

## THE VALUE OF OXYGENATION PRIOR TO INDUCED APNEA

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APNEIC techniques during induction of anesthetic procedures have become commonplace. They are satisfactory if "everything goes well," but there are many instances in which the operator finds distorted laryngeal anatomy; there are times when "the light goes out"; and there are occasions when an unskilled anesthetist attempts to perform endotracheal intubation and requires more time than needed by an experienced individual. If any of these situations occur the apneic patient may be exposed to hypoxia. Adoption of a routine which would protect the patient against prolonged hypoxia would be desirable.

Measurements pertaining to the benefits of administration of oxygen before production of apnea have been made by Lachman<sup>1</sup> and by Nahas.<sup>2</sup> The criteria used were essentially the figures obtained for degree of saturation of blood hemoglobin with oxygen. It occurred to us that the electroencephalogram (EEG) might provide objective information concerning cerebral activity which could indicate impending danger from hypoxia. Although the occurrence of an isoelectric (flat) EEG tracing does not mean that irreversible change has occurred,<sup>3</sup> it does mean that cerebral electrical activity has become dangerously diminished.<sup>4</sup> With the thought in mind that a flat EEG could be used to indicate diminution of cerebral activity due to hypoxia, experiments were undertaken to determine the comparative effects of ventilation (1) with air, (2) with 100 per cent oxygen for a short period, and (3) with 100 per cent oxygen for 30 minutes before apnea. The short period was arbitrarily taken as 30 seconds for it seemed feasible to use this much time in a busy operating room where apneic inductions are frequently used, whereas use of oxygen for one minute might seem unpractical to many persons.

Accepted for publication October 21, 1959. The authors are in the Division of Anesthesiology, University of Colorado Medical Center, Denver, Colorado.

### METHODS

Nineteen mongrel dogs were used. Each was anesthetized with 44 mg./kg. of sodium pentobarbital. Their tracheas were intubated with cuffed Portex tubes, and the cuffs were inflated. Intermittent positive pressure breathing was instituted using a Takaoka respirator. This instrument provides equal phases for inspiration and expiration with a low (not over 15 mm. Hg) inspiratory pressure, and a slight negative pressure on expiration. Frontal-occipital EEG leads were attached. Lead II of a direct-writing electrocardiograph (ECG) was used. Succinylcholine, 1.5 mg./kg., was injected intravenously to produce complete muscular relaxation as well as apnea. During and following the muscle fasciculations due to the succinylcholine, a tachycardia appeared regularly. When the heart rate had returned to normal the respirator was suddenly turned off, and the EEG and ECG were observed until the EEG record was flat for 15 seconds. Rapid pulmonary ventilation with either air or oxygen was followed by a return of the EEG to normal. A second series of experiments using the same animals was identical to those already described except that after the tachycardia following administration of succinylcholine had subsided, the animals' lungs were ventilated for exactly 30 seconds with 100 per cent oxygen before stopping the respirator. A third series, still using the same animals, was identical with the second, except that the time of pulmonary ventilation with 100 per cent oxygen was 30 minutes instead of 30 seconds before stopping the respirator.

### RESULTS

Table 1 shows the average time required for the EEG tracing to become flat after cessation of pulmonary ventilation. After adequate ventilation using air the "safe" time was 4.45 minutes. After 30 seconds of ventilation with oxygen this "safe" period had nearly tripled, the average value being 12.39 minutes. Increasing the period of ventilation with oxygen

to 30 minutes afforded decreasing returns of extension of "safe" time, for this average was 16.5 minutes. As was to be expected, the tongues of the animals became progressively more blue as apnea continued. By the time the EEG tracing became isoelectric, cyanosis was marked in all animals. Carbon dioxide affected the EEG tracing before it became isoelectric in some of the animals whose lungs were ventilated for 30 minutes prior to apnea.

Cardiac activity became abnormal as hypoxia developed. Advanced hypoxia was usually accompanied by arrhythmias as well as by tachycardia. Elevated ST segments and exaggerated T waves generally corresponded to the degree of hypoxia. Typical EEG and ECG patterns are shown in figure 1.

