Phrenic and recurrent laryngeal discharge patterns and the Hering-Breuer reflex

MORTON I. COHEN
Department of Physiology, Albert Einstein College of Medicine, Bronx, New York 10461

COHEN, MORTON I. Phrenic and recurrent laryngeal discharge patterns and the Hering-Breuer reflex Am. J. Physiol. 228(5): 1489-1496. 1975.—In decerebrate, spontaneously breathing cats, whole-nerve recordings of efferent phrenic (Phr) and recurrent laryngeal (Lar) discharge were analyzed with an average-response computer. The Phr inspiratory (I) burst starts abruptly and has an augmenting discharge pattern which reaches a maximum at the end of the I phase. The Lar I burst starts before onset of the Phr burst (median delay 40 ms), reaches a maximum near the start of the I phase, and maintains an approximately constant level till the end of the I phase. These differences are functionally appropriate for the differing respiratory roles of the innervated muscles (diaphragm and glottis dilators). The Hering-Breuer reflex, produced by occlusion at minimum lung volume (start of the I phase), lengthens the I phase and disinhibits I discharge, but differently for Lar and for Phr activity. Lar activity was increased almost from the start of occlusion, indicating that normally pulmonary stretch receptor input inhibits Lar discharge almost from the start of the I phase. In contrast, occlusion produced no change in the Phr augmenting pattern (slope of activity increase) for most or all of the I phase, indicating that normally vagal inhibitory input shuts off Phr activity in a trigger-like manner.

The respiratory act consists of an ordered sequence of neural and muscular events which are temporally coordinated by descending command signals from the brainstem respiratory centers. For example, the activity of muscle groups at different segmental levels (e.g., diaphragm and laryngeal muscles) must occur in the proper time relations. In recent years, the role of the laryngeal musculature in regulating air flow has received increased attention (3, 26). In the present study, some aspects of laryngeal motoneuron activity have been explored by comparison of discharge patterns in the recurrent laryngeal nerve with those in the phrenic nerve with the use of whole-nerve recordings. The observations were made in spontaneously breathing decerebrate cats, both during normal breathing and during the changed breathing produced by manipulation of pulmonary stretch receptor input (Hering-Breuer reflex). Analysis with an average-response computer revealed important differences between the two nerve activities for both the spontaneous and the reflexly evoked discharge patterns. These differences are probably related to: a) differing functions in the respiratory act of the two kinds of motor activity, and b) differing temporal organization of the driving networks.

METHODS

The main series of experiments comprised 14 midcollicular decerebrate cats (weight range 2.8-4.0 kg); another series consisted of 4 cats under urethane anesthesia (1.0-1.25 g/kg). The main observations were made while the cats were breathing spontaneously through a tracheal cannula (inserted caudal to the larynx); ancillary observations were made during neuromuscular block (gallamine triethiodide) and artificial ventilation. Under ether or Fluothane anesthesia, a classical midcollicular decerebration was performed, and the animals were allowed to recover from anesthesia before recordings were taken. All cats had good decerebrate rigidity. After placement of the animals in a supine position, one phrenic (Phr) and one recurrent laryngeal (Lar) nerve were dissected free, desheathed, and sectioned. The central ends of the nerves were placed on bipolar hook electrodes, and the nerves were immersed in a pool of mineral oil through which 5% CO₂-95% O₂ had been bubbled. Arterial blood pressure was monitored and remained at adequate levels (150-100 mmHg systolic). Rectal temperature was maintained at 36-38°C by a heating lamp. The animals had cupric breathing patterns similar to those seen in awake resting cats; thus the section of one phrenic nerve did not seem to impair ventilation, which was adequately maintained by activity in the intact phrenic nerve and by intercostal activity.

Nerve Recordings

Recordings (amplifier band pass, 80-10,000 Hz) were taken from the central ends of the phrenic and recurrent laryngeal nerves. Integrated signals were then obtained after further amplification, half-wave rectification, and passage through a leaky integrator circuit (7). The integrated phrenic signal was further processed to obtain pulses marking the start of the inspiratory (I) and expiratory (E) phases (8). All data were recorded on magnetic tape.

