EXERCISE AND THE SKELETAL MUSCLE

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Introduction

Skeletal muscles pass over joints and are the power units which cause movement as a result of their contraction or shortening. Disuse results in atrophy. This basic fact can be confirmed by anyone who has had a limb immobilised whether mechanically in a plaster cast or by denervation. In fact, bedrest results in an increase in urinary nitrogen (Cuthbertson 1929.) It is probable that most of this nitrogen has its origin in the cytoplasm of the atrophied muscles (Keys 1948.) On the other hand, activity normally contributes towards hypertrophy together with other physiological changes associated with increased performance. It is, therefore, not unnatural that Karpovich 1965 states that muscles stress the slogan that function makes an organ.

Some Factors Affecting the Force of Contraction

All other things being equal, there is a positive linear relationship between the physiological cross-section of a muscle and the amount of force which it can exert (de Vries 1966 and Morris 1948.) Thus hypertrophy increases strength and Huxley 1958 has indicated that excised muscle can exert a force of some 42 pounds per square inch of cross-section. Moreover, it would appear that strength is not the only fitness component to be affected. Several investigators have indicated that the correlation between strength and absolute local muscle endurance is in the range of .75 to .97 (Caldwell 1963 and Start 1964.) Most authorities also agree that these two fitness components are necessary during the performance of any physical activity.

However, it must be realised that strength per unit of cross-section is purely a theoretical concept since in muscle tissue in vivo the force of contraction will largely be dependent on the nutritional status of the muscle together with the degree of fatigue, the length at which the muscle is working, and the angle of pull, the distance the muscle is inserted from the joint, and the co-ordination of innervation.

Asmussen, quoted by Falls 1968, has indicated from length tension diagrams that the muscle fibre attains its maximum isometric tension when slightly stretched above the resting equilibrium state. Wilkie 1949 has also confirmed that tension increases at greater muscle lengths. It has, therefore, become a maxim in Physical Education to put a muscle on stretch immediately before the performance of mechanical work. The sprint start in which the quadriceps are put on stretch

prior to the knee extension is a classical practical example. Muscle consists of a contractile component and an elastic element. The latter component occurs in the tendon and in the connective tissue of the muscle. The contractile component shortens as a result of stimulation and activates the elastic component thus stretching it and setting up tension. Tuttle and Schottelius 1961 therefore state that the pre-stretching probably takes up the excess slack of the elastic component thus allowing the energy of contraction to exert maximum effort on the resistance.

Force is most effective when applied at 90° to a lever and this angle of pull in muscle tissue in vivo only if ever occurs during inner range movement when the force of contraction is greatly diminished. It would therefore appear that the most mechanically advantageous position for intact skeletal muscle is one in which there is a compromise between the length at which the muscle is working and its angle of pull on the bone lever. Clarke 1966 has accordingly ascertained that the joint angles which permit the application of maximal force during the knee extension and elbow flexion are 120° and 115° respectively.

The distance of insertion of the muscle from the joint influences the length of the force arm which in turn regulates the turning moment about a joint. Munrow 1955 states that a change of $\frac{1}{4}$ " in the insertion of the biceps muscle can make a 10% difference in the effective force exerted. The influence of somatotype must be considered and it is generally maintained that the mesomorph has better leverage than the ectomorph (Bunn 1955.)

The Nature of Hypertrophy.

The classical work concerning the nature of hypertrophy in skeletal muscles was presented in 1897 by Professor Morpurgo of Sienna. He removed the sartorius muscle from the left side of 2 dogs and then subjected them to a 2 month running programme. The treadmill was initially turned mechanically but thereafter the dogs ran spontaneously according to their strength. The right sartorius muscles were removed after the experimental treatment. His results indicate that the training resulted in an increased cross-sectional hypertrophy of 53%. The 42% increase in the diameter of each fibre was attributed to an augmented sarcoplasm. No lengthening of the fibres has occurred. Such lengthening would only occur if the distance between the tendinous attachments at the origin and insertion had been increased pathologically. However, the most significant factor was that the number of fibres had not increased. It would therefore appear that the increased cross-section was resultant from an increase in the size of the muscle cells i.e. hypertrophy, as opposed to hyperplasia. The muscle fibres were of varied diameters at the beginning of the experiment whereas at the conclusion they had all grown to nearly the maximal size. This limit is probably determined by rate at which nutrients can diffuse into the centre of the cell. Obviously, the cell cannot be fed to optimum if it is too large.

Morpurgo's data were subsequently confirmed by Kleeberger but his findings were not published. He subjected the left fronto-scutularis muscle, the muscle with which the rabbit prides up its ears, to unipolar stimulation from a Harvard coil for two daily periods of two hours each. He used three rabbits, and the experimental treatment lasted 42 days. The relative cross-section of one of the muscles increased by 90%. Here again there was no increase in the number of muscle cells. The literature does not appear to indicate the age at which muscle cells cease to multiply or whether this age is the same in active and inactive organisms. In contrast, it must be emphasised that many other organs, notably the liver and kidneys, together with the circulatory system exhibit hyperplasia in response to physiological stress.

