Electrothoracic Artificial Respiration*

JEWELL L. OSTERHOLM, M.D., THOMAS HOOKER, B.S.E.E., M.S.E.E.,† AND JACK PYNESON‡
Department of Surgery, Section of Neurological Surgery, Hahnemann Medical College, Philadelphia, Pennsylvania

Artificial respiration has become synonymous with the positive pressure respirator. These mechanical devices successfully ventilate patients with acute respiratory failure. However, when artificial ventilation is necessary for an extended time, alterations occur in the tracheobronchial tree and lung parenchyma; these include retained secretions, atelectasis, pneumonitis, and interstitial pulmonary edema. As obstructive or inflammatory lesions develop in the lungs there is an increase in the physiological dead space and the related ventilation requirements. Pontoppidan and co-workers report the ratio of dead space to tidal volume may become so large that no improvement in alveolar ventilation occurs with increased tidal volumes. They also found impaired oxygenation in all their patients.

Experience indicates that pulmonary complications contribute significantly to the mortality and morbidity of neurosurgical patients, and that these complications are neither minimized nor avoided by our present ventilation techniques. In our search for an improved ventilation technique we have assumed that, if the normal inspiratory muscles could be electrically driven, the chest cage elevated, and the lung uniformly expanded by negative pressure, some reduction in the high pulmonary complication rate might occur.

Little attention was given to electronic driving of the normal neuromuscular apparatus in the work of respiration until the reports by Sarnoff and co-workers in 1948. They demonstrated the feasibility of artificial diaphragmatic respiration by electrical phrenic nerve stimulation. Somewhat later Eisenberg, et al.,1 and Glenn, et al.,2 were successful in the inductive stimulation of phrenic nerves by pulsed radiofrequency waves. In 1966 we described briefly a new technique, based on work over a 3-year period, whereby a single subarachnoid electrode positioned at cord segment T-1 or T-2 and supplied with critical pulsed voltage and frequency can drive the entire respiratory muscular system (intercostal and accessory) in coordinated, useful respiratory movements.

Methods and Materials

To obtain comparable respiratory data for this study, 40 mongrel dogs were selected for similarity of size, weight, and pulmonary tidal volume. A 9-liter Collins spirometer was connected to a cuffed endotracheal tube, and when the system proved to be air tight, data were collected. A femoral artery catheter was led to a three-way stop watch and connected to a Stratham strain gauge or a blood gas syringe. External chest excursion was detected by a leather strap encircling the chest, with a second strain gauge completing the circle. Blood pressure, chest movement, electrocardiogram, and electromyograms were recorded on a six-channel Grass machine or Electronics for Medicine Recorder.

Intercostal nerves were exposed, and electrical activity was recorded during quiet respiration. During inspiration, the intercostal nerve recordings showed increasing voltage and frequency potentials which built to the peak of inspiration. Expiration is electrically silent and occurs by elastic recoil of the stretched chest elements.

A Hewlett-Packard 202A Function Generator was applied to intercostal nerves. Smooth-graded electrically-driven intercostal contractions could be obtained when both frequency (40–800 cps) and voltage (0–5 V) were increased during the stimulating cycle. The stimulating voltage envelope and frequency must both increase in a logarithmic curve to the apex of muscle contraction.

A stimulator built by one of us (TBH)

Received for publication October 14, 1966.
Revision received August 14, 1967.
† Systems engineer, General Electric NASA, Valley Forge, Pennsylvania.
‡ Bioelectronic research associate.
produced the necessary voltage and frequency changes (Fig. 1). The circuit consists of a sinusoidal wave form generator (Hewlett-Packard 202A Function Generator), the output frequency of which is controlled by a linear ramp voltage generated once for each inspiration cycle. The output of this oscillator serves as the input of a gain-controlled amplifier, the gain of which increases simultaneously with the increasing frequency output of the oscillator. Therefore, in the absence of both signals “a” and “b” of Fig. 1, the output stimulation is a constant amplitude, constant frequency signal. With “a” present and “b” absent, the output is a constant amplitude, varying frequency signal. With both control signals present, the output stimulation consists of both an increasing amplitude and an increasing frequency as shown in Fig. 2. Pulse duration, rate, peak voltage, and frequency can be controlled by instrument adjustment.

Thoracic laminectomies were done. Various combinations of intradural electrodes were applied to intercostal nerves. Stimulation produced satisfactory individual intercostal contractions. When four pairs of stimulating electrodes were placed on thoracic nerves four through eight, an electrically driven inspiratory chest movement was obtained, and some air exchange occurred. The spinal cord itself was explored with a single active electrode (with the rectum as indifferent ground). Application of the stimulating electrode on or near cord surface at or between T-1 or T-2 will drive the thoracic cage in coordinated, vigorous, inspiratory movements. All subsequent data in this report were obtained by threading a 30 gauge stainless steel vinyl-coated electrode, exposed for the terminal 3 or 4 mm, through a lumbar puncture needle or a small lumbar incision up the subarachnoid space to lie near the high thoracic cord. The electrode position was determined by the physiological response to stimulation. It was considered correctly placed when low stimulating voltage, large tidal volume, and sequential and coordinated driven inspiratory chest movements all occurred simultaneously. Apnea was induced by barbiturate overdose or spinal cord section at C-2.

**Experimental Results**

*Control Studies.* Control spirometric tracings were made under light barbiturate anes-