Respiration Recording

In spontaneously breathing animals, respiratory motion was monitored by recording the pressure excursions in a chest pneumograph, using a Statham P23BB transducer.
In paralyzed, artificially ventilated cats, the airway pressure changes produced by the ventilation pump were measured through a side tube attached to the tracheal cannula.

Data Processing

The discharge patterns of the whole-nerve recordings were analyzed with an average response computer (CAT 1000). The nerve potentials, after half-wave rectification, were summed in computer sweeps triggered by either the I or E pulses, thus the resultant wave forms showed the average density of discharge in time relation to the start of the I or E phase. In order to process activity which preceded the trigger pulses, the nerve signals were delayed (usually by 100 or 200 ms) by passage through a digital delay circuit. Thus when a delayed signal was summed in computer sweeps triggered by the undelayed pulses, the activity that originally preceded each pulse was passed into the earlier bins of the sweep, and the resultant wave form then showed both pre- and posttrigger activity. The "add-subtract" feature of the computer was used to ascertain the change of activity produced by a test procedure; the method used was to subtract activity in control cycles from activity in test cycles. The computer memory contents were read out in digital form to a printer and in analog form to an inkwriter or X-Y recorder.

Occlusion

Airway occlusion was performed by closing a solenoid-operated valve interposed between the tracheal cannula and room air (10). The added dead space of the valve and its connections (about 10 ml) produced only minor changes (at the most 15%) in the variables of the respiratory cycle (duration and respiratory excursion). The solenoid valve power was switched by means of pulses derived from the I and E pulses, and thus an occlusion could be started or stopped at any predetermined time in the respiratory cycle. Occlusion was complete within 30 ms of the switching pulse. Adequate time for recovery (usually 10–20 respiratory cycles) was allowed between individual occlusion episodes.

RESULTS

Spontaneous Phrenic and Laryngeal Discharge Patterns

Simultaneous recordings from the efferent phrenic and recurrent laryngeal nerves revealed interesting differences between their overall discharge patterns, as illustrated in Fig. 1, which shows typical activity in decerebrate, spontaneously breathing cats.

The oscilloscope traces of Phr and Lar activity during an inspiratory (I) phase (Fig. 1, left) show that: a) the Phr I burst started and stopped abruptly on a background of little or no activity during the E phase; b) in contrast, the Lar I burst was superimposed on a background of "tonic" activity during the E phase. In addition, the pneumograph recording (Pn) shows that respiratory motion had a close temporal relation to Phr discharge, with the pneumograph pressure change starting about 30 ms after the initial Phr volley.

Further details of differences in pattern between the two nerve activities are revealed in the traces of computer-summed activity (Fig. 1, right) time-locked to the start of the I phase (computer sweeps triggered by I pulses).

Onset of inspiratory burst. The Phr I burst started abruptly with a discrete volley of spikes, which can be seen clearly in the oscilloscope trace (left) and which contributed a sharp peak to the summed activity wave form (right). The Lar I burst started less abruptly, as seen in the wave form on a fast time base (bottom right), in the late E phase a gradual increase of activity started and led into the I burst. The delay from the onset of increased Lar activity to the onset of the Phr burst was about 60 ms. In some cases, Lar activity was reached at a time approximately synchronous with the initial Lar volley (as in the recordings of Fig. 2). After reaching its maximum near the start of the I phase, Lar activity remained at an approximately constant level throughout the phase. Usually, there was a gradual slight decrement of activity as the I phase progressed (cf. Fig. 1); in some cases, however, there was a gradual slight increment of activity as inspiration progressed (cf. Fig. 2, state A). In contrast, Phr activity showed an incremental discharge pattern. Following the initial peak of the Phr burst and a subsequent rebound depression, the level of Phr activity augmented throughout the I phase, reaching its maximum at the end. Even when
burst of early E activity appeared in the whole-nerve recording. This phenomenon is shown in the summed activity wave forms of Fig. 2. As was usual in paralyzed cats, the respiratory cycle had lengthened due to the dissociation between the central respiratory cycle and lung expansion, the latter now being independently produced by the pump (10). In state A (top), the central respiratory cycle was locked in a 1:1 relation to the pump cycle. It can be seen that after the decay of the Lar I burst, another distinct burst occurred. The expiratory character of the latter burst is confirmed by its inverse relation to residual Phr discharge during the early E phase. The small Lar E burst started only after Phr discharge had decayed to almost zero level, it reached a peak during Phr inactivity, and finally the residual Phr burst ensued only after the Lar E burst had started to decay. In state B (bottom), which ensued spontaneously after the recordings taken in state A, the Lar E discharge became very prominent and continued for almost all the E phase, while slowly decaying. In this state, the respiratory cycle had further lengthened, every central respiratory cycle now being locked to every two pump cycles, and the I bursts in both Phr and Lar nerves were greatly increased. The associated drastic increase in Lar E activity can probably be explained as a rebound phenomenon consequent to the increased I discharge (10).

