https://ntrs.nasa.gov/search.jsp?R=19940023858 2020-06-16T15:00:11+00:00Z

NASA Conference Publication 3252

Workshop on Countering Space Adaptation with Exercise: Current Issues

(NASA-CP-3252) WORKSHOP ON COUNTERING SPACE ADAPTATION WITH EXERCISE: CURRENT ISSUES (NASA) 234 p

N94-28361
--THRU--
N94-28374
Unclas

H1/52 0207609

Proceedings of a workshop sponsored by the
National Aeronautics and Space Administration
Washington, DC, and held at
Lyndon B. Johnson Space Center
Houston, Texas
1989

NASA Conference Publication 3252

Workshop on Countering Space Adaptation with Exercise: Current Issues

Edited by
Bernard A. Harris and
Steven F. Siconolfi
Lyndon B. Johnson Space Center
Houston, Texas

Proceedings of a workshop sponsored by the
National Aeronautics and Space Administration
Washington, DC, and held at
Lyndon B. Johnson Space Center
Houston, Texas
1989

National Aeronautics and
Space Administration

Contents

Section		Page
PREFACE		v
1	MUSCULAR TRAINING	
	Muscular activity and its relationship to biomechanics and human performance. ..... Gideon Ariel, Ph.D., Ariel Dynamics	1 -1
	Eccentric exercise testing and training. ..... Priscilla M. Clarkson, Ph.D., University of Massachusetts	99 -2
	Exercise detraining: Applicability to microgravity. ..... Edward F. Coyle, Ph.D., University of Texas at Austin	111 -3
2	CARDIOVASCULAR FITNESS	
	Aerobic fitness and orthostatic tolerance: Evidence against an association. ..... Thomas J. Ebert, M.D., Ph.D., Medical College of Wisconsin	121 -4
	Does training-induced orthostatic hypotension result from reduced carotid baroreflex responsiveness? ..... James A. Pawelczyk, Ph.D. and Peter B. Raven, Ph.D. Texas College of Osteopathic Medicine	129 -5
	Cardiac output and cardiac contractility by impedance cardiography during exercise of runners. ..... W. G. Kubicek, Ph.D. and R. A. Tracy, Ph.D. University of Minnesota Medical School	141 - 6
3	SKELETAL COUNTERMEASURES	
	Weightlessness and the human skeleton: A new perspective. ..... Michael F. Holick, Ph.D., M.D., Boston University School of Medicine	153 - 7
	Irreversibility of advanced osteoporosis - limited role for pharmacologic intervention. A. M. Parfitt, M.D., Henry Ford Hospital	169 -8
	Exercise and osteoporosis: Methodological and practical considerations. ..... Jon E. Block, Ph.D., A. L. Friedlander, P. Steiger, Ph.D., and H. K. Genant, M.D., University of California at San Francisco	175 -9
4	ELECTRICAL STIMULATION IN EXERCISE TRAINING	
	Electrical stimulation in exercise training. ..... Walter Kroll, Ph.D., University of Massachusetts at Amherst	183-10

PRECEDING PAGE BLANK NOT FILMED

The value of electrical stimulation as an exercise training modality. 187
Dean P. Currier, Ph.D., J. M. Ray, M.D., J. Nyland, T. Noteboom
University of Kentucky Medical School

5 BIOMECHANICS OF MICROGRAVITY

The use of biomechanics in the study of movement in microgravity. 195
R. J. Gregor, Ph.D., J. P. Broker, and M. M. Ryan
University of California at Los Angeles

6 PSYCHOPHYSIOLOGY OF EXERCISE

Psychophysiology in microgravity and the role of exercise. 205
J. M. Shaw and A. C. Hackney, Ph.D. University of North Carolina

7 SUMMARY OF SOCRATIC DEBATES AND PRESENTATIONS ON THE USE OF PHYSIOLOGICAL COUNTERMEASURES 217

8 FREE COMMUNICATIONS 221

Biomechanics/Bone 223

Cardiovascular/Muscle 229

Body Composition/Energy Metabolism 235

Energy Metabolism/Muscle 239

Preface

The National Aeronautics and Space Administration's continuing goal is to explore the far reaches of the galaxy and universe. With the success of the Space Transportation System and advanced astrological observations, mankind's desire to explore is limitless. However, at the very core of this journey the question is raised, "Can man survive in space?" This certainly not new and has been asked since the onset of the manned space-flight program. Numerous biomedical investigations from the United States and Russian space programs make up the foundation for our knowledge of space-flight physiology. These studies support the hypothesis that the human body can adapt to any environment, even microgravity.

Even though the process of space adaptation is a natural phenomenon, it presents special problems to human performance and long-term survival. If humans were to adapt to a particular microgravity environment and remain in space, the problems in physiological performance would be predictable. Unfortunately, this is not the case. Astronauts and cosmonauts will be required to adapt to many different environments on their travels into space. One example of this would be a trip to Mars. Crewmembers will begin on Earth in a one-g environment, launch into space and stay for a time in a microgravity environment, and then land on Mars that has one third of the gravitational force of the Earth. During the entire mission, crewmembers will be required to maintain an adequate level of proficiency for contingency and/or emergency procedures.

The challenge to life sciences is clear—maintain crew health, performance, and safety in all environments. The tasks are many: (1) understanding how various gravitational fields effect the human body; (2) identifying those changes that will significantly affect crew health and retard crew performance; (3) developing measures to those adverse alterations; and (4) ensuring the appropriate response of the countermeasures, i.e., efficacy.

For many years now, both the United States and Russian programs have extensively used a number of countermeasures to maintain the crew's health and fitness, the premise that maintaining crew fitness results significantly in reducing the adverse effects of prolonged exposure to a microgravity environment. These effects vary from the onset of orthostatic intolerance following short-term space flight to the development of bone demineralization following long-term space flight. One thing is clear and that is the variable gravitational fields and the numerous translations found during space travel underscore the need to be prepared for all contingencies. Only the most trained and fit crewmembers will be prepared for these types of environments.

The countermeasure used most effectively in flight is exercise. Data from numerous ground-based and in-flight studies have shown the benefits of using exercise to mitigate the effects of a microgravity environment on the adaptation of the major human physiological systems.

These studies have led to the development of exercise countermeasures for space flight. However, much more knowledge needs to be gained before exercise can be used effectively and efficiently. For example, recent studies on aerobic conditioning of astronauts in flight have shown a dramatic decrease in heart rate while running on a treadmill in flight when compared to the same activity performed in one g. The study suggests that the basic characteristics of exercise to near maximum effort, particularly in-flight running, may be quite different. Extrapolating from this, other exercise modalities may be different when carried out in a microgravity environment and, perhaps, other variant gravitational fields.

In the fall of 1989, the NASA Johnson Space Center's Exercise Countermeasures Project hosted a workshop to examine the use of exercise as a countermeasure for specific responses. Some of the leading scientists participated in free communication and open debates regarding the use of exercise as a tool to influence physiological systems. This workshop entitled, "Countering

Space Adaptation with Exercise: Current Issues," included topics on: bone demineralization, aerobic fitness and orthostatic tolerance, cardiovascular deconditioning, concentric versus eccentric exercise training, electrical stimulation, biomechanics of movement in a microgravity environment, detraining, the effects of exercise response and rehabilitation, and psychophysiology of exercise and training.

The goal of this workshop was to explore those issued related to the application of countermeasures to increase overall understanding and gain insight into the use of these countermeasures in our nation's space program.

Bernard A. Harris, Jr., M.D.

Section 1

Muscular Training

N94-28362

207610
P-97

MUSCULAR ACTIVITY AND ITS RELATIONSHIP TO BIOMECHANICS AND HUMAN PERFORMANCE

O Investigator, do not flatter yourself that you know the things nature performs for herself, but rejoice in Knowing the purpose of those things designed by your own mind.

Leonardo Da Vinci (118)

INTRODUCTION

The purpose of this manuscript is to address the issue of muscular activity, human motion, fitness, and exercise. Human activity is reviewed from the historical perspective as well as from the basics of muscular contraction, nervous system controls, mechanics, and biomechanical considerations. In addition attention has been given to some of the principles involved in developing muscular adaptations through strength development.

Brief descriptions and findings from a few studies are included. These experiments were conducted in order to investigate muscular adaptation to various exercise regimens. Different theories of strength development were studied and correlated to daily human movements. All measurement tools used represent state of the art exercise equipment and movement analysis.

The information presented here is only a small attempt to understand the effects of exercise and conditioning on Earth with the objective of leading to greater knowledge concerning human responses during spaceflight.

What makes life from non living objects is movement which is generated and controlled by biochemical substances. In mammals, the controlled activators are skeletal muscles and this muscular action is an integral process composed of mechanical, chemical and neurological processes resulting in voluntary and involuntary motions. The scope of this discussion is limited to voluntary motion.

HISTORICAL CONSIDERATIONS

The phenomenon of muscular contraction as a prime mover of animal motion has fascinated men for many centuries. In the fifth century B.C., the Hippocratic collection of writings on medicine and its philosophy, consisted of various treatises of the corpus. It is interesting to note that in these early Greek works the tendons were confused with nerves (320). Today, confusion between tendons and nerves no longer exists. However, there is no precise understanding of the activation of the elastic component of the connective tissue nor their contributions to the total muscular contraction.

Skeletal muscles usually originate on a skeletal segment, span one or more joints, and insert onto another bone. Human motion is composed of the coordinated contraction of these voluntary skeletal muscles. This coordinated movement can be conceptualized as consisting of several components. One of the most basic structural components is the cell. Within the human muscular system these cells include bone, muscle, nerve, and brain. Although these four types of cell make up different appearing structures, they have many common features which all cells posses, eg. protoplasm, mitochondria, membranes, etc. The cells are the components of each organ and the organs constitute the system's production of motion. The coordination of these individual blocks is necessary to create coordinated movement. The movement process, intensity, displacement, speed, and acceleration can be executed by various controlled interactions between the organs.

Aristotle (384 to 322 B.C.), considered animal motion to be caused both by the power of the nerves and with the spirit. He wrote:

The movements of animals may be compared with those of automatic puppets which are set going on the occasion of a tiny movement; the levers are released, and strike the twisted strings against one another... Animals have parts of a similar kind, their organs, the sinewy tendons to wit and the bones; the bones are like the wooden levers in the automation, and the iron; the tendons are like the strings, for when these are tightened or released movement begins... Now experience shows us that animals do both possess conatural spirit and derive power from it ... And this spirit appears to stand to the soul-center or original in a relation analogous to that between the point in a joint which moves, and that which is unmoved. Now since this center in a joint which moves, and that which is unmoved. (160).

Circa 1650, Giovanni Alfonso Borelli (75) showed that the muscles act on the limbs with short lever arms, whereas the part of the body carrying a load utilized a longer lever arm. Consequently, Borelli concluded that the joints transmitted forces which are several times the weight of the supported part of the body.

Borelli's contemporary, Descartes, the mathematician and philosopher, related the muscular action to the nerve which inserted into them. Descartes believed that "spirts" entered the brain, passed into the pores of the substance, and from these pores into the nerves. The "spirits" of Descartes then had the power to change the shape of the muscles in which these nerves were inserted and by this means to make all the limbs move.

Today, there are thousands of research articles dealing with understanding of muscular contraction and many of the mysteries continue to allude investigators. There are more then 600 muscles in the human body. In each movement, even the simplest one, groups of muscles work together to achieve the voluntary task. The intensity of the muscular action can be controlled by neural, chemical and biomechanical coordination.

The goal of efficient movement is to accomplish that action with the minimal amount of energy. For example, an Olympic shotputter tries to throw the longest distance by coordinating all internal and external forces in order to generate maximum velocity on the shot at the proper angle with minimal effort. If the movement is not efficient, energy will be wasted in the wrong direction.

Efficiency of motion obviously incorporates activities within the nervous system. In 1883, Yale College received eighty thousand dollars to investigate and lecture on the subject of "Electricity and Matter". Parts of these lectures were devoted to Charles S. Sherrington (410), who studied the role of electricity in the human body. In his book, "The Integrative Action of the Nervous System", Sherrington wrote that:

in the multicellular animal, especially for the higher reactions which constitute it's behavior as a social unit in the natural economy, it is the nervous system and its reaction which 'par excellence' integrates it, welds it together from its components, and constitutes it from a mere collection of organs.

According to Sherrington, the integration of the animal organism was obviously not the result solely of any single agency at work within it, but of several. Thus, Sherrington believed that the mechanical combination of the unit cells of the individual were not independent but functioned as a single unit.

In muscles, this mechanical integration of the organ may produce a single "cord tendon" by which the tensile stress of myriad contractile cells can be additively concentrated upon a single place of application. Sherrington measured forces at the muscle rather than externally as some subsequent investigators have done.

In addition, Sherrington believed that the integrative action of the nervous system is different from other bodily systems. He believed that connective tissue was merely intercellular material and that the circulation system transferred material in mass. On the other hand, the nervous system worked through living lines of stationary cells along which it "despatches waves of physico-chemical disturbance, and these act as releasing forces in distant organs where they finally impinge." Sherrington described the foundation of reflex activity in the human body and established the knowledge of the integrative mechanisms of muscular activity.

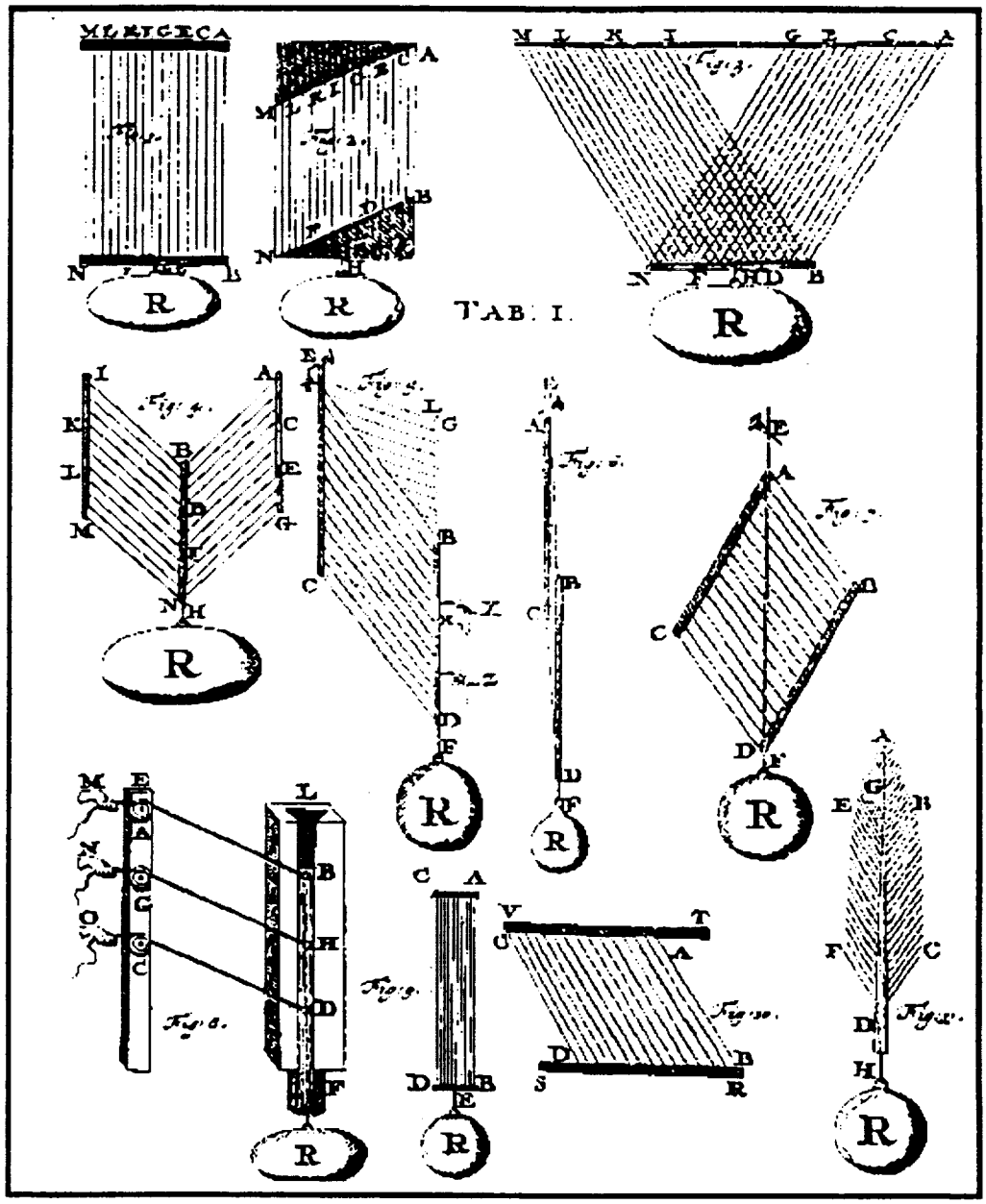


Figure 1

MUSCLE STRUCTURE

Learning about muscular function requires the knowledge of the structure of muscles themselves. Muscular structure can be divided into Macroscopics, which relates the muscle connection to the bones which serve as levers in the different skeletal structure, and Microscopics, which relates the cellular structure and the micro structure of the muscular cell within the fibril structure.

Macroscopic Structure:

In the 1600's, Borelli (75) described the macroscopic structure of the muscular system in his book, "On the Movement of Animals", and presents an exceptionally detailed description. Until some of the recent discoveries in the twentieth Century, there were no other works which provided such detail on muscular function based on mathematical and geometrical relationships. Borelli observed that muscle fibers do not always run parallel to the force which they transmit. This has significant implication for the muscle's internal biomechanics since the resultant force depends on the geometrical relationship of the muscle fibers.

Muscular structure is designed to allow movement and the cell itself is a mechanical structure which causes movement. Borelli (75) stated in his Proposition IV: "Muscle acts by contracting". His illustrated Table (Figure 1) which present the various structures of muscles is amazingly accurate particularly when one realizes that the information dates from the 17th century not the 20th century. His Proposition stated that:

in muscle we see that only the fleshy threads AB, CD, EF and C in the figures 1,2,3 and 4 in Table I contract when the muscle acts. The tendons at the extremities BH to which the fleshy fibers are attached do not contract but retain their initial length. Consequently, only the fleshy fibers AB, CD, EF, GH and C exert a force by contracting when they carry considerable weights. The tendons BH are subjected to a force when moved by the contraction of the fleshy fibers. They undergo this force like a handle to which the fibers are attached.(75)

Hundreds of years after Borelli's pioneering work, the Nobel laureate, Albert Szent Gyorgyi (440) from Hungary, stated that these fleshy parts described in the 17th Century are made of two types of proteins. Skeletal muscle fibers are elongated cylinders containing several nuclei, originally belonging to smaller cells known as myoblasts that merge before birth. Since they are larger than other muscle fibers, many skeletal muscle fibers are visible to the naked eye. Some, like those in the thigh's sartorius muscle, are more than a foot in length and some individual fibers can extend the entire length of the muscle. Usually, however, one end of the fiber attaches to tendon which is the tough tissue that binds muscle to bone while the other end attaches to connective tissue in the muscle.

Borelli (75) believed that when a muscle contracted moving a bone, it created a spherical or circular movement as described in his Table II (Figure 2). It is a humbling experience to realize that, in 1630, Borelli stated what remains as the modern foundation of the relationship of bones, as levers, and the muscles to which they are attached:

But, although the movements of the limbs are circular, the position of the center of rotation of the limbs and bones is not obvious and must be found. If the bones of the limbs were solid lines their contact would be a point which should be considered as the center of fulcrum. However, since the bones have some thickness their extremities cannot easily contact and articulate at one point to rotate about this point of contact. This would be possible if the extremity of one of the bones was pointed, like the apex of a cone or of a pyramid, and if this tip was attached to the cavity of the opposite bone which would be immobile. Then the point of contact would be the fulcrum and the center of rotation. But this would be very inconvenient and fragile. If indeed the ulna ended in a

conical apex and if this conical extremity of the humerus, such a pointed protuberance could easily be crushed and broken and the joint could not be linked strongly enough to avoid unsteady contact at the apex and deviation. Actually, foreseeing and wise nature, to avoid these inconveniences, created a joint which is easy, safe, stable, resistant, and minimally liable to dislocation. She shaped the ends of the bones round, one convex, the other concave so that contact does not occur at one point but over a wide area. This avoids crushing and fracture. This does not give a center of rotation but rather a cylindrical fulcrum over which the bone moves. Any point in which the moving bone rests over the immobile bone is not a fixed or stable point and, therefore, there can be no center of rotation.

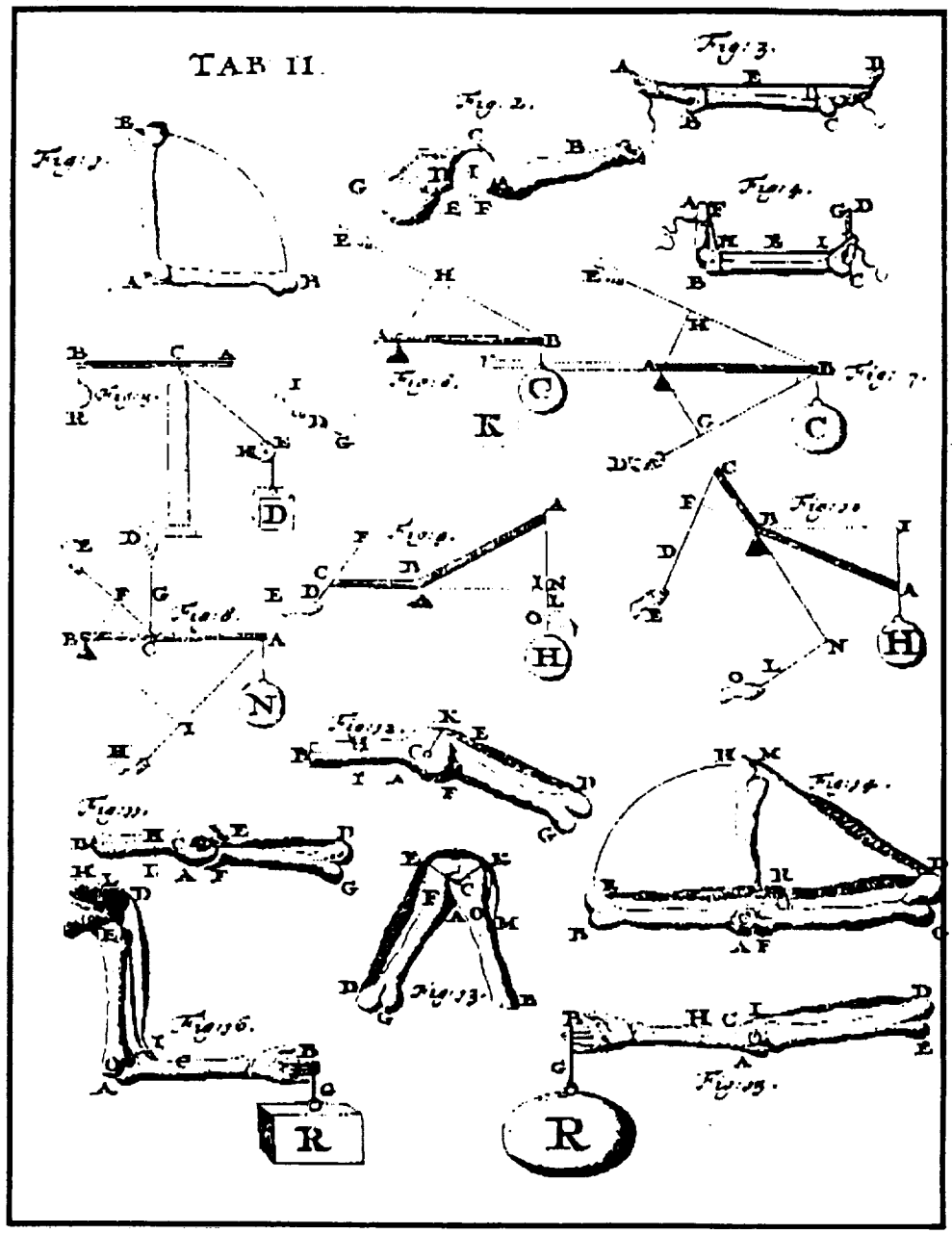


Figure 2

Unfortunately, this explanation from the 17th century has been overlooked by many in our time. The fact that the external force is measured at different angles and at different moment arm lengths is significant in interpreting the muscular force. This concept will be discussed subsequently. Even earlier than Borelli, in the 15th Century, Leonardo Da Vinci (118) wrote: "You may not be versed in geometrical exposition or in the method of calculating the forces and energy of the muscles". Leonardo warned that simple conclusions based on simple assumptions could result in erroneous conclusions.

Another foundation for muscular activity proposed by Borelli, (Proposition VIII) stated:

It is commonly thought that Nature raises considerable weights by using the machines of the muscles with a weak moving force.

The magnitude of the vital force of the muscles must be measured. This force sustains, raises and moves not only an arm or a leg, but the whole animal machine, enabling it even to dance. Besides the mass of the animal, heavy enough by itself, this, this force carries, pulls and pushes considerable weights. Aristotle above all dealt with this matter. He did not recognize the muscles but imagined spirits which pull and push the limbs. This perspicacious author sensibly remarked how difficult it would be for the huge mass of an elephant to be moved and pushed by tenuous spirit or wind. He met the difficulty by saying that Nature moves the joints and limbs of the animal by using very small force. He said that this results from the work of the machine by which motions initially small soon lead to large and multiple displacements. Similarly, small and easy motion of a pole or of the rudder provokes large displacements of the bows and of the boat. Then he considered the nature of a pole from a mechanical point of view and said that the operation is carried out by way of a lever. Therefore, it is not surprising that huge weights can be moved and displaced by a small force. Lucertius used the same example: 'Using pulleys and winding-drums it moves many heavy things and the machine raises them with little effort'.(75)

Despite the antiquity of the statement, the facts are correct. Forces measured at the limbs are related to the muscular force but the relationship is not linear. There is an integration of neuromuscular activity at the muscular site but it does not have a linear relationship to the application of force. It is for this reason that repeating an experiment at different limb positions does not produce the same results in spite of exposure of the segments articulations to the same forces. This calls attention to Borelli Proposition XV which stated:

If two opposite forces are applied at two points of the same lever and have equal moments, their magnitudes are inversely proportional to the distances of their lines of action from the fulcrum (Table II, Fig.8).

The fulcrum of the lever AB is B. Two opposite forces H and E are applied at points A and C in such a way that the lever remains immobile. In other words, the moments of the forces are equal. Their lines of action are AH and CF. Two perpendiculars BI and BF are drawn from the fulcrum to these lines of action. I claim that the magnitudes E and H are inversely proportional to the lever arms BI and BF. Two opposite forces D and N are applied at points C and A. Their lines of action AN and CG are at right angles to the lever BCA. The moment of force D is equal to the moment of force N, to the moment of force H and that of force E. The ratio of the magnitudes D/N is equal to AB/BC. At the end C of the lever BC the fulcrum of which is B, two forces of equal moments are applied, one D at right angles, the other E obliquely. Therefore the ratio of the magnitudes of the forces E/D is equal to BC/BF. Hence, exchanging the terms of the equations, the ratio of the magnitudes E/N is equal to BA/BF. Similarly, the ratio of the magnitude of force N pulling at right angles to the magnitude of force H pulling obliquely is equal to IB/AB. Consequently, exchanging the terms of the equations, the ratio of the magnitudes of the forces E/N is equal to the ratio of the lever arms IB/BF.

Borelli was one of the first mathematicians to reduce the laws of motion into objective, quantifiable relationships. In other words, Borelli was an early pioneering biomechanist. His explanation of fulcrums and levers demonstrated that muscular forces measured at the point of application will not produce the same force quantity at the site of insertion.

Human movement, however, is normally more complicated than a single lever. Most actions involve multiple lever systems with one bone attached to another bone creating a common angular focal point which moves about another angular attachment. These levers are moved by the interaction among different muscle groups. It would be naive to describe muscular activity by considering an isolated joint. Such reality was perceived in the 17th Century as Borelli expressed in his Proposition XVI, which stated (75):

if two opposite forces are applied at the extremities of an angulated balance the fulcrum of which is at the apex of the angulation and if these two forces have equal moments, their magnitudes are inversely proportional to the lengths of their lever arms. Reciprocally, if two forces are inversely proportional to the lengths of their lever arms, their moments are equal (Table II Figs. 9 and 10) (75).

Because of the lever structure of the human skeleton, Borelli evaluated the relationship of muscular force to the external force. He found that different positions of the forearm relative to the upper arm produced different forces applied by the elbow flexors. The various Tables by Borelli III, IV, V and VI (Figures 3, 4, 5, and 6) illustrates the relationships of muscular forces at different body parts. One can observe the complexity of mechanical arrangement and should never forget that these elementary Propositions were proposed in the 17th Century.

In our more modern age, with the advent of sciences and sophisticated instrumentation, it became possible to examine the anatomical structure of the joints and the muscle.

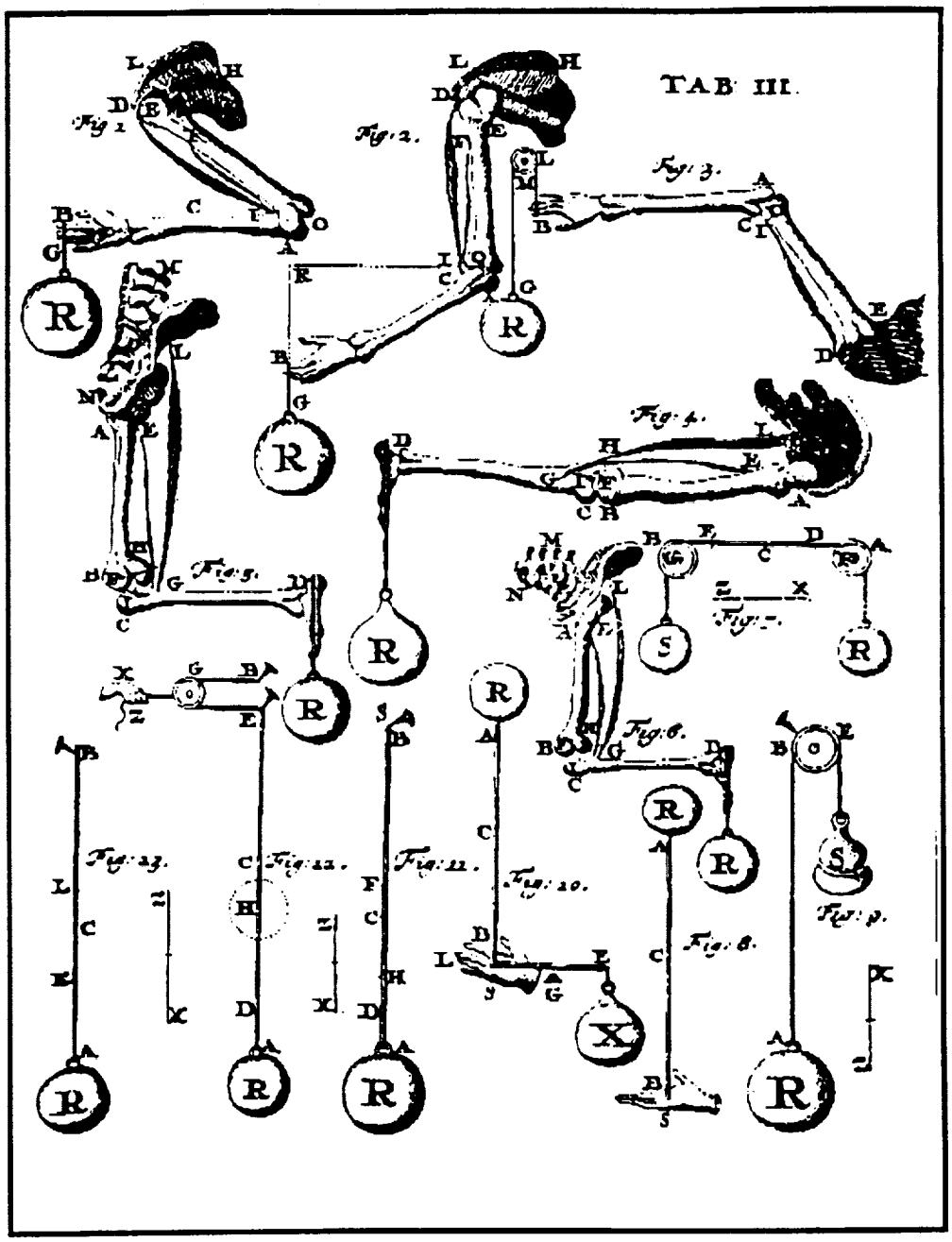

TAB. III.
Fig. 1.
Fig. 2.
Fig. 3.
Fig. 4.
Fig. 5.
Fig. 6.
Fig. 7.
Fig. 8.
Fig. 9.
Fig. 10.
Fig. 11.
Fig. 12.
Fig. 13.
R
S
X

Figure 3

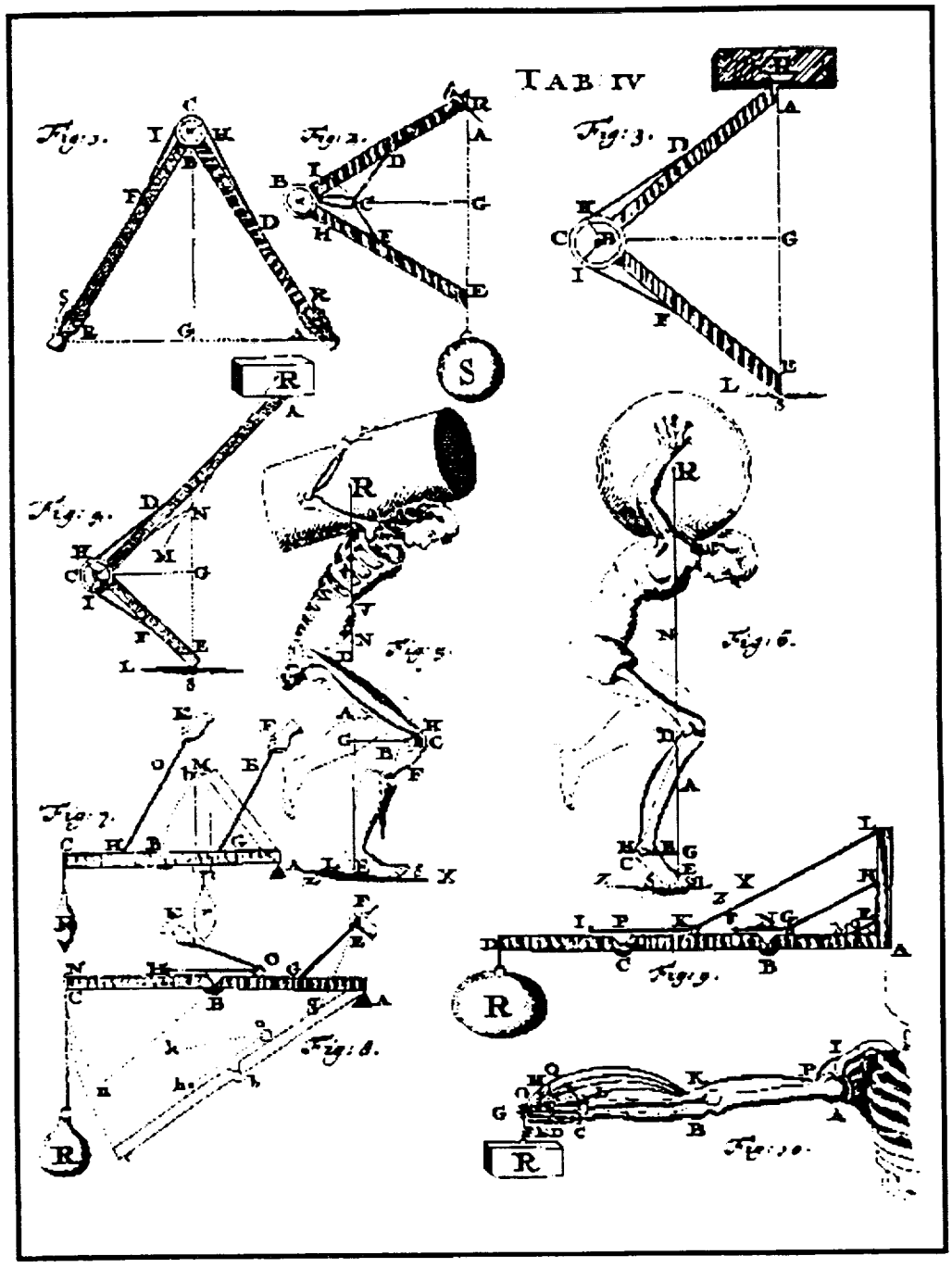


Figure 4

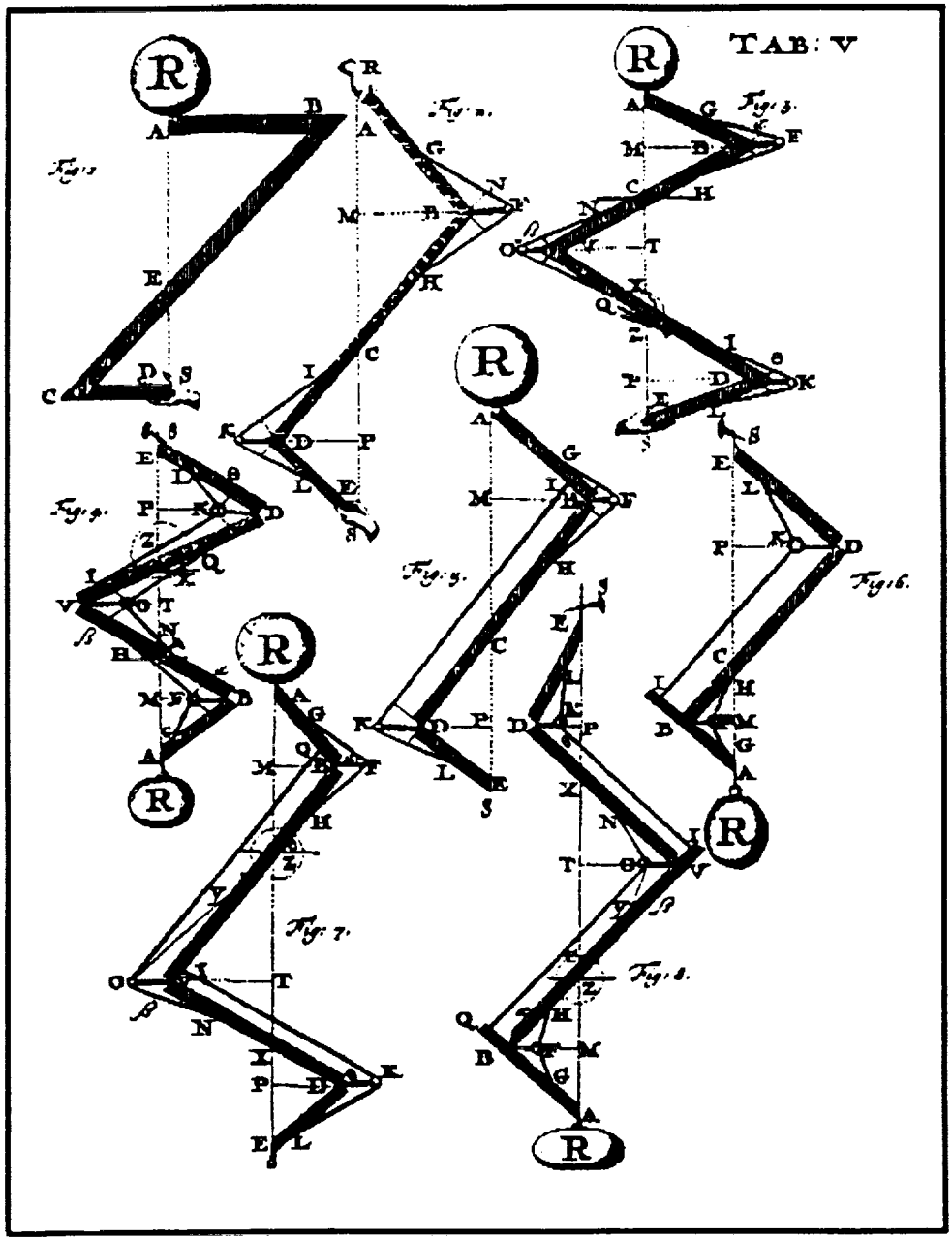


Figure 5

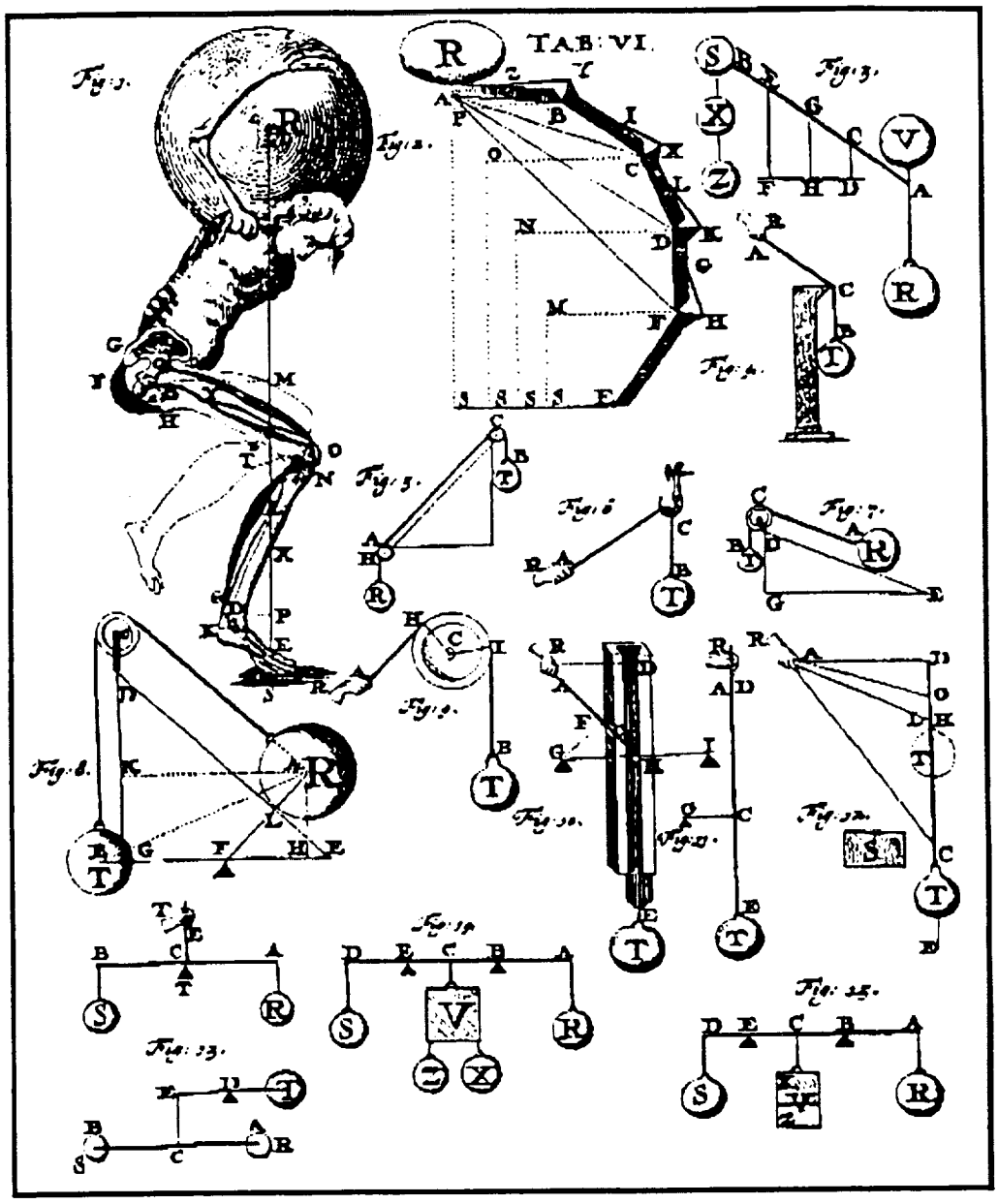


Figure 6

Microscopic Structure:

At approximately the same time that Borelli postulated biomechanical descriptions of motion, a Danish anatomist, Nicholas Stensen, described the motor fibers of the muscles with such detail that one imagines that he had microscopic abilities. Stensen determined that the fibers rather than the tendons were responsible for the muscular contraction.

