

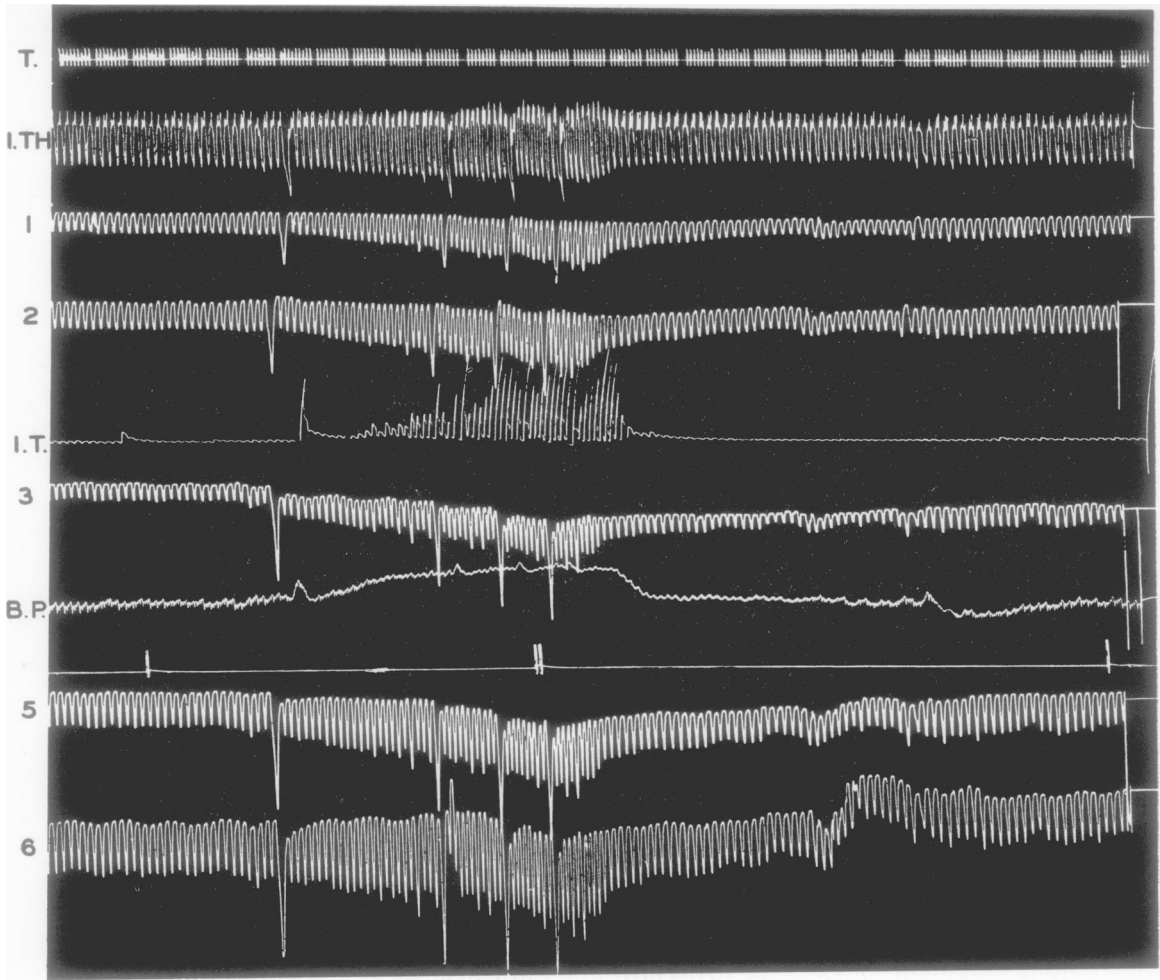
A COMPARISON OF THE EFFECTS OF ANOXEMIA AND CARBON-DIOXIDE SATURATION ON COSTAL AND ABDOMINAL BREATHING. By ROBERT GESELL and CARL MOYER, Department of Physiology, University of Michigan, Ann Arbor, Michigan.