Respiratory Cycle and Hering-Breuer Reflex

The Hering-Breuer reflex effects on the respiratory phase durations were studied in 14 spontaneously breathing decerebrate cats. The reflex was produced by the classical method of airway occlusion (15), which was imposed at the two extreme points of the respiratory cycle: a) at minimum lung volume, occurring at the end of the E phase; b) at maximum lung volume, occurring at the end of the I phase. The occlusions were programmed by means of gates derived from the I and E pulses and were usually

![Fig. 3](http://ajplegacy.physiology.org/)

**Fig. 3.** Effects on respiratory cycle and on phrenic (Phr) and laryngeal (Lar) discharge of 2 kinds of airway occlusion (OCCL, deflection indicates duration of occlusion). Decerebrate, spontaneously breathing cat. Traces show half-wave rectified and integrated whole-nerve discharges (time constant, 100 ms). Top: occlusion at minimum lung volume maintained for one inspiratory phase (started by I pulse, ended by E pulse). Bottom: occlusion at maximum lung volume maintained for one expiratory phase (started by E pulse, ended by I pulse).
maintained for only one respiratory phase. A typical example of each kind of occlusion is shown in Fig. 3.

The occlusion at minimum lung volume (Fig. 3, top) was started by the I pulse and maintained for the duration of the I phase, thereby preventing the lung expansion ordinarily produced by I muscle contraction. During such an occlusion, there is only a very minor change in pulmonary stretch receptor discharge, due to slight lung decompression produced by inspiratory effort against the closed airway (24). The almost complete elimination of normally occurring afferent discharge reflexly produced lengthening of the I phase and increase of the peak amplitudes of both Phr and Lar discharge. The occlusion was released at the start of the E phase, and it can be seen that the immediate postocclusion E phase was longer than the control E phase.

The occlusion at maximum lung volume (Fig. 3, bottom) was started by the E pulse and maintained for the duration of the E phase, thereby keeping the lung in its end-inspiratory position and maintaining a high level of pulmonary stretch receptor discharge. The resultant reflex effects were lengthening of the E phase (with maintenance of Phr silence during the E phase) and an increase of Lar tonic activity occurring during this phase (as shown by the upward deflection in the integrated trace). The occlusion was released at the start of the I phase, and it can be seen that during the immediate postocclusion I phase (which was longer than the control I phase) the amplitudes of both the Phr and Lar discharges were increased in comparison to control levels.

These changes were all mediated by vagal afferent input, since after bilateral vagotomy similar occlusions produced no effects.

The changes of respiratory phase duration produced by the two kinds of occlusion in the population studied (14 decerebrate cats) are shown in Table I. The median duration of the control respiratory cycle was 1.7 s (I duration, 0.7 s; E duration, 1.0 s). The effects of occlusion are shown by the ratios of occlusion-phase duration to control levels. These secondary effects have been explained as rebound phenomena (10, p. 371-372).

The relationship between the durations of the E and I phases for each cat (n = 14) is shown in Fig. 4, both for the control cycles (open circles) and for the cycles in which occlusions at minimum lung volume (during the I phase) were imposed (filled circles). It is apparent that there is a linear relation between E- and I-phase durations, and that the same relation (regression line) holds for both control and occlusion cycles. However, the scatter about regression line is greater for the occlusion than for the control cycles. This relationship, which holds between different cats, is the same as that reported by Clark and von Euler (6) for cycles in the same cat whose durations were varied by experimental inputs.