It is now known that skeletal muscle fibers are elongated cylinders containing several nuclei which developed from cells, known as myoblasts, that merged before birth. In most skeletal muscles, one end of the fiber is attached to a tendon and this tendon is connected to the bone. The other end of the muscle is attached to connective tissue in the muscle itself. This connective tissue, originating in the tendon, spreads into the core of the muscle. However, the materials of which the muscle and the material consist are different from each other. The muscle belly is surrounded by a tissue called endomysium, which is a thin sheath of connective tissue. Another connective tissue is the internal perimysium which bundles the specific muscle into groups which called fasciculi. These bundles are themselves bound together by another layer of connective tissue called the external perimysium or epimysium.

These various connective tissue types and the muscle itself constitute the macroscopic structure of the muscle. However, the dynamics of muscle action is locked within the basic component, the cell. Each individual fiber is surrounded by a thin plasma membrane called the sarcolemma. Figure 7 illustrates these structures. The muscle fiber is filled with tiny fibrils, known as myofibrils, and a jelly-like material called sarcoplasm. Most current scientists agree

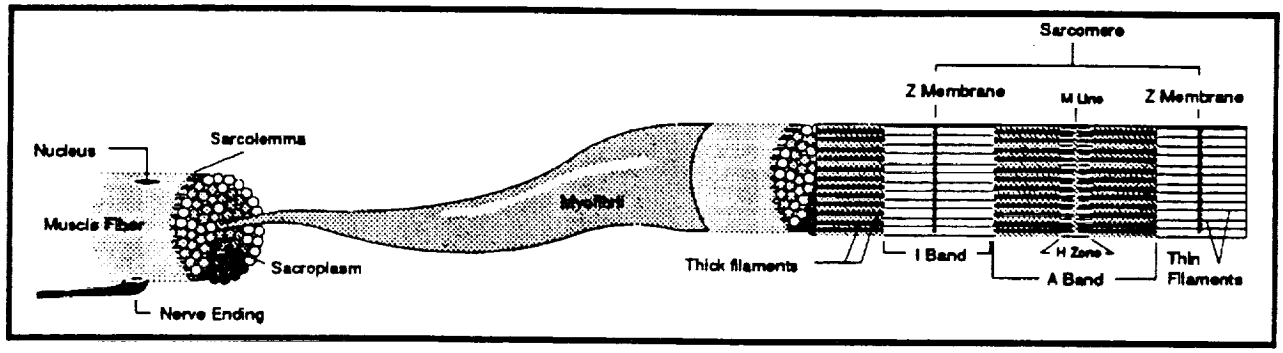


Figure 7

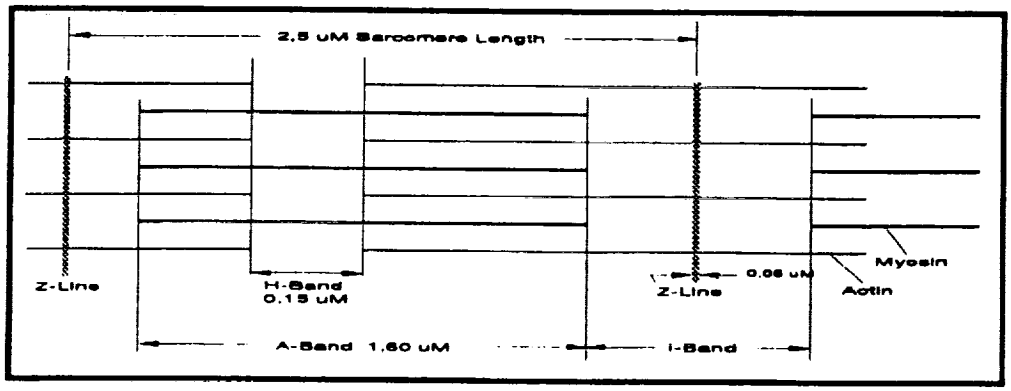


Figure 8

that the fibers contain bundles of fibrils. Each fibril has a diameter of about .5 microns and comprises the hexagonal array of the protein filaments which are directly responsible for the contractile process. Figure 8 illustrates the relative dimensions of the sarcomere. When the fibrils are investigated under the electron microscope, a peculiar structure is evident. In fact, these myofibrils produce the skeletal muscle's striations and cause it to appear striped. It was a seventeenth-century Dutchman, Anton van Leeuwenhoek, who was the first to observe a muscle fiber's striations utilizing a microscope. He predicted that the mechanism of muscular contraction would be in these structures. Leeuwenhoek even entertained the possibility that the striped elements themselves might be enclosed in another membrane and continue into incredibly smaller and smaller filaments.

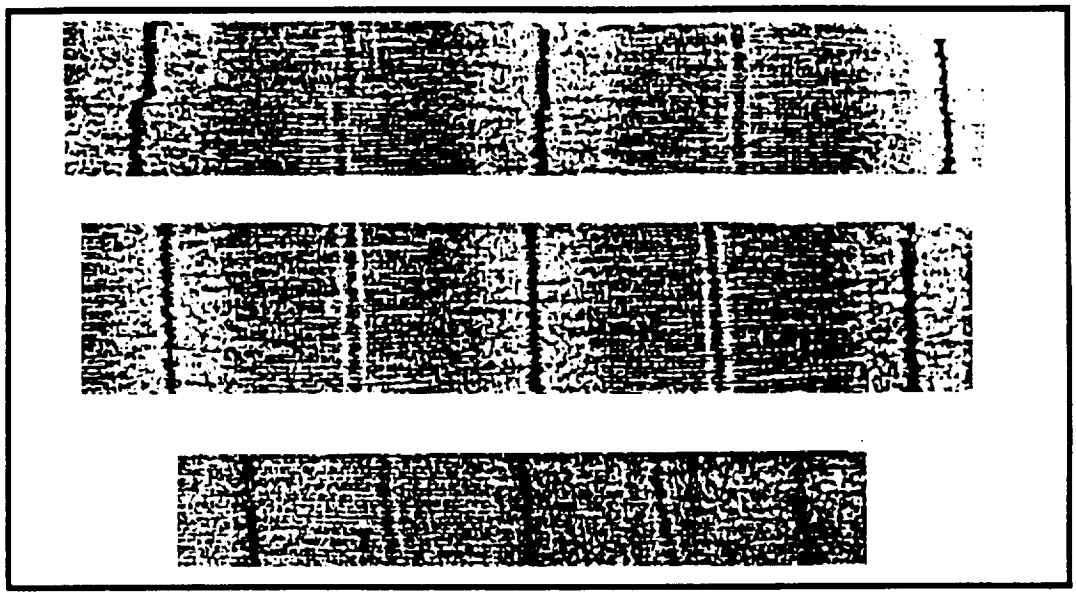


Figure 9

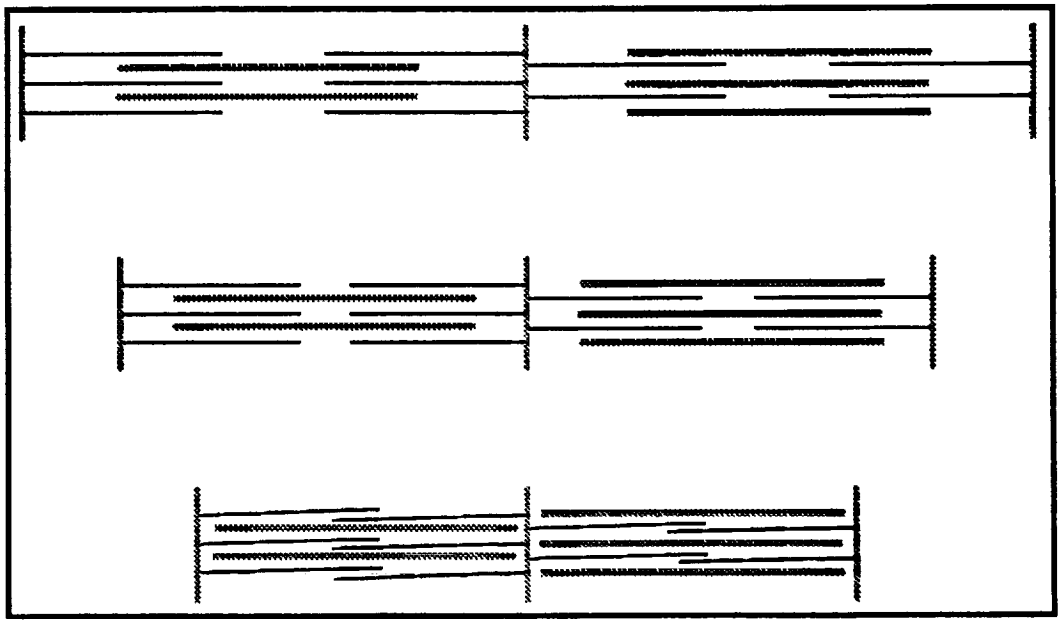


Figure 10

Leeuwenhoek's speculations were not, however, accurate. The striations caused by the myofibrils are subdivided to a unique microscopic structure. The striations are optically made by a dividing band called the Z-line or Z membrane from the German word "Zwischenscheidung". When photographed, the Z-lines appear as dark lines (Figure 9). Two Z lines form a unit of the contractile element and this unit is called a Sarcomere. The Sarcomeres are the basic units of the muscle's contraction. Beside the Z membrane are light regions which are called the I bands. A darker band forms the middle section of the sarcomere and is called the A band. The A band is composed of lighter bands in the middle and darker bands on either side. The lighter center is called the H zone. In the middle of the H zone there is a dark line called the M line (Figure 10). This pattern is repeated throughout the entire length of the fibril.

These striations, then, are due to the composition of the Sarcomeres which are made of thick filaments composed of the protein myosin and thin filaments formed from the protein actin. It is the location of the actin and the myosin which results in the appearance of striations, since the thick filaments originate from the dark regions of the sarcomere and the thin filaments originate in the light areas. The A band, which is darker, consists mostly of the myosin filaments. These filaments are anchored in the middle of the sarcomere in a line called the M line. The actin, the thin filaments, are anchored on the other side, which is the Z membranes. On both sides of the Z membrane, the actin is extended to produce the I band. The thick filaments, the myosin, do not stretch all the way to the center of the sarcomere and, for this reason, the H zone appears lighter in color.

In spite of Leeuwenhoek's discovery of these striations, it was nearly three centuries before the modern knowledge of muscular contraction was postulated. Until the middle of the twentieth century, scientists had assumed that when a muscle shortened, its components also shortened. The theory was that the filaments folded or coiled during contraction. With the invention of electron microscopes and biochemical measuring devices, it became possible to examine muscle as they had never been seen. These 20th Century investigators found that when a fiber contracted, the length of its dark A bands remained constant. This means that the thick filaments, the myosin, do not change their length nor do they contract. However, the two light regions, the I band and the H zone did shorten when the fiber contracted. More evidence suggested that the thin filaments, the actin, did not contract per Se. This was evident from the fact that the Z membrane, where the light actin filaments are anchored, to the H zone where the actin met in the middle of the sarcomere, did not change during contraction. Figure 11 illustrates these relationships.

Investigators from both sides of the Atlantic Ocean independently arrived at the same conclusion, namely that the filaments were actually sliding into each other. M.I.T. scientists, Hugh Huxley and Jean Hanson (275), and Cambridge University researchers, Sir Andrew Huxley and Rolf Niedergerke (272), announced their findings in 1954. Each group postulated the Sliding Filament theory which has served the basic framework for other researchers. Still today, there are many unanswered questions with more mechanisms yet to be discovered. All recently advanced theories of muscular contraction confirm the likelihood that the actual contractile process takes place at the junctions between myosin and actin.

The sliding mechanism is triggered by chain of events which must start with the nervous system. The nerves which terminated near the muscle cell's membrane, secrete specific chemicals. These neurotransmitters start a wave of electrical activity that spreads through the whole fiber. This electrical activity, called the "action potential" causes the fiber's membrane to release calcium ions which initiates the process of contraction. The calcium ions spread throughout the fiber via a specific structure of tubules, diffuse into the myofibrils, and come into contact with the fiber's contractile proteins, the actin and myosin. Two additional types of proteins, Troponin and Tropomysin, work as a team to circle the thin actin filaments. The calcium

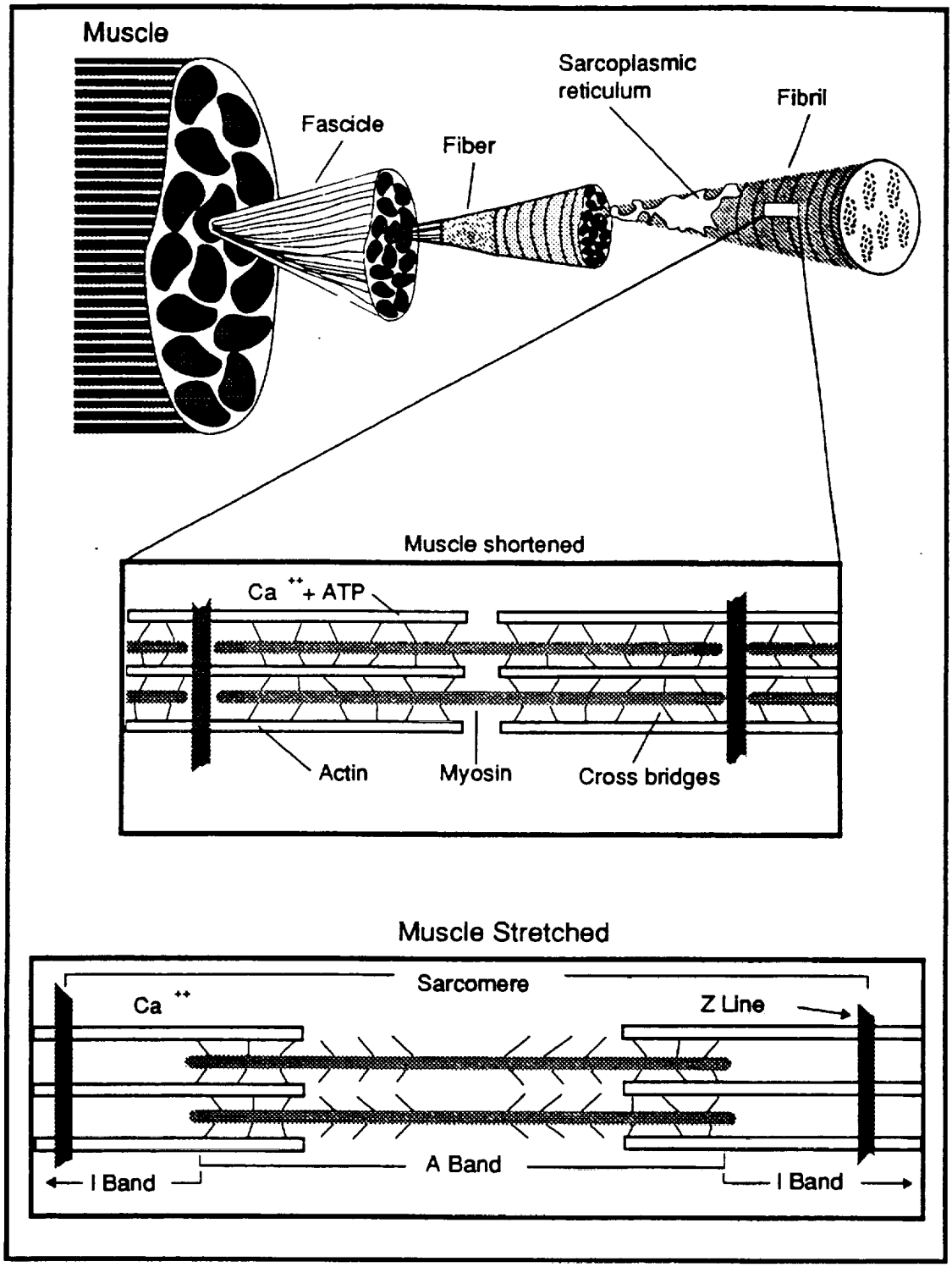

Muscle
Sarcoplasmic reticulum
Fibril
Fascicle
Fiber
Muscle shortened
Ca ++ + ATP
Actin
Myosin
Cross bridges
Muscle Stretched
Sarcomere
Ca ++
Z Line
I Band
A Band
I Band

Figure 11

chemically binds with the troponin and, in a way not yet understood, causes an interaction with the tropomyosin. The tropomyosin threads shift their hold on the actin filament and, with this process, reveal locations along the shaft of the actin filament that are receptive to binding with the myosin filaments. Pairs of rounded extensions, resembling buds are found on these myosin filaments. Each pair forms the head of a single myosin molecule. These buds form bridges to the actin filaments and, hence, these molecules are called "cross bridges" (Figure 12).

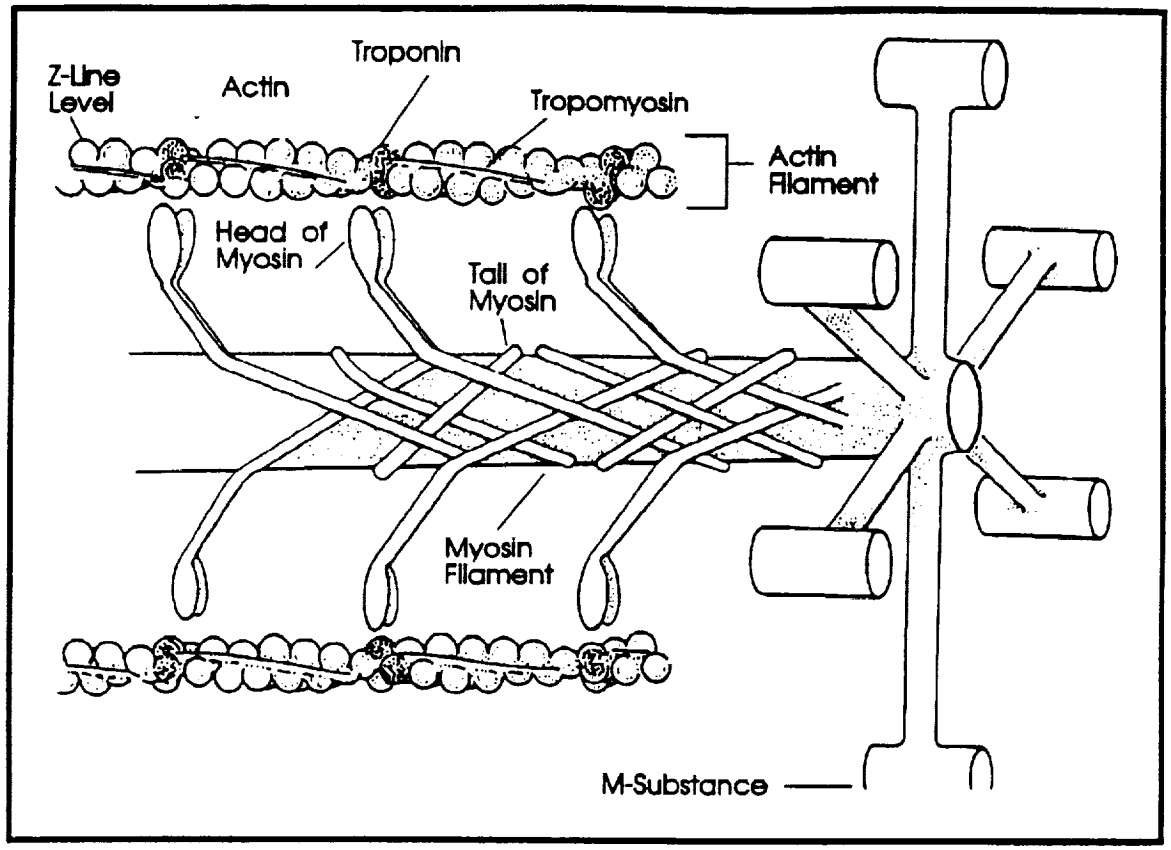


Figure 12

Cross bridges are crowned with a remarkable substance, adenosine triphosphate, or ATP. The ATP molecule is the bases for generating energy for life. In fact, ATP can be referred to as the "Molecule of Life". Reducing the ATP molecule by one phosphate element results in adenosine diphosphate, or ADP. This molecule is able to release energy responsible for muscular contractions as well as many more processes in the human body. Looking at this mechanism from another perspective is that the energy lost in the splitting of the ATP molecule into two lower energy products, the energy lost in the split is then available for use in the body's metabolism.

The ATP molecule has a great affinity to the myosin molecules. Therefore, many of the ATP molecules in the vicinity of the contractile mechanism "choose" to sit on the myosin filament. The higher the number of ATP molecules, the greater the potential for energy requirement during contractions.

The discovery of the enzyme ATPase by Engelhardt (151) gave new light to the research on muscle contraction. Engelhardt proposed a possible mechanism of contraction such that ATP, which is bound in resting muscle to some protein complex, combines with myosin on stimulation. An as yet unknown process within the myosin is initiated and contraction ensues. During this process, the ATP molecule becomes dephosphorylated and yields energy for muscular contraction and ADP, which has a lower affinity for myosin than ATP.

Current theory holds that this "sitting" site of the ATP is comprised of two buds and each bud has a different function. One bud is made of ATPase which is an enzyme responsible for the splitting of the ATP molecule. The other bud is attached to the actin with the ATP molecule. This means that the actin and myosin filaments are "bridged" together with a structure consisting of the enzyme ATPase and the ATP molecule connected with an electrical bond. The bond, by itself, has a unique strength to maintained its position. Figure 13 illustrates schematically these bindings.

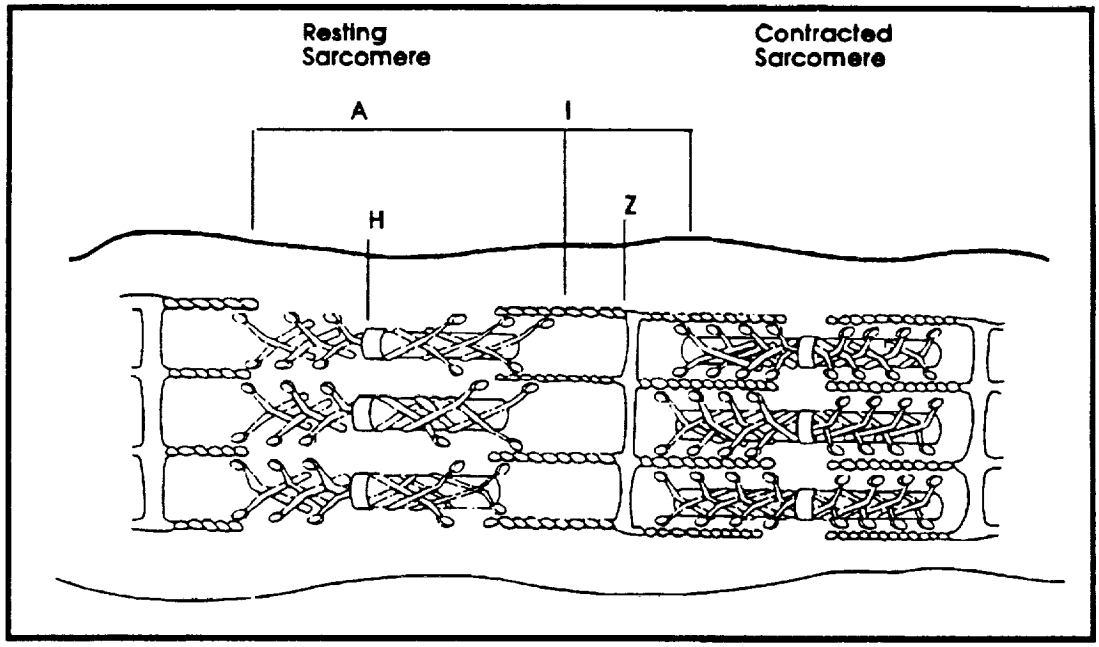


Figure 13

Huxley's model (275), although exceedingly successful at the time of its creation, has lost its attractiveness in the light of more recent experimental evidence. Barany (56) studied the ATPase activity of myosin and found that the rate limiting factor was the ATP splitting. This contradicted Huxley's assumption that one of his rate constant, the constant f, was responsible for the limitation of the reaction rate. Lowey et al. (325) have clearly demonstrated that the side-pieces emerging from the myosin backbone consist of two identical rods of light-metromyosin (LMM) which are connected to two pieces of heavy-meromyosin subfragment.

H.E. Huxley (274) provided electron microscopic proof for the existence of structures linking the myosin and actin molecules. These structures were termed cross-bridges and Huxley suggested in his paper influenced by works of A.G. Szent-Gyorgyi (440), that these bridges consisted of the heavy meromyosin parts of the total myosin molecule. This would mean that the location of the ATPase activity and the site of the contact with actin had been found. This was a tremendous step forward and all subsequent theories had to take this finding into account.

Muscular contraction begins when the tropomyosin shifts away from the binding sites on the actin filaments and the two arms of the myosin filament immediately link with the actin. The ATPase enzyme splits the ATP, liberating energy which provokes cross bridge actions resulting in muscular contraction. In muscular contractions, this activity is continuously repeated with the cross bridges forming and splitting from the actin, causing the actin filament to slide over to the myosin filament, thus, making the sarcomere smaller. The breaking and reattaching of the actin-myosin filaments is, currently, the most accepted hypothesis of muscular contraction. Each sarcomere acts as a distinct unit and the combination of many sarcomeres produces contraction of the whole muscle which pulls on the tendon and its associated bone. Hugh Huxley calculated that the cross bridges would have to execute 50 to 100 cycles per second to accomplish efficient contraction. Other investigators have found that, indeed, the muscle ATP utilization support this rate.

In general, muscular contraction follow the following sequence:

1. The depolarization of the sarcolemma by nervous impulse.
2. The impulse is conducted down the length of the sarcolemma and through the T-tubules.
3. The calcium ions are released from the Sarcoplasmic Reticulum.
4. The calcium ions bind with troponin which stimulates a release of tropomyosin causing inhibition.
5. Actin and myosin interact.
6. Activation of ATPase.
7. ATP is broken down and energy is released.
8. Conformational changes occur at the head of the myosin molecule.
9. Cross bridges are formed.
10. Conformational changes occur at the actin-myosin linkage.
11. The muscle shortens.

The amount of linkage between the cross bridges and the actin relates to the amount of muscular force. The steps listed above allow each sarcomere along the full length of the myofibril to contract. For example, if a muscle is stretched beyond its normal length, there is less overlap between the actin and the myosin resulting in reduced muscular force. On the other hand, when the muscle is compressed or over-contracted, the myosin overlaps itself which reduces muscular force. There appears to be an optimal actin-myosin relationship at which the amount of cross bridges achieves the highest muscular force per sarcomere. However, force in a muscle depends on many factors in addition to the internal sarcomere filament arrangement.

The status at the cross bridges were described by Davies in 1963 (121) such that the cross-bridges were imagined to exist in two different states. In the inactive state, the bridges consist of extended polypeptide chains with fixed negative charges at their bases. One ATP molecule is bound to the top of each bridge in a way which results in a negative unit net charge. The repulsion between the two negative charges, the base and top, keeps the bridge extended. If the muscle is stimulated, calcium moves from the sarcoplasmic reticulum and one calcium ion then provides a link between the ATP ion on top of the cross-bridge and the ADP ion situated on the actin filament. The negative charge on top of the bridge is neutralized, active repulsion no longer exists, and the extended polypeptide chain transforms into a helix-coil. It is believed that contraction is produced by this process. Davies's theory has been challenged by newer studies. Ebashi and Endo (136) have shown that calcium acts indirectly by combining with the troponin-tropomyosin complex.

Based on the preceding information concerning the biochemical and cellular understandings and speculations, the author postulates the following:

Postulate I

Chemical reaction of phosphate molecules result from the splitting of ATP and energy regeneration of these phosphate molecules occurs in the sarcomere. There is, however, no relationship between the chemical processes and the direction of any subsequent limb motion. That is to say, sarcomere activities and the direction of limb movement are independent. Activation levels depend on many external effects such as motor recruiting, motor programming, and the interaction of different muscles. However, the elementary unit of muscular function is the sarcomere and the activity which causes the filament to slide is independent of the external movement.

The sarcomere is the basic functional unit of muscular contraction. However, other factors are associated with the activity of contraction. For example, after the motor unit fires in its motor preprogramming fashion, many sarcomeres contract causing the total fiber to shorten and pulling various connective tissues both internally and externally to the muscle.

This process can be equated with an engineering model. In its simplest form, the model would consists of three-components. A simple model would assume linearity for each component. However, in real life these components are not linear. There are two models which are commonly used to describe these relationships: the Voigt model and the Maxwell model Figures 14 and 15. These models incorporate three distinct elements: elastic, serial and parallel, and damping. Williams and Edwin (473) employed such models in the electronic simulation of frog muscle responses to pulse trains at various selected frequencies of stimulation.

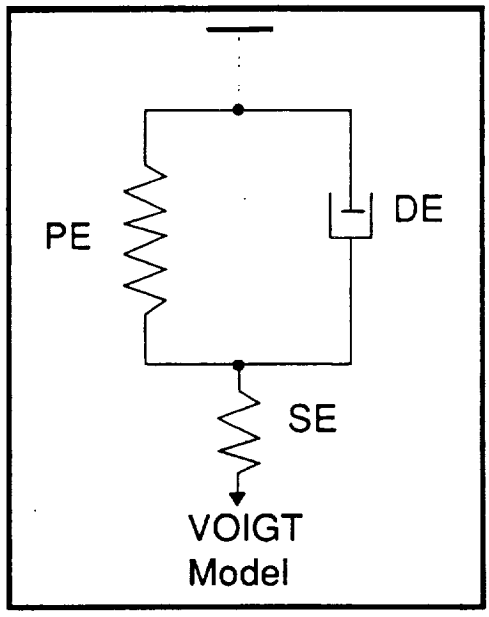


Figure 14

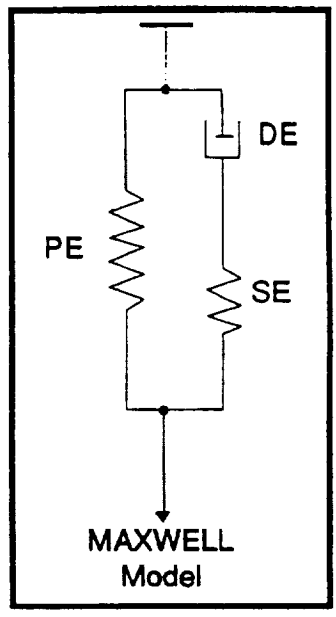


Figure 15

Crowe (116) incorporated the Voigt model to study the responses of intrafusal muscle fibers of mammalian muscle spindles to mechanical stretch and to fusimotor stimulation. In 1969, Green (208) also employed the Voigt version of the three-component model. He recognized the nonlinearity of all the elements involved, thus, accepting the concept that other factors which were not incorporated must ultimately be included.

Many more studies were conducted in order to find a model appropriate for muscular contraction. However, in all cases, the non linearity of the system and the complexity in behavior of muscular contraction precluded an ideal model which functioned properly under all conditions.

Bawa et al. (59) presented a model of muscle consisting of linear springs and a dashpot in parallel with a force generator. This force generator produced a contractile force of constant magnitude each time a stimulus was received. Immediately afterwards, the force was assumed to decay exponentially. According to the authors, the predictions of this model were in good agreement with experimental observations.

If the muscle was not stimulated, its internal tension due to filament binding was small. Therefore, if a muscle was stretched without neural stimulation, any tension must come from the elastic structures of connective tissue which are in parallel with the sarcomeres. At the time of stimulation, the contractile machinery produced tension which was transferred to the endpoints of the muscle. This transference of forces was through the connective tissue lying in series with the sarcomeres.

One can appreciate the mechanical complication associated with this structure and transfer of forces. For example, the system must be described with one type of connective tissue providing non linear resistance in parallel while another type of connective tissue gives non linear resistance at the end points of the muscle in series with the force generator mechanism. In addition to these non linear relationships, the fibrils themselves have specific geometrical relationships to the line of force which causes the contraction. The tendons, as well, have their own nonlinear elastic characteristics.

The concept associated with the "give in the system" provided by the various connective, muscular, and other soft tissues is known as compliance. Various locations for compliances were suggested by various investigators. Jewell & Wilkie (282) suggested various sites. Jewell & Wilkie and Szent-Gyorgyi (283) suggest that the Z-discs in the sarcomere allow a great deal of compliance in the muscular structure. Huxley & Simmons (270) propose that there are two elastic elements with one used as a damper and the other provides compliance for the sarcomere. They suggests that these elements are within the myosin molecule. Flitney and Hirst (170), and Morgan (361) provided conclusive evidence that part of the series elastic structure is indeed located within the cross-bridges. These researchers believe that this part depends on the active state of the muscle and the degree of filamentary overlap.

These research findings indicate that there are elastic elements within the sarcomere and at the end point of the fibers where connective tissues connect the muscle to the bone. The important factor is that when the sarcomere shortens, there is a short latency during which some energy is stored within the elastic components of the muscle. After this short latency, the force is applied at the end points of the muscle and the lever begins moving.

Based on the facts described previously, the author postulates the follows:

Postulate II

All mechanical movement originates at the sarcomere level. Regardless of the movement of any external lever, that is, the bone to which the muscle is attached, the specific chemical reactions within the sarcomere begin the shortening or contracting process. The ATP activity, the ATPase reaction, and the calcium ion flow are independent of the external lever movement. The sarcomeres shorten as the actin filaments are pulled by the myosin cross bridges. Sarcomere activity can only produce contraction. However, bone movement can be either in the direction of the contraction or, if there is a sufficiently large load to overwhelm the contractile strength of the sarcomeres, in the direction opposite to the contraction.

Functional Characteristics and Mechanical Properties of the Muscle:

Skeletal muscle is composed of three distinguishing components: contractile, serial elastic, and parallel elastic. In addition, the muscle also has a blood supply and functions within a semi fluid environment. Attention will not be given here to the vascular factor.It is comparatively easy to determine the constants for the parallel elements of the muscular structure. This can be done by studying the relaxed muscle.

When considering the elastic elements in parallel, Yamada (479), demonstrated its characteristics by utilizing stress-strain curves for muscles and fascia. Figure 16 illustrates this relationship with different muscles having different curves. However, all muscles and fascia demonstrate non linear relationships. Examination of the curves readily confirms that muscles are much more compliant than fascia. This observation seems quite reasonable since fascia consists of connective tissue to transmit forces and for protection whereas muscles are the force generators. Other investigators, including Jewell & Wilkie (282) and Hefner & Bowen (236), have found similar results.

This stress-strain relationship demonstrates that beyond a certain stress level, the fascia will be damaged before the muscle is injured. However, the state of the muscle is the important factor. When the muscle is in a full contractile state, the stress-strain relationship can be shifted. Also, a state of muscular fatigue can bias the relationship resulting in an altered reaction to stress.

Whether a stress-strain relationship can be altered by the forces generated from a combined effort of the sarcomeres alone without external influence is doubtful. That is, it seems unlikely that muscle strength alone can injure the fascia or the muscle tissue themselves. However, high levels of force generated by external means due to gravity or to impact as well as a summation forces originated through movement produced by a combination of levers could exceed the compliance of the fascia or the muscle resulting in internal damage.

Postulate III

Injury to the skeletal system occurs due to external force which exceeds the maximal internal force. Under this condition of excessive external force, the stress-strain relationship may exceed the compliance capability of the connective tissue and, therefore, cause tissue damage.

Examination of the series elastic element in the muscular structure is a more complicated task. The reason is that the state of the muscle alters the relationships. The biological material of which the serial elastic component is composed is not different from the parallel elastic material which consists of elastin and collagen fibers in complex arrangements. Again, investigators such as Yamada (479), Wilkie (471), Hill (247), Bahler (52), Joyce & Rack (290) and others have defined the compliance characteristics of the serial elastic elements of the skeletal muscle. For example, Yamada (479) found that calcaneal tendon tissue ruptures at extensions exceeding a value of .1 relative to the tendon's resting length.

Again, the force generated by the combined sarcomere pool is insufficient to over stress the serial elastic component of the muscular system. Only an external force generated at the contact point of the connective tissue by a lever, such as bone on bone or bone and external object, could transmit a force which would exceed that produced internally. These facts lead to the Author's fourth postulate:

Postulate IV

The muscular system is exposed of two types of forces. The first type is the sum of the forces generated by the individual sarcomeres. The second type of force is generated from the lever through serial and parallel elastic connective tissue. The sarcomeres realize this force but do not contribute to it. The state of the sarcomeres is the determining factor as to the response to this force. If the force is greater then the binding force of the cross bridges, then either the actin filament will be stretched away from the myosin filaments or internal damage will occur in a tissue which is weaker then the binding chemical force occurring at the cross bridges level. The only force generator in the contractile system is the contractile element. The connective tissues, such as the fascia or the tendon, cannot generate force but can only absorb energy and return it to the system in a form of elasticity.

The behavioral characteristics of the contractile element is extremely complicated. Some of the nonlinear factors affecting its force generation capabilities depend on its length, fatigue, the rate of length change, the degree of stimulation, and its temperature. Each of these factors affect the level of force that the contractile element can produce. In physiological terms, these factors can reduce to the active-state function, the filamentary-overlap, and the velocity-dependence functions.

As was discussed previously, the contractile element produces the force due to the actin-myosin cross-bridges. Detailed examination reveals that the cross-bridges are part of the myosin filament, being the heavy-meromyosin subunits projecting out of the light-meromyosin assemblage which constitutes the backbone of the myosin filament. The heavy-meromyosin subunits consist of two rod-like subunits, each of which carries a globular head subunit (325). It is to these globular heads that the energy-providing ATP molecules are presumed to bind in the presence of calcium ions and are thought to provide the direct link with the actin filaments.

The internal source of force by the muscle, therefore, is equal to the sum of the forces produced by all the cross-bridges in one half-sarcomere of the fiber, at any instant of time. Since it was found that the propagation velocity of the calcium ions moving from the terminal cisternae into the sarcoplasm is finite (284), the onset of the contractive cycle of different sets of cross-bridges along the myosin filament upon stimulation will be successive. This fact was verified by Huxley and Taylor (271).

It should be remembered that the appearance of force, measured by an external transducer or calculated from the movement, is not equal to nor does it correlate highly with the internal force. One reason is that in living human subjects, it is impossible to know the number of sarcomeres, fibers, or activation levels within the studied muscle. Another factor is that the leverage system changes during movement as does the interaction between different segments in the body. Inertial forces act on each body segment due to the acceleration of the different segments. Co-activation of antagonistic muscles also plays a major role in producing the net moment around a particular joint.

The force in the skeletal muscle is generated within the sarcomere and is equal to the sum of the forces generated by the cross bridges. The force is equal to the sum of the forces produced by all the cross-bridges in one half-sarcomere of the fiber.

Any attempt to relate the sarcomere force to the force generated by a limb movement will yield a low correlation since the force measured at the force application point depends on the lever arm, the amount of antagonistic muscular co-activation, and the inertial forces due to the movement. The appearance of force on the load arm, such as the hand, wrist, ankle, or at the joints, such as the elbow, knee, etc, does not correlate highly with the forces generated at the sarcomere level.

As early as 1940, Ramsey and Street (396) demonstrated the existence of an internal force tending to extend the fiber and, therefore, presumably removing whatever tension was developed by the filaments during contraction (202). These authors also suggested that this internal force may be attributed to the deformation of the sarcolemma accompanying the increase of the fiber diameter during shortening. Therefore, the amount of force needed for shortening will be smaller on the external measuring device than the amount actually generated at the sarcomere level. The sum of forces generated by the cross bridges is at least 10 times the amount shown on the force application. The efficiency of the force at the point of external application depends upon the position of the lever, the position of the body, the technique in executing the movement, and the utilization of energy derived from other body segments toward the desired movement.

Therefore, the ability to carry a greater load at a particular movement in a specific direction has no bearing on the ability of a single cross bridge at the sarcomere level to produce more force. The sarcomere can only contract and is not effected by the direction of limb movement regardless of the number of cross-bridges which are active at one time.

Training methods were and can be devised to increase the number of active cross bridges needed for a particular movement which would, therefore, increase the force generated for a specific action. However, the net force at the application point might not correlate with the number of cross bridges active at the prime mover muscle.

The number of cross-links formed is a function of the active state of the fiber as well as the degree of filamentary overlap (202) and, presumably, the velocity of shortening or lengthening of the contractile element (273). In addition, the average force output of the cross-bridges is postulated to depend both on the velocity of the interfilamentary movement and on certain intermolecular forces. These intermolecular forces were shown by Huxley (272) to exhibit a cyclic behavior the attachment-detachment-reattachment cycle of the cross bridges. Because this cyclic behavior is asynchronous, individual force fluctuations do not appear externally. Thus, the average force output of an activated cross-bridge becomes a function of the velocity of the interfilamentary movement only.