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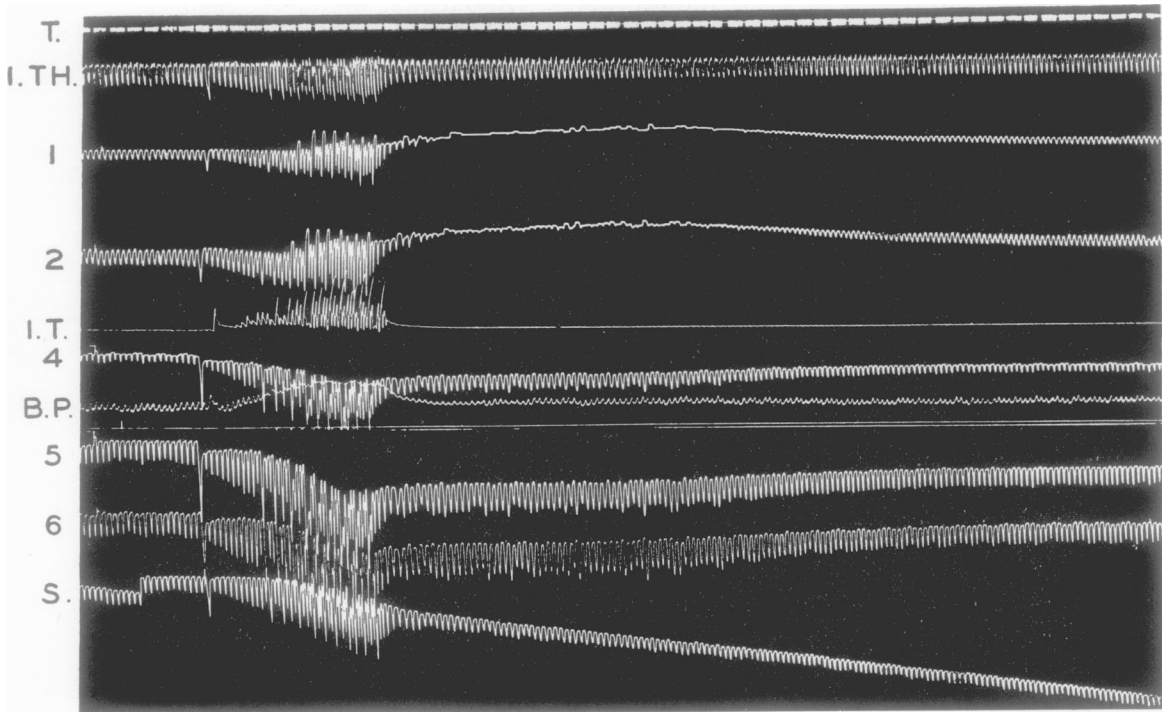
INCREASED breathing elicited by lowered alveolar oxygen pressure usually differs from increased breathing elicited by carbon dioxide. In general, lowered oxygen pressure produces a relatively rapid breathing with relatively small increase in tidal air, whereas carbon dioxide produces a smaller acceleration and a greater tidal air [Haldane, 1922; and Gesell, 1929]. The present study is a further comparison of the effects of anoxemia and carbon dioxide, dealing especially with changes in costal and abdominal breathing and costal and abdominal expiratory circumferences. The methods employed have been described in a preceding paper [Gesell and Moyer, 1934].

The type of results most commonly encountered during anoxic hyperpnea is illustrated in fig. 1. The circumferences of both the chest and abdomen at the end of expiration were decidedly increased, and with readministration of high oxygen mixtures the expiratory circumferences returned to their initial values. Anoxemia, therefore, seems to augment the tension of the diaphragm and of those muscles increasing the circumference of the chest, or possibly decreases the tension of their antagonists. Resupply of oxygen reverses these tensions. During the period of anoxemia the excursions of the chest and abdomen usually tend to increase in the same proportion, but when the chemical processes are suddenly reversed by readministration of high oxygen mixtures a greater reduction of costal excursions almost always occurs. This disproportional decrease is only faintly indicated in fig. 1. It is much more pronounced in figs. 2 and 3.