**Phrenic and Laryngeal Discharge Patterns During Hering-Breuer Reflex**

Occlusion at minimum lung volume during the I phase produced different changes of discharge pattern in the two nerves. Although for both nerve activities the effect of occlusion was disinhibition (i.e., removal of the inhibitory effects of pulmonary stretch receptor input during inspiration), the time course of the changes differed between the nerves.

In the typical example of Fig. 5, occlusion produced lengthened the I phase by 60% (median), while occlusion at maximum lung volume lengthened the E phase by 150% (median). In addition, Table I shows the secondary effects which occurred during the immediate postocclusion phases: both the E phase following the occluded I phase (Fig. 3, top) and the I phase following the occluded E phase (Fig. 3, bottom) were lengthened in duration compared to the control phases. These secondary effects have been explained as rebound phenomena (10, p. 371-372).

**TABLE 1. Duration of respiratory phases and changes produced by occlusion in 14 decerebrate cats**

<table>
<thead>
<tr>
<th>Duration, control cycle, s</th>
<th>Median</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inspiratory phase</td>
<td>0.7</td>
<td>0.4-0.9</td>
</tr>
<tr>
<td>Expiratory phase</td>
<td>1.0</td>
<td>0.5-1.3</td>
</tr>
<tr>
<td>Total cycle</td>
<td>1.7</td>
<td>1.0-2.1</td>
</tr>
<tr>
<td>Occlusion effects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occlusion at minimum lung volume</td>
<td>1.6</td>
<td>1.3-2.3</td>
</tr>
<tr>
<td>1IR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SEFR</td>
<td>1.4</td>
<td>1.2-2.4</td>
</tr>
<tr>
<td>Occlusion at maximum lung volume</td>
<td>2.5</td>
<td>1.6-4.2</td>
</tr>
<tr>
<td>EFR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SIFR</td>
<td>1.4</td>
<td>1.1-2.0</td>
</tr>
</tbody>
</table>

Occlusion at minimum lung volume was maintained for one inspiratory phase. 1IR, inspiratory-inhibitory ratio (occlusion I phase/control I phase). SFR, secondary inspiratory-facilitatory ratio (postocclusion E phase/control E phase). Occlusion at maximum lung volume was maintained for one expiratory phase. EFR, expiratory facilitatory ratio (occlusion E phase/control E phase). SIFR, secondary inspiratory-facilitatory ratio (postocclusion I phase/control I phase).

**FIG. 4. Relation between durations of expiratory and inspiratory phases in 14 spontaneously breathing decerebrate cats: a) in control cycles (open circles); b) during cycles in which occlusion at minimum lung volume was imposed (filled circles). Points are means of 2-40 individual tests. Control cycle immediately preceded occlusion cycle. Occlusion was maintained for one inspiratory phase (started by I pulse, ended by E pulse). Regression line: expiratory duration = 0.3 + 1.0 (inspiratory duration) (g).**
INSPIRATORY DISCHARGE PATTERNS

FIG. 5. Effects of occlusion at minimum lung volume on average discharge patterns of efferent phrenic (PHR) and recurrent laryngeal (LAR) nerves. Decerebrate, spontaneously breathing cat. Occlusion was maintained for one inspiratory phase (started by I pulse, ended by E pulse). Each trace shows summed activity (after half-wave rectification) in 40 control (CONTR) or 40 occlusion (OCCL) cycles. Computer sweeps (20-ms bins) triggered by I pulses (arrow) and terminated before occurrence of succeeding I pulses. EC, ECONTR, EOCCL: median (dot) and range (horizontal line) of times of occurrence of E pulses in control and occlusion cycles, respectively. Bottom traces (OCCL-CONTR): activity in 40 occlusion cycles minus activity in 40 control cycles.

prolongation of the gradually augmenting Phr discharge; but strikingly, there was no change in the rate of increase (slope) of Phr discharge throughout all of the prolonged I burst. This phenomenon is apparent from the parallel rise of the control and occlusion wave forms (top); and it is shown even more convincingly in the wave form of occlusion-minus-control activity (bottom), which remained flat for a time corresponding to the control I phase. The latter wave form started to rise only in the prolonged portion of the I phase, i.e., in the time equivalent to the difference between occlusion and control I-phase durations. In other cats, the Phr activity slope during occlusion started to increase above that during the control state (i.e., the occlusion minus control trace began to lose its flatness) at a time equivalent to about two-thirds of the control I-phase duration; but the magnitude of this increase was small compared to total Phr activity. The position of this point of inflection ranged (in eight cats) from 60 to 100% of the control I-phase duration, with the median value being 80%.