Therefore, the force output of the contractile proteins of a typical muscle fiber is equal to the average force output of a typical cross-bridge, multiplied by the number of cross-links active in a half-sarcomere, at any instant of time. As a formula this fact can be illustrated by the following equation:

$F = N(E) * A(L) * L/t$

F is equal to the force output of the contractile proteins of a typical muscle fiber.

N is equal to the total number of cross-bridges present in a half sarcomere, in a particular state of activation E.

E is equal to the state of activation.

A is equal to the average force output of a typical cross-bridge, at a particular length L.

L is equal to the instantaneous length of the sarcomere.

L/t equal to the Length of the sarcomere per unit of time t, which represents the change in length per unit of time or the velocity of sarcomere contraction.

It is important to realize that it is very unlikely that external conditions of the force application on a particular body segment would effect these relationships in most conditions. Of course, the speed of segment movement and the joint angular changes are directly related to the speed of sarcomere shortening and, therefore, the factor L/t in the sarcomere. If, for example, the elbow joint were restricted, such as in a isometric contraction, then the sarcomeres would remain in a state of unchanging length and, in that state, would function according to the formula. If the elbow joint were free to move, flexing or extending, the sarcomere would produce force regardless of the direction of movement according to the equation. In fact, sarcomere performance is unrelated to direction since there are no sensors of any kind within it to record or detect consequences.

Therefore, the relative number of active cross-links is a function of the active state, the degree of filamentary overlap, and the velocity of shortening or lengthening of the contractile element. This means that there is a condition in which the sarcomere length is optimal and the active state is at maximum stimulation. The sarcomere is in isometric state when this optimal length does not change. At that point the force output will be maximally. However, this optimal state is rarely achieved since the number of cross links varies as well as the distortions caused by the collision between Z-lines and myosin filaments at very short sarcomere lengths and the overlap of actin filaments at intermediate sarcomere lengths (202). In any case, the force output of the contractile element is not identical with the force output as observed externally. Clearly, the force output of the contractile machinery will be reduced by internal resistance, and the externally observable contractile force will be lowered as well. The parallel elastic connective tissue is a main factor in this reduction.

Neuromuscular Dynamics:

The active state of the filaments is one of the factors in the force production formula. Hill (250) reported and defined the active state of the muscle. Hill pictured the active state as some operational ability of the muscle which abruptly appeared when the muscle was stimulated and then slowly disappeared when the stimulus ceased. Gonzalez-Serratos (201) described the events leading to the onset of the contraction with the sequence beginning with the arrival of a nerve signal at the motor end plate of the fiber and the subsequent propagation of the fiber action potential along the fiber surface and down the transverse tubular system. The transverse tubular system which is called the T-system, located at the Z-discs, converts the action potential into a depolarization signal which acts across the walls of the tubular network (10,159). Immediately upon the arrival of the action potential, depolarization of the T-membranes causes the release of calcium ions from the sarcoplasmic reticulum. This phenomenon is described in detailed by Inesi (277). At the instance the calcium ions penetrate the membrane, ATP hydrolysis is initiated and, simultaneously, calcium ions bind to the calcium-binding subunit of the troponin molecule. This cause the myosin head to bind to an actin monomer which caused the shortening (138,222, 386). Simply, the active state relates to the amount of calcium ions bound to troponin. If the maximum number of potential interactive sites on the actin filament are made available by the action of calcium, then the maximal force will be produced by the contractile elements.

It appeared that the number of free calcium ions and the amount of binding is independent of both the external force requirements and the direction of movement. However, neural programming is involve in the recruitment phenomenon to be discussed later.

The amount of calcium ions bound to troponin relates to the amount of contracting elements which are in an active state. If, for some reason, the stimulus is such that the calcium ions do not combine with troponin, there will be a less than active state in the contractile element. In the presence of calcium ions, this binding depends on many factors and one of the most important is the amount of neural stimulation. The amount of calcium concentration varies under different conditions and is the "bottle neck" for the contraction mechanism. Without the

process of the binding of the calcium ions to the troponin-tropomyosin complex, there would be a specific contractile force for a given sarcomere length. This does not occur since the rate of supply of calcium ions varies depending on the amount released from the sarcoplasmic reticulum. Depolarization of the membrane of the sarcoplasmic reticulum results from the depolarizing potential of the T-tubular system.

There is no research to show that the direction of the lever movement or the amount of external load in either direction affects a given sarcomere length or the level of calcium ions. Rather it is the level of electrical stimulation which causes the mobility of calcium ions. With regards to the electrical signal in the interior of the T-system, it was found by Huxley and Peachey (269) that this is due either to direct conduction of the action potential of the surface membrane or to an electronic spread of the surface potential down the T-system. This electrochemical transmission of the nerve impulse arrives at the motor endplate of the fiber to a neuromuscular junction and the T-system. These comprise a very complex electrical networks.

At the motor endplate, Eccles (141) reported that in mammalian muscle the nerve stimulation results in liberation of an acetylcholine transmitter substance. Each stimulation liberates a certain amount of this chemical. The frequency of stimulation varied depending upon the fiber type and ranged from approximately 100 c/s to about 25 c/s.

Needless to say, these activities do not occur simultaneously and various latencies exists. For example, the active state of the contractile element reaches its peak in 10 to 12 milliseconds. The decay of the active state also requires a few milliseconds. Therefore, the dynamics of the contractile element of a muscle fiber is dependent on velocity and the filamentary overlap.

Force Output:

The number of active cross-links in the interfilamentary space and the degree of filamentary overlap are the basic factors which generate force at the fiber. The other factors which effect the force output is the velocity of movement between the actin and the myosin filaments. There is virtually no relationship between the velocity of movement and the force production, however, it should be remembered that only the effect of velocity at the sarcomere level is important. Velocities of levers involve mechanical relationships and inertial forces are external factors which can affect the internal contractile element forces but cannot alter its physiological elements.

All these mechanisms contribute to all body motions. When training for improved movement or for enhanced athletic achievement, muscular activity is the predominant factor for increasing efficiency and optimizing skill.

Muscular Activity in Exercise:

Exercise can be define as physical activity utilized to generate body responses to demands. Exercise enables the body to adapt to certain demands and, therefore, increase or alter the performance capabilities.

There are various factors that affect the type of exercise. These are:

1. Speed of exercise
2. Duration of exercise
3. Resistance

The relationships between these factors determine the various adaptation characteristics of the body's physiological system.

Speed/Duration Factor:

The faster the speed, the shorter the time that exercise can be maintained. The slower the speed of movement, the longer the time the exercise can be performed. Figure 17 illustrates this effect. For example, sprinting can be performed for a short time, approximately 50 seconds, while jogging can continue for hours.

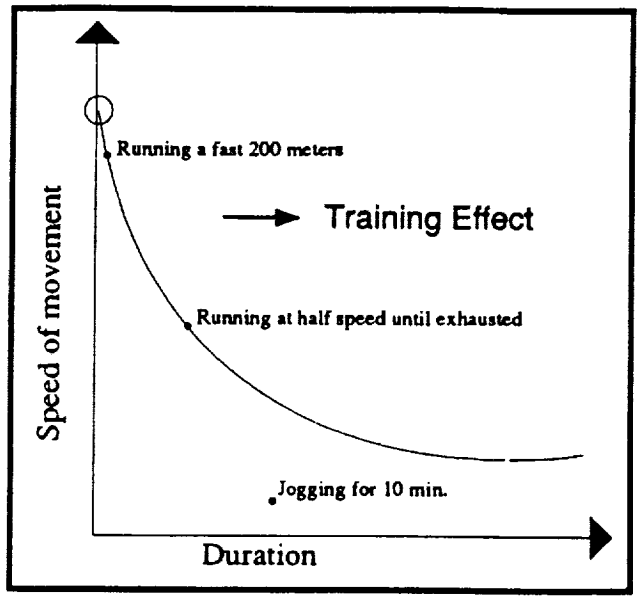


Figure 17

Speed/Resistance Factor:

The higher the resistance, the lower the speed of movement and with less resistance, the movement speed increases. In resistance training, the heavier the load, the slower the movement. Figure 18 illustrates this relationship.

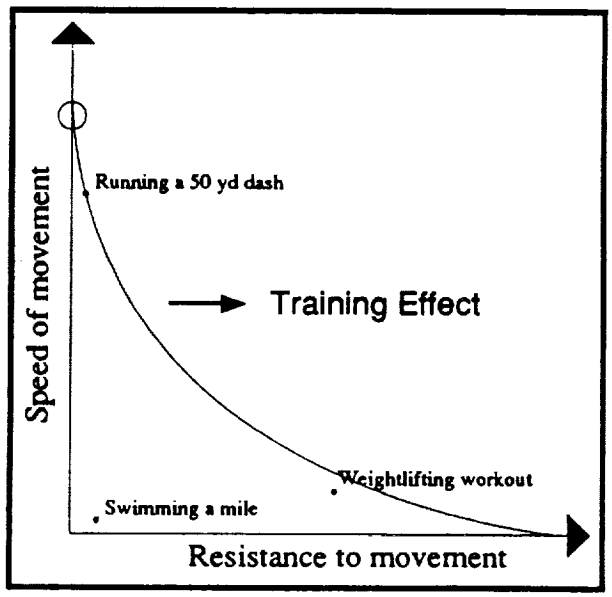


Figure 18

Resistance/Duration Factor:

The greater the resistance to movement, the less time the movement can be endured. Figure 19 illustrates this relationship.

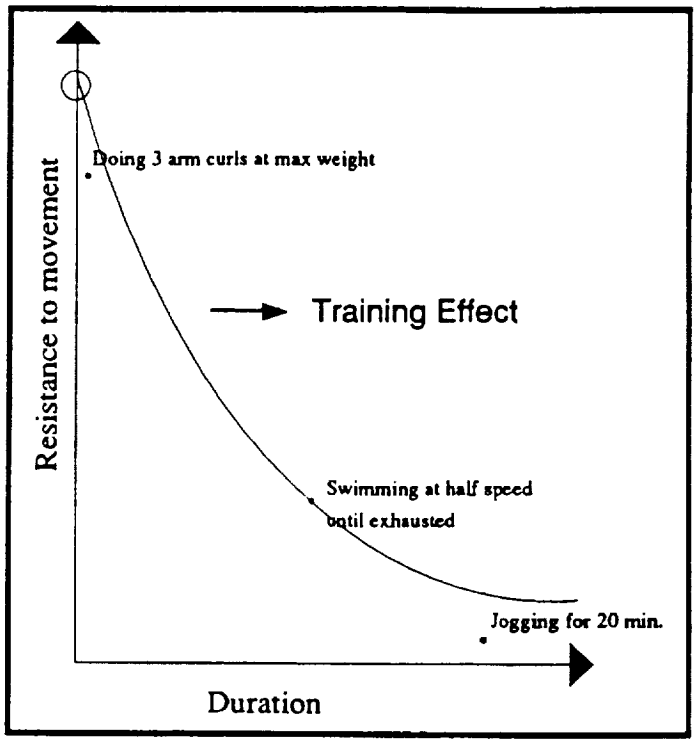


Figure 19

When combining these factors, one can consider the entity as a three-dimensional figure as in Figure 20. Exercises closer to the origin are the least strenuous while the further from the origin, the more difficult the exercise.

If one were to classify exercises for various physiological adaptations, they could be summarized in Figure 21 which shows that strength exercises, as in resistive training, and endurance exercises, such as in distance running.

Exercise is the act of performing a physical activity. In order to elicit specific training characteristics, one must exercise in repeated bouts. Repeated bouts of training will result in biological adaptations over a period of time. This time can vary from days to years.

Periodically repetitive performances of a sequence of exercises at increasing levels of intensity can cause physical adaptations. Adaptation takes place due to some unknown "set up" which occurs within the DNA-RNA protein mechanisms. Protein synthesis takes place during the training period and results in greater muscular strength and endurance. Subcellular components are altered during the training period and it is specific to the type of training regimen. In resistive training, the adaptations primarily occur in the contractile mechanisms on the myofibrillar protein. This results in increased size and number of myofibrils per fiber. On the other hand, endurance adaptations are observed in the enzymatic capabilities of the metabolic pathways. It is important to remember that specific exercises selectively alter specific subcellular compartments causing varying degrees of exercise capability and selectively altering the training adaptations. The training adaptations, therefore, are specifically related to the exercising conditions.

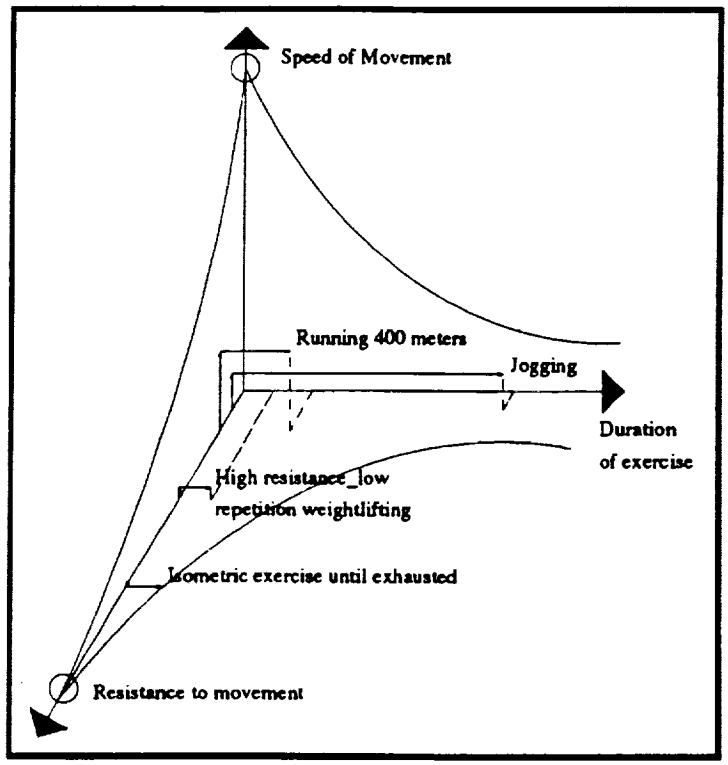


Figure 20

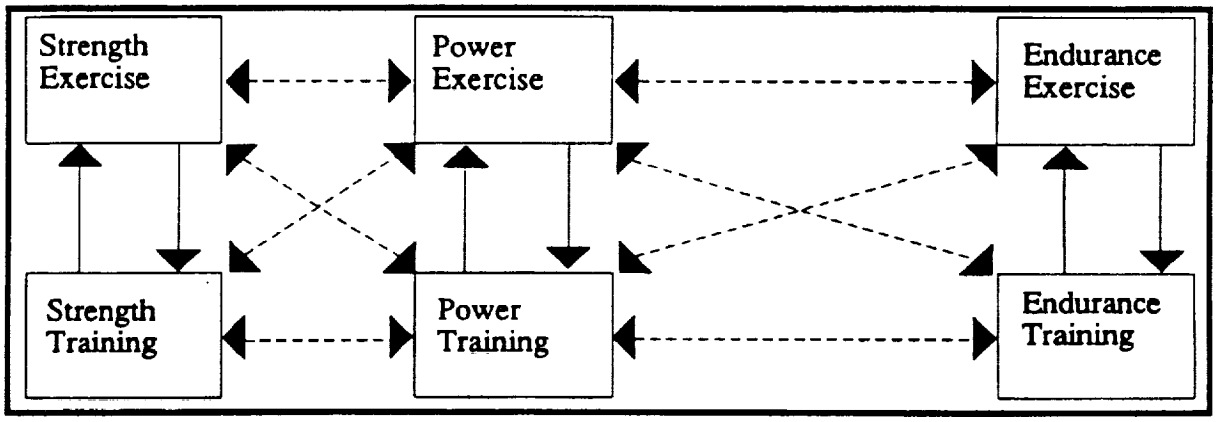


Figure 21

RESISTIVE EXERCISE

The relationship between resistance exercises and muscle strength has been known for centuries. In ancient Greece, Milo, the wrestler, used progressive resistance exercises to improve his strength. His original method consisted of lifting a calf each day until it reached its full growth, and this technique provides probably the first example of progressive resistance exer-

cises. Today, it is well documented in the literature that the size of skeletal muscle is effected by the amount of muscular activity performed. Increased work by a muscle can cause that muscle to undergo compensatory growth (hypertrophy) while disuse leads to wasting of the muscle (atrophy).

This information has stimulated the medical and sports professions, especially coaches and athletes, to try many combinations and techniques of muscle overload. These attempts to produce a better means of rehabilitation or a physiological edge in sporting activities have only scratched the surface of the cellular mechanisms and physiological consequences of muscular overload.

Muscular strength may be defined as the force a muscle group can exert against a resistance in a maximal effort. In 1948, Delorme (125A) adopted the name "progressive resistance exercise" for his method of developing muscular strength through the utilization of counter balances and weight of the extremity with a cable and pulley arrangement and, thus, gave load-assisting exercises to muscle groups which did not perform antigravity motions. McQueen (344A) distinguished between exercise regimens for producing muscle hypertrophy and those for producing muscle power. He concluded that the number of repetitions for each set of exercise determines the different characteristics of the various training procedures.

Based on evidence presented in these early studies, hundreds of investigations have been published relative to "techniques for muscular development including isotonic exercises, isometric exercises eccentric contractions, the Oxford technique, the double and triple progressive super set system, and many others. Each system's effectiveness has been supported and refuted by numerous investigations.

Berger (61A) concluded that 6-7 repetitions three times a week was best for developing dynamic strength. Research conducted by Steinhause (432A) emphasized the need to increase the intensity, not the amount of work, in order to develop maximum strength.

In more recent studies pertaining to exercise, Pipes and Wilmore (388A) compared isokinetic training to isotonic strength training in adult men. According to their findings with isokinetic contractions at both low and high speeds, the isokinetic training procedure demonstrated marked superiority over the isotonic methods. In 1972, Ariel (29,30,31,32,33,34) introduced the Dynamic Variable Resistance exercise principles which resulted the variable resistance exercise equipment. For the first time biomechanical principles were employed in the design of exercise equipment.

Definitions of Terms:

Due to ambiguity in the literature of certain physiological terms and laboratory procedural differences, the following terms are defined:

1. Muscular strength: The contractile power of muscles as a result of a single maximum effort.
2. Muscular endurance: Ability of the muscles to perform work by holding a maximum contraction for a given length of time or by continuing to move a submaximal load to certain level of fatigue.
3. Isometric training: A muscular contraction of total effort but with no visible limb movement (sometimes called static training).
4. Isotonic training: Raising and lowering a submaximal load, such as a weight, a given number of times (sometimes called dynamic training).
5. Isokinetic training (Accommodating Resistance): Muscular contraction at a constant velocity. As the muscle length changes, the resistance alters in a manner which is directly proportional to the force exerted by the muscle.

6. Concentric Contraction: An isotonic contraction in which the muscle length decreases (i.e., the muscle primarily responsible for movement becomes shorter).
7. Eccentric Contraction: An isotonic contraction in which the muscle length increases (i.e., the muscle primarily responsible for movement becomes longer).
8. Muscle Overload: The workload for a muscle or muscle group which is greater than that to which the muscle is accustomed.
9. Repetitions: The number of consecutive times a particular movement or exercise is performed.
10. Repetition Maximum (1RM): The maximum resistance a muscle or muscle group can overcome in a maximal effort.
11. Sets: The number of groups of repetitions of a particular movement or exercise.
12. Variable Resistance Exercise: As the muscle contracts, the resistance changes in a predetermined manner (linear, exponentially, or in a user defined manner).
13. Variable Velocity Exercise: As the muscle contracts with maximal or submaximal tension, the speed of movement changes in a predetermined manner (linear, or exponentially, etc.)

In most existing exercise equipment today and in the previously cited research, resistive training was performed with "tools" which lack intelligence. That means the equipment was "unaware" that a subject was performing an exercise on it. For example, the equipment employed in the study conducted by Pipes and Wilmore assumed certain velocities on the isokinetic modality used. However, verification of the speed was impossible since a closed loop feedback and sensors were not used as they do not exist on the equipment employed. However, with the advent of miniaturized electronics in computers, it is possible today to join exercise equipment with the computer's artificial intelligence. For the first time it is possible for the equipment to adapt to the user rather than for the user to adapt to the equipment.

Another important consideration in both the design of equipment for resistive exercise and the performance of an athlete or a busy executive is that the human body relies on preprogrammed activity by the central nervous system. This control necessitates exact precision in the timing and coordination of both the system of muscle contraction and the segmental sequence of muscular activity. Research has shown that a characteristic pattern of motion is present during any intentional movement of body segments against resistance. This pattern consists of reciprocally organized activity between the agonist and antagonist. These reciprocal activities occur in consistent temporal relationships with the motion parameters, such as velocity, acceleration, and forces.

Hellebrandt and Houtz (236A) shed some light on the mechanism of muscle training in an experimental demonstration of the overload principle. They found that repetition of contractions which place little stress on the neuromuscular system had little effect on the functional capacity of the skeletal muscles; however, they found that the amount of work done per unit of time is the critical variable upon which extension of the limits of performance depends. The speed with which functional capacity increases suggests that the central nervous system, as well as the contractile tissue, is an important contributing component of training.

In addition to the control by the nervous system, the human body is composed of linked segments and rotation of these segments about their anatomical axes is caused by force. Both muscle and gravitational forces are important in producing these turning effects which are fundamental in body movements in all sports and daily living. Pushing, pulling, lifting, kicking, running, walking, and all human activities are results of rotational motion of the links which are made of bones. Since force has been considered the most important component of athletic performance, many exercise equipment manufacturers have developed various types of devices

employing isometrics and isokinetics. When considered as a separate entity, force is only one factor influencing successful athletic performance. Unfortunately, these isometric and isokinetic devices inhibit the natural movement patterns of acceleration and deceleration.

The three factors underlying all athletic performance are force, displacement, and duration of movement. In all motor skills, muscular forces interact to move the body parts through the activity. The displacement of the body parts and their speed of motion are important in the coordination of the activity and are also directly related to the forces produced. However, it is only because of the control provided by the brain that the muscular forces follow any particular displacement pattern, and without these brain center controls, there would be no skilled athletic performances. In every planned human motion, the intricate timing of the varying forces is a critical factor in successful performances.

In any athletic performance, the accurate coordination of the body parts and their velocities is essential for maximizing performances. This means that the generated muscular forces must occur at the right time for optimum results. For this reason, the strongest weight lifter cannot put the shot as far as the experienced shotputter. Although the weight lifter possesses greater muscular force, he has not trained his brain centers to produce the correct forces at the appropriate time.

Because most athletic events are ballistic movements and since the neural control of these patterns differs from slow controlled movements, it is essential that training routines employ programmable motions to suit specific movements.

Resistive Exercising Methods:

There is a significant difference between various resistive training methods. When comparing isotonic and isokinetic exercises, for example, in the isotonic exercises the inertia, that is, the initial resistance, has to be overcome first and then the execution of the movement progresses. The weight of the resistance can not be heavier than the maximum strength of the weakest muscle acting in a particular movement or else the movement can not be completed. Consequently the amount of force generated by the muscles during an isotonic contraction does not maintain maximum tension throughout the entire range of motion. In an isokinetically loaded muscle, the desired speed af movement occurs almost immediately and the muscle is able to generate a maximal force under a controlled and specifically selected speed of contraction. The use of the isokinetic principle for overloading muscles to attain their maximal power output has direct applications in the fields of sport medicine and athletic training. Many rehabilitation programs utilize isokinetic training to recondition injured limbs to their full range of motion. The unfortunate drawback to this type of training is that the speed is constant and there are no athletic activities which are performed at a constant velocity.

In isotonic resistive training, is more than one repetition is to be used, one must use submaximal overload on the initial contractions in order to complete the required repetitions. Otherwise, the entire regimen will not be completed due to fatigue. Berger and Hardage (61B) studied this problem by training two groups of men with 10-RM. One group trained following the standard Berger Technique while the other group used on repetition maximum for each of the ten repetitions. This was accomplished by progressively reducing the weight for the next repetition in a manner which paralleled the fatigue of the muscle. The results showed that the intensity of the work seemed to be the important factor in strength increases, since the maximal overload group showed significantly greater strength gains than did the standard 10-RM group.

Based on these findings it would seem appropriate to assume that a modality which can adjust the resistance so that it parallels fatigue to allow the maximum RM for each repetition would be superior to the currently available equipment. Berger accomplished this function by removing weight from the bar while the subject trained. This is neither the most convenient nor the most practical method. With the aid of the modern computer, this function can be performed automatically.

Another drawback with current isotonic types of resistive exercises is that with the aid of inertia, due to the motion, the resistance changes depending on the acceleration of the weight and the body segments. In addition, since overload on the muscle changes due to both biomechanical levers and the length tension curve, the muscle can only obtain maximal overload in a small portion of the range of motion. To overcome this shortcoming in resistive training, several companies have manufactured strength training devices which have "variable resistance" mechanisms in them. However, these "variable resistance" systems increase the resistance in a linear fashion and this linearity does not truly accommodate the individual. When including inertial forces to the variable resistance mechanism, the accommodating resistance might be cancelled by the velocity of the movement.

There seem to be unlimited training methods and each system is supported and refuted by as many "experts". In the past, the problem of validly evaluating the different modes of exercise was rendered impossible because of the lack of the proper diagnostic tools. For example, in the isotonic type of exercise the investigator does not know exactly the muscular effort and the speed of movement but knows only the weight which has been lifted. When a static weight is lifted the force of inertia is a significant contribution to the load and cannot be quantified by feel or observation alone. In the isokinetic mode, the calibration of the velocity is assumed and has been very poorly verified. The rotation of a dial to a specific location does not guarantee the accuracy of subsequently generated velocity. In fact, discrepancies as great as 40 percent are found when verifying the velocity of the bar.

THE INTELLIGENT EXERCISE MACHINE

In all the previous descriptions of exercise equipment, the user has had to determine the amount of resistance and the number of repetitions desired. The reason the user made the choices was, of course, that the exercise equipment itself was inherently incapable of any intellectual participation. However, with the advent of computers, it became possible to design exercise equipment with artificial intelligence enabling the computerized machine to select the best exercise method based on each individual user. Thus, the user need not be an expert in any biological, physiology, or exercise area since the exercise machine is programmed with information from many scientific fields thus, correctly benefiting the different individual users.

The exercise machine described herein is the result of the application of many unique, innovative features and mechanisms to the long-established fields of resistive exercise or training for athletics, rehabilitation, and physical fitness. The underlying principle behind these innovations is that of a computer controlled feedback of servo-mechanism which is able to maintain any desired pattern of force and motion throughout the range of each exercise, regardless of the magnitude or rate of force applied by the person exercising. The advantages of an intelligent feedback-controlled mechanism over existing resistive exercise mechanisms are many.

First, all systems which employ weights as the mechanism for resistance have major drawbacks in four or more areas: (1) biomechanical considerations, (2) inertia, (3) risk of injury, and (4) unidirectional resistance. The biomechanical considerations are the most important for exercise equipment and have been previously explained. Inertia is the property of resisting any change in motion and, because of this property, it requires a greater force to begin moving

weights than it does to keep them moving in a constant manner. Similarly, when the person exercising slows his motion at the end of an exercise movement, the weights tend to keep moving until slowed by gravity. This phenomenon reduces the required force at the end of a motion sequence. This property becomes especially pronounced as acceleration and deceleration increase, effectively reducing the useful range of motion of weight-based exercise equipment. The risk of injury is obvious in weight-based exercise equipment. When weights are raised during the performance of an exercise, they must be lowered to their original resting position before the person using the equipment can release the equipment and stop exercising. Injury could easily result if the weights fell back to their resting position accompanied by the concomitant motion of the bar or the handle attached to the weights. If the person exercising happened to lose his grip, or was unable to hold the weights due to exhaustion or imbalance, serious injuries could and have resulted. Finally, while being raised or lowered, weights of exercise equipment employing weights offer resistance only in the direction opposite to that of gravity. This resistance can be redirected by pulleys and gears, but still remains unidirectional. In almost every exercise performed, the muscle or muscles being trained by resistance in one direction are balanced by a corresponding muscle or muscles that could be trained by resistance in the opposite direction. With weight-based systems, a different exercise, and often a different mechanism, are necessary to train these opposing muscles.

Exercise mechanisms which employ springs, torsion bars, and the like are able to overcome the inertia problem of weight-based mechanisms and can partially overcome the unidirectional force restriction by both expanding and compressing the springs. However, the serious problem of safety remains. An additional problem is the fixed, non-linear resistance which is characteristic of springs and is usually unacceptable to most users of exercise equipment.

The third type of resistive mechanism commonly employed in existing exercise equipment is that of a hydraulic mechanism. This mechanism is able to overcome the inertial problem of weights and the safety problem of both weights and springs. With the appropriate selection or configuration of hydraulic mechanisms, the unidirectional problem can also be overcome. However, previous applications of the hydraulic principle have demonstrated a serious deficiency that has limited their popularity in resistive training. This deficiency is that of a fixed (although perhaps preselected) flow rate though the hydraulic system. With a fixed flow rate, it is a well-established fact that resistance is a function of the velocity of the piston, and in fact, varies quite rapidly with changes in velocity. It becomes difficult for person exercising to select a given resistance to train with since he or she is usually constrained to moving either slower or faster than he would like in order to maintain this resistance. Additionally, at any given moment, the user is unsure of just what the performing force or velocity actually is. For these reasons, hydraulic mechanisms have found only limited acceptance among serious users of exercise equipment.

Feedback Control of Exercise:

The Computerized Exercise Machine possesses several unique advances over other resistive exercise mechanisms, both fixed and feedback-controlled. The most significant of these advances is the introduction of a stored-program computer to the feedback loop. The computer, and its associated collection of unique programs, allows the feedback-controlled resistance to vary not only with the measured parameters of force and displacement, but additionally, to modify that feedback loop while the exercise is in progress. This modification can, therefore, reflect changes in the pattern of exercise over time. The unique program selection can effect such changes in order to achieve a sequential or patterned progression of resistance for optimum training effect. The advantage of this capability over previous systems is that the user can select the overall pattern of exercise and the machine assumes responsibility for changing the precise force level, speed of movement, and temporal sequence to achieve that pattern.

Consider the following typical examples of exercises which can be performed on this machine which would be impossible on any other exercise machine. A user wishes to select a resistance (weight, in classical terms) starting at 1/2 the body weight, and to have that resistance increase by 10 percent in each successive repetition, until the user reaches a "sticking point" and cannot continue. With a classical weight machine, he would have to initially select weights equal to half his body weight. Then the user would have to stop between each repetition to change weights, with the probability that he would not be able to select the desired unit of increase since weights are normally available in 5,10,25, or 50 pound units only. In addition, the training effect of the exercise is considerably affected because, while stopping to change weights, the muscles "recover". If, with the Isokinetic or other devices, there were a force readout (which is not included on any of the currently available equipment), the user would have to watch that readout and match the force pulled with the desired force as it appeared on the readout. (This is analogous to trying to keep the high performance race "car" on the "road" in the video arcade games.) This would require more control and concentration than most persons are capable of especially with the onset of exercise-induced fatigue, With the Computerized Exercise Machine, the person's weight would automatically be determined by having him support himself briefly on the exercise bar. Then the computer would select the pattern of increasing force, starting at precisely half the body weight, and increasing the resistance by just 10 percent after each repetition until it detected that the user could no longer move the bar. At this point, it would report the final force level, the number of repetitious, and, if desired, the progress the user had made since the last exercise session.

A second example is that of a user desiring to exercise with a constant force or a pre-determined force pattern (i.e. non-linear force through the range of motion). In addition, at the point in the range of motion where his speed is the lowest (the weakest point), the user may want the bar to "lock" for three seconds so that strength could be enhanced through isometric rather than isotonic exercise. After the three second isometric contraction, the motion would be allowed to continue through the next cycle until this sticking point would again be encountered. Experts in various professions believe that such an exercise is a vast improvement over conventional resistive training for developing strength at a person's weakest points. Yet it would be impossible for this exercise to be performed on any other exercise machine known to exist. Not only can the proposed exercise system perform this pattern of exercise, but during and after the exercise it can display the level of strength at the "sticking point" and how this compares both to previous strength levels and to the strength over the entire range of motion. In addition, the programs are then able to adjust ensuing exercise sessions to select the proper range of forces to continue to build strength based on the progress to date. All of this is accomplished without the user having to remember or reenter any data.

As can be seen from the previously cited review of resistive exercise methods, it would seem that the future will rely increasingly on computerized exercise modalities for training and rehabilitation. Current research revealed significantly greater progress in muscular strength for the subjects who trained on the Computerized Exercise Machine. In addition, more efficient and less time-consuming workouts as well as fewer injuries and higher motivation are possible which can produce improved results. The Computerized Exercise Machine is programmed for several training modes. One mode is diagnostic for determining the individual's range of movement as well as the force and speed exerted through that range of movement. On a color CRT, the user can see the force and the velocity curves or print a copy of the display. A second training mode controls a predetermined resistance which can be set in several ways - linear, exponential, user defined and an "ideal" curve. A third mode allows setting "sticking points", or isometric contractions, at any points through the range of movement. The fourth training mode can set a "fatigue level" to which the user exercises until reaching that level. The fifth mode is a power and endurance training which controls the amount of work performed. Another exercise mode is variable velocity training. In this type, the velocity can be predetermined in many possible fashions which also allows the user to exercise in an isokinetic mode. The amount of resistance

can be set as a function of the forces exerted by the user for each repetition. The computer "senses" the changes of forces throughout the range of movement and makes the appropriate adjustments in order to accommodate the user. The Computerized Exercise Machine has many other features which are fully programmable and allow tremendous flexibility for the user.

Some of the capabilities of the Computerized Exercise Machine were studied in several research projects performed at the Coto Research Center at Coto de Caza, California. One study was designed to compare the Computerized Exercise Machine to other existing equipment. The study examined the effect of similar training regimens, using several types of training equipment, on the development of muscular strength. The results of the study demonstrated superiority of the Computerized Exercise Machine over the other types of equipment. The study concluded that the Computerized Exercise Machine was more effective in developing muscular strength. It seems that the Computerized Exercise Machine allows adaptation to the maximum effort of the muscular contraction. In addition the interactive feature of the machine permitted maximum motivation for the user and there was a significant carry over effect to other exercise modalities and other independent athletic skills from training on the Computerized Exercise Machine.

Another study was designed to examine the effect of resistive training on limb velocity. The subjects were tested on the maximum speed they could lift a 32 pound load, and in addition, how fast they could lift loads of 40 and 70 percent of their maximum. Again, the results revealed a significant rate of improvement of the Computerized Exercise Machine users over the other training modalities.

A study was designed to determine the aerobic adaptation to work and fatigue training modes on the computerized Exercise Machine and to compare those effects to the aerobic adaptation of running/jogging. The running group trained three times per week for 40 to 50 minutes of jogging and punning. The Computerized Exercise Machine groups were trained in power/work mode or in a fatigue mode. The results of the Study revealed that, for the same amount of time devoted for exercise, the Computerized Exercise Machine users improved their aerobic capacity by almost 20 percent while the runners improved only 12 percent. The Computerized Exercise Machine users also significantly improved in strength/power and local muscular endurance.

From these results, it can be concluded that the Computerized Exercise Machine is more effective than the other modalities tested for several reasons. The subjects could constantly interact with the machine and receive immediate feedback about their effort. While exercising, the subjects were motivated by the interactive results which reported the average and maximum force produced as well as the velocity associated with the movement. During each session, a comparison to previous sessions was displayed on the CRT, a feature that constantly motivated the subjects to work harder. This motivation contributed to the stimulation for the Computerized Exercise groups to work at their maximum effort. Unlike other modalities, the subjects were not restricted to the range where biomechanically the limb would be at a disadvantage and would have to stop exercising. On the Computerized Exercise Machine, at this point, the intelligence of the machine reduced the resistance and allowed the subject to complete the set at his maximum effort. The Computerized Exercise Machine opens a new dimension in the area of training, rehabilitation, and research.

NEUROMUSCULAR INTEGRATION

In order to understand the computational abilities of the brain, it is necessary to understand something of the basic structure and function of the neuronal substrata. Early descriptions of brain models considered the brain as a randomly connected network of binary neurons. In fact, the brain is highly structured and far from random connections with the neurons functioning as

both analog and digital computing devices. The neurons in the brain are arranged in quite regular patterns and are grouped in functional divisions. Precise and regular mappings exist from the sensory organs to the sensory processing regions of the cortex, as well as from the motor centers to the muscles of various parts of the body. Somehow the neurons from one area know how to locate and establish contact with neurons in other quite distant areas.

The unit of the brain structure is the neuron. It is a living entity, that is, a cell like all the other cells in the body. The neuron, however, is specialized for information processing. Each neuron is shaped by its surroundings, by the composition of the chemical bath in which it swims, by the hormones it detects, by the electrical and chemical fields and gradients it experiences during its growth and maturing, and by the nature and timing of the electrical impulses and chemical transmitters produced in its vicinity as a result of activity of other neurons.

The neuron can be compared, in simplified terms, to a "gate" in a digital computer or to an operational amplifier in an analog device. Any movement executed by the body must start with the neuron. Neurons have four basic parts: a cell body, a set of dendrites, an axon, and a set of terminal buttons. Neuronal information is transmitted to the action site through the axon. However, the axon does not transmit the information as a DC or AC current but in pulses with certain frequencies. All the pulses which are transmitted have approximately the same height and the same duration. In digital computers electronics, these would be considered as binary bits and are analogous to "1"s and "0"s. A pulse is transmitted on the axon whenever the analog voltage in the cell body exceeds a certain threshold voltage value. Using electronic terms, this is an "action potential" which initially generates a pulse. After the pulse occurs, the voltage in the cell body of the nerve returns to the initial base line value. In electronics terms, the nerve acts like a Schmitt trigger.

The cell body interacts with the axon from the preceding neuron at the synapse. The synapse can transmit in only one direction and, therefore, acts like a diode. The synapse is an electrical gate, or valve, whose resistance to the flow of current is controlled by the receipt of transmitter chemicals from the axon buttons of other neurons.

Communication of information across synapses is one-way and flows from the terminal buttons of one neuron to the dendrites or cell body of another neuron. The presence of the transmitter causes an electrical current to flow in the synapse of the receiving neuron. This current may be either positive or negative, depending on the type of transmitter chemical released. As a general rule, a particular neuron releases only one type of transmitter chemical. Thus, neurons can be classified as either excitatory, causing positive current to flow in receiving neurons, or inhibitory causing negative currents to flow. There is a synaptic receptor for every axon button. Thus, there are two types of synaptic receptor sites: excitatory and inhibitory. A single receiving neuron may have both excitatory and inhibitory inputs. Communication of information across synapses is one-way, flowing from the terminal buttons of one neuron to the dendrites or cell body of another neuron.

It is interesting to note that the voltage in the cell body is an analog signal and represents an algebraic sum of the inputs. This means that many small low voltage signals, which by themselves would not have an effect, may sum algebraically to produce a quantity sufficiently large to activate the nerve. When all the input signals on the cell body exceed the threshold, a pulse is initiated down the axon and, it is said, that the neuron has fired. In other words, the neuron has digital inputs which are converted into analog values. These values are processed algebraically in an analog fashion. The arithmetic result is then reconverted into a digital form for transmission. The neuron can behave in different ways depending on the level and frequencies of the incoming signals. For example, activation may require several equally weighted inputs in order to reach the firing threshold. This type of response is analogous to an "AND" gate. A different type of response is that any of several inputs can drive the cell past the firing

threshold; this is analogous to an "OR" gate. A third possibility is that some signals may be subtracted from the sum. This allows both inhibitory and excitatory inputs providing the neuron with characteristics of both "AND" and "OR" gates. Since the individual cell can have thousands of inputs, one can appreciate the nearly infinite possibilities. Thus, the cell must integrate, differentiate, and process other functions through feedback and analog operations.

Interaction of inputs from different axons is called "spatial summation" and interaction of sequential pulses on the same axon is referred to as "temporal summation". In other words, the signals from the neurons can be evaluated by the brain as "what", "where", and "how much". Once the neuron has computed its result, this information is transmitted to its destination which could be another neuron, a muscle, or a gland. Transmission is not a simple problem because the signal voltage is small, less than a tenth of a volt, and the distance may be quite far. The action potential allows the signal voltage of the neuron to be transmitted over long distances by encoding it as string of pulses. This means that the choice of a particular axon specifies "what" and "where". The amount of frequency indicates "how much". This system enables the nervous system to select which muscle to use and how much resistance to create. If a person wants to flex his or her elbow with a certain load held in the hand, the brain must direct the signal through the axons to the proper muscles activate the correct number of fibrils. If the elbow is flexed first, then the summation of signals activates enough sarcomeres to allow the proper muscles to flex the elbow. Once the elbow is extended if the person wants to lower the weight, the summation signal in the brain must calculate the level of contraction necessary to execute the task including computations needed for the gravitational effects which are an integral part of the movement. The net moment around the elbow must consider also the required stabilization between the extensors and flexors and the geometrical orientation of the limbs active in this movement. This ability of the nervous system allows large numerical quantities to be processed with what might be called a temporal byte or the integration, over a brief time period, of a single input line. Thus, the brain structure receiving the information can determine the type and location of the stimulation with a spatial byte, which is a place code allowing determination of the set of active lines, and the intensity of the stimulation with a temporal byte or frequency code.

All muscular activities utilize a basic unit on contraction which consists of spatial and temporal dimensions. Independent sets of information can be encoded in these two dimensions and they can then interact in the receiving structure in a way determined by physical and chemical properties of the cell's membrane. The spatial aspect of the code is essentially digital information and the temporal aspect of the code is essentially analog information, although it is encoded in the frequency of digital pulses.

A simple explanation of these physiological parameters would be impossible. Even mathematical treatments of this sequence represented in various models incur great difficulties. The number of degrees of freedom exceeds the number of simultaneous equations required to quantify all the possibilities. However, logical and experimental results may provide logical conclusions although inconclusive and possibly erroneous. (Remember the statement at the beginning of this article by Leonardo Da Vinci!)

A very important property of the neuron is that many inhibitory inputs are received and processed as well. The total of these inhibitory signals causes the voltage across the neuronal membrane to move away from the firing threshold. This action cancels the combined action of

all the positive inputs. It is also possible to have a negative synapse which negates only a specific input. This type of signal called presynaptic inhibition.

