# Breath holding during and after muscular exercise

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ÅSTRAND, PER-OLOF. *Breath holding during and after muscular exercise.* J. Appl. Physiol. 15(2): 220–224. 1960.—Breath holding was performed *a*) at rest, *b*) at the beginning of muscular work, *c*) during the steady state of work and *d*) immediately after work. End-expiratory air was analyzed for $CO_2$ and $O_2$ tensions. At breaking point the $P_{A_{CO_2}}$ values were similar in *a*) and *b*) (below 52 mm Hg with air breathing) and in *c*) and *d*, respectively. The higher the work load the higher the $P_{A_{CO_2}}$ in *c*) and *d*) (above 70 mm Hg at heavy work load breathing air; after $O_2$ breathing above 90 mm Hg). The $P_{A_{O_2}}$ values obtained were lower during and after work than at rest. These events and the increased 'tolerance' for high $CO_2$ and low $O_2$ in breath holding during and immediately after exercise are discussed in light of the theory that proprioreceptive impulses from working limbs are important in the control of respiration during exercise.

IN SPITE OF NUMEROUS INVESTIGATIONS during the last 50 years, the regulation of respiration in muscular work is still an unsolved problem. From many studies it has been concluded, however, that one factor contributing to the increased pulmonary ventilation during muscular exercise should be afferent impulses from the working limbs (joints and/or muscles) to the respiratory center (see Dejours (1) for references). According to Gray (2) such reflexes, the arterial $Pa_{CO_2}$, $Pa_{O_2}$ and $C_H{}^+$ should cooperate in an additive way. If so, it could be assumed that the breaking point in a breath-holding test during exercise should be at a lower $Pa_{CO_2}$ and/or higher $Pa_{O_2}$ than at rest.

The present study was performed to test this hypothesis.

## METHODS

Breath holding started after a normal inspiration. To obtain alveolar air samples the subject expired rapidly and almost maximally through a 3-way stopcock and the last part of expired air was collected in a small rubber bag by turning the stopcock. Air samples were analyzed by the Haldane technique. Work was performed on a Krogh bicycle ergometer and different work loads were used. Three series of experiments were conducted.

*A.* To follow the changes in composition of alveolar air during breath holding the test was interrupted at different times and end-expiratory air was sampled. At rest and during work with 900 kpm/min.[1] experiments were made on two subjects with air or oxygen breathing preceding the breath holding.

*B.* To determine $P_{A_{CO_2}}$ and $P_{A_{O_2}}$ at breaking point before, during and after muscular exercise with different work loads, the breath was held as long as possible *a*) at rest, *b*) at the beginning of work, *c*) during the steady state of work (at least 5 min. after work started) and *d*) immediately after stopping the work. Three subjects took part and air breathing preceded the breath holding.

*C.* The four experiments (*a–d*) mentioned above were made on one subject exposed to various tracheal $O_2$ tensions (99–700 mm Hg) prior to the test. To obtain a tracheal $P_{O_2}$ lower than at normal conditions, experiments were conducted in an altitude chamber. With few exceptions the work load was 900 kpm/min. One series of breath-holding tests was performed after voluntary hyperventilation of air for 1 minute.

## COMMENTS

The first experiments conducted were those in *series B* and the subject actually terminated the breath holding when first facing an overwhelming desire to breathe. However, when the breath holding was interrupted after various lengths of time, as in *series A*, and the subject predestined a time and tried with every effort to reach it, he could prolong the breath holding compared with the duration in *series B*. (In *series B* and *C* the stop watch could not be seen by the subject.) When able to prolong the breath holding (as in *A* and *C*) the subject first realized some relief but then a second extremely strong dyspnea was felt. Sometimes several such 'waves' of strong dyspnea were experienced. The experiments were quite uncomfortable.

In all experiments the later stage of the breath holding involved involuntary contractions of the respiratory

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[1] kpm = kilopond meter; 1 kp is the force acting on the mass of 1 kg at normal acceleration of gravity.

muscles. The difficulties, especially during exercise, in deciding when further apnea is impossible are obvious from the preceding comments, and are well to keep in mind when evaluating the results of this study.

## RESULTS

*A. Changes in alveolar gas during breath holding.* Figure 1 gives the alveolar $P_{CO_2}$ and $P_{O_2}$ after various periods of breath holding at rest, as well as during work 900 kpm/min.