An examination of muscle fibres under the electron microscope reveals that they are composed of myofibrils which contain the contractile proteins actin and myosin. The proteins are suspended in a nutrient sarcoplasm and arranged in functional units or sarcomeres. Subsequent research therefore began to focus on the effects of exercise on the ratio of protein / sarcoplasm and on the number of myofilaments.

An animal experiment by Helander in 1961 was designed to determine the effect of training and detraining on the ratio of myofilaments to sarcoplasm. Results indicated that muscle hypertrophy resultant from training is accompanied by an increase in the nitrogen component of the myofilament as opposed to an increase in the amount of sarcoplasm. This disproved Morpurgo's original contention of an increase in sarcoplasm. Holmes and Rasch in 1958 ascertained the effect of a 7 week progressive training period on the number of myofibrils of the rat sartorius muscle at 7 different cross-sectional levels. No statistically significant differences were observed between test and control group animals. However, the number of myofibrils per fibre in the experimental animals was more variable than in the control group. The pattern of distribution suggested increases in the number of myofibrils per fibre at the ends of the muscles. A twofold increase in the number of myofibrils per fibre has been reported by Denny-Brown 1964. He removed the gastrocnemius muscle from one side of a cat. Now the cat is a jumping animal and we can therefore assume dynamic work of high intensity during the 3 months training period. These findings were further supported the same year by Goldspink who reported a three to fourfold increment in the number of myofibrils in the biceps brachii of young mice. Here again the Principle of Overload was used and the animals were motivated to lift progressively increasing weights by an ingenious food-rewarding device. Thus the literature would seem to indicate that forceful dynamic work is associated with an increase in the number of myofibrils within the muscle cell. As indicated earlier, the number of muscle cells remains unchanged.

It is a well known fact that most types of progressive physical exercise result in muscular hypertrophy. In 1925 Petow and Siebert designed an experiment to determine how much exercise is necessary to induce hypertrophy. Heavier muscles were found in rats that had run at the faster rate for short durations being dependent on the work rate or power output and not on the total work performed. The muscles hypertrophied to a new plateau until the running speed was increased. This research was the first scientific validation of the Overload Principle or the Principle of Progressive Resistance Exercise. It was not until 1956 that Hellebrandt and Houtz demonstrated that overload training results in an increase in local muscle endurance and power. No such effects were observed when training with an underload. Milo of Crotona was probably the first person to use the modern principle of acquiring strength by gradually increasing the resistance. Gardiner 1930 tells us that during the 6th century B.C. Milo carried a bull calf every day from its birth until maturity when he carried it twice around the stadium at the Olympic Games.

Nowadays athletes realise that muscular hypertrophy is related to the intensity or rate of work and not to the total work performed. Their training programmes are therefore based accordingly. Performers who exert short explosive efforts e.g. sprinters, shot putters, and weightlifters normally have large musculatures. In contrast, athletes who rely on less intensive but more protracted efforts are normally of only average musculature e.g. long distance runners, tennis players, and basketball players. However, the influence of somatotype must also be considered since there will be a tendency for the heavier boned mesomorph to gravitate towards activities requiring strength and local muscle endurance whereas the lighter boned ectomorph is more suited to events requiring circulo-respiratory endurance.

The Effect of Exercise on the Tendons and Ligaments

The connective tissue at the origin and insertion of the muscle merges into a tendon which is attached to the periosteum of the bone by means of Sharpey's fibres. Any tensile force generated by the muscle must accordingly be transmitted to the anatomical lever via the tendon in order for movement to take place. It is therefore pertinent that training animals at an early age has been shown to result in an enlarged cross-sectional area of the Achilles tendon and gastrocnemius muscle (Ingelmark 1945.) Only the muscle tissue hypertrophied when the same experiments were performed on older animals. However, Viidik later in 1968 indicated that training made medium sized tendons stronger when tested alone even though no quantitative changes occurred.

The effect of exercise on the ligaments which reinforce the joints has also been the subject of research. In fact, the knee is one of the most vulnerable joints of the athlete's anatomy in today's high velocity contact sports. Viidik

1968 studied the effects of training in a running machine on 19 rabbits whilst 17 of the same stock were used as controls. The rabbits were 3 months old at the start of the training period and the tests were performed 40 weeks later. Tensile strength of the anterior cruciate ligament of the knee joint was significantly increased as a result of training. The failure site on the specimens was mostly a bony attachment. Deformation at maximum load values also tended to increase with training thus indicating altered viscous properties. Adams 1966 has also indicated with rats that systematic running exercise resulted in an increase in the strength of attachment of the medial collateral ligament to the tibia and femur. Running on an uneven surface promoted a greater increase in ligament strength than running on a smooth surface. Both differences were significant beyond the $\cdot 01$ level.