In fig. 2, as in fig. 1, the circumference of both the chest and abdomen increased with each expiratory pause during the early exposure to low oxygen, but very shortly the expiratory circumference of the chest diminished as that of the abdomen continued to increase. Interpreted solely on the basis of tension of inspiratory muscles the results indicate a progressively increasing tension of the diaphragm and a temporary increase in tension of the inspiratory muscles of the chest, giving way to a final decrease in tension.



4.0% O₂



The disproportionate inhibition of costal respiratory movements during readministration of high oxygen mixtures is the striking point of the experiment. At the level of band 1 respiratory movements have disappeared entirely, at band 2 they are only faintly visible, while at bands 5 and 6 they are greater than the normal movements preceding the period of anoxemia. Only as the abdominal excursions decreased towards the normal did the costal excursions increase back to pre-anoxic amplitude. Why readministration of high oxygen mixtures should produce localised apnea requires further study. Whether it is due to localised chemical reactions within the cord or in the peripheral afferent or efferent end organs remains unanswered. For the present we can only conclude that changes in oxidations are capable of producing profound differential effects on the activity of respiratory muscles.

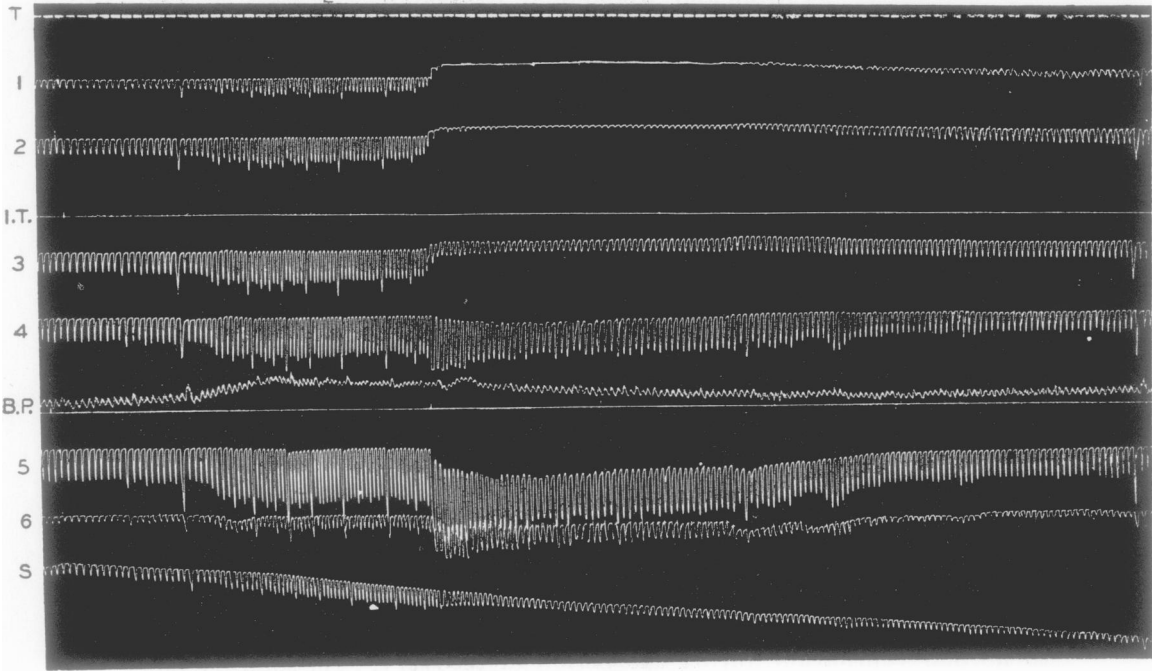
Another and somewhat different combination of results is seen in fig. 3. Here both costal and abdominal expiratory circumferences remained unchanged during the period of anoxemia, but strangely enough when the chemical processes were reversed during recovery the expiratory circumference of the chest decreased while that of the abdomen increased. But of more interest is the peculiar mode of return to normal ventilation during the period of recovery. As soon as the high oxygen pressures took effect there was a simultaneous reduction in costal respiration and an *increase in abdominal breathing above the maximum amplitude of the period of anoxic hyperpnea immediately preceding*. This unique compensatory action of an absolute increase in abdominal excursions during recovery is just sufficient to overcome the effects of relative apnea of the chest and accomplish a slow and even reduction of total ventilation (see the spirometer record).

