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Artero-venous difference of oxygen, cardiac output and stroke volume in function of the energy consumption

By

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With 6 figures in the text

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The cardiac output during muscular exercise was determined with a method previously described (BRANDI[6]).

26 experiments were carried out on two healthy subjects, B. R. and L. V., respectively 27 and 23 years old; they had a mild sport activity and they were familiar with experiments on respiration.

In each experiment the subject walked, or run, on a treadmill; when the steady-state was reached, 40—50 litres of expired air were collected in a Tissot spirometer having a capacity of 180 l and a sensitivity of 1.8 l/cm; this was analized for O_2 and CO_2 to determine $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$. PA_{CO_2} was continuously recorded by an infrared CO_2 meter.

From the rate of increase of PA_{CO_2} during a successive period of rirespiration in a 1 l capacity rubber bag, lasting not longer than 10—15 sec, the value of the artero-venous difference was calculated; dividing this value by R, the correspondent value for oxygen was obtained.

The artero-venous difference has been found to be approximately a linear function of the energy consumption:

$$A—V_{O_2} = a + b\,\dot{V}_{O_2} \tag{1}$$

the values of a and b for each subject were calculated by means of the least square method (Fig. 1 and 2).

Since the cardiac output, $\dot{Q}$, is

$$\dot{Q} = \frac{\dot{V}_{O_2}}{A—V_{O_2}} \tag{2}$$

substituting from (1) we obtain

$$\dot{Q} = \frac{\dot{V}_{O_2}}{a + b\,\dot{V}_{O_2}} \tag{3}$$

The cardiac output, calculated from $A—V_{CO_2}$ values is therefore an hyperbolic function of the oxygen consumption, as it can be seen in Fig. 3 and 4.

In all the experiments the heart rate was measured by means of an electrocardiograph, and the stroke volume found; in the subject L. V. this increases with the oxygen consumption up to 20—25 ml/kg · min while under more intense exercise it remains constant (Fig. 5).

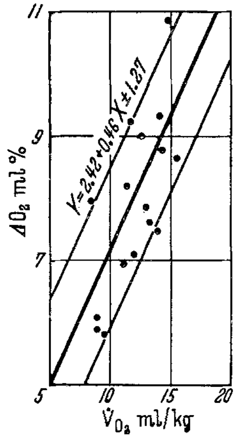

Fig. 1

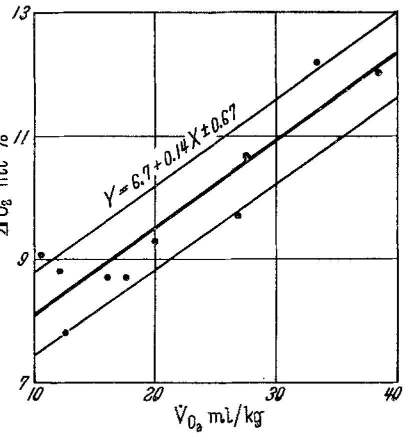

Fig. 2

Fig. 1. Artero-venous difference in O_2 ($\triangle_2$) against O_2 consumption ($\dot{V}o_2$, ml/kg) in subject B. R. (weight 64 kg, height 1,67 m)

Fig. 2. Artero-venous difference in O_2 ($\triangle o_2$) against O_2 consumption ($\dot{V}o_2$, ml/kg) in subject L. V. (weight 82 kg, height 1,78 m)

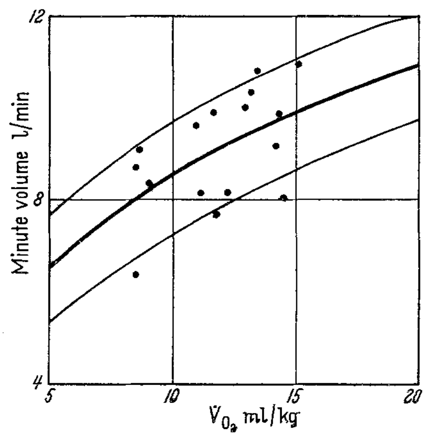

Fig. 3

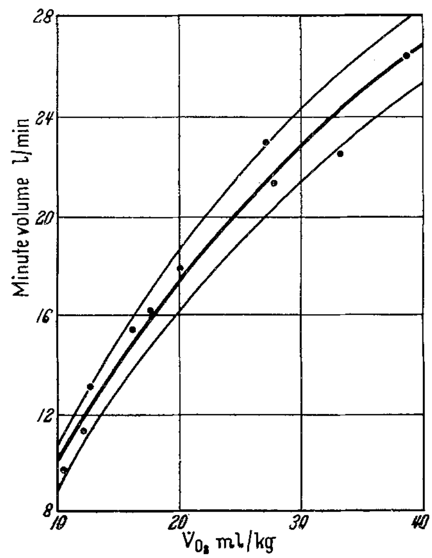

Fig. 4

Fig. 3. Cardiac output (minute volume, $\dot{Q}$, l/min) against O_2 consumption ($\dot{V}o_2$, ml/kg) in subject B. R.

