A TRANSISTORIZED ELECTROPHRENIC RESPIRATOR*

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Abstract—A transistorized, battery operated phrenic nerve stimulator is described. The stimulator supplies 40 pulses per second, of 2 msec duration. The pulses are given in groups, each group causes inspiration. During the interval between stimulation groups, spontaneous expiration occurs. The respiration rate can be regulated in the range of 5-40 inspiration/min, while the duration ratio of 40 per cent inspiration to 60 per cent expiration is kept constant in the whole range.

Implantable stainless steel electrodes on silastic rubber base were developed for chronic connection to the phrenic nerve.

The effectiveness of the electrophrenic respirator was demonstrated on dogs, during apnea caused by respiratory center depression with Pentothal.

The respirator can be used even on animal breathing spontaneously. In this case spontaneous respiration is synchronized to the artificially induced diaphragmatic contraction.

INTRODUCTION

Electrical stimulation of the phrenic nerve was first used as a means of artificial respiration by SARNOFF (1948). Several papers describing the effectiveness of the electrophrenic respirator were published by the same group, CHATFIELD and SARNOFF (1950), but the method was neglected. The method was not accepted into clinical practice because the stimulator was complicated, using a motor driven potentiometer for regulation of the respiratory cycle. No safe method for electrode connection to the nerve was available at that time, and thus the procedure became dangerous as nerve damage could result. Transistor techniques and development of new materials for implantation within the body made possible the building of a compact pulse generator that could be used as an effective and easily controlled electronic artificial respirator.

THE STIMULATOR

Electrical stimulation of the phrenic nerve by 40 pulses/sec., of 2 msec duration and at least 2 V produces an effective diaphragmatic contraction, and inspiration. Cessation of the phrenic stimulation is followed by diaphragm relaxation and passive expiration. In our respirator the inspirium is about 60 per cent of each respiratory cycle, leaving 40 per cent for passive release of the air from the lungs. This ratio was found to be optimal in our experiments on the dog and can be present to other values if required.

A block diagram of the electrophrenic respirator (E.P.R.) is given in Fig. 1, and a schematic diagram in Fig. 2. The stimulator consists of a blocking oscillator T1 serving as 2 msec 40 pps pulse generator. The output of the generator passes to the output circuit through a gate T2 that is controlled by a free running multivibrator.

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T3T4. In this multivibrator, a fixed ratio of on-and-off intervals is maintained in the whole range of respiration rates. The respiration rate is regulated by a single potentiometer. The amplitude of the output pulses is either constant or linear increasing during the inspirium phase. The output of the stimulator is applied to the thoracic or cervical phrenic nerve through stainless steel electrodes on a silastic rubber base (Fig. 3).

**PERFORMANCE**

Stimulation of the phrenic nerve by the stimulator results in a fast tetanic contraction of the diaphragm, we have not succeeded in achieving gradual contraction by the linear increasing mode, and the following results were obtained by constant amplitude (mode a) stimulation.

Figure 4 illustrates the effectiveness of the electrophrenic respirator on a dog with apnea resulting from central depression by 15 mg/kg of Thiopentone. Tidal volume during the electrophrenic respiration (E.P.R.) was about 30 per cent higher than during the spontaneous respiration. The effectiveness of E.P.R. can be demonstrated also during spontaneous respiration. Figure 5 shows that stimulation of the phrenic nerve can increase the tidal volume and also change the respiration rate.

Experiments performed on anaesthetized dogs show that respiration rate can be regulated from 40 to 120 per cent of the spontaneous rate. The regulation of respiration is not performed by depression of the respiratory center (CHATFIELD and SARNOFF, 1950), but by synchronization of the spontaneous respiration with the artificially induced diaphragmatic contraction. This can be proved by the limited range of synchronization and by analysis of the flow and volume curves during phrenic stimulation.

Figure 6 is a fast time based record of pressure, air flow and lung volume curves during E.P.R. It can be seen that the curve is a superposition of spontaneous respiration, and E.P.R. Four phases are recognized in each respiratory cycle: (1) Slow spontaneous inspiration, (2) Rapid, electrically induced inspiration, (3) Spontaneous expirium while diaphragm still contracts and (4) Passive expirium following the end of electrical stimulation.

The spontaneous inspiration of next cycle is locked to the end of electrical inspirium. This locking is the mechanism of the above described synchronization.