The effects of carbon dioxide, most commonly observed, differed from those of anoxemia (see fig. 4). The expiratory circumferences were decreased at all costal and abdominal levels, and there was no differential effect either on the expiratory circumference or the amplitude of respiration during the period of recovery. Again, if we interpret the changes in circumference of the chest and of the abdomen on the basis of muscle tension we must assume that, contrary to the effects of lowered oxygen pressure, carbon dioxide decreased the tension of the inspiratory muscles or increased the tension of the expiratory muscles.

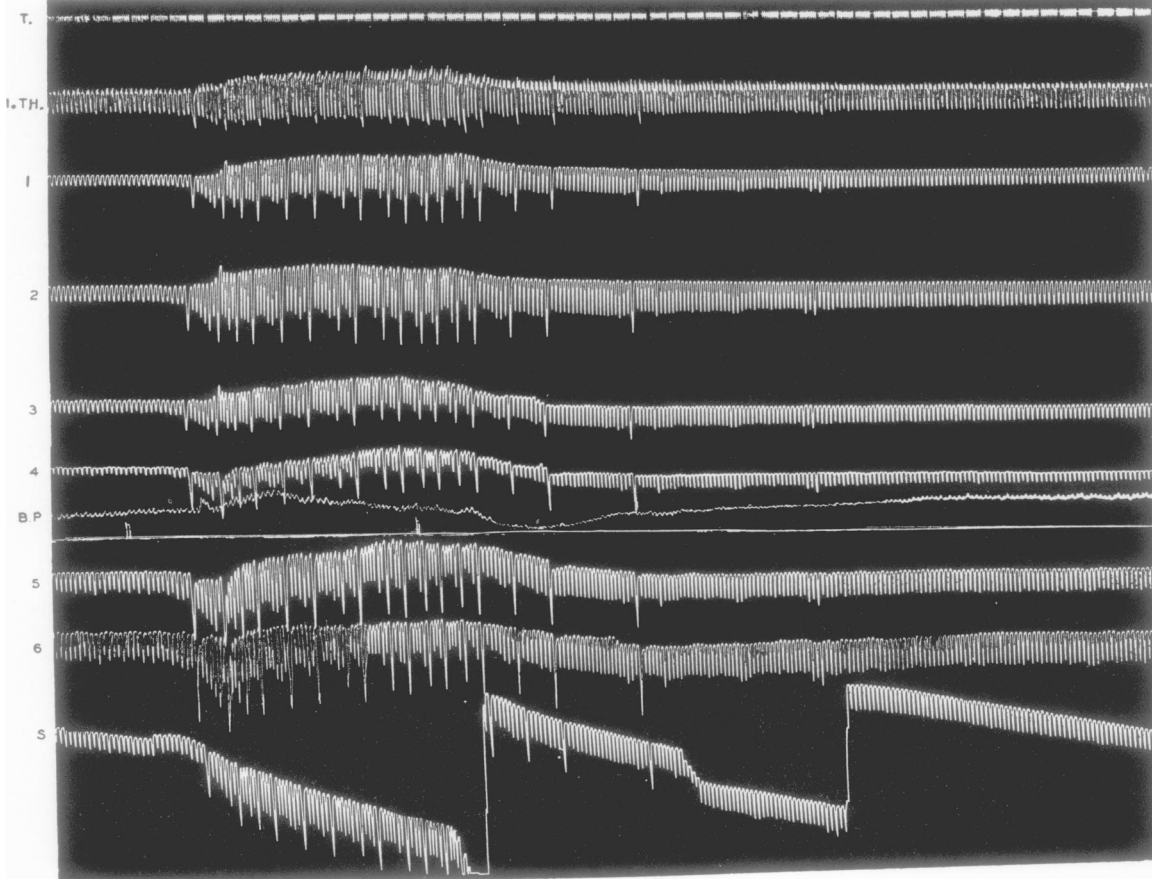
FIG. 1.—Typical effects of anoxemia. During recovery, however, the commonly greater inhibition of costal movements is not pronounced. T., time in seconds and 10 seconds; 1, 2, 3, 4, 5, and 6, respiratory excursions at three costal and three abdominal levels; I.TH., intra-thoracic pressure; B.P., mean blood-pressure; I.T., intra-tracheal pressure.

