygen. In only 2 patients was there an elevation above the normal pH of 7.45. At the time of intubation the hydrogen ion concentration decreased, but in only 7 of 20 patients did it drop below the normal level. In the majority of patients, the hydrogen ion concentration was well within the normal range within two minutes after intubation.

Slow Induction with Cyclopropane and Ether

In this series of 6 patients, anesthesia was produced by using cyclopropane in a closed circle carbon dioxide absorption system with an amount of ether up to 30 cc. Intubation was not attempted until the patient was sufficiently relaxed to permit atraumatic intubation.

TABLE 2

CHEMICAL CHANGES IN ARTERIAL BLOOD WITH INDUCTION OF ANESTHESIA

Rapid Induction with Oxygen and Hyperventilation

Time of Sampling	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH
	Case 20				Case 21				Case 22				Case 23				Case 24			
Sedation	97	49	42	7.44	94	51	44	7.42	95	46	44	7.41	95	47	36	7.48	94	44	32	7.50
After O_2	100	47	39	7.45	100	50	42	7.45	100	43	43	7.41	100	45	34	7.49	100	42	30	7.51
Intubation	100	49	43	7.43	100	51	42	7.45	100	48	50	7.37	98	50	50	7.38	100	47	36	7.48
2 min. after	100	47	40	7.45	100	52	44	7.43	100	42	43	7.40	100	47	44	7.42	100	41	32	7.50
10 min. after	100	44	36	7.40				7.40				7.44					100	41	31	7.50
	Case 25				Case 26				Case 27				Case 28				Case 29			
Sedation	96	47	45	7.40	94	47	52	7.36	97	46	45	7.40	89	48	49	7.38	92	50	46	7.40
After O_2	100	46	45	7.40	100	44	44	7.40	100	39	30	7.49	100	48	49	7.38	100	46	36	7.47
Intubation	100	50	55	7.35	98	52	58	7.33	100	45	38	7.45	100	53	63	7.30	85	53	54	7.35
2 min. after	100	49	50	7.38	100	45	45	7.40	100	43	33	7.48	100	52	59	7.32	100	47	46	7.37
10 min. after	100	49	49	7.38	100	44	43	7.41	100	50	55	7.35	100	56	67	7.28				
	Case 30				Case 31				Case 32				Case 33				Case 34			
Sedation	95	43	41	7.40	96	55	48	7.43	91	45	44	7.38					93	42	38	7.42
After O_2	100	41	39	7.41	100	52	41	7.47	100	45	43	7.39	100	44	43	7.38	100	43	40	7.41
Intubation	100	46	43	7.39	97	57	52	7.38	100	47	47	7.36	100	52	52	7.34	100	44	43	7.38
2 min. after	100	42	39	7.41	100	52	42	7.46	100	46	46	7.36	100	43	40	7.41	100	36	27	7.50
10 min. after		44	41	7.41	100	52	41	7.47	100	47	48	7.34	100	44	42	7.39	100	43	39	7.42
	Case 35				Case 36				Case 37				Case 38				Case 39			
Sedation	91	52	47	7.40	94	44	35	7.48	93	44	31	7.52	91	49	30	7.58	92	47	42	7.43
After O_2	100	50	46	7.40	100	46	43	7.42	100	45	30	7.54	100	45	23	7.66	100	47	43	7.41
Intubation	100	55	62	7.31	100	49	47	7.40	66	53	43	7.45	100	51	34	7.55	100	51	50	7.36
2 min. after	100	50	47	7.40				7.42	100	45	32	7.51	100	51	36	7.52	100	50	47	7.38
10 min. after	100	48	44	7.42	100	50	54	7.36	94	46	41	7.43	100	55	46	7.45	100	46	41	7.43
	Case 40				Case 41															
Sedation	95	46	42	7.41	97	43	40	7.40												
After O_2	100	48	44	7.40	100	38	35	7.42												
Intubation	100	49	44	7.41	100	43	41	7.39												
2 min. after	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]												
10 min. after	100	48	46	7.39	100	43	40	7.40												

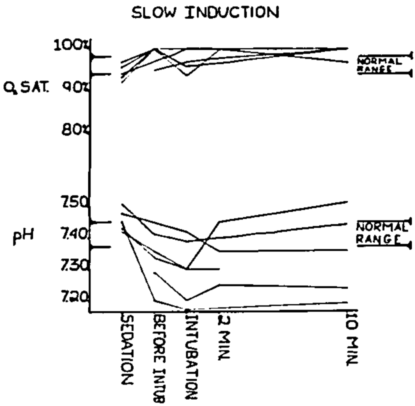

FIG. 4.