Thus, occlusion at minimum lung volume produces no change in the augmentation pattern of Phr discharge for most or all of the time equivalent to the control I-phase duration; therefore, the disinhibitory effect of this occlusion is prolongation of the duration of Phr discharge without change in the basic discharge pattern.

In contrast, the effect on Lar discharge of occlusion at minimum lung volume was to produce disinhibition at a much earlier time in the I phase. As shown in Fig. 5 (bottom), the trace of Lar occlusion-minus-control activity started to increase almost at the start of the I phase (exact measurement being difficult because of variability in the trace). Indeed, average Lar activity during the I phase was converted by occlusion from a plateau-like pattern to an augmenting pattern (Fig. 5, middle). This result indicates that normally vagal afferent inhibition of Lar discharge is operative almost from the start of the I phase. The traces of Fig. 5 display the average of 40 occlusion tests; in other cats (four cases), in which only 10–20 tests were done, it was more difficult to discern the point of inflection (start of increase) in the occlusion-minus-control trace. But in all these cases, the inflection points were easily discernible at times of 200–300 ms preceding the inflection points in the Phr occlusion-minus-control traces.

Thus, vagal afferent inhibition of Lar activity becomes operative at a much earlier time in the I phase than does...
the inhibition of Phr activity. Indeed, a degree of Lar inhibition is probably occurring even at the very start of the I phase.

The effects on Phr and Lar discharge of occlusion at maximum lung volume during the E phase are shown in Fig. 6. The classically observed response, prolongation of the E phase, is accompanied by no change in the normally occurring Phr silence. However, the effect of this occlusion is to increase somewhat the tonic Lar discharge occurring during the E phase; this response is apparent in the trace as a slow rise of activity level during the prolonged E phase. The response is similar to that observed by Barlott et al. (Fig. 6 in ref. 3) in the electromyogram of the posterior cricoarytenoid (an inspiratory muscle) during lung inflation. Similar responses to maintained inflation have also been reported for some individual Lar inspiratory fibers (2). Thus, maintained pulmonary stretch receptor input can exert an excitatory (“paradoxical”) effect on Lar I discharge.

**DISCUSSION**

In the present study, whole-nervc recordings of different phrenic and recurrent laryngeal discharge have been used to compare the functional properties of the two neuronal populations. This approach rests on the assumption that there is some degree of homogeneity within each population, and it must therefore be complemented by observations on individual neurons.

In studies of individual Phr motor fibers (16, 19, 22, 23) it was found that their activity generally has an augmenting pattern, with discharge frequency increasing in an approximately linear fashion as the inspiratory phase progresses. In addition, some of these fibers start to discharge only after a delay from the start of the Phr burst (16). It is apparent then that summation of activity of many fibers will produce an augmenting pattern in the whole-nervc recording.

Several studies have been devoted to activity of individual Lar motor fibers or motor units (1, 2, 5, 13, 14, 17, 18, 20, 21, 27). Three types of discharge pattern have been reported: 1) inspiratory, 2) expiratory, 3) tonic with inspiratory modulation (higher discharge frequency during I). Examination of 1-units’ discharge patterns in published figures reveals that some units had approximately constant discharge frequency throughout the I phase, while others had augmenting discharge patterns. Therefore, depending on which members of the population are firing under different conditions, summated Lar activity could be either plateau-like in pattern (as in Fig. 1) or augmenting in pattern (as in Fig. 2, state B).

The patterns of mechanical change produced by the diaphragm and the Lar muscles resemble the summated activity patterns in the Phr and Lar nerves, respectively. Thus, augmenting Phr discharge (together with similar discharge in external intercostal nerves) results in gradual chest expansion, as monitored in the chest pneumogram, whose temporal pattern closely resembles that of Phr discharge (Fig. 1). This pattern has the obvious function of overcoming the increasing elastic recoil of chest and lung during the I phase by means of gradual increase of I muscle contraction.

