Summary. The effect of acute anoxia on the *economy of effort index* of Wiggers and Katz was studied on medical students by allowing them to rebreath decreasing oxygen mixtures and holding these mixtures at 14, 12 and 10% oxygen, respectively, long enough to record subclavian pulses and determine brachial blood pressures leisurely.

In 20 subjects, the index increased during rebreathing of 14 and 12% oxygen; in 12, it increased further when the oxygen percent was reduced to 10; in 8 it began to show slight signs of decreasing. Three subjects showed no significant increase or a slight decrease. The possible use of the method in testing favorable and unfavorable responses of individuals to low barometric pressures is mentioned.

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Limitations of Myocardial Recovery from Fibrillation Through Countershock.*

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In revival of the ventricles from fibrillation by use of countershock, stress is generally laid on the limited survival of the central nervous system, and particularly of the cerebrum. It is less commonly stressed that the myocardium likewise can survive the anoxia imposed by complete interruption of the coronary circulation for a very limited time.

Experiments were, therefore, carried out to determine how the duration of fibrillation influences recovery and to estimate the maximum period of fibrillation which may be followed by recovery without the aid of cardiac massage.

Procedure. In dogs under sodium barbital anesthesia, the heart was exposed under mild artificial respiration and aortic pressures were recorded optically by a Gregg manometer. Records were taken before and during fibrillation produced by application of a brief tetanic current and at various intervals after recovery following a series of A.C. countershocks. In these records, systolic and diastolic pressures were measured in representative cycles and plotted. Typical results are shown in Fig. 1.

Results. After fibrillation, less than 10 seconds in duration,

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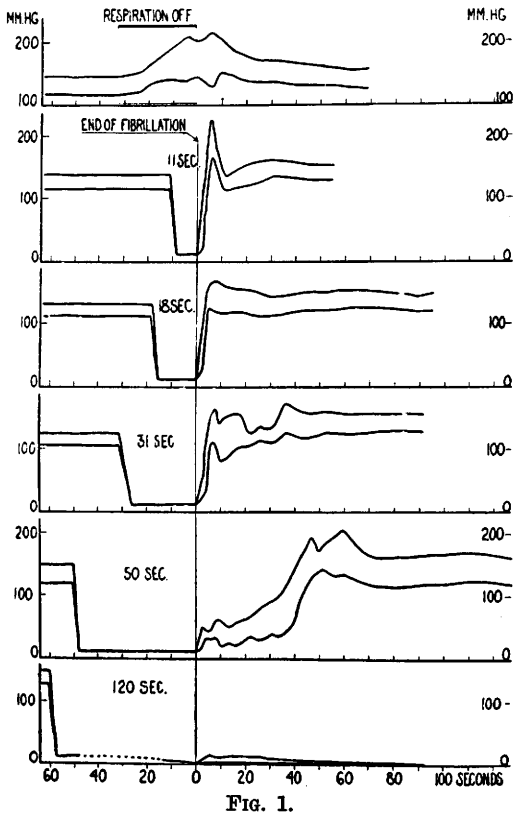

FIG. 1.

Plots showing systolic (upper lines) and diastolic (lower lines) pressures before, during, and after various intervals of ventricular fibrillation.

normal pressures were restored within a few beats. With increasingly longer periods of fibrillation the potential capacity for prompt recovery decreases progressively. The manner of recovery is determined in part by the vigor of the rhythmic beats reëstablished, but also by the type of cardiac rhythm which develops, the degree of venous congestion and the intensity of the peripheral vasoconstriction induced by the period of general asphyxia. As shown in the upper graph of Fig. 1, a short period of asphyxia itself elevates systolic and diastolic blood pressures and increases the pulse pressure. Consequently, if fibrillation does not persist long enough to weaken the contractile power of the myocardium, such an asphyxial effect is reflected in the recovery of blood pressure. This is evident in the fibrillations lasting 11, 18 and 33 seconds in Fig. 1.

If fibrillation is prolonged—roughly more than ½ minute—this initial overcompensation in blood pressure is replaced by a slow

progressive rise. For 20 to 40 seconds the heart struggles along with slow beats. As the coronary circulation and vigor of contractions gradually improve, the pressures reach supernormal levels which gradually fade back to normal ranges.

If fibrillation lasts longer—roughly a minute or more—even such a slow recovery may fail. After 2 minutes of fibrillation, regular beats are started; but under the conditions of our experiments they never become strong enough to give the myocardium sufficient nourishment for revival. Blood pressures remain low and death supervenes.

Conclusions. The dog's ventricles did not generally survive interruption of coronary circulation during fibrillation for more than about 1 minute, in the sense that useful coördinated beats sufficient for recovery are reëstablished without massage. These results caution us not to entertain too high hopes of resuscitating human hearts by the countershock method. Countershock can stop fibrillation; but complete anoxia beyond one minute generally prevents a resumption of beats sufficiently vigorous to reëstablish viable blood pressures.

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Antihemorrhagic Compounds as Growth Factors for the Johne's Bacillus.*

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One of the first bacterial growth factors to be studied in any detail was a substance necessary for the growth of the Johne's bacillus *(Mycobacterium paratuberculosis)*. Twort and Ingram[1] showed that certain other acid-fast bacteria including the tubercle bacillus contained a necessary growth factor for the original cultivation of the Johne's bacillus. Crude concentrates of the active substance were prepared from *Mycobacterium phlei.* We have long been interested in the growth of the Johne's bacillus, and for some time have attempted to isolate the active growth factor. In this paper

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1 Twort, F. W., and Ingram, G. L. Y., *Johne's Disease,* London, 1913.