on leaving (at the top of CC) is collected and measured in a 100-liter Tissot spirometer. This air is then analysed in Haldane apparatus.

Comparison of the results obtained in a few of the more recent "experimental" and "control" determinations is shown in Table 1.

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Studies on Intercostal Nerve Physiology.

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Pathological cases of processes involving the parietal pleura and the diaphragmatic pleura innervated by the lower intercostal nerves, and the attendant referred pain and muscular rigidity in the abdominal region (lower quadrant), have long suggested neural associations between the regions involved. Capps'[1] competent studies upon such clinical cases have yielded valuable information relating the portion of pleural membrane or diaphragm stimulated to site of referred pain.

It is a well established fact known to clinicians that:[2] "The abdominal symptoms of thoracic disease are often so misleading that needless or harmful surgery may be carried out. A pleurisy may give referred pain over the distribution of the 6 lower intercostal nerves, with fever may simulate appendicitis."

In attempting to establish a physiological basis for such phenomena, we have investigated the intercostal nerves of dogs. The effects of our various manipulations upon blood pressure and respiration were kymographically recorded, the blood pressure obtained from the carotid artery, and respiratory movements recorded by the usual pneumograph-tambour method. In cases where a pneumo-thorax was unavoidable, artificial respiration was administered. Isolation of various intercostal nerves and their branches was facilitated by appropriate rib resection. A tetanizing current was used for nerve stimulation.

Stimulation of the central end of any intercostal nerve results in a transient lowering of blood pressure (approximately 25 mm Hg.)

1 Capps, J. A., *An Experimental Study of Pain*, Macmillan, 1932.

2 Nelson, *Living Medicine*, Thomas Nelson and Sons, 1920-1937, 5, 222.

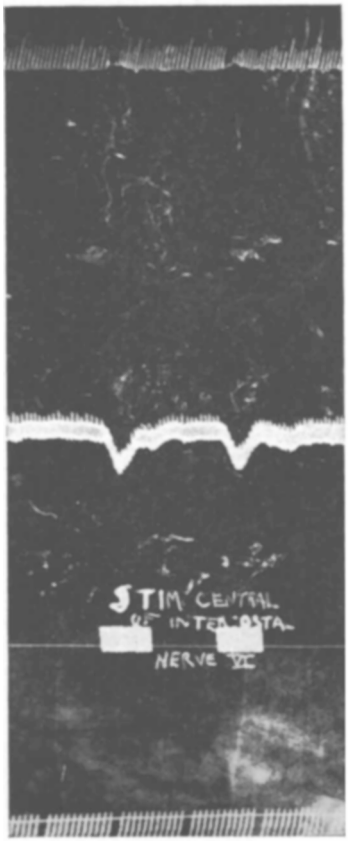

FIG. 1.
Effects of stim. of central end of intercostal nerve VI.

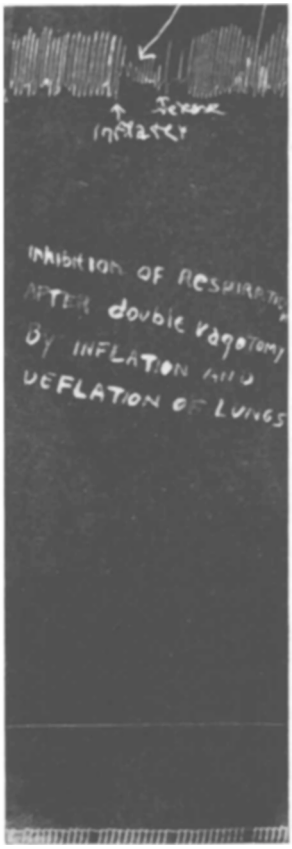

FIG. 2.
After double vagotomy. Inhibition of respiration by inflation and deflation of lungs. Arrow points to artifact of art. resp.

and an inhibition of respiration for a short interval of time. The interval of respiratory inhibition varied in length from 3 to 5 seconds. Normal respiration is resumed after this interval in spite of continuous stimulation. In some cases a decided decrease in amplitude of respiratory excursions was the only respiratory change observed. It was found that the lower intercostal nerves gave rise to more marked respiratory effects than the upper ones.

In attempting to relate the aforementioned effects to specific branches of the intercostal nerves, it was found that the effects on blood pressure and respiration may be elicited by stimulating the central ends of intercostal branches innervating the diaphragm, parietal pleura, and rectus abdominis muscle.