At rest on *subject POÅ* there was a continuous rise in $P_{A_{CO_2}}$ and fall in $P_{A_{O_2}}$, but during exercise there was, after a steep increase in $P_{A_{CO_2}}$ from 43–60 mm during the first 10 seconds, a leveling off at about 65 mm Hg. The $P_{A_{O_2}}$ decreased with prolonged breath holding down to 25 mm after 40 45 seconds. Similar records were obtained with subject *RH.*

A different picture of the alveolar $pCO_2$ was obtained in experiments where oxygen breathing preceded the breath holding. As shown in figure 1, the $P_{A_{CO_2}}$ continued to rise with prolonged breath holding and 90 mm was reached after 47 seconds. The $P_{A_{O_2}}$ was still above 160 mm Hg. These 47 seconds do not represent a maximal effort. To be able to expire in the sampling bag the breath-holding test could not be too prolonged.

*B. Breath holding at different work loads.* Figure 2 gives $P_{A_{CO_2}}$ and $P_{A_{O_2}}$ at breaking point for two subjects after breath holding at rest and in connection with exercise with different work loads. (The trend of the results with a female subject was similar.) Table 1 summarizes the data on *subject POÅ.* The $P_{A_{O_2}}$ values were fairly scattered but the following conclusions can be drawn:

*1)* Breath holding at the beginning of exercise gave a $P_{A_{CO_2}}$ on the same level as at rest independent of the work load. In two of the three subjects $P_{A_{O_2}}$ decreased as the work became heavier, but only to a certain work load. This is most obvious in *subject POÅ* up to 900 kpm/min.

*2)* Breath holding during a steady state of exercise gave $P_{A_{CO_2}}$ that was higher, the heavier the work load. Compared with 50 mm Hg at rest it was for *POÅ* 74 mm during 1500 kpm/min. The $P_{A_{O_2}}$ showed a successive decrease.

*3)* Breath holding from the very moment exercise stopped gave a $P_{A_{CO_2}}$ similar to that obtained in a steady state of work. The $P_{A_{O_2}}$ was lower than at rest experiments, but mostly somewhat higher than in tests during steady-state work.

*4)* The breath-holding time was longest at rest, shortest during steady-state work and after work and of intermediate duration at the beginning of work (table 1).

*C. Breath holding with various $P_{O_2}$ in inspired air.* Table 1 summarizes data from breath-holding experiments at rest and during work (mostly 900 kpm/min.) after breathing gases with various $O_2$ tensions.

The higher the $P_{I_{O_2}}$, the higher was the $P_{A_{CO_2}}$ at breaking point in otherwise similarly conducted experiments (see fig. 3). During steady-state work with tracheal $P_{O_2}$ 205 mm, the breaking point was at a $P_{A_{CO_2}}$ of 71 mm and $P_{A_{O_2}}$ of 40 mm Hg. With tracheal $P_{O_2}$ 99 mm, the values were 51 and 24 mm Hg, respectively.