In addition, the speed of transmission along the axon plays an important role in controlling and processing of the motor nervous system. The speed of the transmission of pulses down the axon can vary over a wide range although it is always the same in any given axon. This means that high speed axons can move data quickly, but low speed axons may be employed as delay lines. Because axons can have branches coming off at any point, delay lines may be introduced. A greater amount of depolarization at the neuron will cause a higher frequency. A lesser amount will cause a lower frequency. Thus, analog voltage in the cell body can be converted into a series of pulses at a particular frequency.

Signal encoding by action potentials unfortunately introduces noise into the information channel. This is because the action potential is a discreet event as is the pulse spacing between action potentials. The encoding of a continuous voltage as a string of pulses forms a noise signal. The brain overcomes this noise by redundancy. This information repetition utilizes many neurons transmitting the same message, each encoded slightly differently, so that the average of a large number of neurons produces the accuracy needed for precise control. This redundancy also provides improved reliability which is important in a structure in which approximately ten thousand neurons die every day due to disease, injury, or old age.

It is beyond the scope of this article to provide an indepth discussion of the complexity of the nervous system in creating muscular contraction. However, in general, the basic architecture of the system is hierarchical. Each of the major functions of the system is partially organized at each level of the system instead of having particular levels devoted to specific functions.

The most obvious partitions of the central nervous system divide it into three levels. At the lowest level is the spinal cord, above that is the brain stem, and finally at the top is the forebrain.

At the lowest levels, there are a multitude of relatively simple processing elements doing similar jobs. At the higher levels, there are a few very complex and powerful processing elements defining system tasks and priorities and organizing the activities of the lower levels to achieve the goals.

On the input side, the lowest levels gather raw data, which is then progressively abstracted, sorted, and refined at each stage according to general guidelines which may be either hard-wired or determined at higher levels. The highest levels then receive abstract symbolic information about the general state of the environment rather than discreet bits of information. Therefore, in order to flex the elbow joint, the higher center recognize the flex and extend movement rather then how many sarcomeres are involved in each of the movements. The output functions begin at the highest levels which determine general goals and strategies and transmit these to the lower levels. The lower levels, in turn, send information about desired actions and timing to the lowest levels for execution.

One of the most important signal from the lower centers is the proprioceptive sensor mechanisms in the muscle spindle organ, shown in Figures 22, 22a, and 22b. The output of the spindle sensor travels to the spinal cord where it enters through the dorsal roots and terminates with excitatory synapses on the dendrites of the alpha motor neurons as shown in Figure 23. When the gamma neuron fires at a rate g1, it shortens the ends of the spindle to a length l(g1). If the muscle bundle attached to the spindle is stretched by more than an amount L(g1), the spindle will fire, sending a signal to the motor neuron that controls the muscle bundle commanding it to resist further stretching.

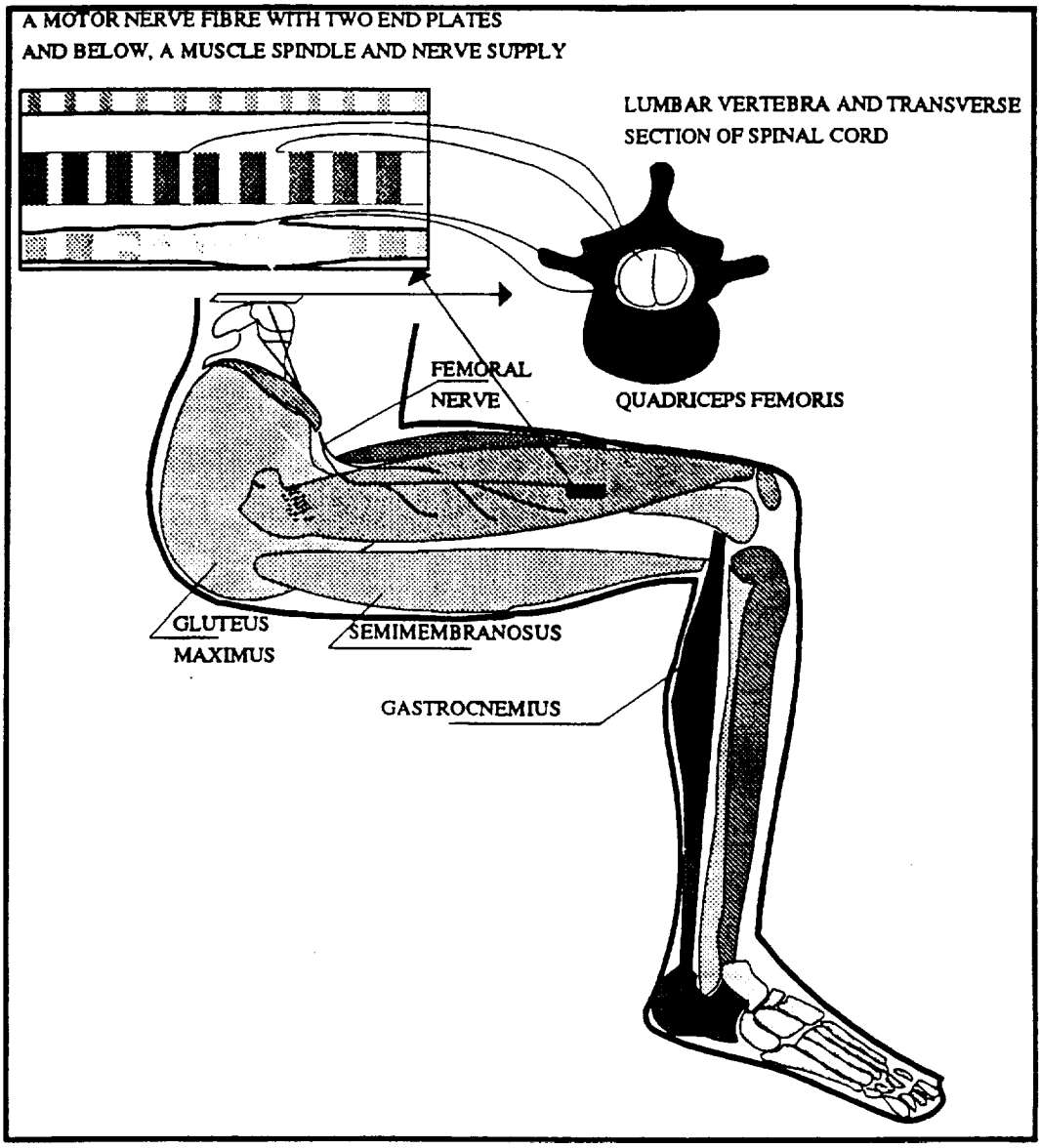


Figure 22

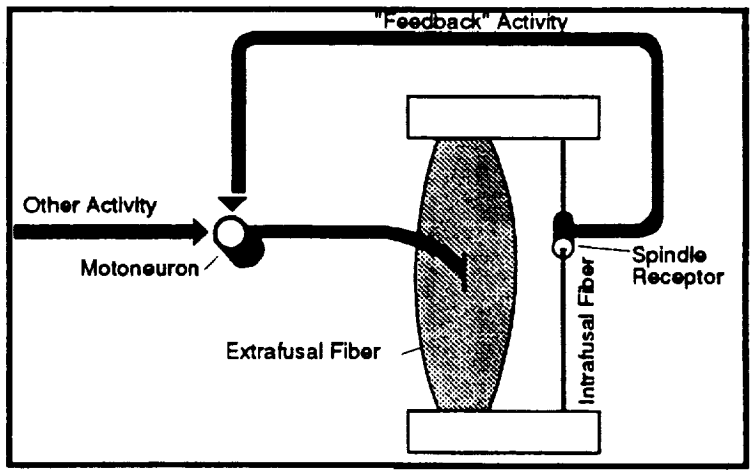
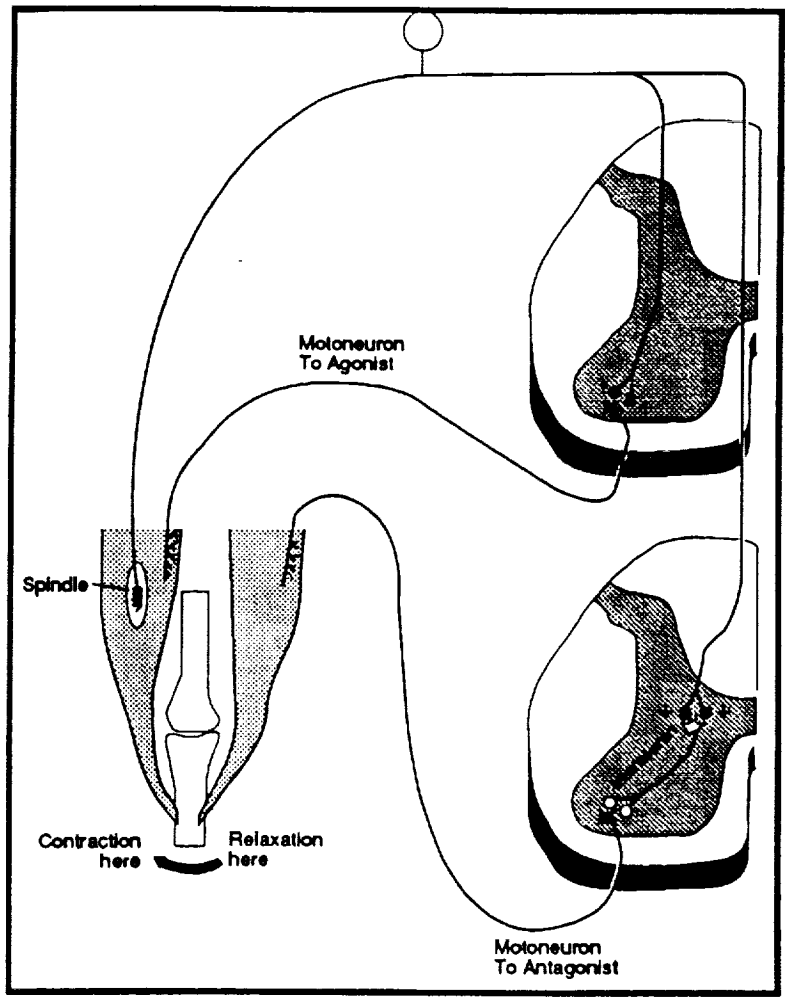


Figure 22a



Fgure 22b

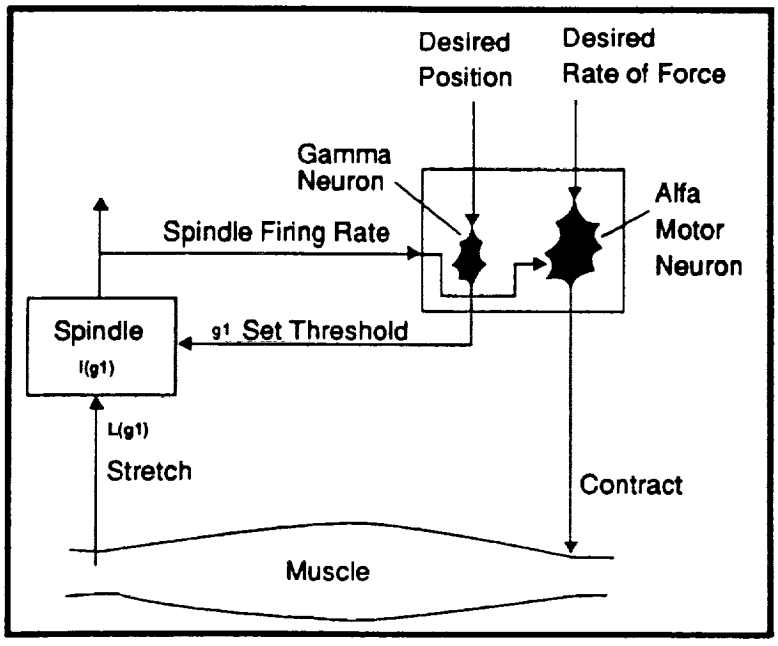


Figure 23

Thus, the gamma neuron can determine the point at which the stretch resists further movement. The result is that the limb moves to a position set by the firing rate on the gamma motor neuron. The gamma neuron, muscle spindle, and motor neuron thus comprise a position servo. A specific firing rate on the gamma neuron tends to produce a particular length of a muscle and, hence, a unique angular position of the joint. Figures 24 and 24a shows a schematic diagram of the computing modules involved in the gamma position servo. The position command enters the motor output module and generates a particular firing rate on a gamma neuron. The gamma neuron sends its indication of what the spindle length should be to the sensory spindle where it becomes an expected position. The spindle compares the actual position with the expected, emits an error signal which is sent to the motor output module as an excitatory feedback signal. The motor output module, whose output is the alpha motor neuron, then uses this feedback together with input from other motor centers to compute its output signal which it encodes as a firing rate to the muscles.

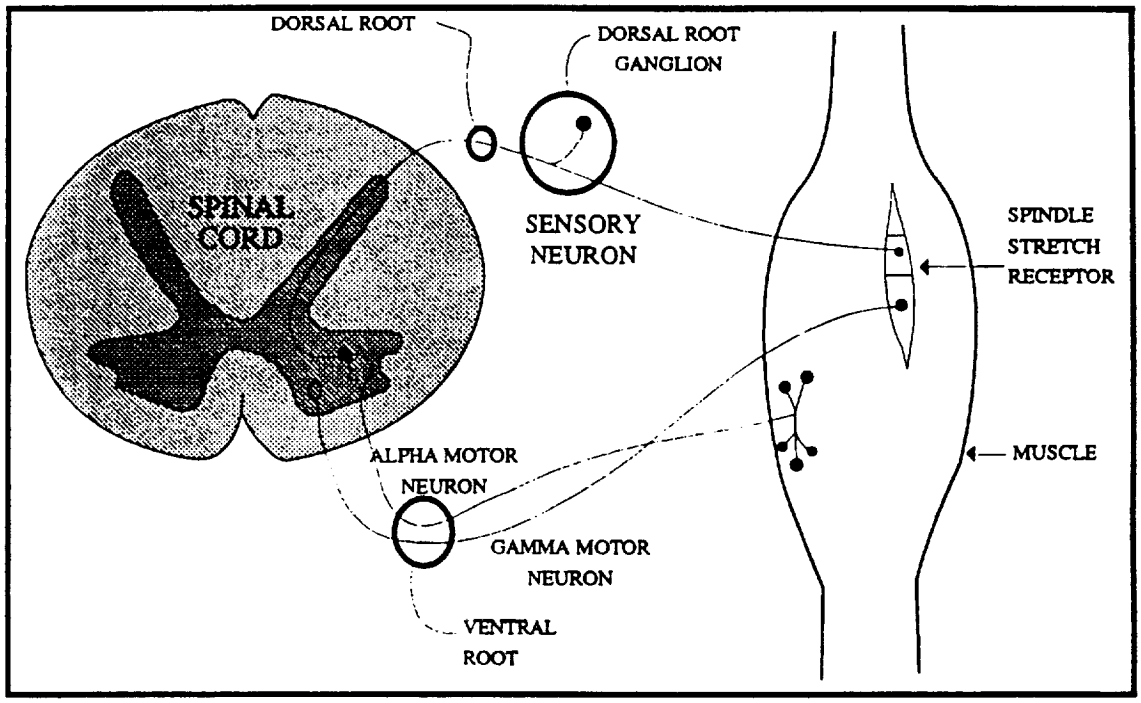


Figure 24

Some of the other motor center inputs come from commands to and feedback from other limbs. The rest of the other inputs come from higher motor centers such as the pyramidal fibers from the motor cortex; extrapyramidal fibers come from the red nucleus, the substantia nigra, subthalamic nucleus, and the vestibular nucleus. All of these synapse directly on the motor neurons. These various inputs essentially command the motor neurons to fire at a particular rate which produce a specific force or rate of contraction of the muscles. This is represented in Figures 25 and 25a.

In addition to the spindles that measure the amount of stretch in the muscles, there are the Golgi tendon organs that measure the tension in the tendons. Axon fibers from the Golgi tendon organs enter the dorsal roots and make excitatory synapses on interneurons. These interneurons then make inhibitory synapses on the motor neurons. The overall effect is to limit the force exerted by the muscle preventing excessive stress from tearing the muscles or tendons. When tension in the tendon organs approaches the danger level, they fire vigorously causing an immediate relaxation of the muscle. Thus, signals from the tendon organs provide the motor output module with the information necessary for controlling the force level in the tendons.

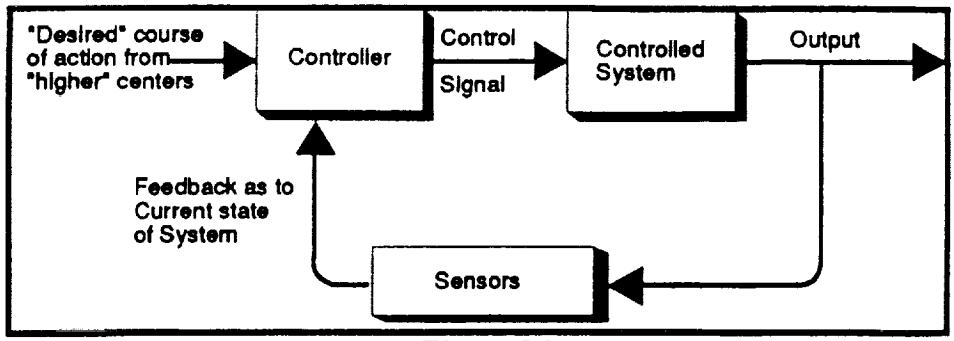


Figure 24a

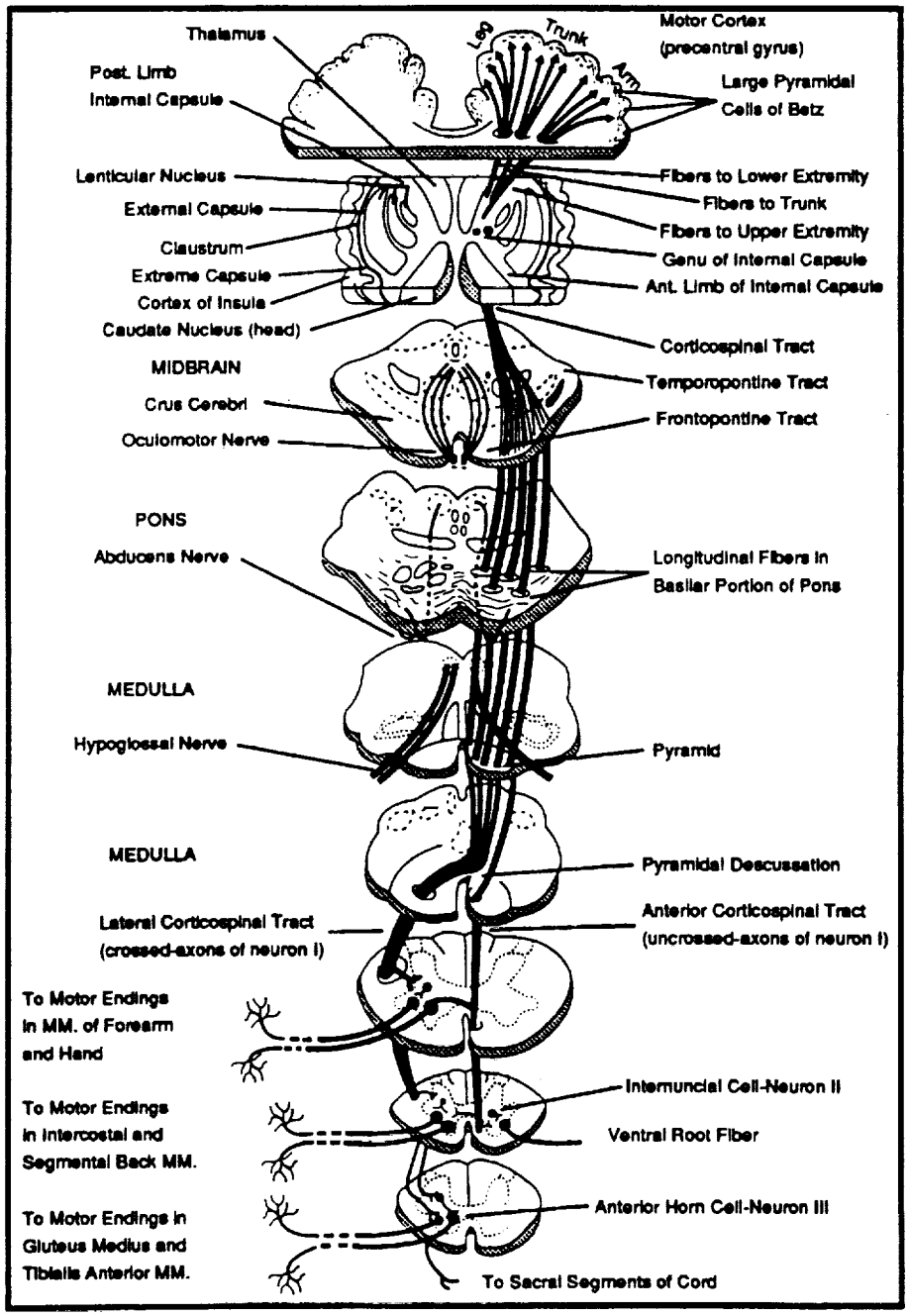


Figure 25

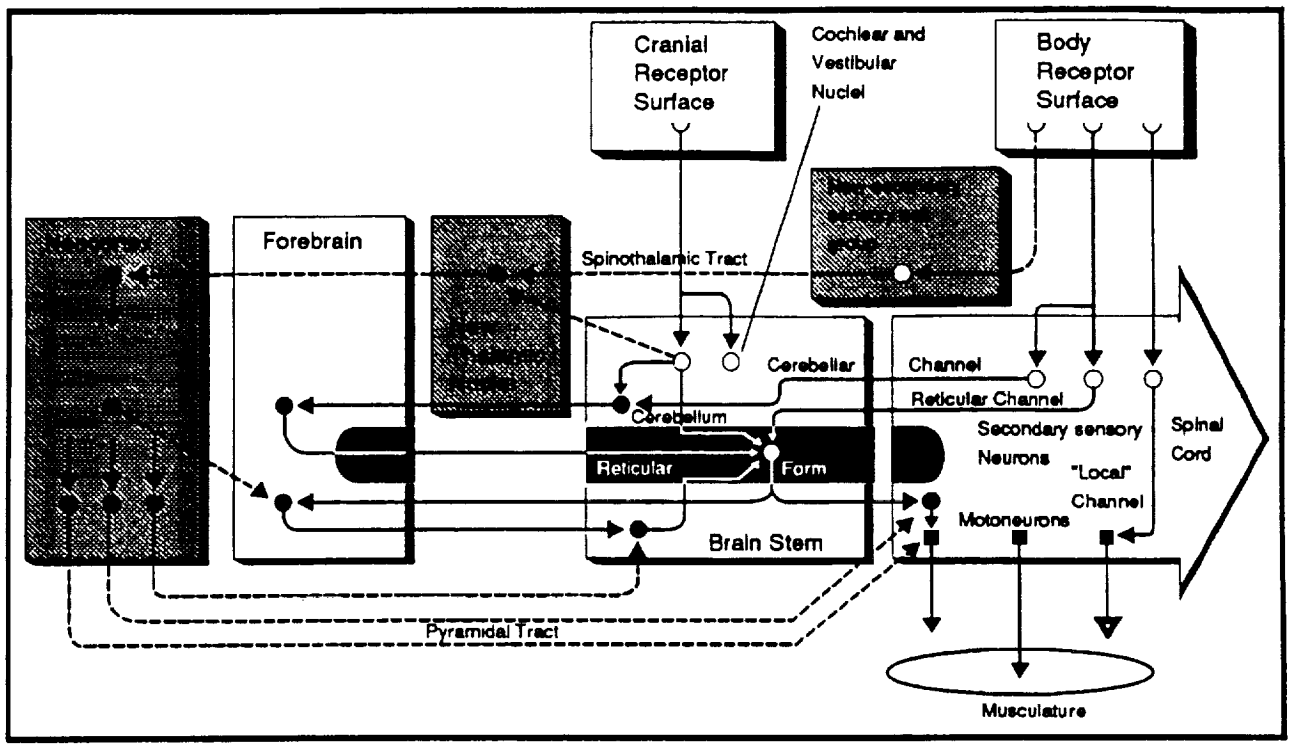


Figure 25a

It is thought local sensory input and commands from higher in the motor system that the gain in the inhibitory portion of the tendon organ loop is modulated. If the inhibitory signal is equal to the difference between the commanded and the observed tension, the motor neuron will increase its firing rate whenever the tension falls below the commanded value. Correspondingly, the motor neuron will reduce its firing rate when the tension rises above the commanded value. The result is that the force in the muscle is servoed to the commanded value.

There are also sensing organs in the joints which measure the position and rate of motion of the joints in addition to the muscle spindles and the Golgi tendon organs. For every sensory input there are specific nerves for each of the different sensations or signals. This nervous system feature has been called the "law of specific nerve energies" or "place encoding." For example, the brain must know the amount of tension on a particular muscle and the position of the joint at that particular angle. If the motor signal is to reduce the angle or flex the joint, the motor system in the brain must know if this task has been accomplished. If the joint center indicates that the limb is moving in the direction opposite to what was required, adjustments must be made. (Attention to such adjustments will be discussed relative to a series of experiments later in this article.)

As a general rule, the intensity of sensory input is encoded by the rate of firing such that each percent increase of stimulus intensity tends to cause an increase in the firing rate by a fixed amount. This leads to a logarithmic or power law relationship between stimulus intensity and firing rate. This is called the Weber-Fechner Law after the two investigators who first carefully measured the effect as shown in Figure 26.

Thus, at each level of nervous system control, there are a number of relatively independent processing elements performing their own jobs in parallel and exchanging information with levels above, below, and laterally. Therefore, any large scale or voluntary task is not processed in a single brain center. Rather, different aspects of the task will be handled by different portions of the functional subsystems within the physical system. For example, consider the case of

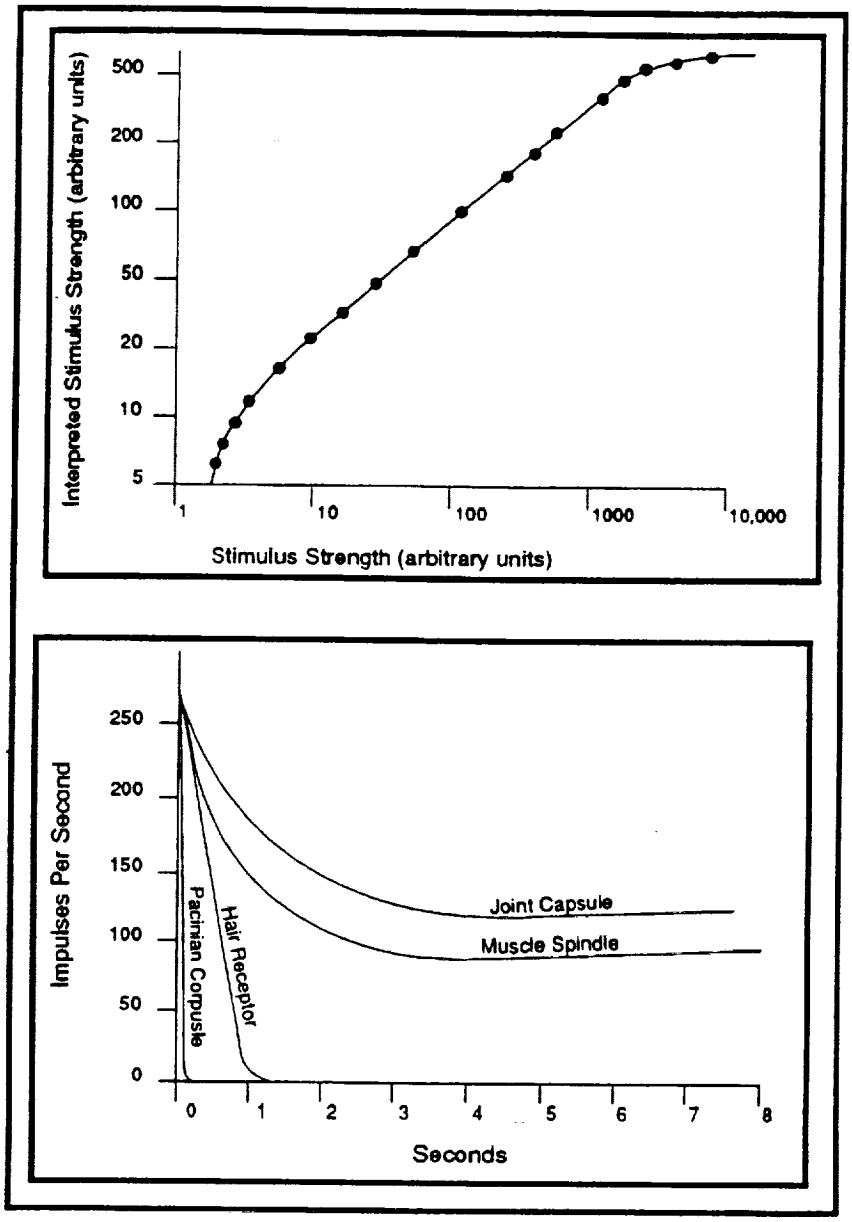


Figure 26

lifting a load by flexing the elbow. There will be continuous computing of forces based on limb positions or velocities requested by higher levels. The brain provides such a processor for each fiber of each muscle and it instructive to examine their approaches to the problem solution involved.

Voluntary limb movements such as this example of elbow flexion necessitates adjustments to be performed quickly and control of the system is made by anticipation of events. This is one of the major principles of the neuromuscular system, the principle of organization. The theory of the principle of organization is that the higher level structures control the lower, not by turning them on when needed, but rather by inhibiting their actions except as desired. Consider running as an example. In running, the muscles involved in plantar and dorsal flexion of the foot which will quickly come into contact with the ground, are fully activated before the impact. In this way, there is force anticipation to absorb the impact. Another example can be found in long jumping. In the analysis we performed on the world's best long jumpers, the results showed that

the man who jumped farther than 8 meters did not use the foot flexors and extensors as driving forces but as blocking forces. This technique enabled the utilization of inertial forces which are much greater than the muscle could generate even though limb muscle contraction was evident prior to takeoff.

The lowest level of the central nervous system is the spinal cord and it is the major route for input and output for the brain. Most of the sensory input from the body and most of the output to the muscles passes through this structure.

The higher centers, including the medulla, the mesencephalon, the thalamus, the hypothalamus, and others, will not be discussed. However, their complexity of regulation and control is an integral part in muscular output. It is interesting to note, however, that the actual physiological mechanisms which are controlled directly by the brain are of only two types: the muscles and the glands. These are the only effector organs to which the brain is connected and, thus, you

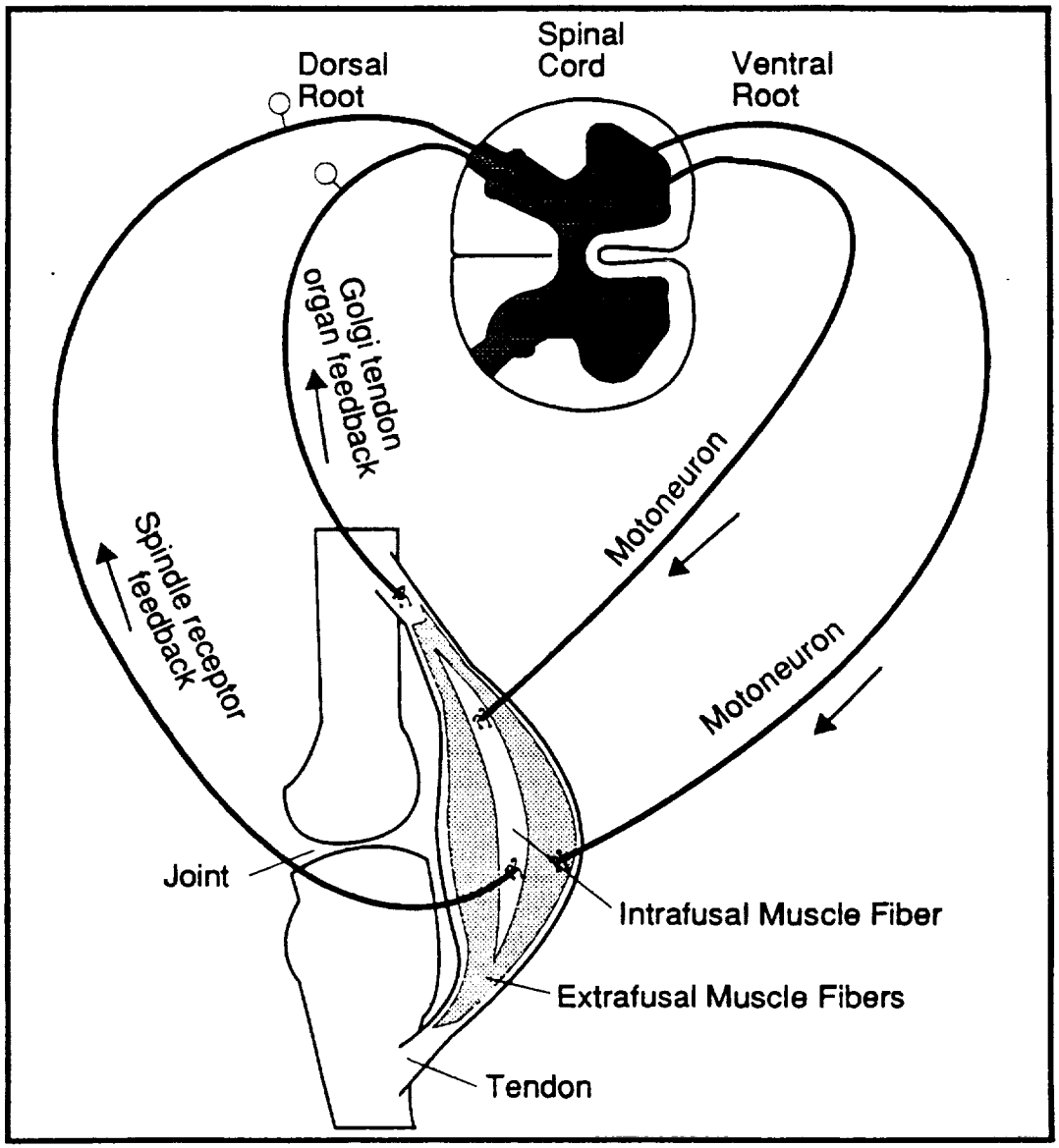


Figure 27

may only contract a muscle and release glandular secretions. The remaining range of physical behavior is only a combination of these two.

Figure 27 provides simplified illustration of the interaction between the high level and the low level controllers. This system governs the servomechanism of muscular contraction.

When it comes to muscular control, there are two fundamental techniques which the brain uses to control muscular contractions. The first mechanism is to equip each level of decision making with subprocessors which accept the commands from higher levels as well as accounting for the inputs from local feedback and environmental information sensors. In this fashion, a descending "pyramid" of processors is defined which can accept very general directives and execute them in the presence of varying loads, stresses, and other perturbations. This kind of input and output control is used for multimodal processes, such as maintaining balance while walking on uneven terrain. However, this type of control is inappropriate for executing deliberate, volitional, higher level goals such as instructing the arm to flex with load held in the hand.

The second technique which the brain utilizes to control muscular contractions applies to the operation of higher level systems which generate output strategies in relation to behavioral goals. These are the categories of output tasks which use informational input rather than the type of generated behavior. The operation of the motor command chain depends upon certain sensory inputs which provide feedback and status information for moment-to-moment operations. The basic required and supplied information relates to the joint angle, muscle loading, and muscle extension or stretch. Various organols at the lower levels of control translate these quantities into neural impulses which are processed by the higher levels of control for the next sequence of instructions by neural transmission.

The functional aspect of muscle control is the motor unit. All skeletal muscle fibers are innervated by the neuron. There are thousands of direct and indirect neural connections to the cell body of the motor neuron, but once an impulse is generated this motor neuron determines the final neural output through which movement is controlled.

All motor neurons transmit impulses from the spinal cord to the muscle fibers they innervate. When the impulse arrives at this point, all of the muscle fibers of the specific motor unit contract. However, motor units differ with regard to speed, force, and endurance. Figure 28 illustrates these differences.

Different populations of motor units account for the speed with which the muscle can react to a stimulus. There are both fast twitch and slow twitch muscle fibers. When considering force production and force-yielding capacity, the force yielding capacity of a motor unit depends on the number of muscle fiber per motor unit, the fiber size, and the type of myofibrils in the particular motor unit.

In different movements, no one type of motor unit is used exclusively. Normally, the requirements for a particular movement will preferentially select slow and fast twitch units in different proportions depending on the movement and the skill desired.

In most cases, muscles work in opposing pairs, that is, one muscle group opens or extends a joint and the other group closes or flexes it. Figure 28 illustrates this kind of arrangement. Local control of this system resides in the spinal cord and it is called the lower motor neuron system. This organization controls the individual fibers of each muscle through input to the motor units that control a varying number of muscular fibers. The organization of the lower motor neuron pool accepts and reconciles commands from a multitude of other systems all of which desire control of the muscle in question.

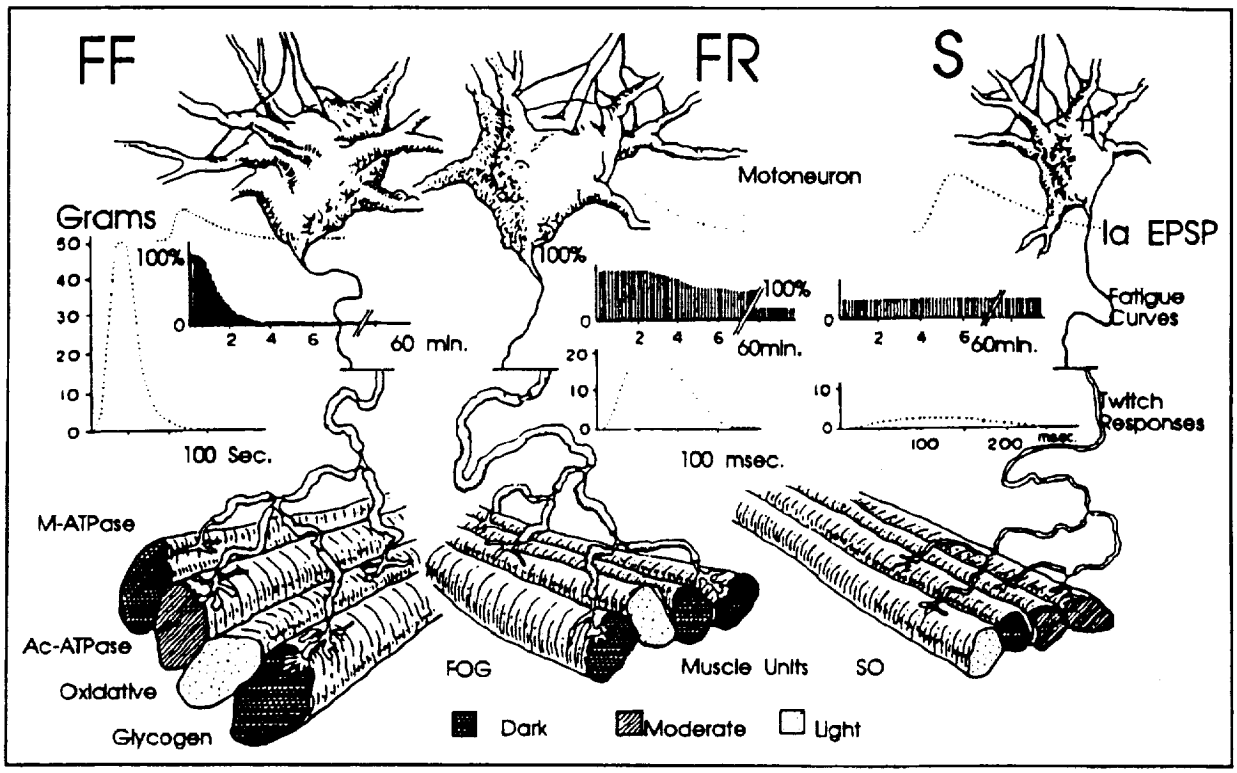


Figure 28

The degree of contraction of the muscle is proportional to the output pulse frequency of the lower motor neuron system. The higher the frequency, the stronger the contraction. In the situation presented in Figure 28, it is apparent that both the extensors and flexors cannot be affected by the lower motor neuron to the same level of intensity simultaneously. There must be a programmable mechanism so that the flexors do not work in opposition to the extensors. Therefore, one group of muscles must be relaxed while the other is active. In addition to the local control system, previously mentioned, there are higher centers of reflex action so the motor system can enact corrective action via other muscles and limbs for the redistribution of weight, to counteract shifts in the center of gravity, etc.

Higher level inputs to the Lower Motor Neuron system may request a variety of actions, such as holding a particular position, moving to a specified position, or moving with a particular velocity. These requests are integrated into the lower motor neuron system according to the following structure. The Lower Motor Neuron system (LMN) attached to the extensor muscle has two functions. One function is to deal with high level instructions while the muscle tension compensates for an external load. The second function is to process information from the muscle itself via the muscle spindle that sends information via the gamma motor neuron. Because the S fiber is mechanically attached to the rest of the muscle, it is passively stretched or relaxed as a result of the neuronal inputs or external forces which extend or contract the main muscle. However, the S fiber's bias, or ambient contractility, is set by its own "private line" input signals from neuron G. The neural component of these stretch receptors is attached to the S fiber, and when this is stretched, the neuron fires at a rate proportional to the degree of stretch.

From a motor integration view, the whole hierarchical motor output system uses a temporal frequency coded as well as analog information derived from the stretch receptors to specify the degree or quantity of an action. In addition, the set of all of the descending input lines to the numerous LMN systems constitutes a spatial byte, or place code, which is essentially digital in character and in which the specified lines select the set of LMN systems which are addressed. This arrangement allows determination of the nature of the movement to be performed, but not its speed, force, or direction.

Figure 29 illustrates some attempts to explain the effect of the higher center on the lower motor neuron pools. Arbib (18) tried to relate the higher center to the control of flexors and extensors system around a particular joint as shown in Figure 29.