The effect of the Lar I discharge is to open the glottis during I. The temporal pattern of glottis dilatation has been monitored in Lar pressure recordings (3, 4). Lar pressure has a trapezoidal wave form: the glottis starts to open before the start of chest motion and rapidly reaches maximum dilatation, which is then maintained throughout the I phase; further, some dilatation continues even into the E phase (cf. Fig. 5 in ref. 3). Thus, the pattern of pressure change closely resembles that of summated Lar activity. The function of this pattern in breathing is presumably to minimize resistance to airflow by producing maximal glottis opening before the onset of inspiratory motion.

Thus, the overall Phr and Lar discharge patterns are functionally appropriate for the differing roles of the innervated muscles in the act of respiration.

The differences between Phr and Lar discharge patterns are of interest in relation to the organization of respiratory center connections. Since both discharges are inspiratory, the neurons of both systems must be driven from a common source, i.e., an inspiratory subpopulation of the brainstem respiratory oscillator; and yet substantially different discharge patterns emerge as outputs.

We can probably exclude from consideration any special properties of Phr motoneurons, since evidence indicates that they are driven by fast projections from neurons of the dorsal infrasolitary complex of the medulla (region ventral to nucleus and tractus solitarius): 1) Lar I neurons in that region have discharge patterns identical to those of Phr motoneurons, as well as similar responses to lung inflation (11). 2) Antidromic stimulation shows that the axons of these I neurons descend to cervical levels (12). 3) Crosscorrelations between activity of the dorsal Lar I neurons and Phr neurons, as well as orthodromic stimulation in the region, indicate that the medullary neurons’ projections to Phr neurons are mono- or disynaptic (10a). Therefore, we may consider the behavior of Phr motoneurons as indicative of the properties of the dorsal medullary I neurons, which will be designated as Phr-driving neurons.

The first major difference between Phr and Lar discharge patterns is the earlier onset (median delay 40 ms) of the Lar I burst. This difference is probably based on the fact, observed by Barlott and Dussardier (2), that an appreciable fraction of Lar I fibers start to fire before the onset of the Phr burst. Indeed, the cumulative histogram of firing onset times of different Lar I fibers relative to onset of the Phr burst (Fig. 2 in ref. 2) closely resembles in shape the wave form of whole-nervc Lar discharge (Fig. 1, this paper).

The earlier excitation of Lar than of Phr-driving neurons could be caused by a) lower firing thresholds, or b) earlier and stronger depolarizing inputs. The timing of depolarizing drives to I neurons has been observed in intracellular recordings from thoracic I motoneurons (25) and from medullary I neurons (19a); these show that in the late E phase there is a slow depolarization which starts well before the onset of spike discharge. Therefore, if on the average Lar I neurons had inputs similar to those of Phr-driving neurons, but had lower thresholds, the former would start to fire earlier. This hypothesis is supported by the observation that many Lar neurons fire tonically (i.e., at low frequency during E and at higher frequency during I), which means...
that these neurons are close to threshold during the E phase. The earlier onset of Lar I discharge would also occur if Lar neurons received stronger or more rapidly depolarizing inputs than Phr-driving neurons; but only by comparisons of intracellular recordings would it be possible to distinguish this mechanism from the mechanism of differential thresholds.

To explain the sharper onset of the Phr than of the Lar inspiratory burst, it is necessary to invoke another mechanism, that of avalanche or positive-feedback excitation of Phr-driving neurons. Though these neurons have delayed onsets of discharge relative to Lar neurons, perhaps due to higher thresholds, a large proportion of them start firing synchronously, as indicated by a) the sharpness of onset of the initial Phr volley (Fig. 1, this paper), and b) the clumping at the start of I of the firing onset times of individual Phr fibers (Fig. 2 in ref. 16). It is therefore suggested that at the onset of the I phase, when a critical fraction of medullary Phr-driving neurons have fired, the reexcitant connections between these neurons cause activation of a much larger portion of the population. Such reexcitant connections between medullary I neurons would also explain the gradually augmenting discharge patterns of Phr-driving and Lar neurons.