Fig. 4. Cardiac output (minute volume, $\dot{Q}$, l/min) against O_2 consumption ($\dot{V}o_2$, ml/kg) in subject L. V.

Also in the subject B. R., whose O_2 consumption was never higher than 16 ml/kg · min, the stroke volume increased with metabolism (Fig. 6). In both cases the maximal increase is about 50% of the resting condition.

The old data of the literature concerning the relationship between stroke volume and work, obtained with indirect methods of determination, based on the rate of absorption of acetylene, on dye dilution, or on the

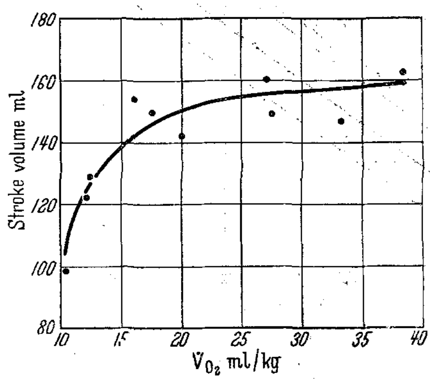

Fig. 5

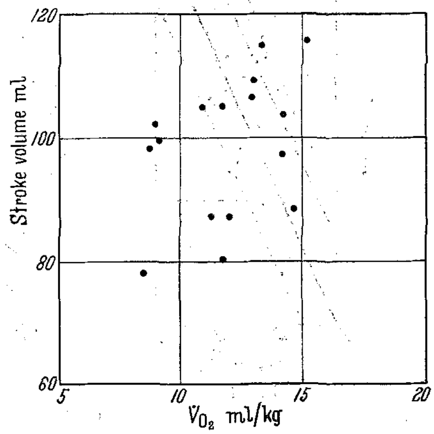

Fig. 6

Fig. 5. Stroke volume, ml, against O_2 consumption ($\dot{V}_{O_2}$, ml/kg) in subject L. V.

Fig. 6. Stroke volume, ml, against O_2 consumption ($\dot{V}_{O_2}$, ml/kg) in subject B. R.

CO_2 pressure of alveolar air supposed in equilibrium with the mixed venous blood, suggest that the stroke volume increases appreciably with the increase of metabolism (KROGH and LINDHARD, 1912[15], BOOTHBY, 1915[5], BOCK et al., 1928[4], CHRISTENSEN, 1931[8, 9]); other authors however (DOUGLAS and HALDANE, 1922[12], ASMUSSEN and NIELSEN, 1952[1, 2]), using the same methods, found that the stroke volume remains constant all through a large metabolic range. HENDERSON et al., 1927[14], found that in subjects athletically trained the stroke volume increased with metabolism, in normal remained constant.

The values of the stroke volume obtained with cardiac catheterism do not seem to show large changes with the increase of energy consumption (DEXTER et al., 1951[10], DONALD et al., 1955[11], BARRATT-BOYES and WOOD, 1957[3], and others[16]); however also with this method a significant increase of the stroke volume has been found (WARNER et al., 1953[17], CHAPMAN et al., 1954[7], FREEDMAN et al., 1955[13]).

It is not possible to evaluate how much of these differencies may be accounted for by errors of measurement and how much is due to individual difference. It seems likely, however, that the stroke volume increases during maximal work; considering that the oxygen consumption can increase 12 fold while the heart rate increases only 3 fold, on the assumption that the stroke volume is constant, the artero-venous difference in oxygen, which in resting conditions is about 4.5 ml%, would increase 4 fold, i. e. should rise to 18 ml% and the saturation of mixed venous blood should be reduced to less than 10%: the highest values of A—V difference obtained with both the dye dilution and the acetylene methods, during strenuous muscular exercise, requiring 3 l/min of oxygen intake and involving a hearth rate of 173, was 14.5% (ASMUSSEN and NIELSEN[1]), and this is still far from the value as calculated above.

There are no data of A—V difference obtained with direct Fick method during maximal work. Also by extrapolation of the data obtained with this method by DONALD et al.[11], it appears likely that the figure of 18 ml% is too high a value even at maximal work; it follows that some increase of stroke volume must take place to account for the observed difference.

Summary

Artero-venous difference and cardiac output have been investigated on two healthy subjects at work and related to the oxygen consumption.

The A—V difference in oxygen has been found to be approximately a linear function of the energy consumption; cardiac output is, therefore, an hyperbolic function of metabolism.

Cardiac stroke volume increases somewhat with increasing metabolism: a further increase of metabolism, from ab. $^1/_3$ of the maximum, takes place without a further increase of the stroke volume.

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