Stimulation of the central end of a branch of the intercostal innervating the diaphragm elicits *a spasm of the rectus abdominis muscle.* Since this reflex is also elicited by stimulation of the central end of the main trunk of a lower intercostal nerve (6-12), and the reflex is not abolished until all of the lower intercostals are severed, regardless of the order in which they are severed, the efferent fibers of the reflex arc must traverse several of the lower intercostal nerves (trunks X-XII especially). This striking reflex is undoubtedly the cause of the increased tonus of the rectus abdominis muscle observed in many cases of lobar pneumonia and pleurisy.

The effect of the parietal pleural nerve fibers upon respiration suggests a normal regulatory mechanism of respiration caused by the mechanical rubbing of the 2 pleural membranes during respiratory movements. Such a regulatory mechanism was also suggested by the work of Scott, Gault, and Kennedy[3] who recorded action potentials from the peripheral end of an intercostal nerve during chest expansion. Pike and Coombs[4] likewise obtained evidence pointing to a regulatory mechanism of respiration by thoracic and cervical dorsal nerve root section. We sectioned the vagi and phrenic nerves and strongly inflated the lungs with air, whereupon respiration ceased for about 20 to 25 seconds. After respiratory movements had started, the lungs were allowed to deflate, and another inhibition of respiration ensued, due to the mechanical stimulation of the parietal pleura by the deflating lungs. The extent of this inhibition with double vagotomy and phrenicotomy was approximately 10-15 seconds. Inflating a balloon in the chest cavity and thus expanding the chest wall internally results in the same respiratory effects. When the phrenic nerves are allowed to remain intact, the intervals of inhibition are greatly reduced (2-5 seconds), and a high cord section (at first thoracic segment) completely abolishes the inhibition. The effect of the phrenic nerves is due to the afferent fibers in them increasing the respiratory rate, as shown by Robb and Deason.[5]

We believe that the intercostal nerves reinforce the Hering-Breuer reflex in normal respiration. The intercostal nerves may also be a factor in regulating the rate of the respiratory excursions with changes in amplitude, that is, the greater the stimulation of the intercostal fibers, the greater the interval to the next respiratory movement.

[3] Scott, Gault and Kennedy, *Am. J. Physiol.*, 1922, **59.**

[4] Pike, F. H., and Coombs, Helen C., *Am. J. Physiol.*, 1922, **59.**

[5] Robb and Deason, *Am. J. Physiol.*, 1911, **28.**

Summary and Conclusions. 1. Stimulation of the central end of any intercostal nerve causes a reflex inhibition of respiration and effects a concomitant drop in blood pressure. 2. The lower intercostal nerves (7-12) elicit a greater response than do the upper ones. 3. Stimulation of the intercostal branches to the parietal pleura, diaphragmatic pleura, and rectus abdominis muscle give the respiratory inhibition and lowered blood pressure (approximately 25 mm of Hg.) 4. Stimulation of sensory or intercostal fibers in the diaphragm causes reflex contraction of the abdominal musculature through reflex connection with other lower intercostal nerves. 5. Among other things, these results furnish the physiological mechanisms involved in referred pain and muscular rigidity in the lower abdominal quadrant as a result of involvement of the base of the lungs, in lobar pneumonia for example. 6. Both expiration and inspiration cause the intercostal nerves to be stimulated and thereby effect reflexly respiratory inhibition. a. Inspiration more strongly inhibits respiration than does expiration. b. At the end of inspiration, the intercostal nerves aid the Hering-Breuer reflex. c. After expiration the intercostal nerves constitute a factor that determines lapse of time before the next inspiration. 7. Abnormal respiration and tightening of the abdominal musculature may be indicative of an irritation in the peripheral region of the diaphragmatic pleura.

I hereby wish to acknowledge that Dr. Arno B. Luckhardt suggested the problem and rendered valuable aid in its prosecution.

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H Ion Concentration of Various Fluids of the Genital Tract of the Cow.

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In a study of certain reproductive phenomena in dairy cattle a few questions were raised which made it necessary to determine the pH of various fluids of the genital tract of the cow. This problem is of considerable scientific and practical interest since it is reported by Warren[1] and others that sex can be controlled by the simple ex-

[1] Warren, Carl, *Animal Sex Control*, Orange Judd Co., 1940.