The second major difference between Phr and Lar discharge patterns is in the wave form of overall activity during the I phase. After its onset Lar I activity reaches an early maximum and is maintained at an approximately constant level throughout the I phase. In contrast, Phr activity augments continually throughout the I phase, reaching its maximum at the end. These differences can result from differences of discharge pattern in individual neurons, as well as differences in proportion of neurons newly activated (recruited) as the I phase progresses. Several causal mechanisms may be suggested: a) greater accommodative properties of Lar neurons, b) lesser degree of reexcitant connections within the Lar or Lar-driving populations, c) greater degree of inhibitory action on Lar neurons. It seems that the last mechanism is of particular importance, since occlusion tests showed that there is a marked difference between Lar and Phr neurons with respect to inhibitory action of pulmonary stretch receptor input.

When the airway is occluded at minimum lung volume and the occlusion is maintained for the duration of the I phase, the normally occurring pulmonary stretch receptor input is eliminated, and the nature of its inhibitory effect is revealed by the pattern of disinhibition, which is manifested as an increase of activity.

The disinhibition of Lar activity by this occlusion is apparent almost from the start of the I phase. Activity starts to increase with little or no delay, and the plateau-like wave form of Lar activity is converted into an augmenting wave form (Fig. 5). Thus, normally the vagal inhibitory effect on Lar neurons is operative throughout the I phase, and the effect seems to be a graded function of lung expansion. The conversion to an augmenting pattern by occlusion suggests that during this test there is an appreciable degree of recruitment of previously silent "late" Lar neurons (2).

In contrast, the inhibitory effect of pulmonary stretch receptor input on Phr discharge operates in a discontinuous (triggerlike) fashion, with graded effects being of secondary importance. This is shown by the fact that occlusion produces no change in the shape of the Phr activity wave form for most or all of the I phase; the rate of increase of discharge (slope of wave form) remains the same, as also shown by the flatness of the occlusion-minus-control activity trace. Similar effects (no change of activity slope resulting from manipulation of vagal input) have been observed for individual phrenic fibers (19) and for medullary I neurons (10, 11).

Thus, pulmonary stretch receptor input seems to act in a triggerlike fashion by determining the level at which the Phr burst ceases. The Phr I burst, once it starts, proceeds in a predetermined pattern which is not markedly affected by stretch receptor input. If this input is removed by occlusion, the effect is to allow the augmenting Phr discharge to continue for a longer time at the same rate of increase. Similar findings on the triggerlike action of stretch receptor input were obtained by Clark and von Euler (6), who found that termination of I by volume pulses occurred in an "all-or-none" manner, with the threshold depending on time of application during the I phase. A possible explanation for their results and those of the present paper is as follows: as the I phase progresses, pulmonary stretch receptor input gradually increases the activity of inhibitory interneurons, e.g., the R6 neurons of the dorsal intrasolitary nucleus (11), but the activity of this group must rise to a critical level in order to produce shutoff of the Phr-driving neurons and of the whole I neuron population.

However, in addition there is a less prominent graded inhibitory effect of stretch receptor input on Phr neuron discharge. It was observed that sometimes during occlusion at minimum lung volume there was a moderate increase of Phr activity during the last third of the time corresponding to the control I phase, as shown by a rise in slope of the occlusion-minus-control activity trace. Thus, in these cases some inhibition of Phr neuron discharge was normally occurring near the end of the control I phase. The observed changes may have arisen from actions on discharge of "late" Phr neurons (16, 22), whose activity starts well after the onset of the Phr burst and which may have higher firing thresholds and be more susceptible to vagally mediated inhibition. But this graded inhibitory action probably affects a relatively small fraction of the Phr motoneuron population, judging by the magnitude of the changes in the occlusion-minus-control traces; the major portion of the Phr population is shut off in a triggerlike manner at the cessation of the I phase.

The further application of the occlusion method to test responses of brainstem respiratory neurons should furnish valuable information on the roles of various neuronal subpopulations in the I→E switching mechanism.

This research was supported by Public Health Service Grant NS-03970.

Preliminary results of this work have been published in abstract form (9).

Received for publication 5 August 1974.
REFERENCES


