in the response of the muscle, but after successive applications of pressure there was no increase in tension above the control value. It was further observed that if a muscle, when immersed in an oxygenated normal Ringer's solution, was fatigued by repeated stimulation, its response under pressure was less than the control, *i.e.*, the action of pressure on the tension developed by the muscle in response to a single stimulus is opposite in direction to its action in a fresh preparation. It seems probable that oxygen lack and acid solutions produce their effects by promoting fatigue. The following points have been established regarding the reversal of the pressure effect in the fatigued muscle: (1) The degree of depressed is roughly proportional to the pressure employed. (2) The tension developed by the fatigued muscle is commonly reduced to about one half by a pressure of 1,600 pounds per square inch. (3) The response very quickly, possibly instantaneously, returns to the control value when the pressure is released.

In the case of cardiac muscle the stimulating action of pressure on the tension can no longer be obtained when the temperature is reduced to 5° C., and we have secured some evidence of a reversal occurring at that point. (In press, *American Journal of Physiology.*) Studies now in progress on striated muscle indicate that there is a critical point at approximately 13° C. at which the stimulating action of pressure passes into depression. At a few degrees below this temperature the depressive action becomes marked and becomes greater as the pressure is increased. A few preparations have shown, in the vicinity of the critical temperature, a stimulation under the lower pressures (1,000 pounds) and depression at higher pressures (2,100 pounds). As the temperature is lowered pressure causes a further reduction in the response until at about 1° C. high pressure (2,100 pounds) almost completely suppresses the development of tension. At room temperature the magnitude of the response of the muscle continues to increase with increasing pressure up to at least 2,100 pounds per square inch, which is the greatest pressure we have employed.

When pressure is released the muscle again responds with the development of tension, the record differing in no way from the control taken before the application of pressure to the cold muscle. When such a muscle is warmed to room temperature, pressure once more causes an increase in the development of tension corresponding closely with the result obtained before the muscle was chilled.

Measurements of the duration of the phases of contraction and relaxation of cardiac muscle show that moderate pressures result in only a slight prolongation of these periods, and the same appears to hold for striated muscle at room temperature. On the other hand, at the lower temperatures pressure causes, along with the depression in tension development, a striking prolongation in the duration of the phases of contraction and relaxation which, for the higher pressures, may reach 100 per cent.

Certain interesting differences of behavior between cardiac and striated muscle are indicated by these results. In the first place, it appears impossible to fatigue cardiac muscle in such a manner as to produce a reversal of the pressure effect such as occurs in striated muscle, and in the second place the critical temperature, in which reversal of the pressure effect occurs, is about 8° C. lower in cardiac muscle.

The significance of these results in relation to the mechanism of muscular contraction will be discussed at a later date in connection with the publication of the complete data.

SUMMARY

The usual augmenting action of pressure on the tension developed by striated muscle in response to a single stimulus changes to depression on fatiguing the muscle. On release of the pressure the tension of the single twitch again reaches the control value.

A similar reversal may be brought about by cooling the muscle, the change usually occurring at about 13° C. Below this temperature the depression is roughly proportional to the pressure. At about 1° C. the tension development may be completely inhibited by high pressure. The muscle responds normally upon release of the pressure, and upon warming pressure again causes augmentation of contraction.

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