Capillarisation

The effect of circulo-respiratory type exercise on the capillaries in gastrocnemius and cardiac muscle was studied as long ago as 1936. Petren, Sjöstrand, and Sylven examined recently killed guinea pigs that they exercised by injecting 1 cubic cm of Histamine into the heart cavity. This resulted in dilation of the capillaries, which facilitated ease of the counting, and the pigs died of cerebral anaemia due to an inadequate blood supply. Training resulted in an approximate increase of 50% in the number of capillaries per sq. mm of muscle not used in the training exercise. Andersen, quoted by Falls, 1968, has stressed that this increased capillarisation would considerably decrease the distance the molecules must diffuse in passing from the blood to the mitochondria of the cells. This then, is one reason why the trained person possesses superior endurance. There are actually more pathways for the blood to come into the muscle. An histological examination revealed that the guinea pig gastrocnemius muscle cells showed no difference in size after training even though the work capacity of the muscles had increased. Muscular hypertrophy is often a consequence of exercise but it does not always occur when the work capacity or endurance is improved by training. Many investigators are of the opinion that improvement in the blood supply to active muscles is a major factor for endurance development and hypertrophy a major factor in strength development. Petren and co-workers stated that these extra capillaries disappeared during a detraining period of 90 days. Thus one of the remarkable facts to be learned about exercise physiology is that all the changes that exercise produces in the body are reversible. It has more recently been indicated by Vanderhoof, Imig, and Hines (1961) that the vascular bed is capable of opening up to a greater extent during and following exercise as endurance training progresses.

Myoglobin

Karpovich (1965) states that trained muscles are darker than untrained muscles due to an increase in myoglobin. It is relevant that the myoglobin of the muscle sarcoplasm has a greater affinity for oxygen than haemoglobin and it also dissociates oxygen faster. The fact that exercise increases the amount of muscle myoglobin has been experimentally demonstrated by Whipple 1926, who showed that hunting dogs possess significantly more myoglobin per 100 gms of muscle tissue than house dogs. This would obviously increase the oxidative potential of the cell. It is unlikely that strength work per se would result in such a large increase in myoglobin when compared with unexercised controls. A system must be stressed before physiological adaptation will occur and the major stressor of the oxygen transport system is progressive circulo-respiratory endurance type work.

It has been postulated that the reoxygenation of myoglobin is one of the factors which contributes towards the alactacid oxygen debt. Åstrand 1960, de Vries 1966, Karpovitch 1965 and Margaria, 1933. Åstrand et al estimated that approximately 100-300 ml of oxygen are required to reoxygenate the muscle myoglobin during the repayment of the alactacid oxygen debt. The oxygen debt, it must be realised enables an athlete to perform exercise that requires far more oxygen than can conceivably be supplied to the muscle cells by the circulo-respiratory systems during the duration of the activity.

The effect of a decreased partial pressure of oxygen in the atmospheric air on muscle myoglobin has been noted by Regnafarjie 1962. Muscle biopsies indicated that persons living at an altitude of 5,000 metres have a greater myoglobin content of the sartorius muscle when compared with sea-level controls. This increased oxygen storage in the muscle was probably a direct result of the decreased diffusion gradient of oxygen across the alveolar membrane.

Adenosine Triphosphate and Creatine Phosphate

According to Hultman et al 1967 human skeletal muscle contains approximately 2.43 mmoles of ATP per 100 gms of dry tissue. This organic compound is the universal energy currency of the cell and the cleavage of the terminal phosphate radical provides the energy for metabolic processes such as muscular contraction, (Bell, Davidson & Scarborough, 1965, Rose, 1966.)

The ATP level is therefore dynamically related to the energy status of the cell. Harris 1964 has concluded that training results in an increase in the base concentration of ATP in the skeletal muscles of exercised rats but this increase was not statistically significant. Unfortunately the amount of ATP available at any moment is so small that it can maintain maximum muscle contraction for only $\frac{1}{2}$ second. The equations indicate how an emergency mechanism, capable of operating immediately, is provided by PC which can transfer its high energy phosphate bond to ADP thus re-synthesising ATP. A training period of 5 days has been shown to result in an increase in the PC content of rabbit skeletal muscle (Palladin et al 1928.) However, Hultman et al have again indicated that the level of PC in human skeletal muscle is approximately 6.78 mmoles/100 gms of dry weight. This emergency mechanism lasts for about 5 seconds until the PC stores are exhausted. Adenosine triphosphate is subsequently resynthesised by the degradation of fat and glucose.