Values obtained when the dogs breathed air were highly significantly different from those found when the dogs' lungs were ventilated with oxygen for either 30 seconds or

TABLE 1  
TIME IN MINUTES FOR EEG TO BECOME FLAT  
AFTER CESSATION OF VENTILATION

| Ventilation with:  | Air  | Oxygen for 30 Seconds | Oxygen for 30 Minutes |
|--------------------|------|-----------------------|-----------------------|
| Mean               | 4.45 | 12.39                 | 16.51                 |
| Standard deviation | 0.30 | 5.57                  | 8.28                  |
| Standard error     | 0.07 | 1.61                  | 2.21                  |

|   | Critical Ratio |                         |
|---|----------------|-------------------------|
| Air--30 seconds oxygen                  | 4.93           | Very highly significant |
| Air--30 minutes oxygen                  | 5.45           | Very highly significant |
| 30 seconds oxygen—<br>30 minutes oxygen | 1.50           | Not significant         |

30 minutes. Differences between the groups getting 30 seconds and 30 minutes of oxygen were not statistically significant.

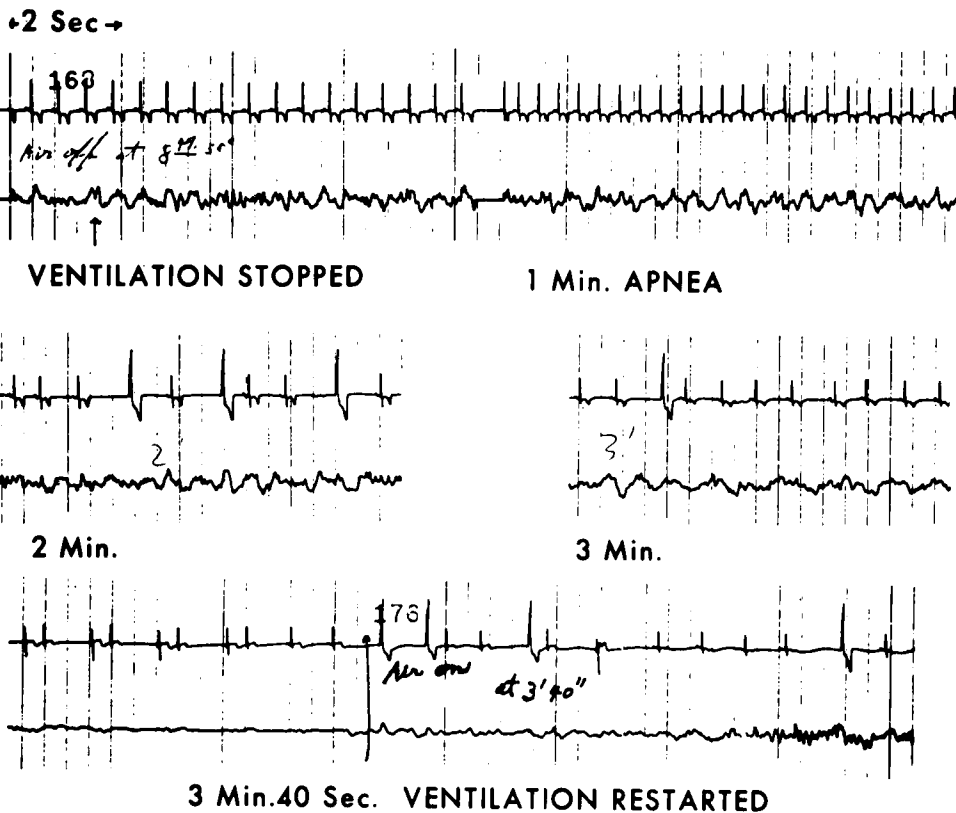


FIG. 1. ECG and EEG tracings during apnea and after beginning artificial respiration. Note (1) arrhythmias and (2) resumption of EEG activity on ventilation with air. Air was used for ventilation prior to apnea.

## DISCUSSION

The advantage of administration of oxygen before production of apnea undoubtedly lies chiefly in having a reservoir of oxygen available in the lungs. The time required for the EEG to become flat when blood flow to the head was clamped off in open-chest animals was 20 to 30 seconds when the animals were respired with either air or oxygen. Our significant differences were therefore not due to oxygen dissolved in the tissues or the blood.

We have made no measurement of the degree of denitrogenation afforded by 30 seconds or by 30 minutes of ventilation with oxygen. The data of Hamilton and Eastwood<sup>5</sup> indicate that the concentration of nitrogen in exhaled air after use of a nonbreathing system with oxygen for 30 seconds is less than 30 per cent. This would indicate that the oxygen concentration is in the neighborhood of 70 per cent at that time. Other methods used by Hamilton and Eastwood such as semiclosed and circle systems resulted in slightly slower denitrogenation. Our object was to find a practical method for enhancing safety, and the results with 30 seconds of administration of oxygen appear to be of considerable value even though appreciable amounts of nitrogen were undoubtedly left in the system.