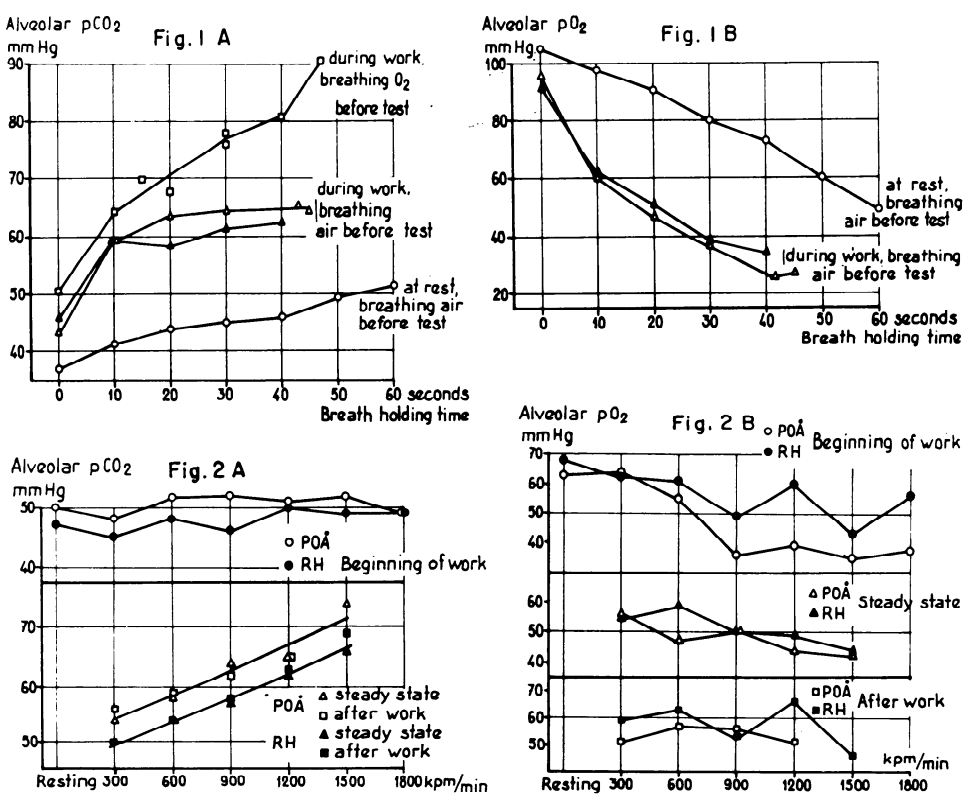



FIG. 1. End-expiratory alveolar $P_{CO_2}$ (*A*), and $P_{O_2}$ (*B*) after different time of breath holding with air- or $O_2$-breathing prior to test. Work load was 900 kpm/min. (*Unfilled dots, POÅ; filled dots, RH.*)

FIG. 2. Alveolar $P_{CO_2}$ (*A*) and $P_{O_2}$ (*B*) at breaking point after breath holding at rest, at beginning of work, during steady state of work and immediately after work. (*Unfilled dots, POÅ; filled dots, RH.*)

TABLE 1. *Breath-Holding Time and End-Expiratory $P_{A_{CO_2}}$ and $P_{A_{O_2}}$ After Breath Holding at Rest, at Start of Muscular Work, During Steady State of Work and Immediately After Work*

| Tracheal $P_{O_2}$, mm | Breath Holding at Rest | | | Work Load, kpm/min. | Breath Holding | | | | | | | | | Steady State | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | At start | | | In steady state | | | After work | | | Pulm. vent., l/min. BTPS | $O_2$ Intake, l/min. STPD | Heart rate | $P_{A_{CO_2}}$, mm | $P_{A_{O_2}}$, mm |
| | $P_{A_{CO_2}}$, mm | $P_{A_{O_2}}$, mm | Time, sec. | | $P_{A_{CO_2}}$, mm | $P_{A_{O_2}}$, mm | Time, sec. | $P_{A_{CO_2}}$, mm | $P_{A_{O_2}}$, mm | Time, sec. | $P_{A_{CO_2}}$, mm | $P_{A_{O_2}}$, mm | Time, sec. | | | | | |
| *Series B* | | | | | | | | | | | | | | | | | | |
| 149 | 50.8 | 63.4 | 61 | 300 | 48.2 | 64.1 | 35 | 53.5 | 56.4 | 25 | 56.0 | 51.3 | 36 | 22 | 0.95 | 90 | 43 | 100 |
| | 49.4 | 62.7 | 61 | | | | | 55.3 | 57.0 | 23 | | | | | | | | |
| | | | | | | | | 54.4 | 55.8 | 25 | | | | | | | | |
| 149 | | | | 600 | 51.5 | 55.4 | 36 | 57.8 | 50.2 | 21 | 58.7 | 56.7 | 24 | 34 | 1.45 | 108 | 46 | 91 |
| | | | | | | | | 59.2 | 47.3 | 25 | | | | | | | | |
| | | | | | | | | 61.3 | 42.9 | 25 | | | | | | | | |
| 149 | | | | 900 | 51.1 | 46.2 | 35 | 63.8 | 52.9 | 14 | 62.1 | 55.6 | 17 | 48 | 2.05 | 124 | 43 | 97 |
| | | | | | | | | 64.0 | 49.3 | 17 | | | | | | | | |
| | | | | | | | | 64.3 | 47.0 | 16 | | | | | | | | |
| 149 | | | | 1200 | 50.7 | 38.6 | 30 | 64.5 | 45.4 | 15 | 65.2 | 50.6 | 16 | 62 | 2.70 | 143 | 45 | 95 |
| | | | | | | | | 64.9 | 42.5 | 16 | | | | | | | | |
| 149 | | | | 1500 | 53.6 | 33.2 | 36 | 74.3 | 42.1 | 14 | | | | 90 | 3.45 | 168 | | |
| | | | | | 50.2 | 36.9 | 29 | | | | | | | | | | | |
| 149 | | | | 1800 | 48.5 | 37.0 | 29 | | | | | | | 125 | 4.20 | 190 | | |
| *Series C* | | | | | | | | | | | | | | | | | | |
| 99 | 42.2 | 42.1 | 25 | 900 | 45.6 | 24.6 | 37 | 50.4 | 24.5 | 25 | 50.4 | 21.5 | 29 | 57 | 2.10 | 140 | | |
| | 44.2 | 36.2 | 34 | | | | | 52.2 | 22.4 | 21 | | | | | | | | |
| 124 | 46.8 | 44.8 | 54 | 900 | 49.6 | 27.3 | 58 | 59.2 | 24.5 | 36 | 59.7 | 29.9 | 31 | 51 | 2.05 | 128 | | |
| | 48.3 | 40.3 | 65 | | | | | 58.7 | 27.3 | 36 | | | | | | | | |
| 149 | 49.5 | 55.8 | 78 | 900 | 51.7 | 36.4 | 59 | 64.3 | 27.7 | 45 | 64.3 | 36.3 | 34 | 48 | 2.05 | 124 | | |
| | | | | | | | | 65.8 | 25.8 | 43 | | | | | | | | |
| 149* | 42.4 | 53.0 | 93 | 900 | 42.0 | 22.7 | 67 | 54.0 | 25.7 | 46 | 53.5 | 29.6 | 58 | | | | 26† | 122† |
| | 40.6 | 46.2 | 125 | | | | | 52.2 | 23.7 | 46 | | | | | | | 28† | 121† |
| | | | | | | | | | | | | | | | | | 29† | 117† |
| 205 | 55.9 | 76.0 | 94 | 900 | 61.1 | 39.6 | 67 | 72.5 | 42.8 | 45 | 73.9 | 66.9 | 43 | 45 | 2.10 | 122 | | |
| | 58.5 | 61.7 | 99 | | | | | 69.4 | 38.5 | 35 | | | | | | | | |
| 99 | | | | 300 | 44.3 | 30.1 | 33 | 46.8 | 27.7 | 27 | 47.5 | 27.0 | 35 | | | 92 | | |
| | | | | | | | | 47.3 | 26.6 | 33 | | | | | | | | |
| | | | | | | | | 48.0 | 25.9 | 31 | | | | | | | | |
| 99 | | | | 1200 | 43.3 | 22.7 | 36 | | | | | | | | | | | |

