ELECTROCARDIOGRAPHIC STUDIES DURING ENDOTRACHEAL INTUBATION: VII. EVIPAL® SODIUM INDUCTION

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THIS is the seventh in a series of studies examining electrocardiographic variations during induction of anesthesia and endotracheal intubation using various anesthetic agents. This report deals primarily with electrocardiographic observations associated with induction of anesthesia using Evipal® sodium in 119 patients.

The area most critically examined, the induction period, was arbitrarily defined for the purposes of this study to extend from the time the inducing agent was injected, to the end of a one minute observation period after the patient lost consciousness.

METHOD

The patients, all adults, received morphine and scopolamine or atropine as preanesthetic medication. After the patient was placed in the supine position on the operating table, electrocardiographic tracings of the first three leads were obtained using a direct writer. Subsequent tracings were all made on lead II: during induction, during endotracheal intubation, one minute after intubation, and five minutes after intubation. While the patient was in the operating room his heart action was continuously monitored on the electrocardioscope and tracings were made of all abnormalities.

In all cases in this series, no test dose of Evipal sodium was given, and the inducing dose was injected, as rapidly as possible through a 22 gauge needle, into the tubing of the intravenous set-up with the tubing clamped distally. The patient was usually unconscious between 15 and 45 seconds after the injection was begun. The patient was then ventilated with oxygen and observed for one minute to detect any possible laryngospasm or coughing.

In 80 cases a muscle relaxant was then given, and the patient was ventilated with oxygen until adequately relaxed. The cords were exposed, sprayed with 4 per cent cocaine, and the patient was intubated with the largest tube that could be comfortably accommodated.

In 16 cases at the end of the one-minute observation period, cyclopropane was added to the circle system. When a sufficient degree of

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relaxation occurred, the cords were exposed, sprayed with 2 cc. of 5 per cent Cyclaine®, and the patient was intubated with the largest tube that could be easily accommodated.

In 14 cases at the end of the one minute observation period, 2 cc. of 5 per cent Cyclaine were injected transtracheally through the cricothyroid membrane. Cyclopropane was then added to the circle system. When a sufficient degree of relaxation occurred, the cords were exposed and the patient intubated with the largest tube that could be comfortably adapted.

In 8 cases at the end of the one minute observation period, the patient was sufficiently relaxed, without the addition of more anesthetic agent, to permit exposure of the cords. The cords were then sprayed with 2 cc. of 5 per cent Cyclaine before intubation.

One patient was intubated while awake using topical anesthesia before Evipal sodium was given for induction.

The dosage of Evipal sodium required to produce anesthesia sufficient for intubation was 500–850 mg. For induction of anesthesia to a depth sufficient to allow a smooth transition to maintenance with an inhalation agent, such as cyclopropane, a dose of 250–500 mg. was usually adequate. A 2.5 per cent solution of Evipal sodium was found to be sufficiently concentrated to insure a rapid induction, yet dilute enough to permit flexible dosage control.

Either tubocurarine or succinylcholine was utilized as the muscle relaxant. The average dose of succinylcholine was 30 mg. in a 2 per cent solution. In many cases a dose as low as 10 mg. was sufficient to secure the desired relaxation. In only one case was it necessary to use as much as 50 mg. of succinylcholine. The average dosage of tubocurarine was 9 mg. in a 0.3 per cent solution.

Results

The most frequent change in the electrocardiographic tracings noted during induction with Evipal sodium was an increase in pulse rate of about twenty beats per minute. The greatest increase was 60 beats per minute. Some patients showed no increase, and in 4 a slowing in pulse rate was observed. In all cases the pulse rate slowed as the maintenance anesthetic drug supervened. In the entire series of 119 patients, no electrocardiographic alterations other than changes in pulse rates were noted during induction (table 1). (Some patients had electrocardiographic abnormalities present before induction; these showed no alterations in the specific abnormalities during induction.)

Before induction, 83 patients had pulse rates of 61 to 99 per minute, 24 had pulse rates over 99, and 18 had pulse rates of 60 or below. Ten patients had abnormal electrocardiograms on the control tracings immediately before induction of anesthesia. These were: auricular flutter-fibrillation; small to absent P waves; myocardial insufficiency; shifting pacemaker; sinus bradycardia; frequent ventricular premature con-

tractions in a case of diagnosed posterior wall myocardial infarction; bigeminal rhythm, premature auricular contractions, bradycardia; nonspecific ST and T wave changes and tachycardia; ST and T wave changes and tachycardia; and tachycardia.

Some of the more interesting sequences are described: In the case of the nonspecific ST and T wave changes along with tachycardia, no change occurred during induction or intubation, but for five minutes after intubation the abnormality was absent and a regular sinus rhythm was present.