Lachman, Long and Krumperman<sup>1</sup> made observations which bear directly on this same situation. They measured blood oxygen saturations in apneic patients who had been sedated with thiopental and given relaxing agents before endotracheal intubation. They observed that when patients inhaled pure oxygen for 3 minutes before this procedure the arterial blood saturation remained at nearly 100 per cent for 10 minutes in 37 of 41 patients. Intubation was difficult in the other 4 patients. They drew attention to the contrast between these results and those of Colon-Yordan, Mackrell and Stone<sup>6</sup> who had reported that oxygen saturation dropped routinely (4 to 26 per cent; average drop: 15 per cent) when the patients had been breathing only room air.

Dillon and Darsie<sup>7</sup> observed that patients breathing room air who were given meperidine and thiopental showed a reduction of

20 per cent in arterial oxygen content when compared with those who were allowed to breathe oxygen for 5 minutes before administration of these drugs. While their patients were not completely apneic, their experiments add weight to the value of giving oxygen before injecting drugs that depress respiration. Nahas<sup>2</sup> sedated dogs with thiopental, administered curare, artificially ventilated their lungs for 30 minutes with oxygen, and suddenly stopped the ventilation. He measured arterial oxygen saturation for the ensuing 90 seconds and found it to remain at 100 per cent in spite of complete apnea. Control animals whose lungs were ventilated with air showed oxygen saturation values ranging from 32 to 68 per cent at the end of 90 seconds of apnea.

Our observations reinforce those of others in emphasizing the enhanced safety by the administration of oxygen for a short period before production of apnea. Even for the experienced anesthesiologist, this extra "safe" time permits him to work deliberately, to minimize the possibility of trauma during tracheal intubation, and to avoid tragedy in unusual situations. It is the patient rather than the physician, who suffers the consequences of "taking a chance" that things will go well if oxygen is not used before production of apnea.

Principles emphasized by this investigation may well be considered at the termination as well as at the beginning of the period of anesthesia. Would not 30 seconds of active pulmonary ventilation with oxygen immediately before tracheal extubation be valuable in prevention of hypoxia due to laryngospasm or other untoward events which might occur at that time?

The reader should not conclude that 4 minutes of apnea with air or 12 minutes with oxygen are innocuous. Cardiac arrhythmias and aberrations of the electroencephalogram occurred regularly before these periods had elapsed. The term "safe" has been put in quotation marks throughout this report to indicate that the situation involved a serious borderline of safety. When hypoxia has progressed to the point of instrumental registration of difficulty in both circulatory (ECG) and central nervous (EEG) systems the sub-

ject is far from enjoying security. Any period of hypoxia should be kept as short as possible.

#### SUMMARY

Nineteen dogs were sedated with sodium pentobarbital and made apneic with succinylcholine. Their lungs were artificially ventilated for 20 minutes with air. Ventilation was suddenly stopped. An average period of 4.45 minutes elapsed before the EEG tracing became isoelectric. The experiments were repeated but 100 per cent oxygen was used for 30 seconds before stopping the respirator. The average time before a flat tracing appeared was 12.39 minutes. The experiments were done a third time, but artificial respiration was carried on for 30 minutes with 100 per cent oxygen before stopping the respirator. The EEG tracing became isoelectric in 16.51 minutes. Using a flat EEG tracing as a criterion for the occurrence of dangerous hypoxia, it appears that 30 seconds of pulmonary ventilation with oxygen rather than air before production of apnea affords materially greater safety for the subject. Increasing the period of oxygenation from 30 seconds to 30 minutes did not significantly alter the results. Cardiac

irregularities were consistently observed during periods of severe hypoxia.

Supported in part by United States Public Health Service Grant H-4077.

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**MISTAKEN IDENTITY** Mixup of patients by the nursing service resulted in the initiation of an operation for a cataract on a patient scheduled for a cholelithotomy. This resulted in a malpractice claim which was settled out of court for \$3,000. There is a

need for constant watchfulness by all persons involved in any part of the complex and vital job of patient care in all parts of the hospital. (*Drumm, R. M.: Mistaken Identity is One Mistake No Hospital Can Afford, Mod. Hosp.* **93**: 78 (Aug.) 1959.)