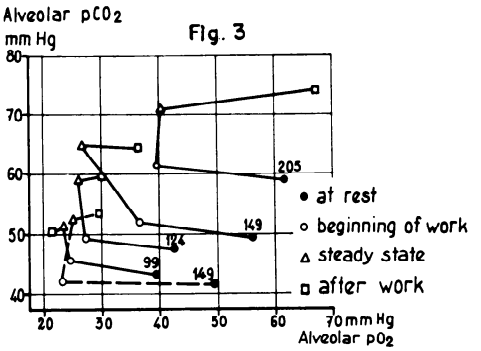
Each figure represents one determination. Some other measurements obtained 5 min. or more after beginning of work are included (Steady State); *subject POÅ.* * Breath holding after 1-min. hyperventilation; before test the alveolar $P_{CO_2}$ at rest was 18 mm, the $P_{A_{O_2}}$, 137 mm. † After 1-min. hyperventilation.

The typical changes of the breaking point in the different cycles of work are evident from figure 3, where the gas tensions at this point are plotted in a $CO_2$ - $O_2$ diagram. At the beginning of exercise, breath holding moved this point from the value at rest to a lower $P_{A_{O_2}}$ but with essentially maintained $P_{A_{CO_2}}$. During steady-state work and with the same $P_{I_{O_2}}$, the breaking point then moved upward to higher $P_{A_{CO_2}}$, but with unchanged $P_{A_{O_2}}$. When this work stopped, breath holding kept $P_{A_{CO_2}}$ as during steady-state work, but with a relatively high $P_{O_2}$ in inspired air there was a shift to the right. When the subject was exposed to hypoxia before these breath holdings after work, the difference in $P_{A_{O_2}}$ compared with steady-state values was less marked. Certainly the $O_2$ debt and $O_2$ uptake during early recovery was higher in such experiments.

One experiment with 1 minute's hyperventilation of air preceding the breath holding follows the same line. In this test convulsions occurred at the end of breath holding and the subject was not far from syncope. The same happened in experiments during hypoxia.