The oxygen saturation of the arterial blood was consistently elevated above the preinduction level and in the majority of cases reached 100 per cent saturation, showing only a slight fall at the time of intubation. In only one patient the oxygen saturation at the time of intubation fell below the preinduction value (fig. 4).

TABLE 3

CHEMICAL CHANGES IN ARTERIAL BLOOD WITH INDUCTION OF ANESTHESIA

Slow Induction

Time of Sampling	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH
	Case 42				Case 43				Case 44			
Sedation	93	47	39	7.45	96	52	48	7.42				
Before Intub.	100	58	74	7.20	100	52	55	7.36	95	48	59	7.29
Intubation	96	57	76	7.17	94	58	67	7.30	97	50	61	7.28
2 min. after					100	48	43	7.45	98	50	65	7.25
10 min. after	100	56	75	7.19	100	48	38	7.51	100	50	68	7.24
	Case 45				Case 46				Case 47			
Sedation	92	49	38	7.51	94	48	40	7.48	97	40	40	7.43
Before Intub.	100	54	52	7.41					100	44	52	7.34
Intubation	100	54	55	7.39	100	51	47	7.42	94	50	60	7.30
2 min. after	100	46	42	7.44	100	52	55	7.36	100	48	59	7.30
10 min. after					97	53	57	7.36				

TABLE 4

CHEMICAL CHANGES IN ARTERIAL BLOOD WITH INDUCTION OF ANESTHESIA

Topical Anesthesia

Time of Sampling	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH	Art. O_2 Sat. %	Tot. CO_2 Vol. %	pCO_2	pH
	Case 48				Case 49				Case 50			
Sedation	90	46	42	7.43	91	49	44	7.44	92	42	37	7.43
After O_2									100	43	42	7.41
Intubation	81	49	49	7.38	100	49	44	7.42	100	47	52	7.36
2 min. after				7.37	100	51	50	7.40	100	47	57	7.32
10 min. after	100	49	48	7.39	100	51	51	7.39	100	45	55	7.32
Control	90	48	41	7.44	91	48	44	7.44				

The carbon dioxide content and plasma carbon dioxide showed a rapid and marked rise from the onset of general anesthesia until the time of intubation. Following the intubation there was a tendency for the carbon dioxide content and plasma carbon dioxide to fall, but in only 3 patients did it reach the preïnduction value. In these 3 cases the respirations were controlled from the time of intubation (table 3).

In this group of patients the greatest changes in values of hydrogen ion concentration were observed. The range was from normal to a

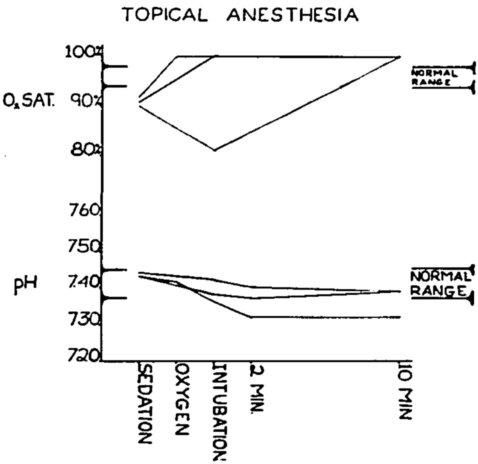

FIG. 5.

low in one case of pH 7.17. Here again the greatest fall occurred at the time of intubation with a tendency for a slight rise after intubation. In those patients in whom respirations were controlled following the intubation, the hydrogen ion concentration returned to normal (fig. 4).

Intubation After Topical Anesthesia to Pharynx, Larynx, and Trachea

There were only 3 patients in this group. In one patient intubation was performed after topical anesthetization of the pharynx, larynx, and trachea. The arterial oxygen saturation fell from the preintubation level of 90 per cent to a low of 80 per cent (fig. 5). In the other 2 patients, intubation was not performed until the patient had become hyperventilated while breathing 100 per cent oxygen and it was observed that the oxygen saturation of arterial blood remained at 100 per cent.

Along with the above observations it was also noted that there was only a slight change in the carbon dioxide content, plasma carbon dioxide and hydrogen ion concentration (table 4 and fig. 5).