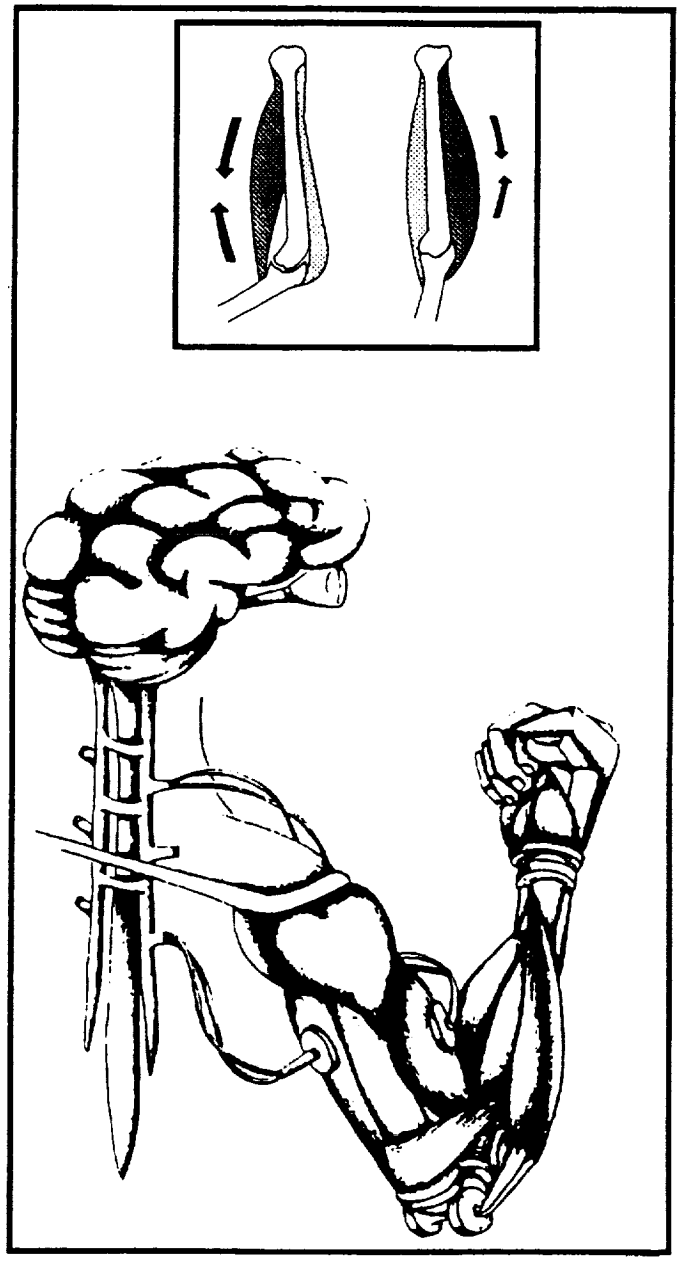


Figure 29

Experiments with rapid movement illustrated a high level of control for a pair of muscles. Studies of isotonic movements, which more correctly should be referred to as "ballistic" or "variable resistance" movements, have shown that muscle activation occupied only a small portion of the movement. In addition, the duration of the activation in an isotonic movement does not seem to be related to the extent of the motion. There is an initial burst of acceleration as the agonist contracts and the antagonist muscle relaxes, an intervening quiet period, followed by a final burst of deceleration as the antagonist contracts. Bizzi (73), experimenting with eye movements, revealed a system in which the initial movement was triggered by an agonist burst and antagonist silence. This activity continued until the end of the movement triggered a return of both muscles to the resting activation level appropriate to the new position.

Braitenberg and Onesto proposed a network for converting space into time by providing that the position of an input would determine the time of the output. This "open loop" system revealed a preset signal in the nervous system which resulted in specific position adjustments. However, the question remained as to how the brain "knows" the joint angle in a cognitive sense. Experiments revealed that the brain processed the angle as a quality and encoded it in terms of which neurons fired. Specific receptors fire at higher frequencies at particular angles. Thus, for each joint angle, there were "assigned" receptors responsible for each specific angle. When the higher center transmitted a command to flex the elbow, for example, feedback information returned from the joint receptors as ascending signals to the higher center for processing and adjusting to the continuous tuning signals to the flexors to contract.

The extent of the kinesthetic receptors' tuning curves suggests that fine discriminations of angle must be made. This is accomplished by establishing "angle detectors" throughout the process of selective inhibitory and excitatory convergence to produce a pattern of activity in a population of inputs which corresponds to the desired action. Activity by any single angle would activate several broadly tuned receptors to varying degrees and not activate others at all. The relative activities could define the angle as finely as desired simply by pulling more of the broadly tuned detectors.

When a body segment is given a signal to move, the agonists muscles are fired to start the contraction. At the same time, the antagonists muscles also receive also a signal of lesser magnitude. Because the activity by the antagonists is smaller, they were believed to be relaxed or inactive. However, it has been found by many investigators that the antagonist functions as a joint stabilizer and, therefore, movement is a product of the net moment around a particular joint. A "burst" of agonist EMG stimulates the required muscular activity to accelerate the segment and this burst of EMG activity is followed by a silent period. The antagonistic activity has reported. This classic illustration of EMG patterning in a ballistic movement utilizing agonist and antagonist muscles is shown in Figure 31.

It is apparent that the brain defines its perceptual qualities so that the nature of the quality is encoded in active cells and the rate of their firing determines the "goodness of fit" between the stimulus and the desired action. The encoding process at the receptors achieves higher intensities with increased firing rates.

A situation to address concerns the rate of firing when the resultant movement is opposite to feedback signal loop such as in a maximal eccentric contraction. For example, the contractile direction may be flexion but the external load exceeds the muscular force in that direction. In this situation, the different joint receptor outputs do not match the required or the anticipated signal. The neural and muscular mechanisms and the various effects have not as yet been investigated.

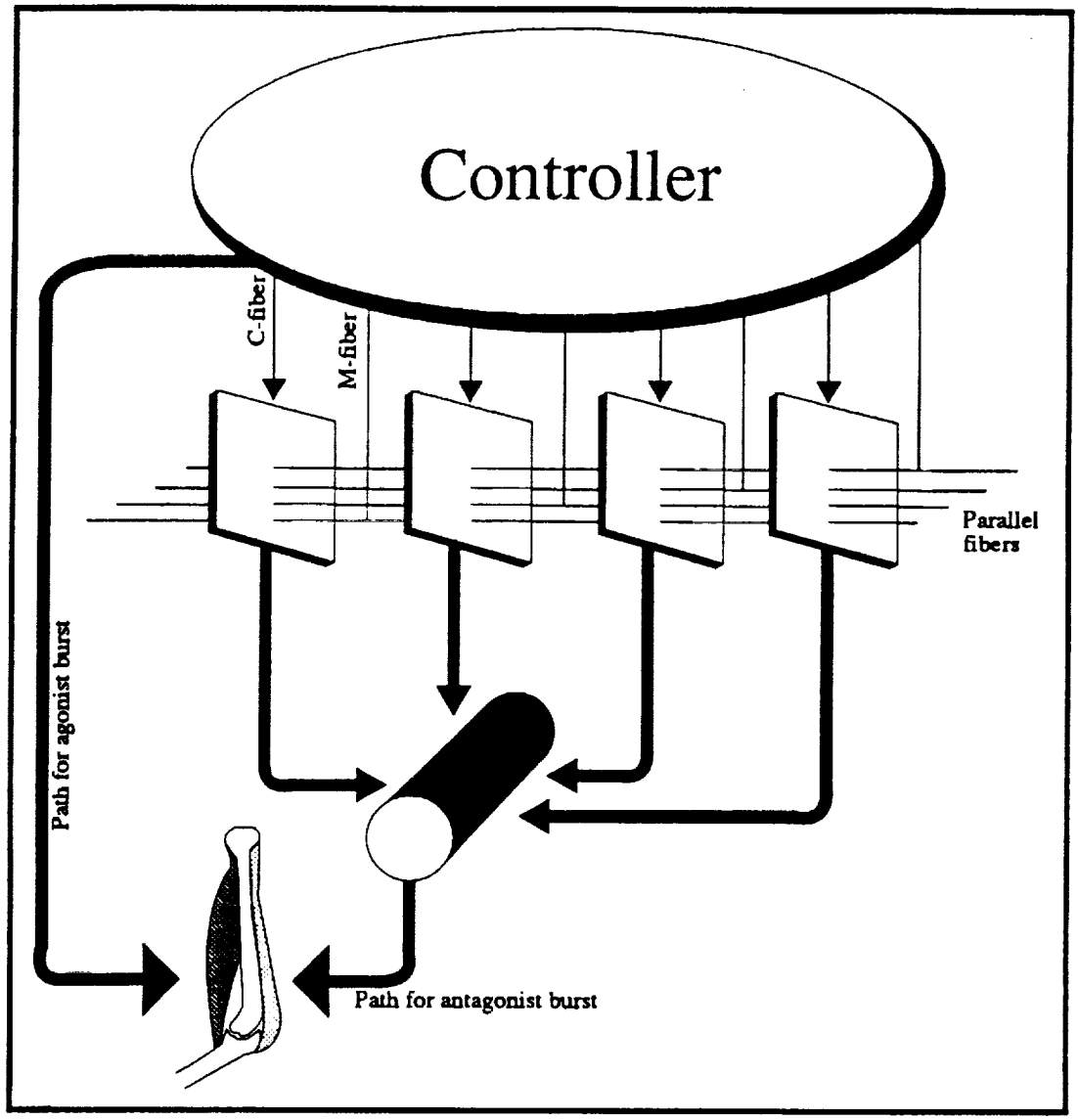

Controller
C-fiber
M-fiber
Parallel fibers
Path for agonist burst
Path for antagonist burst

Figure 30

Types of Muscular Contractions:

When muscles contract, the limbs may appear to move in different ways. One type of motion is a static contraction, known as an isometric type of contraction. Another type of contraction is a shortening or dynamic contraction which is called an isotonic contraction. Dynamic contractions are accompanied by muscle shortening and by limb movement.

Dynamic contractions can exhibit two types of motion. One activity is a concentric contraction in which the joint angle between the two bones becomes smaller as the muscular tension is developed. The other action is an eccentric contractions in which, as the muscles contract, the joint angle between the bones increases.

These two types of dynamic contractions exhibit various characteristics and produce a level of confusion. It was thought that a person could generate more force with an eccentric contraction then with a concentric one. Many studies have focused on this point, however, to date, there has been only confusion and disagreement with the results. To adequately investigate this phenomenon, the following areas must receive consideration:

1. Physiological
2. Neuromuscular
3. Biomechanical
4. Skill and Technique

The physiological characteristics of muscular contraction have been previously discussed. The interactions among the remaining factors, the biomechanics, neuromuscular control, and the level of preprogrammed skill and technique are essential if one is to determine the source of the causes and the discrepancies in force measurements.

Komi (307), a notable researcher in the area of muscular function, concluded his research findings with the following statement: "It would be an overstatement to conclude that the available scientific information guarantees full understanding of the factors involved in production of force and speed by human skeletal muscle." Such sentiment was probably aroused by the fact that, in skilled activities, the muscle does not behave in a manner that is logically explained. For example, Komi observed that the greatest EMG activity occurred before the force requirement. The level of force was greater in the eccentric phase as compared to the concentric phase. The concentric contractions appeared to be less efficient with minimal energy production as compared to the eccentric contraction.

It appear to the author of this manuscript that some of the findings resulted from not amalgamating the total complex of requirements needed to generate force. For example, sufficient time must be allowed for the flow of calcium ions to interact with the cross bridges. Time is also necessary for neuromuscular integration and for the entire system to generate the required potentiations. All the biochemical processes require some activation time. Thus, muscular contractile activation must start before an impact occurs such as in running or walking. These actions must be preprogrammed to allow a sophisticated timing mechanism to initiate a chain reaction of events.

The level of performance is dependent on the integrative programming of the nervous system, the status of the biochemical state of the skeletal muscle, and the biomechanics of the muscles, joints, levers, and external physics affecting the body.

When a limb moves, a sophisticated chain of events occurs before, during, and after the movement is completed. The fineness of control depends upon the number of motor nerve units per muscle fiber (207). The more neurons, the finer the ability to maneuver, as in the case of the

muscles that operate the eye. When there are fewer motor nerve units involved, the action becomes less fine or precise. The individual muscle fibers that make up a muscle contract and relax in an elaborate synchronization. Consider the operations required of the human eye and arm. Eye muscles must operate with great speed and precision in quickly orienting the eyeball close or distant focusing as well as tracking. At the same time, the eye muscle does not have to contend with such external demands as lifting weight. The fine control needed in eye movement calls for a high innervation ratio (the ratio of the number of neurons with axons terminating on the outer membrane of muscle cells to the number of cells in the muscle). For eye muscle, the innervation ratio is about one to three, which means that the axon terminals of a single motor neuron release their chemical transmitter to no more than three individual muscle cells.

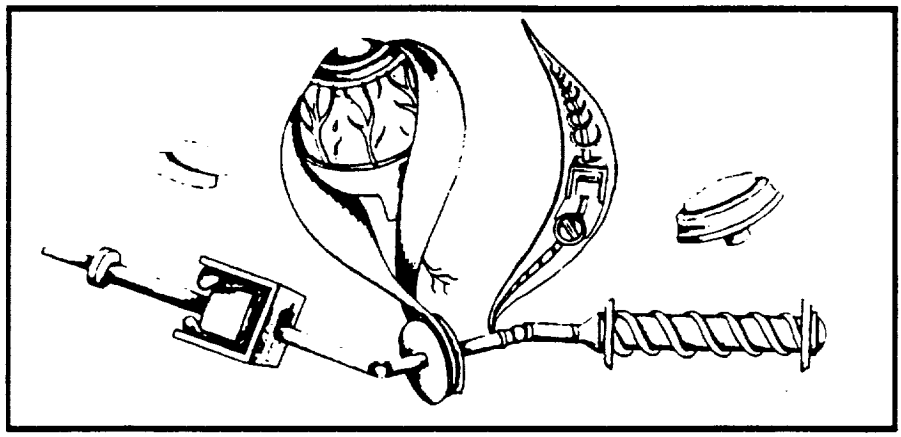


Figure 31

In contrast to this high innervation ratio, the axon terminals of a single motor neuron that innervates a limb muscle, such as the biceps of the arm, may deliver a chemical transmitter to hundreds of muscle fibers. The muscle may, therefore, have a low ratio of one to many hundreds. As a result, the output of the motor unit in limb muscle is correspondingly coarse.

One of the most elementary movements for humans is walking (113,139, 245). It seems as if all mammals and other land animals are born with the ability to walk and run. In experiments with babies between four and six weeks of age, it was found that these infants start to walk when supported, raised to a standing position, and placed on a treadmill. It seems that a baby at this early age possesses and can utilize the built-in walking mechanism with which it was endowed by its genes. The nerve cells controlling the mechanism come from the spinal cord.

Consider one of the most common activities in modern civilization, handwriting, and the execution of one's own signature. Whenever John Smith signs his name, it always looks the same (or enough to be so recognizable) and different from what any other person can write, even if trying to sign the name of John Smith. Even if Mr. Smith uses chalk and signs his name on a blackboard, the signature appears the same though he used different muscles than those employed when writing on paper. The individuality remains.

In this complex handwriting movement, there is a preprogrammed control mechanism. Optimum performance depends on the control efficiency. It does not matter how strong the muscles are or the efficiency of the metabolism. The control of these processes is the most important factor. Most people believe the brain is primarily used for thinking, yet research shows it to be first and foremost a control system. Although, very complex, the task of handwrit-

ing incorporates all of the required components: neurological, biomechanical, and biochemical. To begin a handwriting analysis, it should be remembered that there are many ways to move the joints in order to accomplish the task. However, for any given function there seems to be a best way to code the information and receive the quickest response from the system. Therefore, the way in which we activate the muscles and the bones in order to perform any volitional task will greatly affect the amount of computation that is required to assure smooth operation of joints and easy interaction of the organism with the world.

BIOMECHANICS

Biomechanics is an integration of the two disciplines of biology, "bio", and physics, "mechanics". It recognizes that all bodies on earth, animate and inanimate, are affected in the same way by gravity and provides a better understanding of performance. In other words, a bridge, a car, a baseball player, or a horse must all adhere to the laws of mechanics. The additional factors which must be included to more accurately assess motion for the biological entities include such things as bone capacity, neuromuscular coordination, and physiological attributes. From the understanding of each component will come greater appreciation of the integrated result that is called biomechanics.

Da Vinci (118) once observed that while drops of rain are in fact independent of one another, they appear to the human eye as "continuous threads descending" from the clouds, and that therein lies the truth of how the eye "preserves the impression of moving things which it sees." Therein also lies the visual distortion that allows us to see motion pictures. Because of the properties of the human eye and the visual system, a series of separate images on film becomes a smoothly flowing image when projected onto a screen at a certain speed - a movie. Movement of the human body is, also, a series of separate, individual actions. They begin with minute electrochemical processes infinitely swifter and more complicated than any set of film images traveling at 24 frames, or 1.5 feet, per second. Our muscles are thin strands of fibers which, when inactive, have all the strength of jelly. But they contract or relax because of these electrochemical reactions. The result is movement of the body with a fluidity that defies even the sharpest eyes to distinguish the separate actions. For instance, the simplest of human movements, such as crooking a finger or raising an eyebrow, involves a complex of neuromuscular happenings that cannot be duplicated by artificial means. In fact, the best robot still moves in jerks and stops when compared to the subtle, flowing pace of a human (207).

Man fathoms the nature of things by tracking their motion. All motion follows mechanical principles. Like machines he makes, man is a set of levers whose movements copy the geometry of classical mechanics. These levers are powered by muscles, whose actions can be as simple as their characters are complex. Each of the more than six hundred muscles is abundantly supplied with nerves which link the muscles to the brain and spinal cord and which often follow labyrinthian circuits, humming with signals, to control the ebb and flow of muscular energy. Many muscles must work harmoniously together in order to perform even the simplest task (115).

It is biomechanics that seeks to understand how these neuromuscular events occur and to analyze a series which the naked eye only dimly sees and the mind often fails to comprehend. Biomechanics is a science that depends upon the known facts of biology, physics and, to a lesser extent, chemistry. It can be called the study of the structure and function of biological systems by the methods of mechanics. To reap the rich rewards of the more satisfying, fulfilling life which this science can offer, a through understanding of both the "bio" and the "mechanics" of biomechanics must be gained.

The "bio" part of Biomechanics has been covered at length in the beginning portions of this paper. Therefore, attention will be directed at the second half of the word, the "mechanics".

THE MECHANICS

The second half of the science of biomechanics concerns itself with the physical laws that can be applied to the human body, the "mechanical" consideration. Unlike the "bio" portion, which is affected by biological structure, anatomy, physiology, genetics, nutrition, activities and environment, the mechanics portion is governed by mechanical laws which are universal tenets throughout the Earth.

The Italian scientist, Galileo Galilei (1564-1642), found experimentally that different balls of different weights roll down an inclined plane at the same rate (43) If the plane were tipped more sharply, the balls would roll more rapidly, but all the balls would increase their rate of movement similarly; in the end, all would cover the same distance in the same time. This means that freely falling bodies fall through equal distances in equal times, regardless of their weight. In other words, a heavy body will not fall more rapidly than a light body. The importance of the falling masses experiment lies in understanding acceleration.

Galileo determined that the distance traversed by a body rolling down an inclined plane grows greater and greater in successive equal time intervals. This means that the rate of speed is changing. Acceleration is precisely the change in rate of speed or, in more correct terms, velocity. In the falling masses example, each second the velocity of the mass increased by the same amount for this particular time interval. Such a change in velocity with time is called acceleration. On Earth, the acceleration of free falling bodies is a constant of 32 feet per second per second.

It was absolutely necessary to understand acceleration in order for the English scientist, Sir Isaac Newton (1642-1727), to formulate the laws of motion (409). As stated by the Newtonian law, acceleration produced by a particular force acting on a body is directly proportional to the magnitude of the force and inversely proportional to the mass of the body. In other words, the greater the acceleration, the greater the force and, if the mass is greater for the same force, the acceleration will be reduced. From a practical point of view, the greater the mass or the weight of an object, the greater the force necessary to accelerate the object. Also, to produce a greater acceleration with a given mass, a greater force is required.

The importance of discussing acceleration and forces lies in the fact that movement has to start with force. It is impossible to begin movement without applying force, whether it is external force, such as gravity, or an internal force, such as muscular. For example, the force applied to a hockey puck on the ice will create an acceleration and sets the puck moving faster and faster as long as the force is applied. The length of time that force is applied on the puck is important. The muscular forces needed to swing the stick and skate down the ice are also forces requiring consideration.

In measuring external and internal forces acting on the body, the mechanical models can be classified as static or dynamic. The static models are simpler since no forces due to movement occur.

For example, consider a static model with a person standing motionless holding an iron weight in his hand, like in the shot-put event, and assumed that the shot has a 10-kg mass. In this case, the forces acting on the shot is vertical. The gravitational attraction of the earth creates the weight of the mass, and its magnitude is proportional to the mass by the gravitational factor which is the gravitational acceleration in meters per second per second. In other words, the weight of the shot is equal to the mass of the shot times the gravitational acceleration.

Weight equal to
Mass times Gravitational Acceleration

One should bear in mind that forces are vector quantities and, therefore, have a magnitude, a direction, a line of action, and a point of application. In Figure 32, the magnitude of the force is 10 kg times 9.8067 meter per second per second, or 98 Newtons. Newtons represents the force rather than the mass which is represented by the weight of the load which is 10 kg. Since the gravitational acceleration is 9.8067 meter per second per second, then the product of these two numbers yield 98 Newtons. Since it is a static analysis without motion, the direction of the force is vertically down and the line of action is vertical. The point of application of the gravitational force is at the center of the mass.

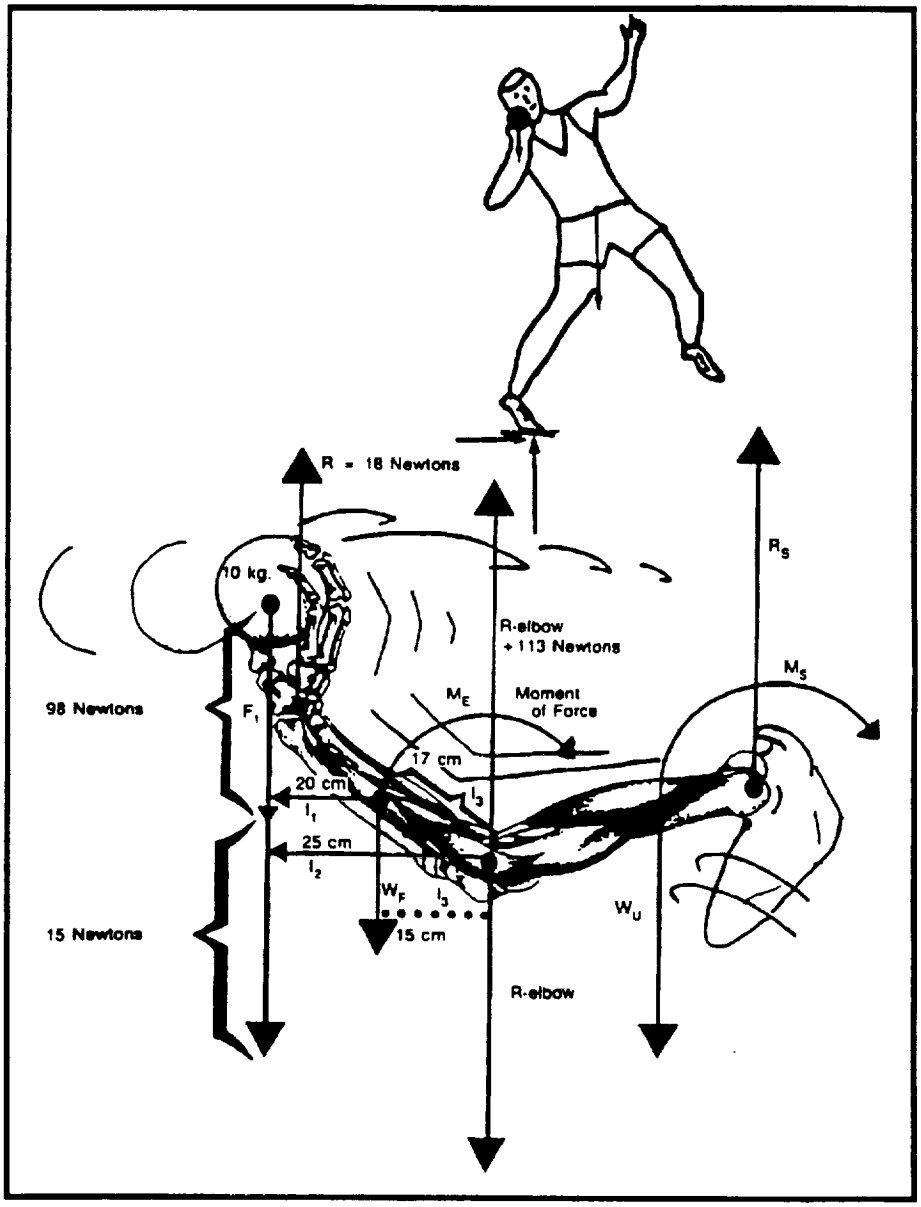


Figure 32

In Figure 32, the load is stationary since the person holding the shotput is motionless. Therefore, the downward force has to be cancelled by an opposite and equal upward force. The force, in this situation, is provided by the hand supporting the mass or shot. The upward force cancels the downward force and the load is said to be in equilibrium. The opposite force is called a reactive force and it is designated in Figure 1a as "R". Since the forces act along the same line of action, no rotation occurs at the wrist, elbow, or shoulder joints.

This example can be described using the Engineering analytic concept which is called a Free-Body-Diagram. In a free body diagram, a graphical representation is given with the magnitude of forces. In this example, the system is in equilibrium so the summation of forces must be equal to zero.

Summation of forces equal to 0

That means:

The force in the down direction is equal to -98 Newtons. The reason for the negative sign is to indicate the downward direction.

The force in the upward direction is equal to positive 98 Newtons since it pushes upward against the gravitational pull. Therefore:

-98 Newtons plus 98 Newtons equal to 0

From this simple equation, it can be determined that the hand has to apply 98 Newton of force in order to keep the shot from falling down.

A more complicated problem is to calculate the static force at the elbow. For this calculation, the length, weight, and the center of mass location of the forearm must be known. Calculations using this information will yield the forces at the elbow joint which resist, or are exerted against, the combined weight forces of the load, the forearm, and the hand. If the forearm and hand segment are assumed to weigh 15 Newtons, then the combined effect at the elbow joint will be:

-98 Newtons -15 Newtons plus the counter force at the elbow equal to zero

(-98N -15N +R elbow = 0)

That means that:

the resultant force R at the elbow is equal to 98N + 15N or 113 Newton in the positive direction or upward to order to cancel the force of the shot, the forearm, and hand which are in the negative or downward direction.

In the above example, the forearm and the hand were considered as one segment for simplicity. A more through analysis would divide this segment into two segments consisting of the hand segment with its center of mass location and the forearm with its center of mass location.

Where, then, does the force at the elbow joint come from to counteract the weight of the shot with the forearm and hand segment. The answer is that this reactive force is generated by the muscles around the elbow joint with some additional force provided by the ligaments and connective tissue around the elbow joint.

In mechanics, one must consider not only the force(s) but also the product of force and time which is called an impulse. For a given mass, a given impulse will result in a particular velocity. The heavier the object, the greater the impulse needed to achieve the same velocity. From that it is obvious that velocity and mass are related to each other and, in fact, the product of mass and velocity is referred to in physics as momentum. This law of momentum is most important in contact sports where different masses collide at different velocities. This law is what allows a smaller football player with greater velocity to block a heavier football player with lesser velocity.

In a hockey game, the puck which possesses a certain mass and is speeding across the ice at a given velocity has momentum equal to its mass times its velocity. If along its travels another hockey puck of the same mass moving at the same speed, but in the opposite direction, collides with it, they will then come to an instant stop. One momentum was canceled by the other. This principle of conservation of momentum is an important component in the game of billiards where solid balls hit others at different velocities.

In running and jumping activities, the forward force applied to the body depends on the force the foot applied to the ground and the amount of time that this force was applied. In other words, the product of the two or the impulse will determine the energy applied to the performance. The combination of greater velocity, magnitude of force, and time of contact with the ground are the essential factors which determine the speed of an athlete along the horizontal or vertical direction.

Consideration of these linear movements, where objects displace all their dimensions at the same rate are important in biomechanical considerations. However, the anatomy of the human body dictates that the parts move primarily in a rotational fashion. A good example for understanding rotational movement is the wheel in which the center remains stationary while the other parts move around it.

Understanding rotational movement necessitates appreciation of torque. A force that gives rise to rotational movement is called a torque. The amount of torque or, as it is also called, moment, depends on the force and its distance from the center of the rotational object. The product of force and distance is equal to torque.

The examples considered thus far have assumed translational motion such as when the forces around the elbow joint react to forces across a line of direction in a particular plane. However, these forces do not stop the segment tendency to rotate around the joint, thus, creating or counteracting a rotational motion. The force on the hand and forearm joint create a moment around the elbow joint forcing it to rotate about its joint. Moments of force are very significant around the body joints since the human structure consists of long bones and, the further the force applied to these long segments, the greater is the moment around the joints of the body.

The Moment of Force around a joint is equal to the product of a force and the perpendicular distance from its line of action to the point of rotation. In Figure 1a, the moment around the elbow joint is equal to the perpendicular distance of the elbow joint center to the line of force which represents the vector of force from the center of the mass at the hand designated as F1 times the combined static force calculated before as 113 Newtons. That means:

Distance from Elbow to Line of force (L2) times 113 Newtons or 25 Centimeters times 113 Newtons equal to 2825 Newton-Centimeters

Moments, like forces, are vectors and, therefore, direction about a point of rotation as well as magnitude must be considered.

In the previous example, we considered the static equilibrium of forces which are called the first condition of equilibrium. When considering the equations for moments of force, the second condition of equilibrium is assumed which states that the sum of the moments around a joint in static analysis is equal to zero. This means that:

The perpendicular distance from the elbow joint to the center of mass of the forearm (L3) times the weight of the forearm which is the moment due to the forearm and hand segment combine must be added to the moment due to the load at that hand. This moment is equal to the perpendicular distance from the elbow joint to the line of force from the load designated as L2 times the weight of the load. These combine moments must be equal to the counter moment by the elbow flexors muscles designated as Me. In equation form it looks like:

$$(L3 \times -Wf) + (L2 \times -WL) + (Me) = 0$$

From this equation, it is possible to calculate that static moment about the elbow joint as:

$$Me = (L3 \times Wf) + (L2 \times WL)$$

This means that the Moment around the elbow is equal to the combined moments due to the load and the weight of the forearm with the hand. To describe this example numerically, the weight of the shot is assumed to be 10 kg. which is approximately 98 Newtons of downward force. The force due to the weight of the forearm and hand are assumed to be 15 Newtons. The distances of the center of mass of the forearm which is the distance from the elbow joint center is assumed to be 17 centimeters. The perpendicular distances to the lines of force were calculated as follows:

The perpendicular distance from the elbow joint center to the line of force from the center of mass of the forearm is calculated by the product of the cosine of the elbow angle times 17 Centimeters which yield approximately 15 centimeters and the perpendicular distance from the elbow joint center to the line of force of the mass at the hand was calculated from the total forearm and hand length times the cosine of the elbow angle and was found to be approximately 25 centimeters. Using this information, it is possible to calculate the moment around the elbow joint as:

$$-(15cm \times 15N) + -(25cm \times 98N) + Me = 0 \text{ therefore:}$$

$$Me = 2675 \text{ Newton-Centimeters.}$$

This is the value that the muscles around the elbow joint must exert to keep the arm from rotating due to gravitational force.

The importance of the concepts presented thus far is that there are two types of forces acting on the joint in a static analysis. The translational force affects the tendency to move in the same line of action of the force. The second effect is the moment or the torque that tends to rotate the segments about the supporting joints. To combat the first effect, the joint has to counteract the translational force with tensile forces in ligaments and muscles to hold the joint together as well as the shearing and compressive forces which also act on the joint contact surfaces This is why a business man can develop "tennis elbow" by holding briefcase in his hand since the tensile force at the joint may cause micro tears in the ligaments and connective tissues at the elbow joint.

On the other hand, the rotational moment is a function of the strength of the muscle to move or rotate the joint. When a person plays tennis, the muscles move the racket as a result of muscular contractions. At the same time, ligaments and tendons react to the shearing forces. These shearing forces, which occur during the movement and the impact, can create forces which could result in tissue injury and, therefore, create problems in and around the joint.

Thus far, consideration has been limited to analysis of forces around one segment consisting of the hand and forearm. The problem becomes more complicated when additional segments are considered. It is possible to treat each segment separately and then add the effects of the previous segments to the present segment. In this way, a kinetic chain from one segment to the attached segment is created. The analysis begins at the point of application of the external load and proceeds in sequence, solving the equilibrium equations for each body segment, until reaching the segment that supports the body, which is usually the feet.

To analyze the forces on the upper-arm, all of the external forces and moments operating on the arm must be considered. In this case, the weight of the upper arm and the resultant elbow force and moment caused by the weight of the forearm and hand link must be considered. In this way, it is possible to calculate the static equilibrium equations at the shoulder which result in a reactive force Rs and a moment Ms for the person analyzed.

The principle of momentum that applied to linear movement also applies to rotational movement, and the conservation of angular momentum is one of the key principles in athletic performance. Angular momentum is a function of the mass and the rotational acceleration as well as the square of the distance from the center of rotation. In rotational motion, therefore, the quantity of mass times the square of the distance from the center of rotation is analogous to mass alone in linear motion.

These basic physical concepts are essential to the understanding of human movement and the principles of physical performance. The fact that it is harder to hold a weight further from the center portion of the body is related to torque. The fact that a ballet dancer on her toes and a figure skater on ice can generate high rotational speed is because both performers are affected by the conservation of angular momentum.

The product of a turning body's moment of inertia and its angular velocity is called its angular momentum. According to the law of conservation of angular momentum, a turning body isolated from external forces will have a constant angular momentum; that is to say, the product of moment of inertia and angular velocity about the axis of rotation is constant. If, for example, a man is standing on a revolving turntable without friction, he may increase his resistance to turning threefold by stretching his arms sideways (Figure 33). By the same token, if a man rotating on the same frictionless turntable pulls his hands toward his body, the rotational velocity will increase threefold because the moment of inertia has decreased.

A figure skater makes use of these laws on ice. At first, as rapid a spin as possible is produced with arms extended. The arms are then brought down, and the body spins on the point of one skate with remarkable velocity. The same principle allow throwers, such as discus throwers or shotputters, to generate higher speed across the circle of throwing.

These various laws of motion are critical when applied to the muscles and bones of the body. Muscles and bones constitute a form of basic tool, the lever. To understand the application of motion, therefore, we must first understand the use of the lever.

The combination of muscle and bone forms a lever system which is one of the most basic mechanical systems for performing work. A lever is a machine by which force applied at one point does work at another. Each joint in the human body is the fulcrum of a particular lever.

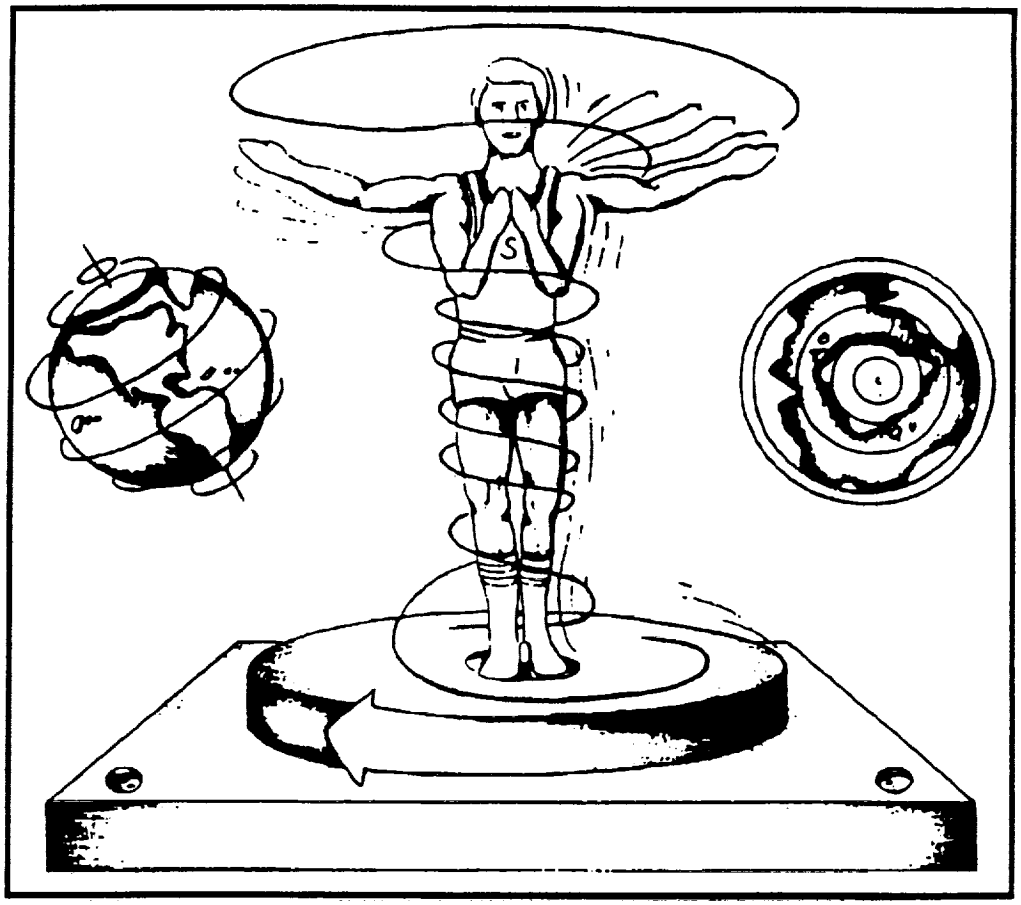


Figure 33

There are different types of lever systems and the human body uses all types. The forces on the different levers are applied by the muscular system. It must be remembered that muscles do all of their work by contraction or shortening. Activity occurs because the muscles contract or pull the various bones, or levers.

Muscles, like many other components of our anatomy, operate in pairs known as "agonists" and "antagonist". Bending the arm at the elbow, for example, requires the biceps to contract while the extensors, the triceps, relax. In order to stop this bending motion, the biceps must stop contracting at the appropriate time and the triceps begin contracting in order to slow and subsequently stop the action. The neural coordination of this system was previously discussed in the "bio" section. It is the bones which constitute the levers employed in the mechanical properties of motion.

Physics has divided levers into three classes (348) In the first of these, the force is applied at one end of the lever and the resistance to be overcome or the work to be done lies at the other end. The fulcrum, or pivotal point, lies between. Two children bouncing up and down on a playground teeter totter exemplify a class one lever.

In the second class, the force is applied at one end but the resistance is located above the pivot or fulcrum. A crow bar underneath a tree stump is an example of class two leverage.

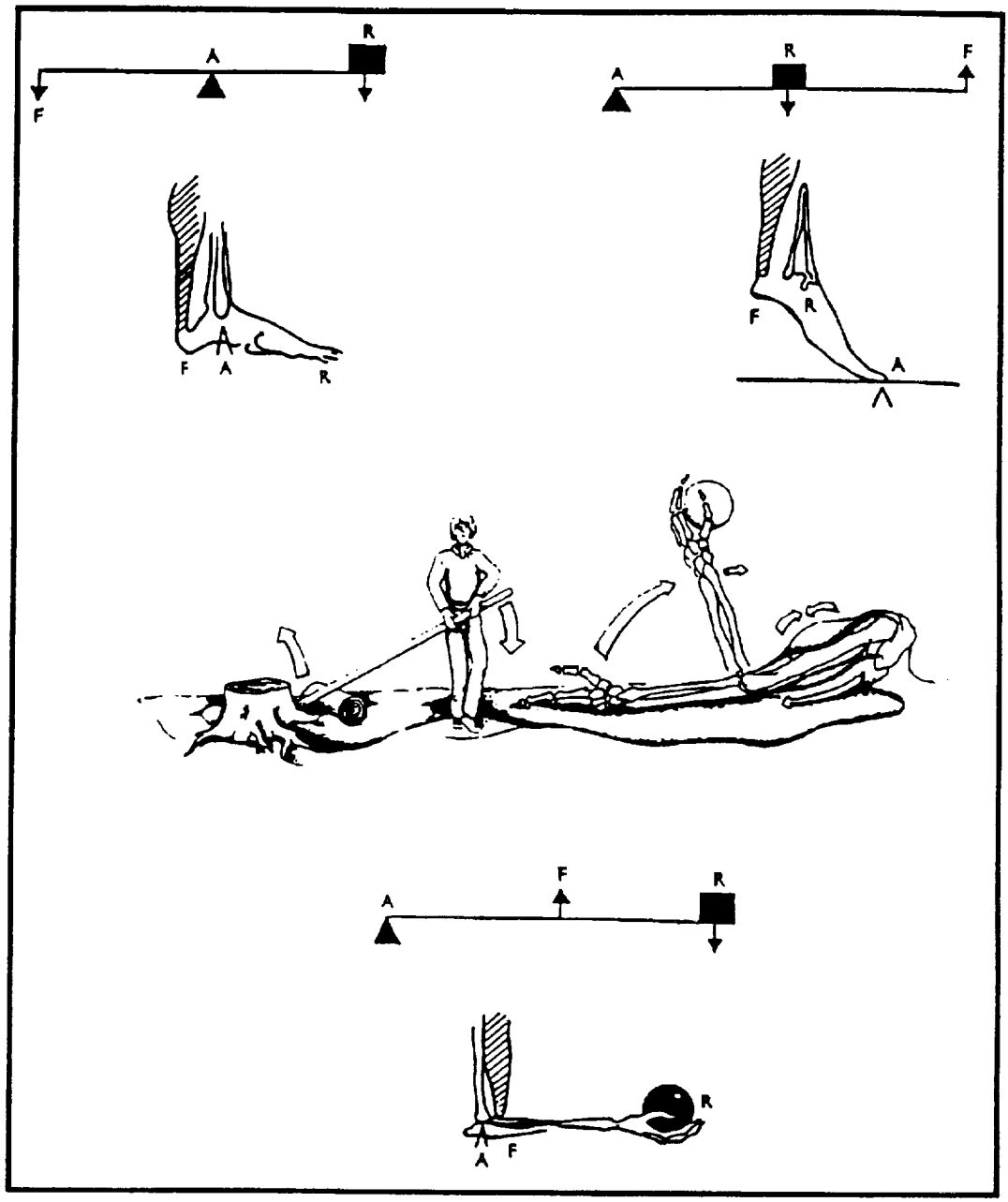


Figure 34

The third type has the force exerted between the pivot point and the resistance, and this is the common lever system within the human body. When you lift a weight with your arm, the pivot is the elbow joint, the force is exerted between your elbow and your hand by your biceps muscle, and the weight in your hand is the resistance (Figure 34).