## DISCUSSION

The factors determining the breaking point when holding the breath, are complex and certainly other factors than alveolar $P_{CO_2}$ and $P_{O_2}$ are involved. The tolerance to low $P_{O_2}$ and high $P_{CO_2}$ is higher in rebreathing experiments than at actual breath holding (3). The larger the tidal air in such rebreathing experiments the higher this tolerance (4). Single or repeated breaths after breath holding, without change in alveolar air composition, make new breath holding possible and higher $P_{A_{CO_2}}$ and lower $P_{A_{O_2}}$ are obtained compared with the first trial (5, 6). In those experiments Hering-Breuer reflexes are not supposed to be the important factor (6, 7). There are suggestions that circulation improves with the respiratory movements.



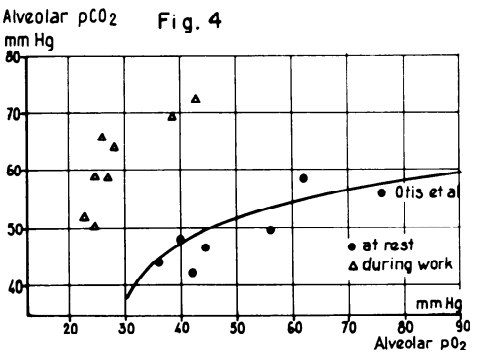




FIG. 3. Same experiments as in fig. 2 on *subj. POÅ* but with constant work load (900 kpm/min.) and breathing air with various $P_{I_{O_2}}$ prior to breath holding. The tracheal $P_{O_2}$ in mm Hg is marked on each line. *Broken line* represents breath holding after 1 min. of hyperventilation.

FIG. 4. End-expiratory $P_{A_{CO_2}}$ and $P_{A_{O_2}}$ at breaking point after breath holding at rest and during steady state of work (900 kpm/min.) and with various $P_{I_{O_2}}$, *subj. POÅ*. *Curve* is from Otis *et al.* (17), representing data obtained at rest.

Douglas and Haldane (8) pointed out that any muscular movement during breath holding is a relief. On the other hand, breath-holding time during muscular exercise is shorter than at rest (9–11), which is also the case immediately after work (12). From experiments with breath holding during running Hill and Flack (13) at the breaking point obtained a $P_{A_{CO_2}}$ above 55 mm with air breathing preceding the test compared with 50 mm at rest. Their explanation for the higher $P_{A_{CO_2}}$ during work is an improved circulation and/or that the work 'takes off' attention. Muxworthy (14) analyzed the alveolar gases after breath holding before, during and after step-up work and concluded a decreased sensitivity (or greater tolerance) of the respiratory system to $CO_2$ during work (see also 11).

The present study shows a striking increase in $P_{A_{CO_2}}$ and decrease in $P_{A_{O_2}}$ in breath holding during or after exercise compared with tests at rest, thus confirming the few data found in earlier literature. The higher the work load the larger the difference. One important question is how well the changes in alveolar gas tensions reflect the events in the respiratory center and the chemoreceptors in the arteries. It can be assumed that the $P_{a_{CO_2}}$ and $P_{a_{O_2}}$ are not too far from the values obtained on the end-expiratory samples. During exercise the circulatory time is shorter than at rest, but the changes in $CO_2$ and $O_2$ content of the blood and alveolar air eventually occur more rapidly and pronouncedly. Figure 1 shows the fast increase in $P_{A_{CO_2}}$ during the first 10 seconds of breath holding during work, but from then on the changes are, however, small. This smaller increase is partly explained by the high $CO_2$-storing capacity of blood and tissues during breath holding, which is evident from other studies (15–17) and the probably reduced $CO_2$ production when $O_2$ supply becomes insufficient. Furthermore, it should be emphasized that the concentration of $CO_2$ in the lungs will increase as oxygen is diffusing into the blood and the total volume of gas becomes diminished. This, per se, will cause an increase in $P_{A_{CO_2}}$. There will be a gradual decrease in the gradient $P_{A_{O_2}}$–$P_{V_{O_2}}$ and the reduction in lung volume will be less the longer the breath is held. In experiments with about 80% nitrogen in the alveoli the effect will be less marked than in experiments with oxygen-enriched air inhaled before the breath holding. However, it is concluded that, with the prolonged breath-holding times in the later experiments (*series C*), the blood reaching the respiratory center had a $P_{a_{CO_2}}$ similar to that in the lungs at the actual breaking point. (In experiments where oxygen-enriched air was inspired prior to the breath holding the situation was somewhat different. Part of the continuous rise in $P_{A_{CO_2}}$ can be explained by the reduced $CO_2$-combining capacity of the blood as the hemoglobin in the lungs becomes saturated with $O_2$ and a continuous, normal $CO_2$ production.) The $P_{A_{O_2}}$ decreased during the breath holding and the chemoreceptors and respiratory center were surely exposed to the lowest $P_{a_{O_2}}$ some seconds after the breath holding ended.