In the patient with normal sinus rhythm and premature ventricular contractions the electrocardiogram showed no change during induction; but the premature ventricular contractions disappeared with intubation and nodal rhythm occurred five minutes later.

TABLE 1

CARDIAC CHANGES OBSERVED BEFORE, DURING, AND AFTER ENDOTRACHEAL INTUBATION FOLLOWING INDUCTION WITH EVIPAL ® SODIUM

Time	Increase* in Rate	Decrease* in Rate	No Change	Remarks
Induction	86	4	28	1 Not recorded
Intubation	58	31	23	6 Not recorded 1 Nodal rhythm
1 Min. after intubation	23	38	18	38 Not recorded 2 First degree heart block
5 Min. after intubation	12	85	16	2 Not recorded 1 First degree heart block 3 Nodal rhythm

* All changes in rates computed relative to last recorded rate.

In the patient with shifting pacemaker the electrocardiogram showed no change during induction, but nodal rhythm occurred with intubation and disappeared after 5 minutes.

In *none* of the 119 patients was coughing or laryngeal spasm observed during induction. In the one patient who was intubated while awake before Evipal sodium was used for induction, no abnormalities were detected by electrocardiography.

The changes observed at the moment of endotracheal intubation are interesting. There was the usual high incidence (52 per cent) of transient sinus tachycardia at the time of intubation. However, the only cardiac arrhythmia observed at the time of intubation was one case of nodal rhythm. This result contrasts sharply with the first series in this study in which induction of anesthesia was accomplished by various usual techniques, the majority of which consisted of inhalation anesthesia (1). In that series, it will be recalled, in addition to

the 43 cases of sinus tachycardia, there were 10 cases of premature ventricular contractions with bigeminal or trigeminal rhythm, 5 cases of nodal rhythm, 4 cases of sinus bradycardia, 4 cases of decrease in the voltage of the T wave, 3 cases of increase in the PR interval, 2 cases of sinus arrhythmia, 2 cases of ventricular tachycardia, and 1 case of auricular fibrillation: a total of 31 cardiac arrhythmias in a series of 109 cases.

Discussion

In some cases, Evipal sodium sufficed to permit intubation with ease, but it is felt that most patients require additional anesthesia with inhalation agents. As with all barbiturates, constant attention is required to insure proper ventilation with sufficient oxygen. Five per cent Cyclaine used as topical anesthesia to the glottis and larynx is recommended (2).

A muscle relaxant serving to diminish the amount of anesthetic agent involved was found desirable in most cases. Succinylcholine was preferred because of its rapid onset, brief action, and apparent lack of toxicity in the dosage used.

The tachycardia associated with intravenous barbiturates could be a direct effect of the drug on the myocardium, on the conduction system, or on the vagal mechanism. Oxygen-lack due to hypopnea seemed to be another plausible cause, but it was ruled out by adequately assisting the inspiratory phase of pulmonary ventilation. Certain studies have intimated that the effect is primarily vagal and that the tachycardia is selflimited and followed by bradycardia (3). It was seen to persist in our cases until the maintenance anesthetic agent had time to supplant the Evipal sodium.

The use of Evipal sodium with agents such as cyclopropane or ether for intubation caused no increase in number of electrocardiographic changes, and indeed showed some protective action when compared statistically with similar series wherein Evipal sodium was not used (1). With Evipal sodium there was an incidence of less than 1 per cent of cardiac arrhythmias on intubation, without Evipal sodium there was an incidence of 28 per cent. This protective action has been previously observed with Evipal sodium (4), and with other barbiturates (5). Recent studies (6) have suggested that the incidence of cardiac arrhythmias may be related more strictly to technical rather than pharmacological factors. For this reason our original work (1) is being repeated with emphasis on technical standardization, especially in regard to oxygenation.

Since there is some indication that this drug, like other barbiturates, may produce liver damage when given in large doses, it is recommended that its use be limited to induction of anesthesia (7). It has no apparent toxic effects in the dosage used, and is quite transient in its action.

This series strengthened the impression we formerly entertained that laryngeal spasm and coughing are not as likely to appear with Evipal sodium as with Thiopental® (7, 8).

There were no local reactions observed in any case (phlebitis or subcutaneous tissue irritation), reinforcing earlier indications that Evipal sodium is less irritating than Thiopental sodium (9).

Summary

The electrocardiograms of 119 patients were studied during induction with Evipal sodium and subsequent anesthetic procedures. The most frequent change found during induction with Evipal sodium was an increase in the pulse rate. Even in patients with previously abnormal hearts, no deleterious effects attributable to the Evipal sodium were seen. Some protective action by Evipal sodium against cardiac changes during endotracheal manipulations was deduced from statistical comparisons.

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