A lever may either increase the amount of work that can be done with a given input of force or it may cause the work to be done at a faster rate than the application of the force. Archimedes once bragged that he could move the world if given a long enough lever (43). To perform the feat, Archimedes would have used a second class lever with his fulcrum very close to the Earth while he himself would have dangled on his elongated lever somewhere deep in outer space.

The concept to be mastered is that a mechanical advantage is gained with use of a lever, the amount of force is multiplied many times over to produce greater output at the other end. This is true if the fulcrum is situated closer to where the force is to be applied than to where the force is executed. If the reverse is true, and the fulcrum is nearer to the point where the force is executed than to the point of force application (a third class lever), then the result is a mechanical disadvantage.

For humans, the fulcrum usually falls closer to the point where the force is executed or initiated than to where it is applied, or where the work is done. The biceps attaches to the radius bone quite close to the elbow. Therefore, to lift a one pound weight with your hand, your biceps operates at a mechanical disadvantage of approximately seven to one. That is to lift one pound requires a force of seven pounds.

However, the same principle that governs mechanical advantage and disadvantage has its compensations. The hand at the end of the lever of the arm will move seven times faster than the point where the biceps attaches to the radius. It is easily seen that only a slight movement upward of the forearm near the elbow, causes the hand to move several inches during the same time interval. Obviously, the hand traveled considerably faster than the elbow.

For most human joints, the length of the lever does not produce a mechanical advantage. None the less, there is still more potential for production of speed if the human levers are longer. The knees are particularly vulnerable to injury not only because of their limited range, but because, in some instances, the whole body becomes one long lever applying its force at the knees. A situation to illustrate this point is on the ski slopes. If the boot does not release when the shear forces are excessive, the long skis and the body can create an exaggerated and destructive lever ultimately resulting in injury.

While Archimedes required a very long lever to move a very large ball (the Earth), Hannibal needed a shorter lever to throw large balls from his catapult. Individuals concerned with much smaller spheres, such as golf and baseball, can still apply more force to the object or their intentions with a longer lever. Golfers should play with the longest clubs they can comfortably manage, as should batters in baseball.

However, the longer the lever, the less fine the control and the greater the requirement for muscular force. Thus, the putter, the club most concerned with a deft, accurate stroke, is the shortest in the bag. Many good golfers further reduce the potential margin for error by shortening up on the putter and holding it lower on the shaft.

In addition to some of the important components of mechanics, it is also essential to understand the other laws of motion. Newton neatly encapsulated these laws of motion into three principles. The first law states that an object remains at rest until some force acts upon it. If the object is already in motion, it continues at a constant speed unless some outside force is brought to bear. In other words, until the levers of foot and leg are applied to a soccer ball, it remains at rest on the ground. Once having been kicked, however, the ball will continue to roll at a constant rate until it is acted upon by the outside force of friction, from ground or air, or contact from another system of levers in the form of an opposing soccer player.

When two or more forces act upon an object, the subsequent force is known by physicists as the resultant force. If Player A kicks a soccer ball due north simultaneously with Player B's kick of the same ball due west, the ball will travel northwest along a path that will be determined by which athlete delivered the most force. The route taken by the ball and its velocity is the resultant force supplied by Players A and B.

Newton declared that the main task of mechanics was to learn about forces from observed motions. The physics behind movement is related to the law of momentum, which is part of Newton's second law. Momentum is a concept that consists of velocity multiplied by the mass of the moving object. Momentum, in terms of physics, is distinguished from force, which is defined as mass multiplied by acceleration, or the rate of change in velocity. Alteration of momentum, or a change in motion, declared Newton, is governed by the force brought to bear upon the object, which then follows the straight line in which the force acts (Figure 35).

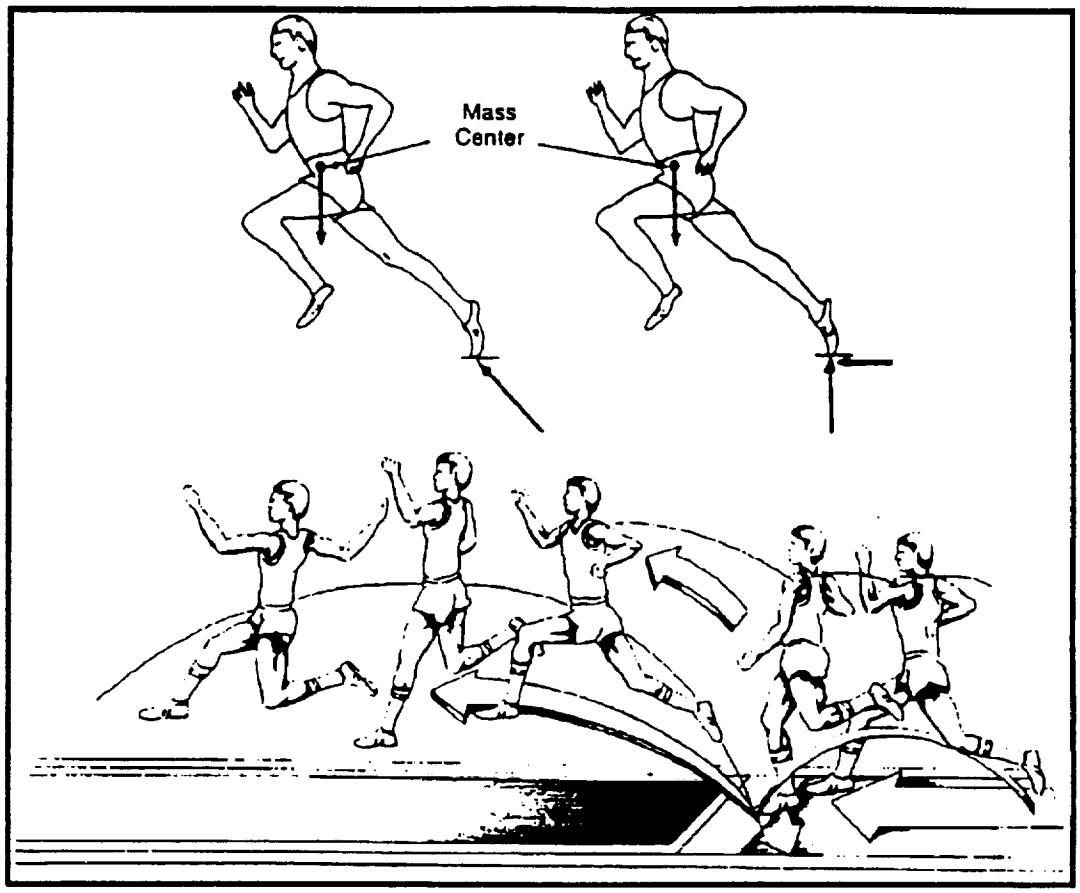


Figure 35

Consider the problem of a man leaping over a small puddle. He runs towards the puddle creating many forces including horizontal ones. As he nears the water, the central nervous system, the movement coordinator, orders the muscles the feet and legs to contract generating a vertical force for the jump. The height depends upon the ability to generate enough force to temporarily exceed gravitional force. If the man weighs 150 pounds and produces only 140 pounds of vertical force, he will have wet feet. Once airborne, he can no longer add any force to the jump. The force of momentum, the velocity at takeoff multiplied by the man's weight, must be enough to overcome the demands of gravity in order to jump over, not in, the puddle. The vertical force combines with the horizontal force for the leap. That is to say, the body does not travel in either a purely vertical direction or continue to move in a direction solely horizontal. Rather the path becomes a combination of horizontal and vertical forces, a direction that physicists call a resultant.

The third law of Newton was that for every force that acted upon an object, the object itself exerted an equal and opposite amount of force. When you kick a ball with your bare foot, the painful sensation in your big toe confirms Sir Isaac's third law. The recoil of a fired rifle is also an example of equal and opposite reactions. A car striking a bridge abutment at 60 miles per hour is demolished while one that nudges the wall at 5 miles per hour remains intact. The wreck is an example of a much greater degree of equal and opposite force.

Another principle affecting your body is derived partly from theories of Einstein. An old bedroom farce joke uses the punch line, "Everybody's got to be someplace," and Albert Einstein said that energy can neither be created nor destroyed. In other words, energy or a visible manifestation of it in the form of force also always must "be someplace."

This means that when one generates a force by swiveling his back, and then suddenly tries to stop the movement of the back, the force developed in the trunk of the body does not simply disappear. It must go someplace. The secret to efficient use of a body for work or sport, for fitness or injury prevention, depends to a great extent on where these forces go or how well they are exploited.

In the human body, the bones, or levers, move in a rotational manner. These angular movements create linear movement for the total body. The same laws that govern linear motion also govern angular motion. The only difference is that the length of the lever also plays an important part. If the body begins a rotation, it will continue to turn on its axis until the movement is altered by either a change in body position or the application of some other force. Consider the previous example of the ice skater and imagine that this skater begins a spin with the arms abducted or outstretched, building up angular momentum by the maneuver. If this athlete suddenly drops the arms to the side, the velocity of the spin will increase because the momentum that was initially generated is constantly maintained around the axis. The change in the arms' distance from the center of the body transfers this angular momentum to the body itself.

Angular momentum can be redistributed throughout the body. When a long jumper leaves the ground, he or she is propelled forward and angular momentum is developed. Unless that momentum can be redirected, however, the jumper will land flat on his or her face. Such a disaster is prevented by transferring some of the angular momentum to the arms, which explains the wild flailing exhibited by long jumpers.

Angular momentum can be expressed in terms of two other important parameters of rotation: angular velocity and moment of inertia. Angular velocity is represented by the body's rotational speed and direction. For example, if a diver performs a forward double somersault in one second, the magnitude of his average angular velocity is two revolutions per second.

The moment of inertia of a body about an axis is the body's tendency to resist changes in angular velocity about that axis. It is obvious that massive and extended bodies have a larger moment of inertia than do lighter and smaller ones. In fact, the contribution of each particle or segment in a body to the total moment of inertia about an axis is equal to the mass of the segment times the square of its distance from the axis of rotation. For example, a typical diver with this body straight and his arms at his sides has a moment of inertia of 14 kilograms times meters squared about his somersaulting axis, but a moment of inertia of only one kilogram times meters squared about his twisting axis.

Angular momentum is the product between the angular velocity and the moment of inertia about a specific axis. In the case of the diver, that will be the sum of angular velocities around the two axis and the moment of inertia around these axis.

The analogy between angular momentum (moment of inertia times angular velocity) and linear momentum (mass times linear velocity) is not perfect. The reason is that the body can change its body segment lengths while performing angular movement, such as the diver straightening from a tuck position into a straight body. That changes the moment of inertia about his somersaulting axis. In linear momentum, this does not occur.

Any mechanical phenomenon created by the human body must be initiated by the energy produced by the skeletal muscles. This energy allows the movement of a body segment in rotational fashion to create movement. The faster the movement the more powerful is the motion.

There are some limitations to any analogy between power created by muscles and that produced by an engine. An engine will be rated as having a certain amount of horsepower, meaning that it will produce a specific amount of work each second that it is in operation. It will lift or push a number of pounds a certain distance. We can measure sustained human efforts the same way. A woman pedaling a bicycle can be rated for the horsepower she produces in transporting her weight and that of the bike over a certain distance within a certain amount of time. This type of power rating is valid for a rhythmic and sustained amount of force. But it does not serve as a useful description for impulsive actions.

Consider for a moment what happens when one fires a rifle with the barrel pointed straight up into the sky. Most of the power to speed the bullet on its way is produced before the bullet is actually moving. Because of the confines of the rifle barrel, there may be some power added as the explosive charge pushes toward the muzzle behind the bullet. There is no way to accurately compute a value for the amount of horsepower generated. The force is not constant and gravity and friction constantly alter the velocity.

The same is true for an impulsive action by a human, such as in a high jump. It may be said that one jumper will actually generate more force than another jumper, but still not leap as high, because of a failure to coordinate all of the force into as large a single impulse. The key measurement, therefore, is not how much "horsepower" was developed as the athlete sprinted to the launching point and then hurled himself into the air, but only the amount of ultimate ballistic muscle force that was generated for the actual liftoff.

Another factor necessary to understand movement is appreciation of the classification of mechanical energy, which is defined as the capacity to perform work. Kinetic energy is that which the body possesses by virtue of its motion. During the motion of the windup of a pitch, the arms of the baseball pitcher contain kinetic energy. Potential energy consists of that which owes its existence to the position of the body. A diver at the edge of the platform possesses a certain amount of potential energy through the imminent application of gravity.

Sometimes the types of energy can be totally separate aspects of a movement. For example, the instant that a person begins to rise from a trampoline, the kinetic energy begins to diminish while the potential energy increases. At the highest point of the maneuver, the instant of zero velocity when the gymnast is neither ascending nor descending, the kinetic energy is zero and the potential energy is at its maximum. During the descent, the potential energy in effect is transformed to kinetic energy. At the deepest penetration of the trampoline bed, the strain energy reaches its maximum.

These mechanical, physical precepts are useful in the analysis of human movement because they permit examination of the forms of energy, forces, directions, or speeds that comprise an activity. Quantification can also assist in determining the most efficient use of effort, that is, the optimum way to do something.

THE BIOMECHANICS

Quantification of an action, regardless of whether for evaluative purposes or to attempt optimization, can be accomplished through biomechanical analysis. Biomechanical assessments normally begin with the quantification of the kinematic portion. This is usually accomplished by utilizing high speed cinematography or videography which allows careful scrutiny of even the fastest movements of humans or other animals, such as dogs, cats, horses, etc. The films or videos are traced and the resulting data stored in a computer which calculates the results by applying the principles of physics and mechanical engineering. Tables and graphs can then be generated which give a precise profile of what actually occurred during the execution of the movement. The researcher then carefully examines this output in order to understanding the motion and, in the case of an athlete for example, to determine which patterns are most important in distinguishing championship from average performances.

Biomechanics is a science still in its adolescence with many discoveries yet to be made. Hand analysis of high speed films is a slow and tedious process, and it is only recently that the computer has been harnessed to make the process more efficient. Development of this technology in the United States has meant that many complex analyses can be executed in a relatively short time.

In the past, athletic achievement depended mainly on the individual's talent, although skill was often enhanced or ruined by existing facilities, equipment and, undeniably, coaches. Athletes with superior genetic compositions who successfully interacted with the available facilities dominated the list of world records. Continual improvement of equipment and techniques has complemented raw talent.

However, with the advent of new measurement tools and knowledge in the field of sport science, athletic achievement has attained a new dimension. The athletic teams of the United States, which for years had dominated amateur sports, were no longer the leaders. Countries such as those of Eastern Europe and Cuba, which have relatively small populations, have achieved a spectacular level of success in athletic events. Current evidence suggests this trend may continue through the remainder of the 1980s and 1990s. Such domination stems from the application of science to the realm of athletic performance.

Modern coaches can use biomechanical means to optimize the human body in each event. Since the human body obeys the same physical laws as all other earthly objects, the laws of motion govern its performance. In order to throw, run or jump, physical laws must be obeyed. It is impossible to throw the shot 20 meters if the shot velocity and angle of release do not attain certain values. These values do not differ for different athletes, since for each particular shot velocity there is one specific optimal angle.

For the jumper to leap 8 meters, it is necessary to produce certain forces on the ground in order to propel the body with a specific reaction force at a particular angle. This force is unique, and it is impossible to cover the same distance with only a fraction of the force since gravitational pull acts uniformly regardless of the jumper.

The concept to be reemphasized here is that all bodies, athletes, implements or machines, are affected by and must adhere to the laws of motion. The science of biomechanics specifically deals with motion of the body and the resultant forces. A number of scientists have long recognized these facts of force and motion and their relationship to humans. But the kind of equipment that could measure and analyze the motion and forces involved was lacking and, thus, impeded further research.

The field of biomechanics can be divided to Kinematics, which describes the motion of the body and its segments without reference to the forces that cause the motion, and Kinetics, which describes the forces which caused the movement. The kinematic parameters includes linear and angular displacement, velocity, and acceleration. The kinetic parameters include the external and internal forces acting upon the body segments.

In order to measure the kinematic and kinetic parameters, it is necessary to make a few assumptions. If it were possible to disassemble and reassemble the human body like other machines, then it would simplify and make the measurements more accuracy. However, this is obviously impossible. Therefore, some of the measurements are derived from cadaver and additional assumptions are made upon the linkages of the human body. This is not different from any other field involved with living bodies. In the field of physiology, for example, many assumptions are made on the ability of the body to consume oxygen. In the conversion of energy measurements from external measurements to internal measurements, many reasonable assumptions are made. In determining the composition of different muscles and their classification into fast and slow twitch muscles, many assumptions are made concerning the chemical staining methods and the counting methods. Of course all statistical methods, which are the bases for most behavioral research, utilize assumptions about the normality of the populations and the distributions of the data samples.

Therefore, to view the human as a machine made of links is an oversimplification but it is possible to create a humanoid model as a representation of the body made of rigid segments. In this way, it is possible to facilitate quantitative analysis of the movement. The links which represent the body's limbs are a series of interconnected rigid segments which demonstrate independent motion.

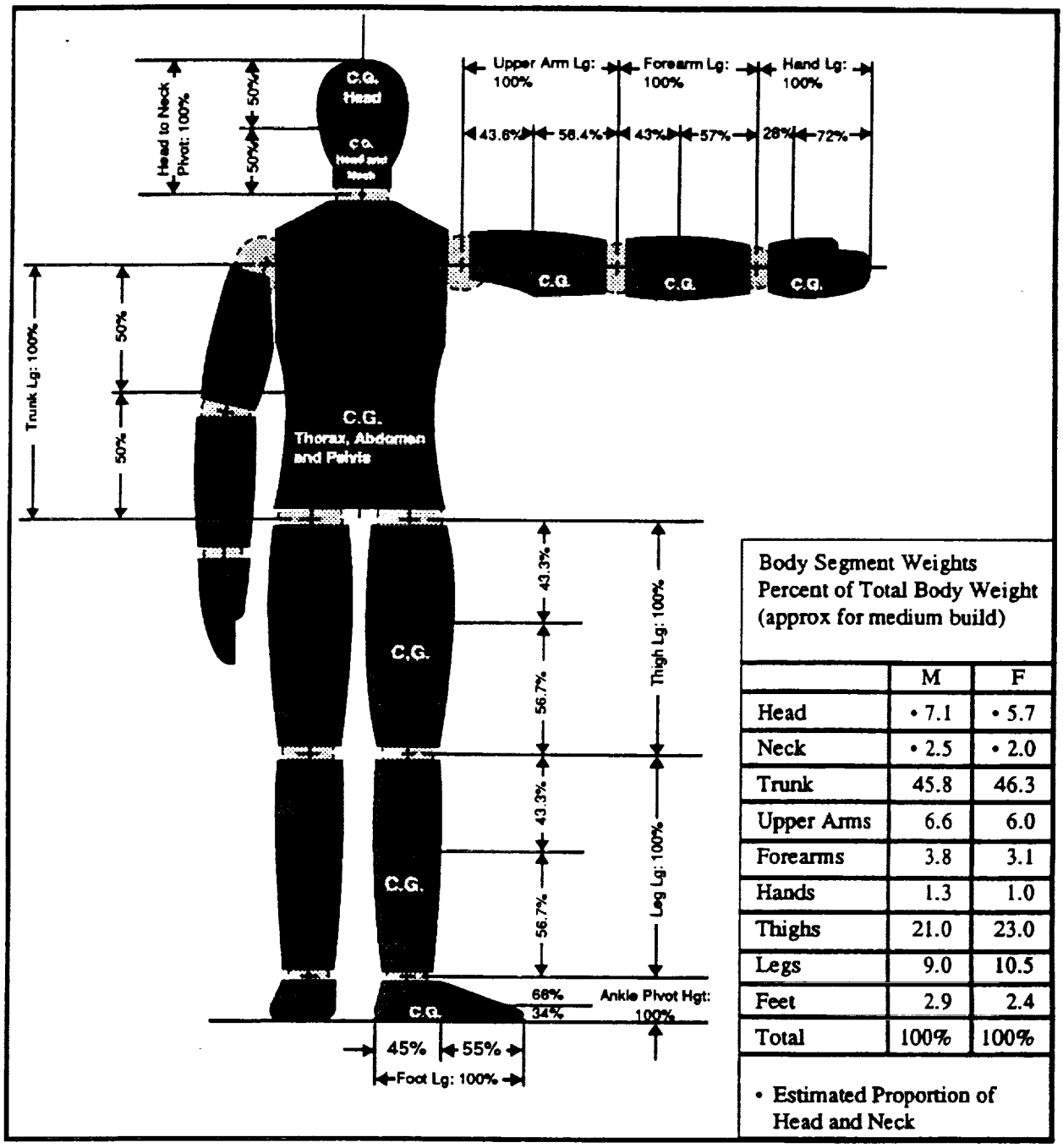
To more accurately facilitate the above mentioned system, it is important to realize that there are different body types. It is clear that body differences in shape occur between ages, sexes, and within individuals. The field which deals with different body shapes is the field of Anthropometry.

Anthropometry is the science that deals with the measure of size, weight, and proportions of the human body (96). Anthropometric data is fundamental to biomechanics because some of the assumptions made in the calculation of movement parameters made based on its data. In performing a biomechanical analysis of any movement, the human body is considered to be a system of mechanical links with each link of known physical size and shape given by anthropometric measurement.

After adapting the anthropometric measurements to the different body segments, the next assumption is that this link system is connected at identifiable joints. Since the body landmarks or the body segments are covered by muscle, fat and skin tissues, it is sometimes difficult to identify joints such as the hips and shoulders. However, with the aid of statistical and numerical methods it is possible to filter some of the errors.

This field of anthropometry has made tremendous contributions by dissecting cadavers and measuring the location of joint centers. Some of the pioneers are Braune and Fischer (81), Dempster (126), and Snyder, Chaffin, and Schulz (419). From the cinematographical data, it is possible also to use tracing methods which connect the intersection of the long axis line of the segments during movement. Some of the landmarks used in the field of biomechanics are illustrated in Figure 36.

In addition to landmarks required for biomechanical tracing, it is important to predict the segment mass and location of the center of the mass. Body-segment mass and volume are related to the density of the segment. From the field of Anthropometry, these measurements were



Body Segment Weights
Percent of Total Body Weight
(approx for medium build)

	M	F
Head	• 7.1	• 5.7
Neck	• 2.5	• 2.0
Trunk	45.8	46.3
Upper Arms	6.6	6.0
Forearms	3.8	3.1
Hands	1.3	1.0
Thighs	21.0	23.0
Legs	9.0	10.5
Feet	2.9	2.4
Total	100%	100%

• Estimated Proportion of Head and Neck

Figure 36

determined by immersing cadaver body segments in water and measuring the volume of the water displaced. From the equation of:

AVERAGE DENSITY IN MASS PER UNIT VOLUME EQUAL TO THE MASS OF THE BODY SEGMENT DEVIATE BY THE VOLUME OF THE WATER DISPLACED.

$$(D = M/V)$$

The values for the different body segments are available in various biomechanical text books.

In addition to the segment weight and its volume, the distribution of the mass within the segment is necessary in order to compute the kinetic information. From this distribution of the mass, it is possible to calculate the location where the gravitational effect on the segment occurs. In other words, where the segment can be considered to be in its gravitational balance so that if one were to hang the segment at this point, it would be in balance regardless of the orientation of the segment in space. This point is known as the mass center or the center of gravity of the segment.

There are a few methods to calculate the segment center of gravity. One method involves the distribution of forces by allowing a person to be suspended between two force platforms and calculating the change in vertical forces by moving various segment to different angles. Another method involves submersion of the segment in water. Regardless of the method used, there exists a sufficient data base from various investigations which have calculated the center of mass for different segments for different populations.

Knowledge of the center of gravity location of each segment and its weight and length allow the calculation of the static force and torque at each body joint for a given posture. However, in athletic performances and in normal human life, we are seldom concerned with static posture. More realistic are the dynamic performances where the forces due to the motion in addition to the forces due to gravity act upon the body.

For these dynamic analysis of the human body, it is necessary to know the inertial property of the segment. This property is referred to as the Moment of Inertia. The formula which describes the moment of inertia considers the following:

MOMENT OF INERTIA OF THE SEGMENT IS EQUAL TO
THE MASS OF THE SEGMENT TIMES THE
SQUARE OF THE PERPENDICULAR DISTANCE FROM A GIVEN AXIS.

$$I = M X R X R$$

There are different methods for calculating the Moment of Inertia of the individual body segments and these methods are described in various Biomechanics text books.

It is important also to calculate the location at the body segment where the moment of INERTIA effect the segment. This point is called the Radius of Gyration.

THE RADIUS OF GYRATION IS EQUAL TO:
THE SQUARE ROOT OF THEMOMENT OF INERTIA DIVIDED BYTHE MASS OF THE SEGMENT

According to Drillis and Contini (134), the radius of gyration is "the distance from the axis of rotation to an assumed point where the concentrated total mass of the body would have the same moment of inertia as it does in its original distributed state". This information is necessary to quantify the dynamics of human motion in conjunction with other parameters.

Another important contributor to biomechanical analysis was the National Aeronautics and Space Agency which have also made detailed measurements of human body composition, the relative mass for segments such as arms, legs and hands, given the overall height and weight of the individual. The specifications may not be exactly accurate for each individual, but they are close enough to humans in general for the purposes of even the most exacting scientists.

It should be remembered that all of these estimated parameters are used in calculating the dynamic forces rather than merely describing movement in qualitative terms. These parameters are available in numerous sources and the normal analysis of human movement does not require the individual calculation of all of these parameters. The information resides in tables and charts which can be accessed at will. The normal analysis of movement relies on this information in the same way that physiologists depend on the tables presenting characteristics of different gasses and their coefficients at various temperatures and pressures.

Without a computer to store information, retrieve it, and perform the myriad computations, such calculations can place the scientist in an impossible position. But before a computer can perform its job, whether it is to guide a robot, print a check or retrieve a space vehicle, it needs a program, that is, a sequence of instructions which tell it how and what to do.

Programming a computer involves hundreds and, at times, thousands of hours of work. But once the instructions have been fed into the computer, it will automatically execute the hundreds of steps without any need to restock the computer with further instructions, and the execution will only take a few seconds. The beauty of a computer is that it can play the great game of "what if?". In sports, one could ask, "What if I hold the shot down here and then whirl in this fashion?" The computer will calculate the distance the shot will travel, applying the amount of force developed in previous analysis. Through use of the computer, then, biomechanics can write equations and construct models which will result in optimal performances.

Another critical element is the camera which can be either a high speed movie or sufficiently fast video system. It provides sequences of the body in motion. Knowing the speed with which film or video tape travels through the camera allows calculation of the velocity and acceleration of body segments using its joints as points of reference. For example, if the shutter speed on the camera is 200 frames per second, one can identify the location of the right knee at the start of a sprint, and then compare the position of the right knee in frame 20 of the film, thereby learning how far the right knee has moved in one tenth of a second. The data can be further utilized to determine velocity, acceleration and, with some additional information, even the forces involved. The forces can be calculated by measuring the length of the leg, for example, from knee to ankle, and by using the NASA specifications, determine the mass of that segment as well as determining the center of gravity. Using these values, quite reasonable estimates of the exact forces and torques around the joint center can be calculated.

Along with analyses based upon films taken during actual events, highly sensitive force plates have been developed for precise impact measurements. These allow controlled laboratory testing of forces, such as when an object like the human foot strikes the plate during a sprint or the vertical component of a monkey leaping from the plate onto a table. The plate is capable of recording three different components of forces: vertical, horizontal, sideways or lateral, as well as the moment or torque.

Any kind of athletic movement or work action which can be photographed with a high speed motion picture or video camera can be fed into the computer. Forces can be plotted for each segment of the body as the accelerations and lengths of the segments are measured. The maximum amount of force that can be generated using a particular approach in an activity can be calculated (19). For instance, it is feasible to calculate how high a jumper might go if he changed from his customary form of the roll to the flop style, assuming he was able to generate the same amount of force for the flop as he did for the roll. Analysis have shown that the flop happens to be a more efficient use of forces for athletes who do not possess extremely powerful legs.

It is important to remember that because of both gross and subtle variations in the neuromuscular system of each human, the biomechanical actions of individuals are as unique as their fingerprints. The shades of difference from one person to another are, in fact, great enough to permit the development of a foolproof method for guaranteeing a signature. A person could file his or her signature in a computer bank. The information in the computer would contain not only the shapes of the letters, but the amount of force the individual applied to every loop, line and curve. With this device, a buyer in a store need only sign the chit on a force plate or use a pen with a force sensitive transducer which transmitted the information directly to the computer. The patterns of force would be compared instantly and, if not the same, the new one would be rejected.

Similarly, detection of variations or errors in human movement has always been one of the most difficult problems facing coaches, trainers and physicians in athletic situations. If the error detection is inaccurate or non-specific, the quality of correction will be poor. Failure to recognize the causes of error stems from an inadequate understanding of the mechanisms of human motion. Impacts in sports, automobile accidents, falls, and other movements involving forces can be accurately quantified through biomechanical applications.

The designer of protective equipment for sports, such as hockey and football, must have an understanding of biomechanics since it is necessary to comprehend before the equipment is designed how the human head reacts to impact forces or how a skier's leg reacts to twisting forces. The forces produced by the human body cause a change in acceleration or speed. The change might involve the entire body, as in sprint starting, skating, or volleyball (in a vertical jump to block). It may also be a body segment or combination of segments, as with a boxer's upper arm and forearm, a golfer's arms, or a soccer player's thighs and lower legs. Through use of biomechanical analysis, it is now possible to scientifically detect errors which are beyond the visual capabilities of the human eye.

When the muscle contracts and there is a change in limb position, work is performed. When time is required to perform the physical work, then units of power measurement are employed, since the rate of work is power. In human performance, striving for excellence on the athletic field or in recreation, it is important to be able to sum the forces exerted on the various joints. This principle is called the summation of joint forces.

For example, in swinging the golf club in the drive, the amount of force exerted by the club on the golf ball depends on how much of the forces totaled by the body actually reach the club. If there is any loss of force due to bad timing, the golf club head will not move at the same velocity. Any violation of the principle of summation of joint forces can result in too small a force being exerted by the golf club.

Another important biomechanical principle is that of continuity of joint forces. This is illustrated by the fact that not only must the golfer use all body joints efficiently, but he must also time their use so that the motion begins at the larger segment (such as the thigh), and then continues and is overlapped by motion of the hip and trunk. There must be no pauses in the flow of motion from the legs to the trunk and to the club. It must be continuous. A violation of this principle not only results in too small a force, but also in bad timing and a poor "feel" on the golf club.

Because club speed is determined by the force applied and the length of time of force application, the best combination of force application should be determined — a large force in a short time or a small force for a longer time.

There is an optimal combination for each activity. The size of the force multiplied by its time of application is called "impulse" and it is actually this force/time combination which produces the golf club velocity. Therefore, the impulse in any activity should be determined to result in optimum efficiency. Although compromises in the size of force and duration of application often have to be made in sport to achieve an optimum combination, one such combination to be avoided is a small force applied for a short time.

The size of the force an athlete can produce is determined by his or her ability to comply with the principles of summation and continuity of joint forces. In the absence of measuring devices, assessing whether the force application time is as great as possible presents yet another problem. In general, if each joint has gone through a complete range of motion, one can be assured that the maximum time available has been used.

It was previously pointed out that not only must the range of motion of the joint be complete, but the joint must straighten fast and the combined joint motion be continuous. The concept of the combined effect of force and duration of application in producing speed changes is called the principle of impulse. Violation of this principle causes further errors in performance.

Direction of force application is another important principle. Not only is the direction of the application of force to the golf club vital, but, in addition, from the club head to the golf ball. In an optimal situation, the force is exactly 90 degrees to the club and the club head hits the ball exactly at its center. However, some deviation is at times necessary if the flight of the ball has to be changed in a predetermined pattern. Incorrect direction of force can be disastrous in events such as gymnastics and diving. A good technique implies that the principle of direction of force was followed.

A final principle is the summation of body segment speeds. Especially in any throwing, kicking and striking events, it is important to obtain as high a hand, foot, stick blade, racket head, or club head speed as possible at the instant of impact or release. The speed of last segment in the chain is built by adding the individual speeds of all the preceding segments with appropriate timing. If any of the segments contribute low or negative values, the resultant measured for the last segment will be less than optimum. This principle is similar to summation of joint forces and is closely related to it.

To summarize, any motion to seek optimum should obey the principles of summation of joint forces, continuity of joint forces, impulse, and the direction of joint forces. Through the use of biomechanics, all of these principles can be quantified and optimized so that better and safer results will be obtained.

EXPERIMENTAL STUDIES

To evaluate the behavior of some of the factors previously discussed, the author administered a series of experiments. The experiments were conducted with a small sample as these preliminary studies were intended as pilot projects only. Sophisticated measuring devices were utilized to quantify displacements, velocities, accelerations, forces, and EMG activities in single and multi joint movements. The equipment is described in Appendix A, B, and C.

METHODS

Subjects:

Ten subjects, 8 males and 2 females, ranging in age from 25 to 51 years took part in the study. They were healthy, active, and regularly participate in fitness exercises. Subject training programs generally consist of jogging 3 to 7 miles 3 to 5 times weekly and engaging in resistance exercises 2 to 5 times weekly.

Exercise:

To activate the elbow flexors and extensors concentrically and eccentrically, the subjects stood holding the bar of a computerized exercise device. The position used was the same position in which the Subject had performed the curl exercise with the elbow in full extention initially, then flexed to approximately 135 degrees, and then extended to the start position. The Subject was not restrained and the technique did not differ from the method used during the preceding two years of training on the equipment. Therefore, learning had previously been accounted for and would not contaminate the study.

Equipment:

The computerized exercise devise used in the present study is described in the Appendices. Its validity and reliability have been tested and presented. One of the unique features of these experiments was that a sophisticated programmable servo valve was utilized to control the speed of the mechanical member held by the hand. All the forces were measured with a pressure transducer and processed by the computer in real time. Special calibration procedures were used and described in Appendix A. Positions, velocities, and accelerations were measured and calculated simultaneously. EMG activity at the elbow flexors (biceps motor point) and at the elbow extensors (long head of the triceps) were measured simultaneously with the other parameters. Other parameters which were measured were forces for various muscular contractions, EMG activity during various motions against resistance, and various physiological parameters representing muscular contractions.

Different velocities were set for the concentric and eccentric contractions. Concentric contractions were performed by each subject as he or she tried to move the resistance bar as fast as possible while the bar motion was restricted to a specific velocity. For the eccentric contraction, an external force was applied on the mechanical bar to a level that exceeded the subject's ability to resist it. Since the bar was moving down at a specific velocity, the eccentric phase was restricted to that velocity and, therefore, could not damage the subject's arm. Each subject was encouraged to try as hard as possible to stop the bar from moving down throughout the specific range of motion. The range was set at 125 degrees for the elbow joint and 25 degrees for the axes of rotation of the machine.

Experimental Conditions:

The experimental design consisted of three basic conditions. In Condition One, the subjects performed concentric contractions in both directions, flexion and extension. In Condition Two, the subject flexed his elbow concentrically and extension was done eccentrically with

a super-maximal force when the subject could not resist the extension at a specific velocity. In Condition Three, the subject was subjected to a sub-maximal force during the eccentric extension of his elbow. In other words, the subject could resist the force of extension but could allow the elbow to extend.

RESULTS

Condition One - Concentric contractions:

In this phase, the subjects were required to exert concentric contractions for both flexion and extension at various velocities. Figure 37 illustrates the force curve for flexion and extension. The displacement curve illustrates the flexion, passive delay where the subject was pausing between actions, and then the extension phase. In the same Figure, the associated EMG signals for the flexor and extensor muscles can be observed. During the flexion phase, the biceps generated high EMG activity while the triceps produced less EMG activity. However, it is important to note that EMG activity was present in both antagonistic muscles. To determine whether this was due to an artifact or "crosstalk", EMG activity was measured in a resting position with the arm motionless on a table. Figure 37 illustrates the EMG secured in a resting condition. Comparison of the EMG activity during an exercise and during a passive, motionless situation revealed that the EMG activity was significantly higher during exercise than at rest. It should be noted that the EMG electrodes were not removed or displaced at any time.

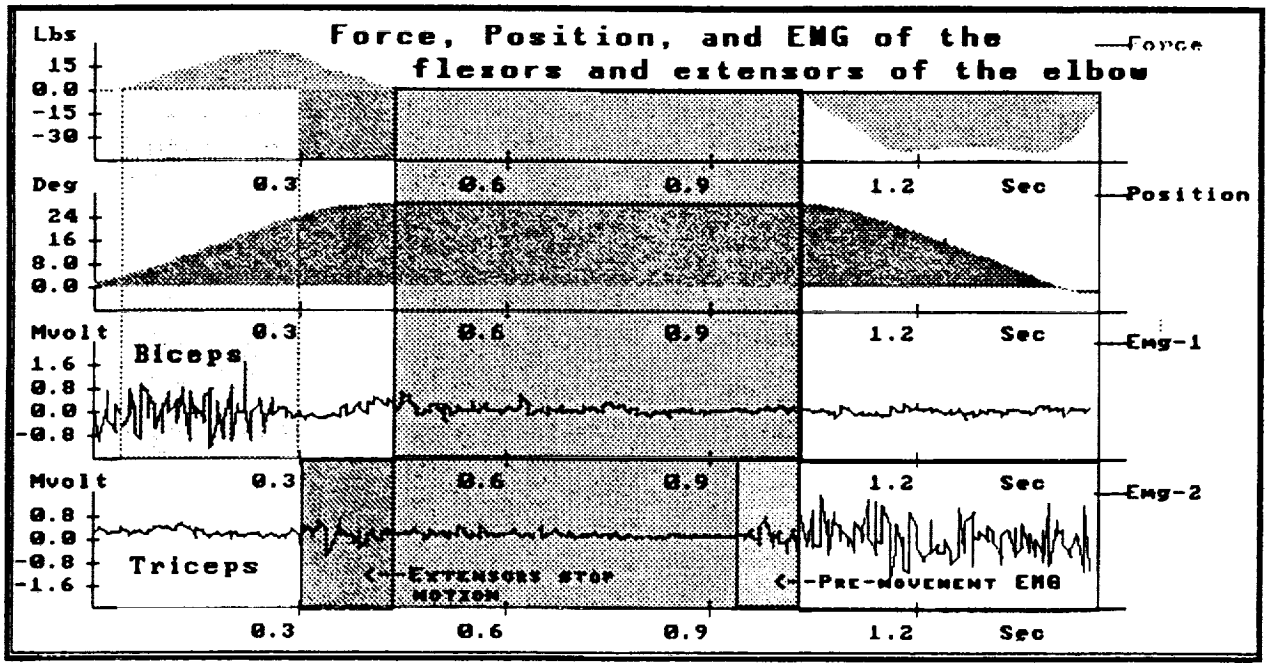


Figure 37

Figure 37 also illustrates that in the tricep's concentric phase, the muscle began firing, as indicated by the elevated EMG activity, despite the fact that the movement had not started. This phenomenon has been well documented by other investigators (70,72, 73) It can also be seen that during the concentric extension, the biceps muscle produced EMG activity as well.

In addition, it was found that during the flexion phase, the triceps showed elevated EMG activity before the end of movement. This phenomenon is well documented (223,305,306, 307,308) and illustrates the anticipatory effort needed to decelerate the segment to a resting position. Therefore, while the biceps fired the most at the beginning of the movement, the EMG activity lessened near the end of the movement. Although the triceps showed significant activity during the beginning of the movement, it demonstrated higher activity near the end of the movement.

Figure 37 illustrates the time relationship between the forces, displacements, and EMG activity. While each subject produced unique results, the general pattern displayed is like that shown in Figure 37 which represents one of a specific subject's trials.

Figure 38 illustrates typical displacement and force curves. The elbow was flexed concentrically and produced a force at maximum flexion of 25 degrees on the bar corresponding to 125 degrees at the elbow joint. Then the force dropped to zero with a delay at that maximum angle. Approximately one second later, the subject extended his elbow utilizing the elbow extensors (triceps) in a concentric phase for the extensors. This type of exercise is called a "double concentric exercise".