The Glycogen Stores

Karpovich informs us that as much as 7 ounces of glycogen may be stored in the liver. It is generally maintained that during exercise the glycogen is mobilised from the liver by the action of epinephrine and nor-epinephrine from the adrenal medulla and post-ganglionic fibres of the sympathetic nervous system respectively. However, Gollnick et al 1967 have reported that both liver and muscle glycogen are depleted in the rat during exercise after adrenalectomy, adrenomedullectomy and ganglionic blockage. It is therefore probable that the mobilisation of the glycogen stores during exercise is resultant from the operation of several mechanisms. Nevertheless, liver glycogen is released into the blood as free glucose by the action of the enzyme glucose 6-phosphatase and then transported to the active muscle. This enzyme is not contained within the skeletal muscle. The glycogen reserves of non-working muscles cannot therefore be transferred to active muscles.

Respiratory quotient measurements and muscle biopsies on man during heavy physical exertion indicate that glucose is readily used as a fuel for the resynthesis of ATP (Borgstrom et al 1967.) Wakabayashi 1928 has indicated that the livers of the untrained rats were almost completely depleted of glycogen after exhaustive treadmill running whilst the same workload did not materially affect the liver glycogen content in the trained rats. This is of significance since Rowell, Masoro, and Spencer 1965 have estimated in man that the glucose output from the liver is 300mg/min during prolonged exercise. It has also been experimentally demonstrated with animals that physical activity increases the glycogen content of the exercised muscles (Emben and Habs 1926.) More recently Bergstrom et al have reported that the glycogen content of human skeletal muscle can be controlled by the diet. The normal average muscle

glycogen content is 1.5 gms% (Borgstrom et al 1967.) The mean value for 3 subjects after 3 days on a carbohydrate diet was 2.53 gms%. Six subjects who were on a fat/protein diet for 3 days exercised to exhaustion and then went on a carbohydrate diet for 3 days. They registered mean muscle glycogen content of the sartorius muscle of 3.70 gms%. Thus a period of carbohydrate free diet followed by exercise to exhaustion seems to further stimulate glycogen synthesis when carbohydrates are given. The enhancement of glycogen syntheses was localised to the exercised muscles and did not affect other muscle groups. The subjects who were on the fat/protein, mixed, and carbohydrate diets exhibited mean maximal work times of 57, 114, and 167 minutes respectively at workloads requiring 75% of the maximal aerobic capacity. It would therefore appear that an individual's ability to delay fatigue at this work level is a function of the glycogen content of the muscles and liver which in turn depend on the pre-exercise diet.

Exercise and the Mitochondria

The mitochondrion is the site of aerobic energy release and consequently has become known as "the powerhouse of the cell". It is therefore significant that Gdlnick & King 1969 have indicated that the number and size of mitochondria in rat skeletal muscle is increased as a result of a circulo-respiratory endurance type programme of 12 weeks duration. Such a training effect would obviously increase the oxidative potential of the cell. The authors postulated that this could be a contributory factor towards the lower blood lactates exhibited by fit subjects when compared with unfit controls after a standardised workload. Nevertheless, this effect has not been observed in man. Saltin et al 1968 have indicated that the size and number of mitochondria in the quadriceps muscle of man seem to be un-affected by bedrest or training. However, the duration of the physical conditioning programme in this study was only 55 days. It would appear that further research on man should be done in this area.

The Myoneural Junction

Research seems to have concentrated on the effects of training on the muscle tissue itself. Little work appears to have been conducted on the effect of exercise on the neurological apparatus which innervates the skeletal muscle. The only study located indicated that 14 days of femur immobilisation resulted in a large decrease of the myoneural junction area in the gastrocnemius muscle of the male rat, (Cole 1960.) This is in accordance with Karpovich's suggestion that training increases the facility of transmission of the nerve impulse across the motor end plate in the muscle fibre.

Conclusion

This paper has attempted to discuss some of the effects of exercise on skeletal muscle and to indicate how these effects are conducive to increased performance. A review of the literature indicates that most of the experimental work has been conducted on guinea pigs and rats. The big advantages here are that the experimental treatments can be carefully controlled and the animals killed at the end of the experiments. However, the extent to which the research findings can be fully extrapolated to man is debatable.

In conclusion it must be noted that the level of performance is not solely a physiological phenomenon. Ikai and Steinhaus 1961 have postulated that performance is limited by the psychological limit whereas capacity is governed by the physiological limit. There is considerable inter-individual variability in the difference between these 2 limits but capacity is almost always greater than performance. These investigators indicated that the ergogenic aids of shot and shout stimuli, hypnosis, and the administration of amphetamine resulted in statistically significant increases in the expression of human strength. It was maintained that the ergogenic aids resulted in the occurrence of complex changes in the nervous system whereby the inhibitory mechanism was overcome.

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