Metabolism of Oxygen-15

OXYGEN-15 has been prepared by deuteron bombardment of nitrogen molecules in the Medical Research Council cyclotron at Hammersmith Hospital. Gases labelled with this isotope, which has a half-life of only 2 min., can be inhaled into the lungs and detected by external scintillation counters. The initial counting-rate in a given region of the lung is proportional to the regional ventilation[1], and the rate of removal from the region during breath-holding is a measure of the regional blood-flow[2].

During some of these experiments the radioactivity in the blood was measured at intervals after the breath was taken. Blood samples were taken from a forearm vein in four subjects between 15 sec. and 4 min. after the beginning of the breath of air, the oxygen of which was labelled with oxygen-15. The samples were heparinized and centrifuged for 2 min. to separate the cells from the plasma. The radioactivity of 2-ml. samples of red cells and plasma was measured in a sensitive 'well' type crystal scintillation counter. In a further study, 20 ml. fresh blood at 37° C. was labelled with oxygen-15 by bubbling a stream of the isotope in air through it. A sample was taken from the blood immediately after labelling and after 5-min. incubation at 37° C. and the radioactivity in both red cells and plasma was measured.

A typical example of the changes in the radioactivity in the plasma and red cells for 4 min. following the breath of oxygen-15 (corrected for radioactive decay to a common zero time) is shown in Table 1. The radioactivity in the plasma increased rapidly and the red cell activity fell during the 4 min. of observation. In consequence the proportion of the radioactivity in the plasma increased from 10·6 per cent after 30 sec. to 53 per cent after 240 sec. The results in all four normal subjects followed a similar pattern.

When blood was labelled *in vitro* only 1 per cent of the radioactivity appeared in the plasma, and this proportion did not alter significantly after 5 min. incubation. This is the proportion which would be expected from the known partition of molecular oxygen between red cells and plasma.

The rising plasma radioactivity found *in vivo* must represent the return to the plasma of oxygen-15 which has been metabolized in the body cells. The results show in a striking way how rapidly this takes place. An appreciable fraction of the inhaled molecules of oxygen-15 is transported by the blood to the tissues, diffuses into the tissue cells, is metabolized and then diffuses back into the blood within 30 sec. of the beginning of the breath.

An attempt was made to fractionate the radioactivity in the plasma into carbon dioxide and water by acidifying and shaking under vacuum, but none of the radioactivity could be removed. This is because oxygen-15, which is metabolized to carbon dioxide, becomes freely exchangeable with the oxygen in water when bicarbonate is formed in the blood, and therefore cannot be separated.

Table 1. RED CELL AND PLASMA RADIOACTIVITY CORRECTED FOR RADIOACTIVE DECAY TO A COMMON ZERO TIME

Time after breath (sec.)	Red cell radioactivity (counts/min./ml.)	Plasma radioactivity (counts/min./ml.)	Ratio plasma/red cell activity (per cent)
30	102,000	10,800	10·6
60	104,000	12,500	12
120	78,000	21,600	28
240	55,000	29,000	53

C. T. DOLLERY
J. B. WEST

Department of Medicine,
Postgraduate Medical School of London,
Hammersmith Hospital,
Ducane Road,
London, W.12.

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The Membrane Potential of the Anterior Byssus Retractor Muscle of *Mytilus edulis*

As yet no reports of intracellular recording from the anterior byssus retractor muscle appear to have been made. The membrane potential can be measured with coventional glass micro-pipettes filled with 2·5 *M* potassium chloride, but satisfactory recordings have only been obtained with electrodes possessing tips of about 0·5μ or less in external diameter. Very few such electrodes are free from large tip potentials; but some satisfactory recordings have been obtained from the muscle *in situ*, using selected electrodes.

The animal was opened and the muscles of one side cut through leaving the byssus attached to one anterior byssus retractor muscle in one half of the shell. The shell was mounted horizontally and filled with sea water, and tissue covering the anterior byssus retractor muscle was carefully cleaned away, leaving the muscle exposed. The recording electrode was connected direct to the grid of a cathode follower (6*BS*7 selected for low grid current) through a silver–silver chloride/sea water–agar bridge. The sea water in the shell was earthed via a similar bridge. Calibration voltages were applied across a 50-ohm resistor between earth and the shell. Signals from the muscle were taken through a d.c. amplifier and displayed on a pen-writing oscillograph. Membrane potentials recorded fell within the range 30–50 mV., but potentials from the same muscle were within a range of 7 mV. or less. The mean value was 39 $\pm$ 2 mV. (*S.E.* of 14 observations).

D. H. PAUL

Department of Zoology and
Comparative Physiology,
The University,
Birmingham.

Effect of Size of Pupil on Visual Acuity

DENTON[1] has suggested that a possible function of the pupil light reflex might be to provide apertures optimum for visual acuity at different light intensities. At low levels of luminance visual acuity is probably limited by the rate at which light quanta activate the retinal receptors[2], and thus a large pupil admitting the maximum amount of light would appear to be advantageous. As the luminance is increased into the photopic range, loss of image contrast due to optical aberrations, diffraction, and stray light also tend to limit visual acuity, although the fineness of the retinal cone mosaic may ultimately become the limiting factor. The decrease in pupil size which normally accompanies increase of illumination should reduce to some extent the deleterious effects on visual acuity of the optical aberrations. However, it has never