lowing a single inspiration of radioactive gas allow comparison of the relative ventilation and blood flow of the two regions being compared. These are not discreet anatomical regions as measured during bronchospirometry. Tests must, of course, be conducted in the immediate vicinity of a cyclotron. (*Dyson, N. A., and others: Studies of Regional Lung Function Using Radioactive Gas, Brit. Med. J. 1: 231 (Jan. 23) 1960.*)

INTERCOSTAL MUSCLES Intercostal muscle function in man has been investigated by electromyographic techniques capable of distinguishing the activity in the various layers. The external intercostal muscle is purely inspiratory in action. The internal layer is an expiratory muscle with the exception of its parasternal part which is inspiratory. The transversus thoracis contributes to expiratory effort and is well developed only in those regions where there is a deficiency in one or another of the layers of intercostal muscle. In fully relaxed subjects in the supine position, activity occurs only in two restricted parts of the intercostal muscles; namely, the internal intercostal in the parasternal region during inspiration and in the lower, lateral part of the chest during expiration. More vigorous respiratory effort brings the layers of muscle into reciprocal action all over the chest wall, the external layer being inspiratory and the internal layer expiratory. (*Taylor, A.: The Contribution of the Intercostal Muscles to the Effort of Respiration in Man, J. Physiol. 151: 390 (May) 1960.*)

NITROGEN DESATURATION Nitrogen elimination from the tissues during oxygen breathing seems to be released in an exponential way in three stages. First, there is a rapid phase with an elimination half-time of about 15 minutes, then a slower phase with a half-time of 12 to 13 minutes and lastly a slow phase with a half-time of about 110 to 200 minutes. It is probable that the first phase corresponds to the nitrogen from highly vascular tissues such as liver, brain, heart, intestines, etc., the middle phase to nitrogen mainly from the muscles, and the slowest to nitrogen mainly from the fat. Probably the free intestinal nitrogen escapes with the third fraction. The nitrogen desaturation rate of the body can be followed by measuring the nitrogen content of end-tidal air during oxygen breathing. The desaturation curves may be separated into exponential fractions which can be used to calculate the amount of fat and muscle tissue in the subjects. The blood flow through these tissues may also be calculated from these data. (*Lundin, G.: Nitrogen Elimination from Tissues During Oxygen Breathing and Its Relationship to the Fat : Muscle Ratio and the Localization of Bends, J. Physiol. 152: 167 (June) 1960.*)

CARBON MONOXIDE POISONING The effect of cytochrome C on the oxygen consumption of the brain, liver and heart muscle of guinea pigs poisoned with carbon monoxide has been investigated manometrically. Carbon monoxide causes a decrease in the oxygen consumption of slices from these three organs. Cytochrome C, when added *in vitro*, can reverse this effect. The increase in oxygen consumption produced by cytochrome C in poisoned tissues was 28 per cent for brain, 31.2 per cent for liver and 56.4 per cent for heart muscle. In brain, normal oxygen consumption was restored by 3.0 μg. of cytochrome C/g. of wet weight tissue. A level of 6.0 μg. increased it above normal. This may be explained by supposing that a situation exists in the carbon monoxide poisoned slices that is analogous to the phenomenon of oxygen debt accumulated after severe exercises. In both cases there is a disproportion between the oxygen requirements of the cells and the amount of oxygen which is available. Cytochrome C has the effect of reducing this disproportion in carbon monoxide poisoning by increasing the availability of oxygen. Thus this effect of carbon monoxide poisoning is reversible. Cytochrome C when added *in vitro* had no effect on the oxygen consumption of normal brain slices. This is because there is no lack of available oxygen for the cellular oxidation process. (*Dutkiewicz, J. S., and others: Effect of Cytochrome C on Oxygen Consumption of Tissues of Normal and CO-Poisoned Animals, J. Physiol, 152: 482 (July) 1960.*)