In all of the cases the indirect technique of observing the patient's blood pressure was used. The pulse rate was obtained by palpation of the temporal artery. During the procedure the blood pressure and pulse rate were observed frequently, and with this technique no marked alteration in either was noted. Additional valuable information could have been obtained if a continuous recording of blood pressure, pulse rate and electrocardiogram had been available.

DISCUSSION

The changes in patient's arterial oxygen saturation, carbon dioxide content, plasma carbon dioxide and hydrogen ion concentration have been observed during various types of induction and endotracheal intubation.

It becomes apparent that sedation alone reduces ventilation to some extent, the plasma carbon dioxide increases above the control level and the arterial oxygen saturation falls below normal (table 1 and 2).

Stone *et al.* (2) have shown that when rapid induction is accomplished by the use of decamethonium bromide and pentothal sodium, during the period of apnea accompanying the intubation the arterial oxygen saturation routinely drops below normal and the hydrogen ion concentration falls markedly (fig. 1).

It has always been our policy to administer 100 per cent oxygen to the patient before apnea is produced and endotracheal intubation is performed. This obviates the drop in arterial oxygen saturation with any type of rapid induction and intubation (figs. 2 and 3). The arterial oxygen saturation is maintained at a normal level except in those patients who have long periods of apnea as a result of difficult intubations (*a* in fig. 2; *a* and *b* in fig. 3).

It was observed that even when the patients breathed 100 per cent oxygen before induction, plasma carbon dioxide began to rise and the hydrogen ion concentration began to fall, and that these two factors reached their greatest change at the time of intubation. The induction technique was then modified further. The patient was asked to hyperventilate during the breathing of 100 per cent oxygen. With this technique, the plasma carbon dioxide then remained stable or fell slightly. It was also revealed that there was little alteration in the hydrogen ion concentration, and the changes that did occur were within the normal range.

It can be seen from figure 4 that, with the slow induction and intubation technique which has been used for years and which is still considered by many to be the safest method to employ, the greatest variations were in the carbon dioxide content, plasma carbon dioxide and hydrogen ion concentration. These changes occurred in spite of what was thought to be adequate respiratory exchange, using assisted respirations when indicated.

The use of topical anesthesia applied to the pharynx, larynx and trachea without any additional narcosis before intubation is reserved for the old and poor risk patient. From our study, the changes in plasma carbon dioxide and hydrogen ion concentration are minimal as compared with the changes associated with the above described procedures. It is also apparent that unless the patient inhales 100 per cent oxygen there may be considerable decrease in arterial blood oxygen saturation during intubation (fig. 5).

This technique of rapid induction and intubation should be avoided in patients with marked aortic stenosis, shock, and in those with recent myocardial infarction. The one common factor present in these conditions is a fixed cardiac output and it has frequently been observed that the rapid intravenous administration of sodium pentothal may result in a marked and persistent hypotension. It has been observed that patients with advanced cardiac disease other than aortic stenosis tolerate this type of induction well (3).

The following conclusions may be drawn from these observations. The use of rapid induction and intubation causes much less alteration in the arterial blood gases than does the older accepted technique of inhalation anesthesia. This, however, is true only if the patient is requested to hyperventilate while breathing 100 per cent oxygen immediately preceding the induction and intubation. The aged or extremely poor risk patient should be intubated while awake, after the pharynx, larynx and trachea have been anesthetized adequately and the patient has become hyperventilated while inhaling 100 per cent oxygen just before the endotracheal intubation.

Future studies of this nature are indicated and should include a method of recording the oxygen content and the hydrogen ion concentration of arterial blood throughout the entire anesthetic and operative period. If these alterations can occur early during anesthesia with the patient under close observation and before operation has begun, what changes occur during and at the finish of a long operative procedure?

SUMMARY

The effects of various techniques of induction and endotracheal intubation upon arterial blood oxygen, plasma carbon dioxide and hydrogen ion concentration have been observed on 50 patients.

The greatest alteration in the gases of arterial blood occurred during a slow induction.

There was deviation from the normal values of the arterial blood gases with rapid induction and intubation. The changes that occur with this technique can be reduced to a minimum which will remain within the normal range if the patient is permitted to hyperventilate while inhaling 100 per cent oxygen before the induction and intubation.

From these observations, it would appear that the so-called rapid technique could be used for all patients who are anatomically suitable and who must receive endotracheal anesthesia.

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