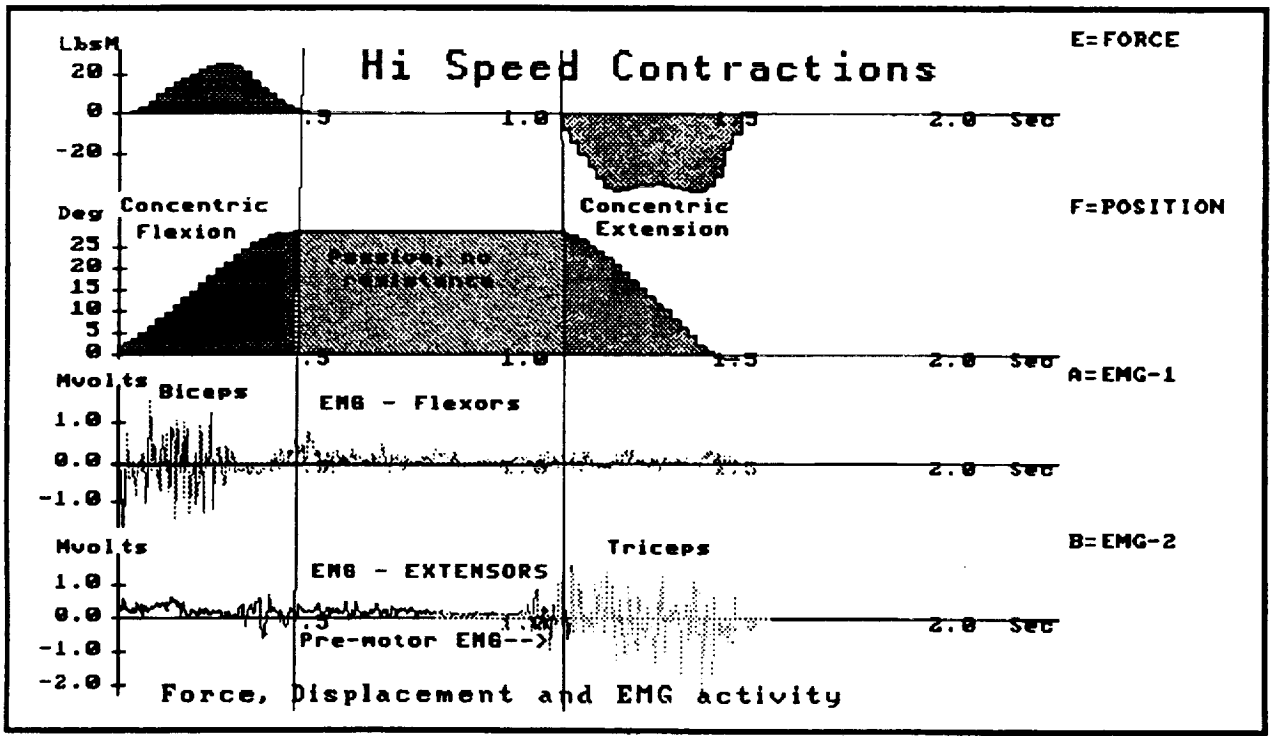


Figure 38

Figure 37 illustrates typical force, displacement and EMG curves obtained simultaneously in the double concentric exercise. It can be observed that the elbow flexors (biceps) became active before any displacement or force was produced. At that point, maximal EMG activity was observed at the flexors. At the same time, some EMG activity was recorded from the extensors indicating co-contraction. Before the end of the movement, more EMG activity was observed at the extensors apparently in anticipation of cessation of movement by the flexors. Also, the flexors reduced the level of firing indicated by a lower EMG activity before the end of the movement. When the muscle was in the delay phase before the extension portion of the exercise, the flexor force dropped to zero and both the extensor and flexor EMG activity decreased significantly. Approximately 50 milliseconds before the extension phase began, the extensor EMG increased significantly indicating potentiation or firing of the motor units of the extensors. This phenomenon has been reported in the literature and is referred to as the electro-mechanical delay. The level increased when the extensor force reached a peak. The flexor EMG activity indicated co-contraction in this phase as well.

In this phase of the study, the subjects were exposed to concentric contractions at various velocities in both flexion and extension directions. It is significant to note that for both concentric activities, the agonist and the antagonist worked in a coordinated pattern. That is, when the biceps was the prime mover, the triceps, as the antagonistic muscle, was active; when the triceps was the prime mover, the biceps, which functioned as the antagonist, was active as well.

Condition Two - Supermaximal Concentric Eccentric Contractions:

In the eccentric phase, a supermaximal external force was applied to the bar which was moved at a specific speed under servo valve control. The subject initially raised the bar (elbow flexion) in a concentric fashion. During the second part of the exercise, elbow extension, the subject tried to resist the downward movement of the bar until the position was returned to the origin.

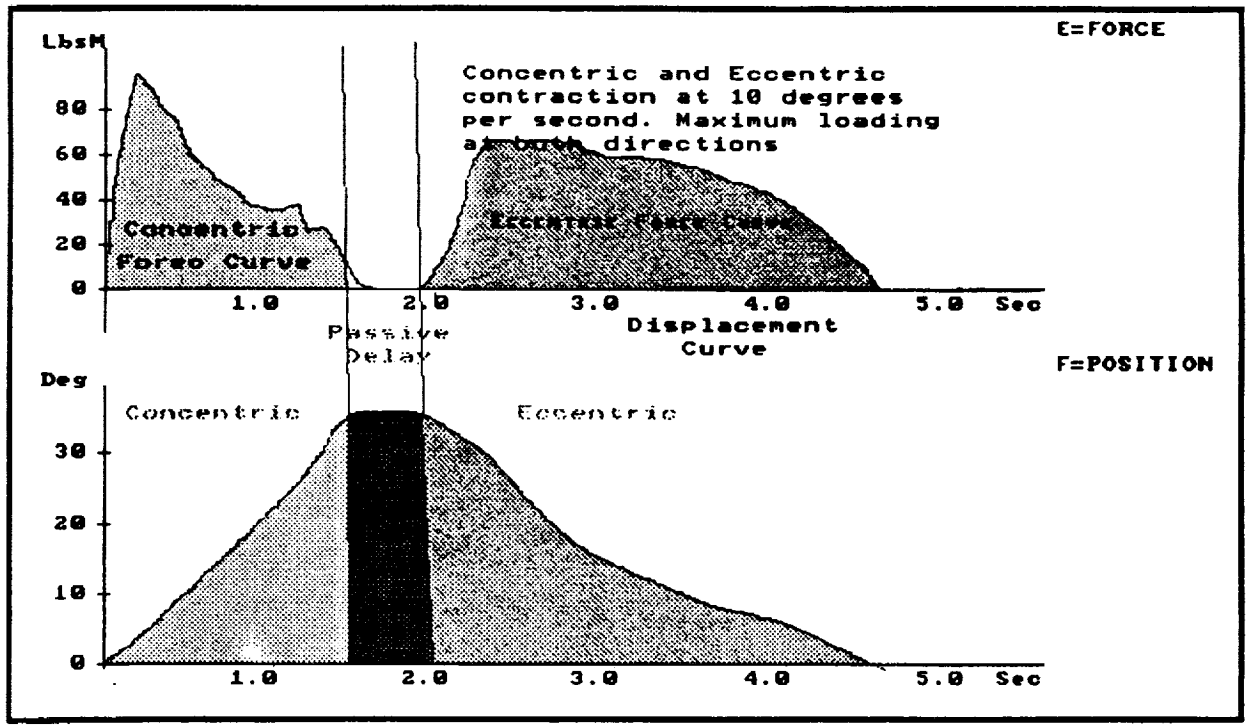


Figure 39

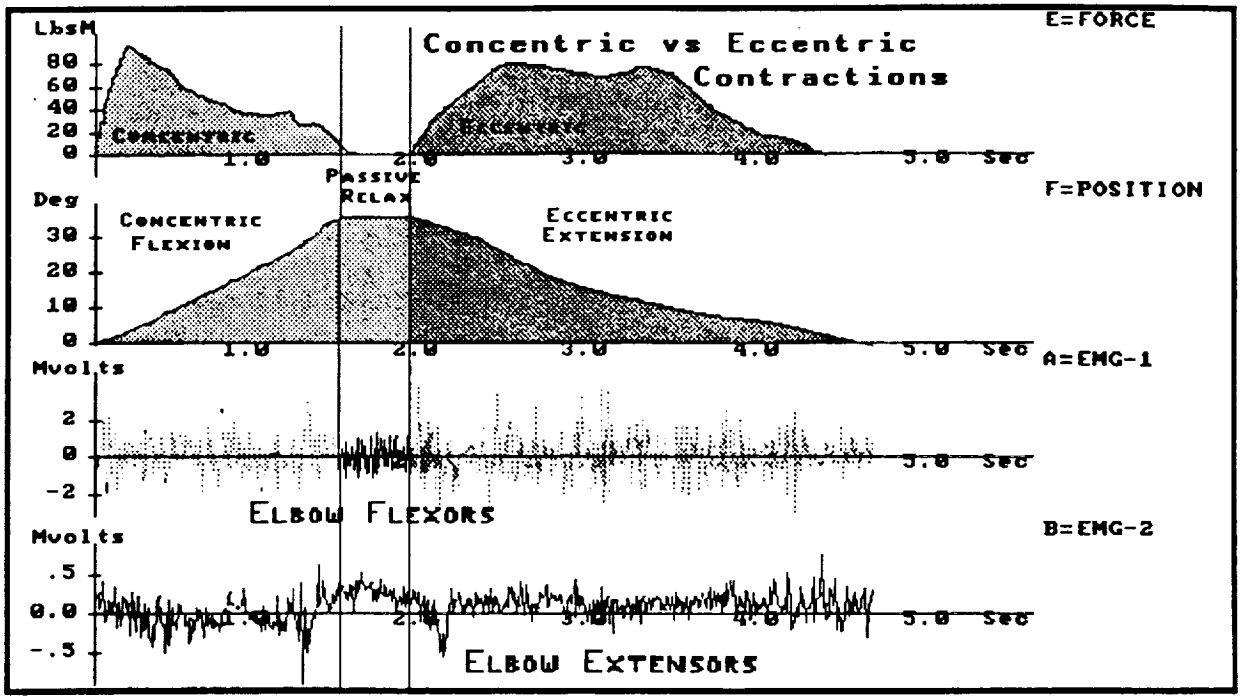


Figure 40

Figure 39 presents a typical force curve throughout the range of movement. Figure 40 shows the force and displacement curves as well as the corresponding EMG curves from the flexors and extensors of the elbow in the concentric and eccentric phases.

When a subject applies muscular force to an external device, a few factors must be considered. The first factor is the pattern of velocity. In this situation, the movement began ate "0" velocity, accelerated to the assigned velocity, and the equipment recorded the velocity of the mechanical bar. This is a very important factor since much of the previous research reported in the literature was conducted with isokinetic type equipment which did not report the instantaneous velocity throughout the range of movement but rather yielded an average quantity. This means that at an assigned velocity of 100 degrees per second beginning from a point of zero velocity, the investigators would be unable to determine the time it took to reach the designated velocity and at what point the segment returned to "0" velocity.

This limitation was overcome in the present experiment by selecting a different measurement device. Examination of Figure 41 illustrates a control curve with the velocity programmed to be 25 degrees per second in the flexion direction and 125 degrees per second in the extension phase. As can be seen, the subject needed approximately 5 degrees before reaching the selected velocity of 25 degrees per second and the subject never attained the 125 degrees per second during the extension phase.

Controlled velocity curves with data throughout the range of motion were recorded in the present set of studies. In addition to the velocity curve data, a second factor which must be considered is the acceleration pattern. In other words, consideration must be given to the amount of acceleration needed to achieve the required speed. When acceleration occurs, there are inertial forces affecting the movement. Even if the subject does not reach the assigned velocity, force is created by the muscle and is related directly to the level of acceleration. In

Figure 41, although the subject did not reached the velocity assigned during the extension phase, a force curve was calculated. It can be seen in the flexion phase that the velocity was reached and, therefore, the force level was measured to approximately 50 pounds. In the downward direction, due to acceleration of the bar, a force of approximately 25 pounds was achieved during the acceleration phase and then dropped to 0 during the deceleration phase.

The ability to report displacements, velocities, and accelerations throughout the range of movement is essential for the correlation of test results. For instance, two different subjects exercising under the same condition may yield different force curves results due to the pattern of

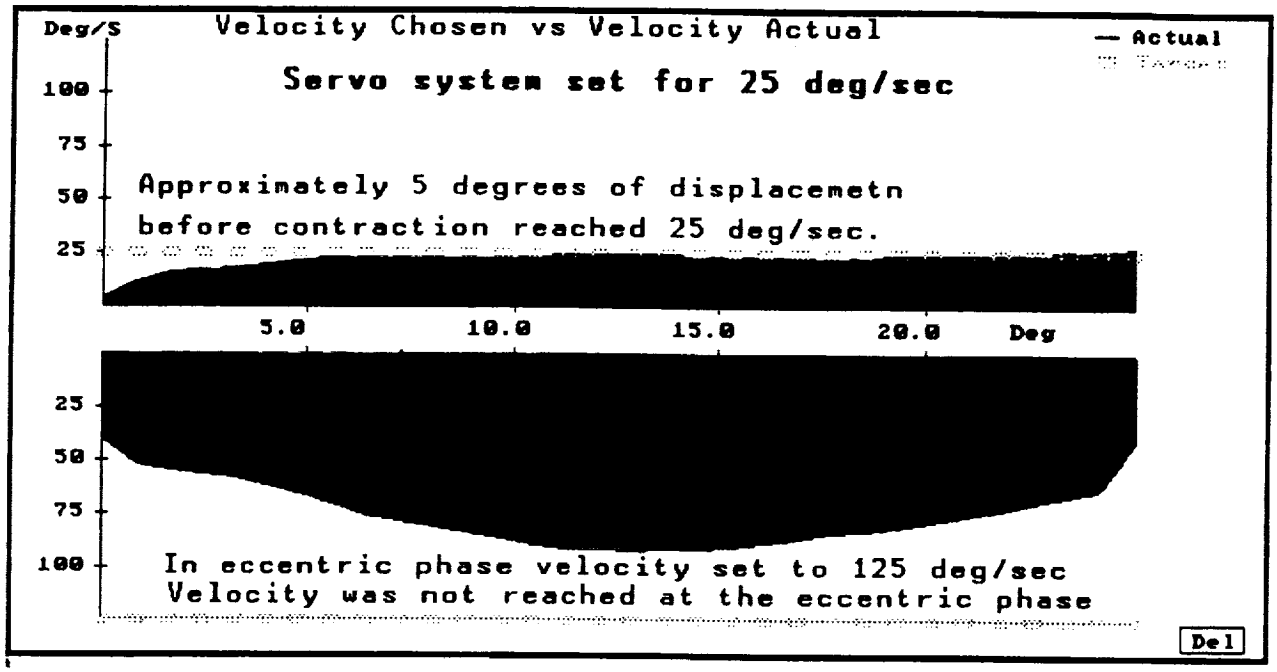


Figure 41

their movement. Figure 41 shows different force curves resulting from different levels of acceleration with the same assigned condition of 25 degrees per second in the flexion and 125 degrees per second in the extension phases. Obviously, EMG activity will vary significantly depending upon these conditions. Figure 42 illustrates that in a high speed exercise, it takes time to reach the assigned velocity as it cannot be attained instantaneously.

Condition Three - Submaximal Concentric Eccentric Contractions:

In this procedure, the subjects flexed the elbow concentrically to maximum flexion, paused approximately one second at the end of the range, and then the machine applied a submaximal force to extend the subject's arm so that the elbow extended. The subject tried to resist this extension against the active force of the machine. The machine mechanism moved the bar at a given velocity at a very high force, thus, preventing the subject from stopping the bar's movement. Since the velocity was controlled at a given speed, the subject was able to sustain the movement without injury. As in the previous study, the machines mechanism used was providing limited force to prevent injury. In the present study, the force applied was to exceed the ability of the subject to resist it by at least 500 percent. The only safeguard against injury was the fact that the bar moved at manageable velocities.

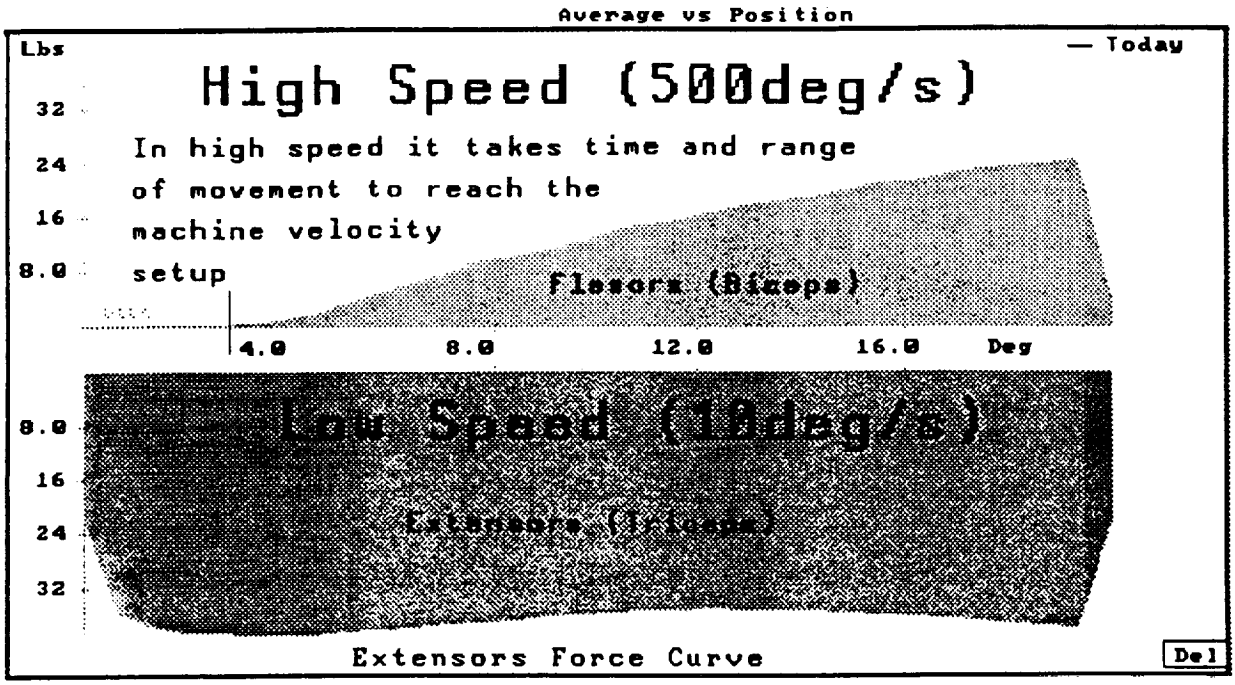


Figure 42

Figure 39 illustrates the concentric phase followed by the eccentric phase. The concentric phase by the flexors illustrates the same typical results as previously observed. In the eccentric phase, Figure 40 illustrates the displacement curve as well as the EMG activity of the flexors and the extensors. One can observe that in both the concentric and the eccentric phases, the flexors and extensors demonstrate EMG activity. As expected, the flexors demonstrate higher activity. It is interesting to note that during the passive rest phase, the extensors EMG shifted above the base line. Also, at the onset of extension, the extensors exhibited a sharp negative spike. This may indicate the result of a pre-stretch reflex which augmented the antagonistic muscle, the extensors, at the beginning of the eccentric contraction.

Figure 43 illustrates a typical concentric eccentric combination utilized in this experiment. The force curves, the displacements, and the EMG activities are presented. One of the goals of this experiment was to determine if the firing characteristics of the extensors and/or the flexors change during the concentric and/or eccentric phases. A typical result is presented in Figure 44. A fast Fourier Transformation revealed that the frequency characteristic of the EMG signal did not change for the same muscle group during the concentric and eccentric contractions. However, the amplitude of the EMG signal did vary. Amplitude is referred to as "power" and the power of the EMG activity measured in the current study did change. Higher power signals were observed during the eccentric contractions.

A third factor to consider provides information about the EMG activity and this is known as the "value reset integration". A value reset integration provides insight into the relationships of the EMG activity between the two phases of contractions. A specific level of EMG activity was set in this study as 100 millivolt seconds for the value reset integration. The reason for the selection of this type of integration was to determine if the activity level changed during the time of contraction for the concentric and eccentric phases.

Figure 45 illustrates a typical concentric and eccentric contraction with the associated displacement, raw EMG and value reset integration for the flexor and extensor muscles. As can

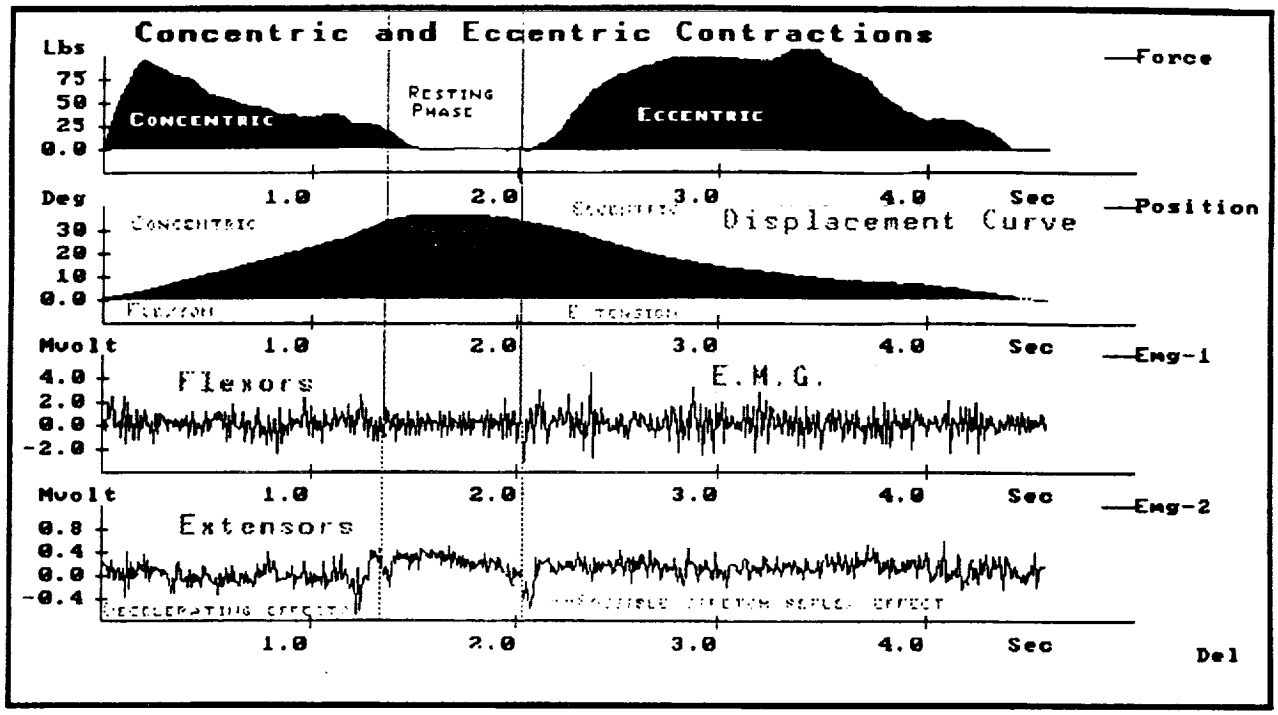


Figure 43

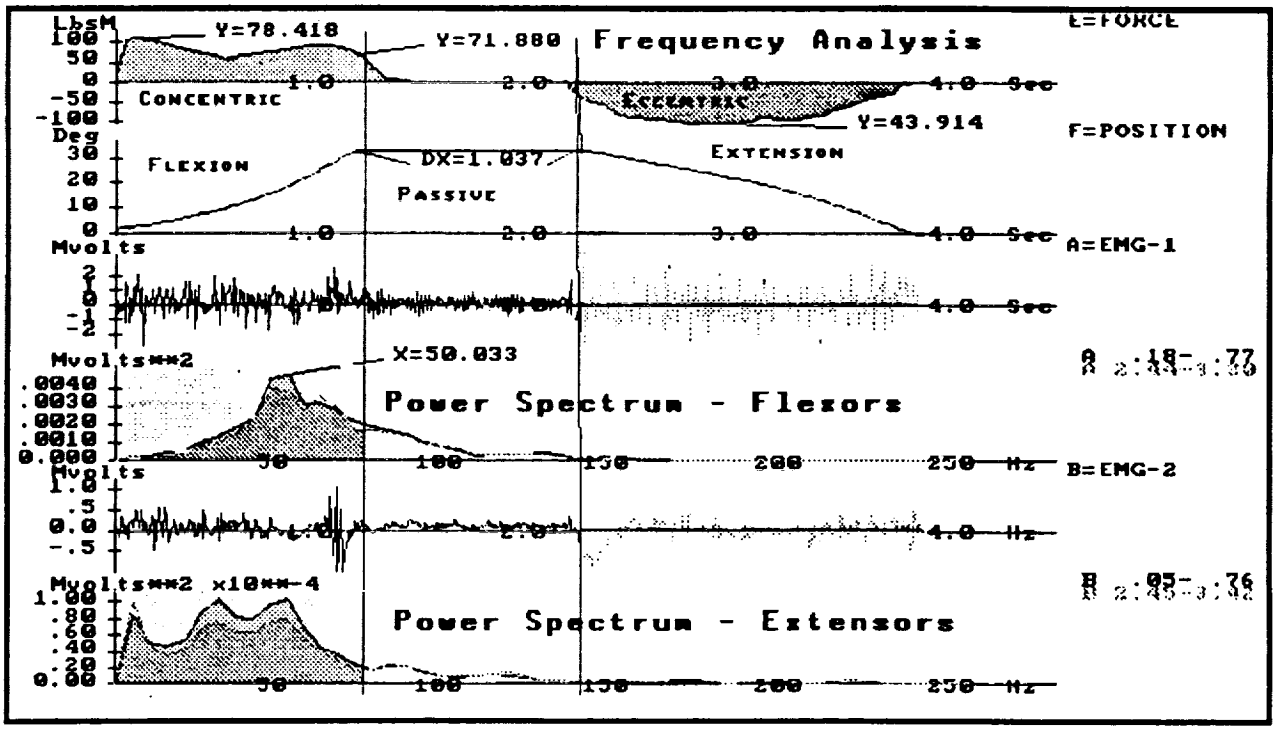


Figure 44

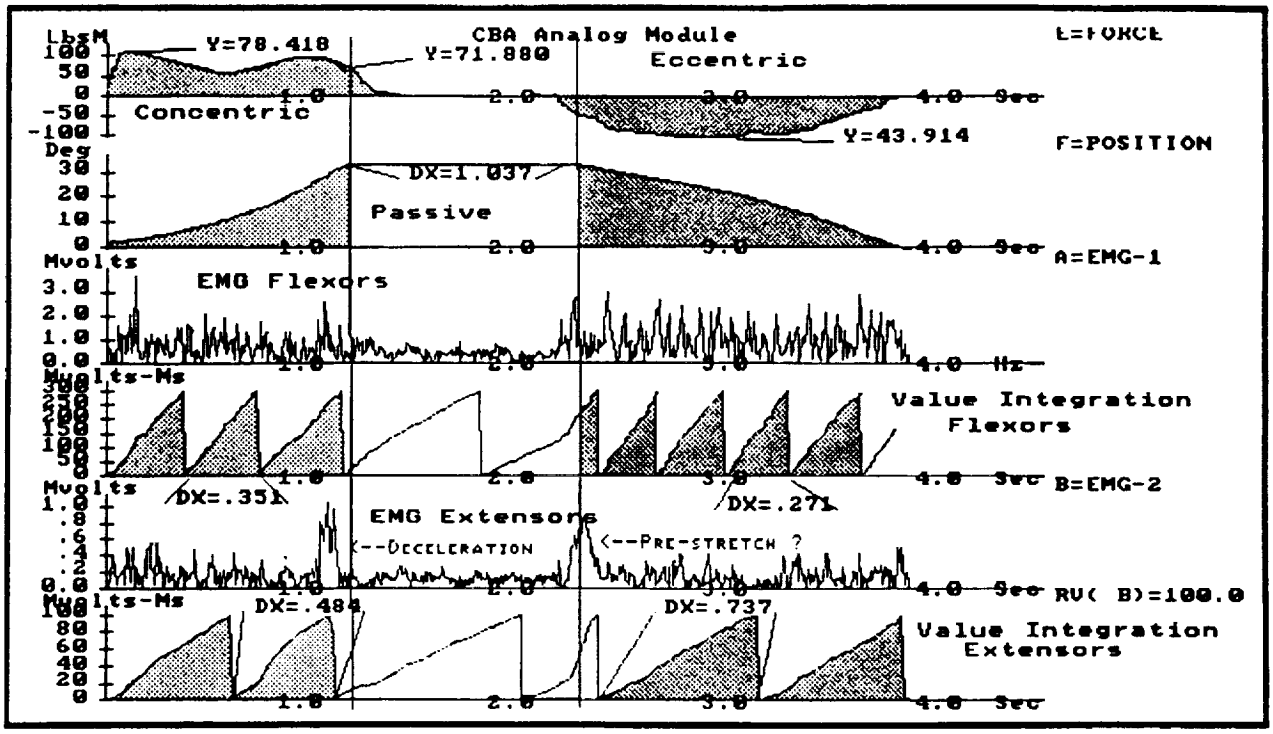


Figure 45

be seen in this Figure and, in general for all cases, the flexors were more active during the concentric phase as indicated by the shorter time required to reach a particular EMG level of activity indicated by the area under the integral curves. In the eccentric phase, the value integration of the flexors muscles shows even more activity as indicated by the smaller time interval to reach the chosen value for the reset integration of the EMG activity. However, the extensors muscles indicated less activity to reach the selected level of EMG activity as revealed by the longer base line. This phenomenon was repeated in most of the super maximal eccentric contraction trials. When measuring the same parameters during sub-maximal eccentric contractions, the extensor activity did not change significantly but the flexors showed less activity.

DISCUSSION

The present studies show that during dynamic contractions, whether concentric or eccentric, there is a control mechanism which simultaneously regulates the amount of EMG firing in the flexors and extensors. The level of activity appears to be regulated by some higher level neural control program. The net effect is responsible for the level of force produced in each movement.

It seems that there are differences between maximal and submaximal eccentric contractions. In the maximal eccentric contraction, where the external force was significantly greater than that generated by the muscle, a regulator mechanism was initiated so that the activity level of the antagonistic reduced, thus, enabling the agonist to perform in a more efficient manner. That is to say, the antagonistic effect was reduced in order to allow better "net" activity by the agonist. By reducing some of the antagonistic muscle activity, the agonist force produced a greater net moment. However, if the individual continues to try to overcome a large externally applied force, the net force may reach a level where internal injury to the tissue occurs. This situation can only occur in an eccentric condition.

When the eccentric contraction was sub-maximal, the antagonistic activity was not effected. In that situation, the elongation of the agonist muscle was achieved by "progressive relaxation" as the number of motor units firing decreased as indicated by the level of EMG and the force associated with this type of contraction.

It appears that eccentric contractions should be classified as two types. In both cases, the agonist is elongated during a dynamic contraction. In the case with a super-maximal external resistance, the regulatory mechanism of the agonist-antagonist relationship "turns off" or reduces some of the antagonistic activity allowing the agonist to exert a greater net moment. In the case of the sub-maximal eccentric contractions, the relationship of force production is changed by simply "turning off" some of the agonist muscle and progressively relaxing the amount of agonistic activity and allowing the limb to extend.

When examinating activities, such as walking and running, it appeared that the body tended to use progressive relaxation to achieve the proper level of shock absorption and gait efficiency. However, in a task involving jumping from a height in excess of 3 feet, it seemed that this mechanism gradually changed to a maximum eccentric contraction. An activity such as high jumping would probably incorporate both types depending on the intensity of contraction and external resistance.

Application to Training:

The literature is filled with contradictory findings concerning the muscular strength level which can be achieved with both methods of training, eg. concentric and eccentric. There seems to be a "carry over" effect regardless of the system of training whether concentric, eccentric, or a combination of the two.

Based on the literature and the experimental findings presented here, this author believes that for efficient and safe training, exercise should be done concentrically. This assures neuromuscular regulation of the desired movement. If one elects to train eccentrically, these exercises should be performed only at super-maximal contraction levels which will produce a higher net moment. Any level lower then the super-maximal will result only in progressive relaxation of the agonist muscle. A complete training regimen should include exercises with the body weight utilized in a way that mobilizes the parallel and serial connective tissues to absorb elastic energy and to maintain a full range of motion for each joint. These types of exercise necessitate eccentric contractions at some level; however, at a maximal resistance level, the risk of injury increases significantly.

Training should always include the natural movements of the human body. The body will not gain maximum benefit with training on mechanical machines which cannot reproduce the number of degrees of freedom the body requires for movement. Therefore, a healthy exercise program should include running, jumping, gymnastics, calisthenics, swimming, and rhythmic activities. These essential programs can be supplemented with exercise machines which, preferable, possess artificial intelligence to evaluate and coordinate the program.

In the particular situation of microgravity, in order to simulate "natural" resistance to the body, only a machine can perform adequately to stimulate the desired responses in an "artificial" environment. An exercise device properly designed should be able to accommodate the limitations of microgravity, since the body can be restrained in its environment and can execute activities providing high resistance externally thereby simulating exercise in 1-G condition.

Needless to say, more research is needed to determine the ideal training conditions for people to achieve the most efficient fitness level for performing specific activities on earth and in microgravity conditions.

REFERENCES

1. Abbot BC, Aubert XM, Hill AV: The absorption of work by a muscle stretched during a single twitch of a short tetanus. Proc R Soc Lond (Biol) 139: 86-104, 1951.
2. Abbot BC, Aubert XM: Changes of energy in a muscle during very slow stretches. Proc roy Soc (Lond.) 139: 104-117, 1951.
3. Abbot BC, Bigalnd B, Ritchie JM: The physiological cost of negative work. J Physiol 117: 380-390, 1952.
4. Abbott BC: The heat production associated with the maintenance of a prolonged contraction and the extra heat produced during large shortening. J Physiol 112: 438-445, 1951.
5. Abbs JH, Gracco VL: Control of complex motor gestures: Orofacial muscle responses to load perturbations of lipsduring speech. J Neurophysiol 51: 705-723, 1984.
6. Abend W, Bizzi E, Morasso P: Human arm trajectory formation. Brain 105: 331-348, 1982.
7. Abraham WM: Factors in delayed muscle soreness. Med Sci Sports 11-20, 1977.
8. Abramowitz M, Stegun IA: Handbook of mathematical functions. Dover Publishers. New York. 1968.
9. Adrian ED, Bronk DW: The discharge of impulses in motor nerve fibres. Part II. The frequency of discharge in reflex and voluntary contractions. J Physiol. (Lond) 67: 119-151, 1929
10. Adrian RH, Chandler WK, Hodgkin AL: The kinetics of mechanical activation in frog muscle. J Physiol 204: 204-230. 1969.
11. Al-Amood WS, Pope R: A comparison of the structural features of muscle fibres from a fast-and a slow-twitch muscle of the pelvic limb of the cat. J Anat, 113: 49-60, 1972.
12. Alexander RS, Johnson PD: Muscle stretch and theories of contraction. Am J Physiol 231: 87-104, 1973.
13. Andersson O, Forssberg H, Grillner S: Peripheral control of the cat's step cycle. I Phase dependent effects of ramp movements of the hip during "fictive locomotion." Acta Physiol Scand, 118: 229-239, 1983.
14. Andersson O, Grillner S: Peripheral control of cat's step cycle. II. Entrainment of the cen. pattern generators for locomotion by sinusoidal hip movements during "fictive locomotion" Acta. Pysiol. 118: 229-239, 1983.
15. Angel RW, Eppler W, Iannone A: Silent period produced by unloading of muscle during voluntary contraction. J. Physiol (Lond.) 180: 864-870, 1965.
16. Angel RW: Antagonist muscle activity during rapid arm movements: central versus proprioceptive influences. J. Neurol Neurosurg Psychiat 40: 683-686, 1977.
17. Angel RW: Electromyography during voluntary movement: The two burst pattern, Electroenceph. Clin. Neurophysiol. 36: 493-498, 1974.
18. Arbib, Michael A., The Metaphorical Brain, Wiley-Interscience, New York, 1972.
19. Ariel, Gideon, Computerized Biomechanical Analysis of Human Performance, Mechanics and Sport, Vol. 4, The American Society of Mechanical Engineers, p. 267 - 275, 1973.
20. Ariel, Gideon, The Effect of Knee Joint Angle on Harvard Step Test Performance, Ergonomics 12: 33-37, 1969.
21. Ariel, Gideon, Effect of Anabolic Steroids on Reflex Components, J. Applied Physiol., 32: 795-797, 1972.
22. Ariel, Gideon, Anabolic Steroids: The Physiological Effects of Placebos, Medicine and Science 4: 124-126, 1972.
23. Ariel, Gideon, The Effect of Anabolic Steroid upon Skeletal Muscle Force, J. Sports Medicine and Physical Fitness 13: 187-190, 1973.
24. Ariel, Gideon, Prolonged Effects of Anabolic Steroid upon Muscular Contractile Force, Medicine and Science in Sports 6: 62-64, 1974.
25. Ariel, Gideon, Computerized Biomechanical Analysis of Human Performance, In Thomas P. Martin, Biomechanics of Sport. State University of New York at Brockport, 228-229, 1975.
26. Ariel, Gideon, Computerized Biomechanical Analysis of the Knee Joint during Deep Knee Bend with Heavy Load, Biomechanics IV. Edited by Richard C. Nelson and Chaucey A. Morehouse, Fourth International Seminar on Biomechanics Volume IV. The Pennsylvania State University, 1973.
27. Ariel, Gideon, Shear and Compression Forces in the Knee Joint during Deep Knee Bend, XXth World Congress in Sports Medicine Handbook, Melbourne, Australia, 1974.
28. Ariel, Gideon, Computerized Biomechanical Analysis of Human Performance. XXth World Congress in Sports Medicine Handbook, Melbourne, Australia, 1974.

29. Ariel, Gideon, Computerized Biomechanical Analysis of Human Performance: An Application for Exercise Equipment Design and Athletic Performance, Technical Report Uni-1, Universal Fitness Research Department, 1974.
30. Ariel, Gideon, Computerized Biomechanical Analysis of the Bench Press and the Leg Press Exercise, Universal Fitness Research Department, Technical Report Uni-3, 1974.
31. Ariel, Gideon, Variable Resistance Exercise: A Biomechanical Approach to Muscular Training, Universal Fitnes Research Department, Technical Report Uni-3, 1974.
32. Ariel, Gideon, Intra-articular Shearing Factor during Resistance Exercises, Universal Fitness Research Department, Technical Report Uni-4, 1974.
33. Ariel, Gideon, Principles of Ballistic Motion in Resistance Exercise Training, Universal Fitness Research Department, Technical Report Uni-5, 1974.
34. Ariel, Gideon, Computerized Biomechanical Analysis of the Variable Resistance Exercise Machine, Universal Fitness Research Department, Technical Report Uni-7, 1974.
35. Ariel, Gideon, Computerized Biomechanical Analysis of Human Performance, Athletic Journal 54: 54-84, 1974.
36. Ariel, Gideon, Method for Biomechanical Analysis of Human Performance, Research Quarterly 45: 72-79, 1974.
37. Ariel, Gideon, Principles of Ballistic Motion in Resistive Exercise Training, Scholastic Coach 43: 80-108, 1974.
38. Ariel, Gideon, Understanding the Scientific Bases Behind Variable Resistance Exercises, Fresno: Universal Athletic Sales, 1974.
39. Ariel, Gideon, Biomechanical Consideration in the Design and Construction of Resistance Exercise Equipment, Proceedings of the 1st Annual Meeting, Canadian Society for Biomechanics. University of Alberta, Edmonton, Alberta, 25-34, 1974.
40. Armstrong BW, Hurt HH, Workman JM: Downhill walking as a possible form of negative work. Am J Physiol 211: 1264-1268, 1966.
41. Armstrong RB, Ogilvie RW, Schwane JA: Eccentric exercise-induced inury to rat skeletal muscle, J Appl Physiol 54: 80-93, 1983
42. Arshavsky, Y.I., Y.M. Kots, G.N. Orlovsky, I.M. Rodionov, and M.L. Shik, Investigation of the Biomechanics of Running by the Dog, Biophysics: 10, p. 737 - 746, 1965.
43. Asimov, Isaac Understanding Physics: Motion, Sound, and Heat, New American Library, New York, 1966.
44. Asmussen E: Observations on experimental muscular soreness. Acta Rheum Scand 2: 109-116, 1952.
45. Asmussen E: Positive and negative muscular work. Acta Physiol Scand 28: 365-382, 1953.
46. Astbury WT, X-ray studies of muscle. Proc R Soc (Biol.) 137: 58, 1950.
47. Aura O, Komi PN: Effects of prestretch on mechanical effiency of positve work and on elastic behavior for skeletal muscle in stretch shortening cycle exercise. Int J Sports Med 7: 137-143, 1986.
48. Bagust J, Knott S, Lewis DM: Isometric contractions of motor units in a fast twitch muscle of the cat. J Physiol 231: 87-194, 1973.
49. Bahler AAS, Fales JT, Zierler DK: The active state of mammalian skeletal muscle J Gen Physiol 50: 2239-2253, 1967.
50. Bahler AS, Fales JT, Zierler KL: The dynamic properties of mammalian skeletal muscle. J Gen Physiol 51: 369-384, 1968.
51. Bahler AS: Modeling of mammalian skeletal muscle. IEEE Trans Bio-Med Eng BME-15: 249-257, 1968.
52. Bahler AS: Series elastic component of mammalian skeletal muscle. Am J Physiol 213: 1560-1564, 1967.
53. Baldissera F, Hultborn H: Integration in spinal neuronal sys. Handbook of Physiology, sec. 1, vol II, The Nervous System: Motor control, Part I, Brooks VB ed. Bethesda: Amer. Physiological Soc.:509-595, 1981.
54. Ballereich R: An analysis of long-jump. In: Biomechanics III, Medicine and Sport, Karger.Basal vol. 8: 394-402, 1973.
55. Banus MG, Zetlin AM: The relation of isometric tension to length in muscle. J Cell Comp Physiol 12:403-420, 1938.
56. Barany M: ATPase activity of myosin correlated with speed of muscle shortening. J Gen Physiol 50: 197-218, 1967.
57. Barnett JG, Holly RG, Ashmore CR: Sretch induced growth in chicken wing muscles: biochemical and morphological 'characterization. Am J Physiol 239: C38-C46, 1980.