This study was undertaken to see whether or not there was a marked change in the alveolar gases at breaking point in transition *a*) from rest to work and *b*) from work to rest. If some sort of proprioreceptors in the working limbs took part in the regulation of respiration during exercise they should start firing at *a* and cease such firing at *b*. However, the $pCO_2$ remained at the previous level in both cases (figs. 2 and 3). The $P_{A_{O_2}}$ showed larger changes but the values obtained were lowest in tests during work when the possible afferent impulses should be present. The circulation and blood components at the beginning of work (situation *a*) are to some degree related to that at rest. At the end of work (*b*) the increased circulatory rate continues at high level for a while, with blood composition similar to that during work. The breaking point thus seems to be based mainly on circulatory and metabolic factors. Judging from the breath-holding tests there is some sort of 'unawareness' in the respiratory center whether the muscular exercise, changing those factors, is actually going on or not.

The much higher 'tolerance' for $P_{A_{CO_2}}$ during breath holding in exercise than at rest cannot be explained. It is not a simple 'taking off' of the attention as the response is so different in different cycles of work. There must be some physiological mechanism involved. The alveolar $P_{A_{CO_2}}$ during exercise and normal breathing is actually some millimeters higher than at rest, especially the $P_{CO_2}$ of the end-tidal alveolar air, but the pulmonary ventilation is also higher.

The significance of the findings for the understanding of the regulation of respiration during work may be

obscure. Certainly breath holding is a rude interference in the respiration as well as in the circulation. From a theoretical viewpoint the results have, however, some special interest. Figure 4 presents alveolar $pCO_2$ and $pO_2$ at breaking point plotted in a $pCO_2$-$pO_2$ diagram. It summarizes data obtained at rest by Otis *et al.* (17) and the results from the present experiments at rest and with a fixed work load, but various $P_{I_{O_2}}$ inspired prior to the breath holding. Otis *et al* (17) suggested that exercise, either active or passive, should displace the curve obtained from rest experiments downward and to the right if reflexes from the working limbs contribute to the increased pulmonary ventilation during exercise. If this argument is valid, the present finding of a shift upward and to the left definitely speaks against this reflex theory.

## REFERENCES


1. DEJOURS, S. P. *J. physiol., Paris* 51: 163, 1959.
2. GRAY, J. S. *Pulmonary Ventilation and Its Physiological Regulation. III.* Springfield: Thomas, 1950, p. 82.
3. HILL, L. AND M. FLACK. *J. Physiol.* 37: 77, 1908.
4. MITHOEFER, J. C., C. D. STEVENS, H. W. RYDER AND J. MCGUIRE. *J. Appl. Physiol.* 5: 797, 1953.
5. FOWLER, W. S. *J. Appl. Physiol.* 6: 539, 1954.
6. CAIN, S. M. *J. Appl. Physiol.* 11: 87, 1957.
7. CHAPIN, J. L. *J. Appl. Physiol.* 8: 88, 1955.
8. DOUGLAS, C. G. AND J. S. HALDANE. *J. Physiol.* 38: 420, 1909.
9. RODBARD, S. *Am. J. Physiol.* 150: 142, 1947.
10. CAIN, S. M. *Fed. Proc.* 13: 22, 1954.
11. CRAIG, F. N. AND E. G. CUMMINGS. *J. Appl. Physiol.* 13: 30, 1958.
12. CRAIG, F. N. AND S. M. CAIN. *J. Appl. Physiol.* 10: 19, 1957.
13. HILL, L. AND M. FLACK. *J. Physiol.* 40: 347, 1910.
14. MUXWORTHY, J. F., JR. *WADC Tech. Rep. 6528*, 1951, pp. 479–482.
15. ADOLPH, E. F., F. D. NANCE AND M. S. SHILING. *Am. J. Physiol.* 87: 532, 1929.
16. STEVENS, C. D., E. B. FERRIS, J. P. WEBB, G. L. ENGEL AND M. LOGAN. *J. Clin. Invest.* 25: 723, 1946.
17. OTIS, A. B., H. RAHN AND W. O. FENN. *Am. J. Physiol.* 152: 674, 1948.