FIG. 2.—Increasing expiratory circumference during lowered alveolar oxygen which gives way in the chest to decreasing expiratory circumference while the abdomen continues to expand. The greater costal inhibition during recovery is typical. S., spirometer tracing.

4% O₂



29% CO₂



These changes in tension with their respective effects upon lung volume would be expected to decrease the intra-thoracic pressures during anoxemia and increase them during carbon-dioxide hyperpnea. Such are the more common results encountered in our experiments (see figs. 1 and 4) and agree with the results obtained by Prinzmetal and Brill [1932].

In the light of experiments published in a fourth paper of this group, indicating that carbon-dioxide saturation inhibits all prevailing reflexes and that anoxemia and carbon-dioxide depletion have an augmenting effect, it may not be rash to suggest that reflexes tending to increase the volume of the lungs predominated under the conditions of our experiments. In accordance with this conception a reduction of this tendency by a weakening of the reflexes from carbon dioxide saturation would permit the chest and abdomen to constrict, whereas an augmentation of the prevailing reflexes would lead to expansion of the torso.

It is possible, however, that all our results cannot be interpreted solely on the basis of muscle tension. Other factors, such as elastic pull of the lungs and resistance to the passage of air, are capable of influencing the changing configuration of the chest and abdomen. For the present we have no evidence that anoxemia produces such effects and only one experiment in which a strong carbon-dioxide mixture appears to decrease the inward traction of the lungs. In this experiment, contrary to the usual effects of carbon dioxide, there was an increase in circumference at all costal and abdominal levels. Despite the increase in lung volume, which must have occurred, there was an unquestionable increase in intra-thoracic pressure.

SUMMARY.

The effects of lowered alveolar oxygen and of increased alveolar carbon dioxide on costal and abdominal breathing were recorded with six encircling bands.

Lowered alveolar oxygen most commonly increased the circumference

FIG. 3.—Unusual co-ordination (?) of costal and abdominal breathing during recovery from preceding anoxemic hyperpnea in which a marked reduction of costal excursions below the normal amplitude is compensated by an augmentation of abdominal movements above the anoxemic amplitude. A later increase in costal excursions is accompanied by a decrease in abdominal excursions. T., time; 1, 2, 3, 4, 5, and 6, respiratory excursions at corresponding recording bands; I.T., intra-tracheal pressure; B.P., mean blood-pressure; S., spirometer record.

FIG. 4.—A common response to administration of carbon dioxide showing a decrease in expiratory circumference in both chest and abdomen accompanied by an increase in intra-thoracic pressure (I.T.H.).

of the chest and abdomen at the end of expiration and decreased the intra-thoracic pressure.

This expansion of the chest and abdomen was tentatively attributed to an increased tension of the diaphragm and of the inspiratory muscles of the chest.

Sometimes anoxemia increased the expiratory circumference of the abdomen and decreased that of the chest.

This differential effect was tentatively attributed to a greater development of tension in the diaphragm which, through the lungs, exerted an inward traction on the chest.

During recovery from anoxemia costal respiratory movements were inhibited more than abdominal respiratory movements. The upper costal segments were inhibited more than the lower costal segments. Upper costal apnea and abdominal hyperpnea were not uncommon. In one experiment upper costal apnea was accompanied by abdominal hyperpnea *greater* than the hyperpnea of the immediately preceding anoxemia.

As abdominal respiratory movements diminished during recovery the costal movements increased. This unusual co-ordination (?) of costal and abdominal breathing led to a smooth return to normal ventilation.

The effects of carbon dioxide differed importantly from those of anoxemia. In most experiments there was a decrease in circumference of the chest and abdomen at the end of expiration and a corresponding rise in the intra-thoracic pressures.

It was tentatively suggested that carbon dioxide decreases the tonus of the inspiratory muscles by inhibiting prevailing reflexes which tend to increase lung volume, and that anoxemia increases the tonus of the inspiratory muscles by augmenting these same prevailing reflexes.

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