58. Basmajian, J.V., Muscles Alive, Williams and Wilkins, Baltimore, 1978.
59. Bawa P, Mannard A, Stein RB: Predictions and experimental tests of a visco-elastic muscle model using elastic and inertial loads. Biol Cybernetics 22: 139-145, 1976.
60. Belen'kii VYi, gurfinkel; VS, Pal'tsev YeI: Elements of control of voluntary movements. Byophysics 12: 135-141, 1967.
61. Bendall JR: Muscles molecules and movement. Heinemann Educ. Books Ltd. London, 1969.
61A. Berger RA: Optimum Repetitions fr the development of Strenth. Res. Quarterly 33: 334-338, 1962.
61B. Berger RA and Hardage B: Effect of Maximal Load for each of ten repetitions on Strength Improvement. Res. Quarterly 38: 715-718, 1966.
62. Bernstein J: Experimentelles und eine kritsche Theorie der Muskelkontraktion Arch f ges Physiol 162: 1, 1915
63. Bernstein N: The Coordination and Regulation of Movements. New York: Pergamon, 1967.
64. Bertolotto A, Palmucci L, DoriguzziC, Mongini E, Gagnor M, del Rosso M, Tatone G: Laminin and gibronection distribution in normal and pathological human muscle. J Neurol Sci 60: 377-382, 1983.
65. Bigland B, Lippold OCJ: Motor unit activity in the voluntary contraction of human muscles during voluntary contraction. J Physiol.
66. Bigland B, Lippold OCJ: The relation between force, velocity and integrated electrical activity in human muscles. J Physiol 123: 214-224, 1954.
67. Bigland-Ritchie B: EMG/Force relations and fatigue of human voluntary contractions. In Miller DI (ed): Exercise and Sport Science Reviews. Philadelphia: The Franklin Institute, Vol. 9: 75-117, 1981.
68. Bischoff R: A satellite cell mitogen fromcrushed adult muscle. Dev Biol 115: 140-147, 1986.
69. Bischoff R: Proliferation of muscle satellite cells on intact myofibers in culture. Des Biol 115: 129-139. 1986.
70. Bizzi E, Abend W: Posture control and trajectroy formation in single and multi-joint arm movements. Motor Control in Health and Disease, Desmedt JE, ed. New York: Raven, pp 31-45, 1983.
71. Bizzi E, Accornero N, Chapple W, Hogan N: Arm trajectory formation in monkeys, Exp. Brain. Res. 46: 139-143, 1982.
72. Bizzi E, Polit A, Morasso P: Mechanisms underlying achievement of final head position. J. Neurophysiol. 39: 434-444, 1976.
73. Bizzi, E. "Coordination of Movements", Neural Control of Locomotion, Editied by Herman, Grillner, Stein, and Stuart, Plenum Press, New York, pp 798-804, 1976.
74. Bolstad G, Ersland A: Energy metabolism in different human skeletal muscles during voluntary contraction. Acta Physiol Scand 95: 73A-74A, 1975.
75. Borelli, Giovanni Alfonso, On the Movement of Animals. (translated) By Paul Maquet. Springer-Verlag, Berlin.
76. Borg ATK, Caufield JB: Morphology of connective tissue in skeletal muscle. Tissue Cell 12: 197-207, 1980.
77. Borg ATK, Klevay LM, Gay RE, Siegel R, Bergin ME: Alteration of connective tissue network of striated muscle in copper deficient rats. J Mol Cell Cardiol 17: 1173-1183, 1985.
78. Bouisset S, Lestienne F, Maton B: The stability of synergy in agonists during the execution of a simple voluntary movement. Electroenceph. Clin. Neurophysiol. 42: 543-551, 1977.
79. Boylls CC: Prolonged alterations of muscle activity induced in locomoting premammillary cats by microstimulation of the inferior olive. Brain Res. 159: 445-450, 1978.
80. Brandell BR: An analysis of muscle coordination in walking and running gaits. In Biomechanics III, Medicine and Sport, vol. 8: 278-287, Karger. Basel. 1973.
81. Braune, W., and O. Fischer, "The Center of Gravity of the Human Body as Related to the Ger Infantryman", Leipzig, Germany, 1889, sited in Occupational Biomech. by Chaffin and Andersson, John Wiley & Sons, N.Y., 1984.
82. Brett JR: The swimming energetics of salmon. Scient Amer 213: 76-84, no. 2, 1965.
83. Brink E, Jankowska D, McCrea D, Skoog B: Inhibitory interactions between interneurones in reflex pathways from group Ia and group Ib afferents in the cat. J. Physiol. (Lond.) 343: 361-373, 1983.
84. Broer MR, Houtz SJ: Patterns of Muscular Activity in Selected Sport Skills: An electromyographic study. Springfield: Charles C. Thomas, 1967.

85. Brzank BKD, Pieper KS: Die Awirkung intesiver, kraftbetonter Trainingsbleastungen auf die Feinstruktur der menschilichen Skelettmuskelkapillare. Anat Anz 16: 243-248, 1985.
86. Buchthal F, Kaiser E: The rheiolgy of the cross striated muscle fibre with particular reference to isotonic conditions. Dan Biol Med 21: 318, 1951.
87. Buller AJ, Eccles JC, Eccles RM: Differentiation of fast and slow muscles in the cat hind limb. J Physiol 150: 399-416, 1960.
88. Buller AJ, Lewis DM: The rate of tension development in isometric tetanic contractions of mammalian fast and slow skeletal muscle. J Physiol 106: 337-354, 1965.
89. Burke D: Muscle spindle function during movement. Trends Neurosci. 3: 251-253, 1980.
90. Burke RE, Levine DN, Zajac FE, Tsairis P, Engel WK: Mammalian motor units: physiological-histochemical correlation in three types in cat gastrocnemius. Science 174: 709-712, 1971.
91. Burke RE: Motor units: anat., physiology, and functional organization. Handbook of Physiology, 1, vol. 2, The Nervous Sys: Motor Control, Part 1. VB Brooks, ed. Bethesda: Amer. Physiological Soc., pp 345-422, 1981.
92. Burr DB, Martin RB, Schaffler MB, Radin EL: Bone remodeling in response to in vivo fatigue microdamage. J Biomech 18: 189-200, 1985.
93. Byrnes WC, Clarkson PM, White JS, Hsieh SS, Frykman PN, Maughan RJ: Delayed onset muscle soreness following repeated bouts of downhill running. J Appl Physiol 5: 719-715, 1985.
94. Carlson BM, Faulkner JA: The regeneration of skeletal muscle fibers following injury: a review. Med Sci Sports Exerc 115: 187-198, 1983.
95. Cavanagh PR: On "muscle action" vs "muscle contraction." J Biomech 21: 69, 1988.
96. Chaffin, D.B., and G.B.J. Andersson, Occupational Biomechanics, John Wiley & Sons, New York, 1984.
97. Chandler C, Hewit J, Miller S: Computers, brains and the control of movement. (Letter to the editor) Trends in Neurosci. 5: 372, 1982.
98. Chao EY, Rim K: Application of optimization principles in determining the applied moments in human leg joints during gait. J Biomech 6: 497-510, 1973.
99. Chapman AE: The mechanical properties of human muscle . In Terjung RL (ed.): Exercise and Sport Sciences Review New York, Macmillan, Vol 13, pp 443-591, 1985.
100. Chow CK, Jacobson DH: Studies of human locomotion via optimal programming. Math. Biosci. 10: 239-306, 1971.
101. Clarkson PM, Byrnes WC, Gillisson E, Harper E: Adaptation to exercise-induced muscle damage. Clin Sci 73: 383-385, 1987.
102. Clarkson PM, Tremblay I: Rapid adaptation to exercise induced muscle damage. Med Sci Sports Exerc 19: 36, 1987.
103. Close RI: Dynamic properties of fast and slow skeletal muscles of the rat after nerve crossunion. J Physiol 204: 331-346, 1969.
104. Close RI: Dynamic properties of fast and slow skeletal muscles of the rat during development. J Physiol 173: 74-95, 1964.
105. Close RI: Dynamic properties of mammalian skeletal muscle. Physiol Rev 52: 129-197, 1972.
106. Close RI: The relation between intrinsic speed of shortening and duration of the active state of muscle. J Physiol 180: 542-559, 1965.
107. Coddington EA, Levinson N: Theory of ordinary differential equations. McGraw-Hill, New York, 1955.
108. Constantin LL, Podolsky RJ: Evidence of depolarization of the internal membrane system in activation of frog semitendinosus muscle. Nature 210: 483-486, 1966.
109. Cordo PJ, Nashner LM: Properties of postural adjustments associated with rapid arm movements. J. Neurophysiol. 47: 287-302, 1982.
110. Cordo PJ, Rymer WZ: Contributions of motor unit recruitment and rate modulation to compensation for muscle yielding. J Neurophysiol.47: 797-809, 1982.
111. Cotes JE, Meade F: Energy expenditure and energy demand in walking. Ergonomics 3, 1960.
112. Crago PE, Houk JC, Hasan Z: Regulatory actions of human stretch reflex. J. Neurophysiol. 39: 925-935, 1976.
113. Craik, R., R. Herman, and F.R. Finley, "Human Solutions for Locomotion: Interlimb Coordination", Neural Control of Locomotion, Edited by Herman, Grillner, Stein, and Stuart, Plenum Press, New York, pp 51-64, 1976.

114. Creed RS, Denny-Brown D, Eccles JC, Liddel EGT, Sherrington CS: Reflex Activity of the Spinal Cord. London: Oxford University Press, 1932.
115. Crosby, Elizabeth C. Tryphena Humphrey, and Edward W. Lauer, Correlative Anatomy of the Nervous System, The Macmillan Company, New York, 1962.
116. Crowe A: A mechanical model of muscle and its application to the intrafusal fibres of the mammalian muscle spindle. J Biomech 3: 583-592, 1970.
117. Curtin N, Gilbert C, Kretzschmar KM, Wilkie DR: The effect of the performance of work on total energy output and metabolism during muscular contration. J Physiol 238: 455-472.
118. da Vinci, Leonardo: An Artabras book, Reynal and Company, New York.
119. Darling WG, Cooke JD: The influence of practice on movement dynamics and muscle actvity. Soc. Neurosci. Abstr. 9: 1032, 1983.
120. Davies CT, White MJ: Muscle weakness following eccentric work in man. Pflugers Arch 392: 168-171, 1981.
121. Davies RE: A molecular theory of muscle contraction: calcium dependent contractions with hydrogen bond formation plus ATP-dependent extensions of part of the myosin-actin cross-bridges. Nature 199: 1068-1074, 1963.
122. Davis, J. Central Activation of Movements. Neural Control of Locomotion, Edited by Herman, Grillner, Stein, and Stuart, Plenum Press, New York, pp 804-808, 1976.
123. Del Castillo J, Katz B: Biophysical aspects of neuromuscular transmission. Progr Biophys 6: 121-170, 1956.
124. Deleze JB: The mechanical properties of the semitendinosus muscle at lengths greater than its length in the body. J Physiol 158: 154-164, 1961.
125. Deliagina TG, Orlovsky GN, Perret C: Efferent activity during fictitious scratch reflex in the cat. J. Neurophysiol 45: 595-604, 1981.
125A. Delorme TL, Ferris BG, Gallgher JR: Effect of Progressive Resistance exercise on muscle contraction time. Arch. Phys. Med. 33: 86-92, 1952.
126. Dempster, W.T. "Space Requirements of the Seated Operator", WADC-TR-55-159, Aerospace Medical Research Laboratories, Ohio, 1955.
127. Denny-Brown D, Pennybacker JB: Fibrillation and fasciculation in voluntary muscle. Brain 61: 311-333, 1938.
128. Desmedt JE, Godaux E: Ballistic contractions in fast or slow human muscles: discharge patterns of single motor units. J. Physiol. (Lond.) 285: 185-196, 1978.
129. Desmedt JE, Godaux E: Ballistic contractions in man: characteristic recruitment pattern of single motor units of the tibialis anterior muscle.
130. Desmedt JE, Godaux E: Spinal motoneuron recruitment in man: Rank deordering with direction but not with the speed of voluntary movement. Science 214: 933-936, 1981.
131. Desmedt JE, Hainaut K: Kinetics of myofilament activation in potentiated contraction: Staircase phenomenon in human skeletal muscle. Nature 217: 529-532, 1968.
132. Dick RW, Cavanagh PR: An explanation of the upward drift in oxygen uptake during prolonged submaximal downhill running. Med Sci Sports Exerc 19: 310-317, 1987.
133. Dragomir CT: On the nature of forces acting between myofilaments in resting state and under contraction. J Theor Biol 27: 343-356, 1970.
134. Drillis, R., and R. Contini, "Body Segment Parameters", BP174-945, Tech. Rep. No. 1166.03, School of Engineering and Science, New York University, New York, 1966.
135. Duncan CJ: Role of calcium in triggering rapid ultrastructural damage in muscle a study with chemically skinned fibers. J Cell Sci 87: 581-594, 1987.
136. Ebashi S, Endo M: Calcuim ion and muscular contraction. Progr Biophys Mol Biol 18: 125-183, 1968.
137. Ebashi S, Kodama A, Ebashi F: Troponin. 1. Preparation and physiological function. J Biochem 64: 465, 1968.
138. Ebashi S, Kodama A: A new protein factor promoting 'aggregation of tropomyosin. J Biochem 58: 107, 1965.
139. Eberhart, H.D. "Physical Principles of Locomotion", Neural Control of Locomotion, Edited by Herman, Grillner, Stein, and Stuart, Plenum Press, New York, pp 1-11, 1976.

140. Eberstein A, Goodgold J: Slow and fast twitch fibres in human skeletal muscle. Amer J Physiol 215: 535-544, 1968.
141. Eccles JC: The understanding of the brain. McGraw-Hill, New York. pp 20, 1973.
142. Eccles RM, Lundberg A: Integrative pattern of IA synaptic actions on motoneurons of hip and knee muscles. J. Physiol (Lond.) 144: 271-298, 1958.
143. Edman Kap, Elizinga G., Noble MM: The effect of stretch on contracting skeletal muscle fibers. In Sugi H, Pollack GH (eds.): Cross Bridge Mechanisms in Muscle Contraction. Balt. Univ. Park Press, pp 297-309, 1979.
144. Edman KAP, Mulieri LA, Scubon-Mulieri B: Non-hyperbolic force velocity relationship in single muscle fibres. Acta Physiol Scand 98: 143-156, 1976.
145. Edman KAP: The velocity of unloaded shortening and its relation to sarcomere length and isometric force in vertebrate muscle fibres. J Physiol 291: 143-156, 1976.
146. Edwards RHT, Hill DK, Jones DA, Merton PA: Fatigue of long duration in human skeletal muscle after exercise. J Physiol 272: 769-778. 1977.
147. Edwards RHT, Jones DA: Diseases of skeletal muscle. In Handbook of Physiology: Vol. 10 Skeletal Muscle. Baltimore, Williams & Wilkins, pp 633-672, 1983.
148. Edwards RHT: Muscle fatigue and pain. Acta Med Scand(Suppl)
149. Elliot GF, Rome EM, Spencer: A type of contraction hypothesis applicable to all muscles. Nature 226: 417, 1970.
150. Enberg I, Lundberg A: An electromygraphic analysis of muscular activity in the hidlimb of the cat during unrestrained locomotion. Acta Physiol. Scand. 75: 614-630, 1969.
151. Engelhardt VA, Lyubimova MN: On the mechanochemistry of muscle. Biokhimiya 7: 205, 1942.
152. Enoka RM, Hutton RS, Eldred E: Changes in excitability of tendon tap and Hoffmann reflexes following voluntary contractions. Electroenceph. Clin. Neurophysiol. 48: 664-672, 1980.
153. Enoka RM, Stuart DG: Henneman's "size principle": Current issues. Trends Neurosci. 7: 226-228, 1984.
154. Enoka RM: Muscular control of a learned movement: The speed control system hypothesis. Exp. Brain Res. 51: 135-145, 1983.
155. Evans WJ. Meredith CN, Cannon JG, Dinarello CA, Frontera WR, Hughes VA, Jones BH, Knuttgen HG: Metabolic changes following eccentric exercise in trained and untrained men. J Appl Physiol 61: 1864-1868, 1986.
156. Evarts EV: Sherringtons's concept of proprioception. Trends Neurosci. 2: 44-46, 1981.
157. Evoy WH, Ayers J: Locomotion and control of limb movements. Biology of Crustacea, vol. 4. New York: Academic Press, pp 61-105, 1982.
158. Falk G, Fatt P: Linear electrical properties of striated muscle fibres observed with intracellular electrodes. Proc Roy Soc Biol.
159. Falk G: Predicted delays in the activation of the contractile system. Biophys J 8: 608-625, 1968.
160. Farquharson, A.S.L. Aristotle's De Motu Animalium (translation). In: The Works of Aristotle. Edited by Smith & Ross, Vol. 5. Claredon Press, Oxford, 1912.
161. Fatt P: Skeletal neuromuscular transmission. In: Neurophysiology I, pp 204. Am Physiol Soc, Washington. 1959.
162. Feldman AG, Orlovsky GN: The influence of different descending systems on the tonic stretch reflex in the cat. Exp. Neurol. 37: 481-494, 1972.
163. Feldman AG: Change in the length of the muscle as a consequence of a shift in the equilibrium in the muscle load-system. Biophysics 19: 534-538, 1974.
164. Feldman AG: Functional tuning of the nervous sys. during control of movement or maintenance of a steady posture. III. Mechanographic analysis of the execution by man of simple motor tasks. Biophysics 11: 667-675, 1966.
165. Feldman AG: Functional tuning of the nervous system with control of movement or maintenance of a steady posture. II. Controllable parameters of the muscles. Biophysics
166. Feldman AG: Superposition of motor programs. I. Rhythmic forearm movements in man. Neuroscience 5: 81-90, 1980.
167. Field EJ: The development of the conduction system in the heart of sheep. Br Heart J 13: 129-147, 1951.
168. Fitzhugh R: A model of optimal voluntary muscular control. J Math Biol 4: 203-236, 1977.

169. Fleshman JW, Lev-Tov A, Burke RE: Synaptic organization in FDL and FHL motor nuclei: A search for mechanisms underlying the functional disparity in strict anatomical synergists. Soc. Neurosci. Abst. 7: 689, 1981.
170. Flitney FW, Hirst DG: Cross-bridge detachment and sarcomere "give" during stretch of active frog's muscle. J Physiol 276: 449-465.
171. Forssberg H: Phasic gating of cutaneous reflexes during locomotion. Muscle Receptors and Movement. A Taylor and A Prochazka, eds. London: MacMillan, pp 403-412, 1981.
172. Freund HJ, Budingen HJ: The relationship between speed and amplitude of the fastest voluntary contractions of human arm muscles. Exp. Brain Res. 33: 1-12, 1978.
173. Friden J, Sfakianos PN, Hargens AR: Muscle soreness and intramuscular fluid pressure: comparison between eccentric and concentric load. J Appl Physiol 61: 2175-2179, 1986.
174. Friden J, Sjostrom M, Ekblom B: A morphological study of delayed muscle soreness. Experientia 37: 506-597, 1981.
175. Friden J, Sjostrom M, Ekblom B: Myofibrillar damage following eccentric exercise in man. Int J Sports Med 4: 170-176, 1983.
176. Friden J: Changes in human skeletal muscle induced by long-term eccentric exercise. Cell Tissue Res 236: 365-372, 1984.
177. Fritz VK, Stauber WT: Characterization of muscles injured by forced lengthening: II. Proteoglycans. Med Sci Sports Exerc 20: 354-361, 1988.
178. Froese EA, Houston ME: Torque-velocity characteristics and muscle fiber types in human vastus lateralis. J Appl Physiol 59: 309-314, 1985.
179. Fukami Y: Responses of isolated Golgi tendon organs of the cat to muscle contraction and electrical stimulation. J. Physiol. (Lond.) 318: 429-443, 1981.
180. Fukson OI, Berkinblit MB, Feldman AG: The spinal frog takes into account the scheme of its body during the wiping reflex. Science 209: 1261-1263, 1980.
181. Fung TCB: Chapter 7 of Biomechanics. Its foundations and objectives. New Jersey: Prentice-Hall, 1972.
182. Fung TCB: Mathematical representation of the mechanical properties of heart muscle. J Biomech 3: 381-404, 1970.
183. Fung YCB: Biomechanics: Its scope, history and some problems of continuum mechanics in physiology. Appl Mech Rev 21: 1-20, 1968.
184. Gallistel CR: The Organization of Action: A New Synthesis. Hillsdale: L. Erlbaum, 1980.
185. Garnett R, Stephens JA: Changes in the recruitment threshold of motor units produced by cutaneous stimulation in man. J. Physiol. (Lond.) 311: 463-473, 1981.
186. Garrett WE, Nikolaou PK, Ribbeck BM, Glisson RR, Seaber AV: The effect of muscle architecture on the biomechanical failure properties of skeletal muscle under passive tension. Am J Sports Med 16: 7-12, 1988.
187. Georgopoulos AP, Kalaska JF, Caminiti R, Massey JT: Interruption of motor cortical discharge subserving aimed arm movements. Exp. Brain Res. 49: 327-340, 1983.
188. Ghez C, Martin JH: The control of rapid limb movement in the cat. III. Agonist-antagonist coupling. Exp. Brain Res. 45: 115-125, 1982.
189. Ghez C, Shinoda Y: Spinal mechanisms of the functional stretch reflex. Exp. Brain Res. 32: 55-68, 1978.
190. Ghez C, Vicario D, Martin JH, Yumiya H: Sensory motor processing of target movements in motor cortex. Motor Control Mechanisms in Health and Disease. JE Desmedt, ed. New York: Raven, pp 61-92, 1983.
191. Ghez C, Vicario D: The control of rapid limb movement in the cat. II. Scaling of isometric force adjustments. Exp. Brain Res. 33: 191-202, 1978.
192. Ghez C: Contributions of central programs to rapid limb movement in the cat. Integration in the Nervous System. H Asanuma and VH Wilson, eds. Tokyo: Igaku-Shoin, pp 305-320, 1979.
193. Gibbs CL, Gibson WR: Energy production of rat soleus muscle. Amer J Physiol 223: 864-871, 1972.
194. Gielen CCAM, Houk JC: Nonlinear viscosity of human wrist, J. Neurophysiol, 52: 553-569, 1984.
195. Gillis JM: The site of action of Ca in producing contraction is striated muscle. J Physiol 200: 849-865, 1969.
196. Glantz SA: A constitutive equation for the passive properties of muscle. J Biomechanics 7: 137-145, 1974.
197. Goldberg AL, Etlinger JDS, Goldspink DF, Jablecki C: Mechanism of work-induced hypertrophy of skeletal muscle. Med Sci Sports 7: 185-198, 1975.

198. Goldberger ME: The extrapyramidal systems of the spinal cord. II. Results of combined pyramidal and extrapyramidal lesions in the macaque. J. Comp. Neurol. 135: 1-26, 1969.
199. Goldspink G: Growth of muscle. In Goldspink DF (ed): Development and Specialization of Skeletal Muscle. London, Cambridge University Press, pp 19-35, 1981.
200. Gollnick PD, King DW: Effect of exercise and training on mitochondria of rat skeletal muscle. Am J Physiol 216: 1502-1509, 1969.
201. Gonzalez-Serratos H: Inward spread of contraction in striated during a twitch. J Physiol 185: 20-21, 1966.
202. Gordon AM, Huxley AF, Julian FJ: The variation in isometric tension with sarcomere length in vertebrate muscle fibres. J Physiol 184: 170-192, 1966.
203. Gordon J, Ghez C: EMG patterns in antagonist muscles during isometric contraction in man: Relations to reponse dynamics. Exp. Brain Res. 55: 167-171, 1984.
204. Goslow GE, Stauffer EK, Nemeth WC, Stuart DG: Digit flexor muscles in the cat. Their action and motor units. J. Morphol. 137: 335-352, 1972.
205. Gottlieb GL, Agarwal GC: Response to sudden torques about ankle in man.III. Suppression of stretch-evoked responses during phasic contraction. J. Neurophysiol. 44: 233-246, 1980.
206. Granit, Ragnar "Demand and Accomplishment in Voluntary Movement", Control of Posture and Locomotion, Edited by Stein, Pearson, Smith, and Redford, Plenum Press, New York, pp 3-18, 1973.
207. Granit, Ragnar The Basis of Motor Control, Academic Press, London and New York, 1970.
208. Green DG: A note on modeling muscle in physiological regulators. Med & Biol Eng 7: 41-48, 1969.
209. Greene PH: Problems of organization of motor systems. Prog. Theor. Biol. 2: 303-338, 1972.
210. Grillner S: Control of locomotion in bipeds, tetrapods, and fish. Handbook of Physiology, sec.1, vol. II, The Nervous System: Motor Control, Part 2, VB Brooks, ed. Bethesda: American Physiological Society, 1981.
211. Grillner S: Locomotion in vertebrates: Central mechanisms and reflex interaction. Physiol. Rev. 55: 247-304, 1975.
212. Grillner, S. Some Aspects on the Descending Control of the Spinal Circuits Generating Locomotor Movements. Neural Control of Locomotion, Edited by Herman, Grillner, Plenum Press, New York, pp 351-375, 1976.
213. Grimby L, Hannerz J, Hedman B: Contraction time and voluntary discharge properties of individual short toe extensor motor units in man. J Physiol 289: 191-201, 1979.
214. Grimby L, Hannerz J: Firing rate and recruitment order of toe extensor motor units in different modes of voluntary contraction J Physiol 264: 864-879.
215. Gurfinkel VS, Shik MS: The control of posture and locomotion. Motor Control, Gydikov AA, Tankov NT Kosarov DS, eds. New York: Plenum, pp 217-234, 1973.
216. Guyton, Arthur C. Textbook of Medical Physiology, 3rd edition, W.B. Saunders Company, Philadelphia, 1966.
217. Gydikov AA, Kosarov DS: Physiological characteristics of the tonic and phasic motor units in human muscles. In Motor Control. AA Gydikov, NT Tankov, DS Kosarov (eds) New York: Plenum, 1973.
218. Hansen KN, Bjerre-Knudsen J, Brodthagen U, Jordal R, Pauley PE: Muscle cell leakage due to long distance training. Eur J Appl Physiol 48: 177-178, 1982.
219. Hardt DE: Determining muscle forces in the leg during normal human walking - an application and evaluation of optimization methods. J Biomech Eng Trans ASME 100: 72-78, 1978.
220. Harrington WF: On the origin of the contractile force in skeletal muscle. Proc Natl Acad Sci USA 76: 5066-5070, 1979.
221. Harrison PJ, Jankowska E, Johannisson T: Shared reflex pathways of Group I afferents of different cat hind-limb muscles. J. Physio. (Lond.) 338: 113-127, 1983.
222. Hartshorne DJ, Dreizen P: Studies on the subunit composition of troponin. Cold Spring Harb symp Quant Biol 37: 225-234, 1973.
223. Hasan Z, Enoka RM, Stuart DG: The interface between biomechanics and neurophys. in the study of movement: some recent approaches. In Terjung RI (ed.): Exercise Sports Sci. Rev. Macmilan, Vol. 13, pp 169-234, 1985.
224. Hasan Z, Stuart DG: Mammalian muscle receptors. Handbook of the Spinal Cord, vol. 3, RA Davidoff, ed. New York: Marcel Dekker, pp 559-607, 1984.
225. Hasan ZA: A model of spindle afferent response to muscle stretch. J. Physiol. (Lond.) 338: 113-127, 1983.

226. Hatze H, Buys JD: Energy-optimal controls in the mammalian neuro-muscular system. Biol Cyb 27: 9-20, 1977.
227. Hatze H: A complete set of control equations for the human musculoskeletal system J Biomechanics 10: 799-805, 1977.
228. Hatze H: A mathematical model for the computational determination of parameter values of anthropomorphic segments. J Biomech 13: 833-843, 1980.
229. Hatze H: A model of skeletal muscle suitable for optimal motion problems. In Biomechanics IV, Basel: Karger, pp 417-422, 1974.
230. Hatze H: A new method for the simultaneous measurement of the moment of inertia, the damping coefficient and the location of the centre of mass of a body segment in situ. Europ J Appl Physiol 34: 217-226.
231. Hatze H: A telelogical explanation of Weber's law and the motor unit size law. Bull Math Biol 41: 407-425, 1979.
232. Hatze H: A theory of contraction and a mathematical model of striated muscle. J Theor Biol 40: 219-246, 1973.
233. Hatze H: The use of optimally regularized Fourier series for estimation higher-order of derivatives of noisy biomechanical data. J Biomech 14: 13-18, 1980.
234. Haut RC, Little RW: A constitutive equation for collagen fibres. J Biomech 5: 423-430, 1972.
235. Hayes KC, Hatze H: Passive visco-elastic properties of the structures spanninig the human elbow joint. Europ J Appl Physiol. 37: 265-274, 1977.
236. Hefner LL, Bowen TE: Elastic components of cat papillary muscle. Am J Physiol 212: 1221-1227, 1967.
236A. Hellebrandt F and Houtz S: Mechanism of Muscle Training in Man: Experimental Demonstration of Overload Principle. Physiol. Ther. Rev. 36: 371-376, 1956.
237. Hemami H, Farnsworth RL: Postural and gait stability of a planar five link biped by simulation. IEEE Trans Auto Contr AC-22: 452-458, 1977.
238. Hemami H, Golliday CL: The inverted pendulum and biped stability. Math Biosci 34: 95-110, 1977.
239. Hemami H, Weimer FC, Koozekanani SH: Some aspects of the inverted pendulum problem for modelling of locomotion systems. IEEE Trans Automat Contr AC-18: 658-661, 1973.
240. Henneman E, Mendell LM: Funct. organiz. of motoneuron pool and its inputs. Handbook of Physiology, sec. 1, vol. II, The Nervous system: Motor Control, Part I. VB Brooks, ed. Bethesda: Amer. Phys. Soc., pp 423, 1981.
241. Henneman E, Olson CB: The relations between structure and function in the design of skeletal muscle. J Neurophysiol 28: 581-598, 1965.
242. Henneman E, Somjen G, Carpenter DO: Excitability and inhibitability of motoneurones of different sizes. J Neurophysiol 28: 597-620, 1965.
243. Henneman E: Peripheral mechanisms involved in the control of muscle. In VB Mountcastle (ed.): Medical Physiology. St. Louis: Mosby, pp 1697-1716, 1968.
244. Heppenstall RB, Sapega AA, Scott R, Shnton D, : Rhe compartment syndrome. An experimental and clinical study of muscular energy metabol using phosphorus nuc. mag. reson. spectro. Clin Orthop 226: 138-155, 1988.
245. Herman, R., R. Wirta, S. Bampton, and F.R.Finley: Human Solutions for Locomotion: Single Limb Analysis. Neural Control of Locomotion, Edited by Herman, Grillner, Plenum Press, New York, pp 13-49, 1976.
246. Hikada RS, Staron RS, Hagerman FC, Sherman WM, Costill DL: Muscle fiber necrosis associated with human marathon runners. J Neurol Sci 59: 185-203, 1983.
247. Hill AV: A discussion of muscular contraction and relaxation: their physical and chemical basis. Proc Roy Soc (Lond.) B137: 40, 1950.
248. Hill AV: Muscular exercise. Nature 112: 77, 1923.
249. Hill AV: The abrupt transition from rest to activity in muscle. Proc Roy Soc (Biol.) 136: 399-420, 1949.
250. Hill AV: The heat of shortening and the dynamic constants of muscle. Proc. R. Soc. B126: 136-195.
251. Hill AV: The instantaneous elasticity of active muscle. Proc Roy Soc (Biol.) 141: 162, 1953.
252. Hill AV: The series elastic component of muscle. Proc Roy Soc (Biol.) 141: 161, 1953.
253. Hill DK: Tension due to interaction between the sliding filaments in resting striated muscle. The effect of stimulation. J Physiol 199: 637-684, 1968.

254. Hill L: A band length striation spacing and tension change on stretch of active muscle. J Physiol 266: 677-685, 1977.
255. Hodgkin AL, Horowicz P: Potassium contractures in single muscle fibres. J Physiol 153: 386-403, 1960.
256. Hoffer JA, Andreassen S: Regulation of soleus muscle stiffness in premammillary cats: intrinsic and reflex conponents. J. Neurophysiol. 45: 267-285, 1981.
257. Hollerbach JM, Flash T: Dynamic interations between limb segments during planar arm movement. Biol. Cybern. 44: 67-77, 1982.
258. Hollerbach JM,: Computers, brains and control of movement. Trends Neurosci. 5: 189-192, 1982.
259. Hollerbach JM: A recursive Lagrangian formulation of manipulator dynamics and a comparative study of dynamics fomulation complexity. IEEE Trans. Sys. Man. Cyb. 10: 730-736, 1980.
260. Hough T: Ergographic studies in muscular soreness. Am J Physiol: 76-92, 1902.
261. Houk J, Henneman E: Responses of Golgi tendon organs to active contractions of the soleus muscle of the cat. J. Neurophysiol. 30: 466-481, 1967.
262. Houk JC, Rymer WZ, Crago PE: Dependence of dynamic response of spindle receptors on muscle length and velocity. J. Neurophysiol. 46: 143-166, 1981.
263. Houk JC, Rymer WZ: Neural control of muscle len and tension. Handbook of Phys., sec. 1,vol. II, The Nervous Sys: Motor Control, Part 1. BN Brooks, ed. Bethesda: Amer Phys. Soc., pp 257-323, 1981.
264. Houk JC: Regulation of stiffness by skeletomotor reflexes. Ann. Rev. Physiol. 41: 99-114, 1979.
265. Howell JN, Chila AG, Ford G, David D, Gates T: An electromyographic study of elbow motion during postexercise muscle soreness. J Appl Physiol 58: 1713-1718, 1985.
266. Hudgson P, Field EJ: Regeneration of muscle. In Bourne GH (ed): The structure and Function of Muscle, ed 2. New York, Academic Press, Vol. 2, pp 312-363, 1973.
267. Hughston JC, Walsh WM, Puddu G: Patellar Sublaxation and Dislocation. Philadelphia. WB Saunders, Vol. 5, pp 1-191, 1984.
268. Humphrey DR, : Sep. cortical sys for control of joint movement and joint stiffness: Reciprocal activation and coactivation of antagonist muscles. In Motor Control Mech. Hlth and Dis.: New York: Raven, 347-372, 1983.
269. Huxley AF, Peachey LD: Local activation of crab muscle. J Cell Biol 23: 107a, 1964.
270. Huxley AF, Simmons RM: Proposed mechanism of force generation in striated muscle. Nature 233: 533-538, 1971.
271. Huxley AF, Taylor RE: Local activation of striated muscle fibres. J Physiol 144: 425-441, 1958.
272. Huxley AF: Muscle structure and theories of contraction. Progr Biophy 7: 255, 1957.
273. Huxley AF: The double array of filaments in cross-striated muscle. J Biophys Biochem Cyt 3: 631, 1957.
274. Huxley HE: General discussion. In Sugi H, Pollack GH, (eds): Cross-Bridge Mechanism in Muscle contracting. Baltimore: University Park Press, pp 638, 1979.
275. Huxley HE: The mechanism of muscular contraction. Science 164: 1356-1366, 1969.
276. Huxley, AF: Muscular contraction. (Review Lecture.) J. Physiol.(Lond.) 243: 1-43, 1974.
277. Inesi G: Active transport of calcium ion in sarcoplasmic membranes. Ann Rev Biophys Bioeng 1: 191-210, 1972.
278. Ishikawa H, Sawada H, Yamada E: Surface and internal morphology of skeletal muscle. In Handbook of Physiology: Section 10. Skeletal Muscle, Bethesda, MD, American Physiological Society, pp 1-22, 1983.
279. Jankowska E, McCrea D, Mackel R: Pattern of "nonreciprocal" inhibition of motoneurones by impulses in group Ia muscle spindle afferents in the cat. J. Physiol. (Lond.) 316: 393-409, 1981.
280. Jankowska E, McCrea DA: Shared reflex pathways from Ib tendon organ afferents and Ia muscle spindle afferents in the cat. J. Physiol. (Lond.) 338: 99-111, 1983.
281. Jankowska E, Odutola: Crossed and uncrossed synaptic actions on motor neurons of back muscles in the cat. Brain Res. 194: 65-78, 1980.
282. Jewell BR, Wilkie DR: An analysis of the mechanical components in frog's striated muscle. J Physiol 143: 515-540, 1958.
283. Jewell BR, Wilkie DR: The mechanical properties of relaxing muscle. J Physiol 152: 30-47, 1960.
284. Jobsis FF, O'Connor MJ: Calcium release and reabsorption in the sartorius muscle of the toad. Biochem Biophys Res Com 25: 246-252, 1966.
285. Jones DA, Jackson MJ, McPhail G, Edards RHT: Experimental mouse muscle damage: the importance of external calcium. Clin Sci 66: 317-322, 1984.

286. Jones DA, Newham DJ, Clarkson PM: Skeletal muscle stiffness and pain following eccentric exercise of the elbow flexors. Pain 30: 233-242, 1987.
287. Jones DA, Newham DJ, Round JM, Tolfree SEJ: Experimental human muscle damage: morphological changes in relation to other indices of damage. J Physiol 375: 435-558, 1986.
288. Jorgenson K: Force-velocity relationship in human elbow flexors and extensors. In Komi PV (ed.): International Series on Biomechanics: Vol. IA. Biomechanics VA. Baltimore: University Park Press, pp. 145-151, 1976.
289. Joyce GC, Rack PMH, Westbury DR: The mechanical properties of cat soleus muscle during controlled lengthening and shortening movements. J Physiol 204: 461-474, 1969.
290. Joyce GC, Rack PMH: Isotonic lengthening and shortening movements of cat soleus muscle. J Physiol 204: 461-474, 1969.
291. Julien FJ: The effect of calcium on the force-velocity relation of briefly glycerinated frog muscle fibres. J Physiol 218: 117, 1971.
292. Kandel ER,Schwartz JH: Principles of Neural Science. New York: Elsevier/North-Holland, 1981.
293. Katz AM : Physiology of the Heart. New York: Raven Press, pp 132-136, 1977.
294. Katz B: The relation between force and speed in muscular contraction. J Physiol 96: 45-64, 1939.
295. Katz EP, Wachtel EJ, Maroudas A: Extrafibrillar proteoglycans osmotically regulate the molecular packing of collagen in cartilage. Biochim Biophys Acta 882: 136-139, 1986.
296. Kelly DL: Kinesiology. New Jersey: Prentice Hall, 1971.
297. Kelso JAS, Tuller B: A dynamical basis for action systems. Handbook of Cognitive Neuroscience. MS Gazzaniga, ed. New York: Plenum, pp 321-356, 1984.
298. Kelso NAS, Holt KG: Exploring a vibratory systems analysis of human movement production. J. Neurophysiol. 43: 1183-1196, 1980.
299. King SW, Statland BE, Savory J: The effect of a short burst of exercise on activity values of enzymes in sera of healthy young men. Clin Chim Acta 72: 211-218, 1976.
300. Klausen K, Knuttgen H: Effect of training on oxygen consumption in negative muscular work. Acto Physiol Scand 83: 319-323, 1971.
301. Kniffke K-D, Mense S, Schmidt RF: Muscle receptors with fine afferent fibres which may evoke circulatory reflexes. Circ. Res. (Suppl. I) 48: 25-31, 1981.
302. Knutsson E: Analysis of spastic paresis. In Proceedings of the Tenth International Congress of the World Confederation for Physical Therapy. Sydney, pp 629-633, 1987.
303. Knuttgen HG, Kraemer WJ: Terminology and measurement in exercise performance. J Appl Sport Res 1: 1-10, 1987.
304. Knuttgen HG: Human performance in high-intensity exercise with concentric and eccentric muscle contractions. Int J Sports Med (Suppl) 7: 6-9, 1986.
305. Komi PV, Buskirk ER: Measurement of eccentric and concentric conditioning on tension and electrical activity in human muscle. Ergonomics 15: 417-434, 1972.
306. Komi PV: Measurement of the force-velocity relationship in human muxcle under concentric and eccentric contractions. In Biomechanics III. A Cerquiglini, A Venerando. Basel: Karger, pp 224-229, 1973.
307. Komi PV: Phys. and biomechanical correlates of muscle function: effects of muscle structure and stretch shortening cycle on force and speed. Exercise and Sports Sci Rev. Collamore Press, Vol. 12, pp 81-121, 1984.
308. Komi PV: Relationship between muscle tension, EMG, and velocity of contraction under concentric and eccentric work. In Desmedt JE (ed): New Developments in Emg and Neurophysiology. Vol. 1, pp 596-606, 1973.
309. Kovanen V, Suominen H, Heikkinen E: Mechanical properties of fast and slow skeletal muscle with special reference to collagen and endurance training. J Biomech 17: 725-735.
310. Kovanen V. Suominen H. Heikkinen E: Connective tissue of "fast" and "slow" skeletal muscle in rats-effects of endurance training. Acta Physiol Scand 108: 173-180, 1980.
311. Kuffler SW, Nicholls JG: From neuron to brain. Sinaur Assoc. Sunderland, MA, pp 183-188, 1976.
312. Kumazawa T, Mizumura K: Thin-fibre receptors responding to mechanical, chemical and thermal stimulation in skeletal muscle of the dog. J Physiol 273: 179-194, 1977.
313. Lamarre Y, Spidaliere G, Busby L, Lund JP: Programming of initiation and execution of ballistic arm movements in the monkey. Prog. Brain Res. 54: 157-169, 1980.

314. Lammert O, Jorgensen F, Einer-Jensen N: (AMG) I: method for measuring mechanical vibrations from isometrically contracted muscles. In PV Komi (ed): Biomechanics V-A. Baltimore: University Park Press, pp 152-158, 1976.
315. Lance JW, McLeod JG: A Physiological Approach to Clinical Neurology, 3rd ed. London: Butterworths, 1981.
316. Lane RJM, Turnbull DM, Welch JL, Walton SJ: A double-blind, placebo-controlled, crossover study of verapamil in exertional muscle pain. Muscle Nerve 9: 635-641, 1986.
317. Lestienne F, Polit A, Bizzi E: Functional organization of the motor process underlying the transition from movement to posture. Brain Res. 230: 121-132, 1981.
318. Lestienne F: Effects of inertial load and velocity on the braking process of voluntary limb movements. Exp. Brain Res. 35: 407-418, 1979.
319. Levine WS, Zajac FE, Belzer MR, Zomlefer MR: Ankle controls that produce a maximum vertical jump when other joints are locked. IEEE Trans. Automat. Control AC-28: 1008-1016, 1983.
320. Littre, E., On the Nature of Bones. (translation) Vol. IX of Oeuvres Completes d'Hippocrate. Paris, 1839-61.
321. Loeb GE: The control and responses of mammalian muscle spindles during normally executed motor tasks. Exercise and Sport Sci. Rev. 12: 157-204, 1984.
322. Loeb GE: The control and responses of mammalian muscle spindles during normally executed motor tasks. In Terjung RL (ed): Exercise and Sport Sciences Reviews. Lexington, MA: Collamore Press, Vol 12, pp 157, 1984.
323. Loeb GE: Finding common ground between robotics and physiology (Letter to the editor) Trends Neurosci. 6: 203-204, 1983.
324. Loewy AG: A theory of covalent bonding in muscle contraction. J Theor Biol 20: 164, 1968.
325. Lowey S, Slayter HS, Weeds AG, Baker H: Substructure of the myosin molecule. J Mol Biol, 42: 1-29, 1969.
326. Luhtanen P, Komi PV: Mechanical power and segmental contribution to force impulses in long jump take-off. Europ J Appl Physiol. 41: 267-274, 1979.
327. Luhtanen P, Komi PV: Segmental contribution forces in vertical jump. Eur. J. Appl. Physiol. 38: 181-188, 1978.
328. Lundberg A: Multisensory control of spinal reflex pathways. Prog. Brain Res. 50: 11-28, 1979.
329. MacDougall JD, Sale DG, Alway SE, Sutton JR: Muscle fiber number in biceps brachii in bodybuilders and control subjects. J Appl Physiol 57: 1399-1403, 1984.
330. MacDougall JD, Sale DG, Elder GCB, Sutton JR: Muscle ultra-structural characteristics of elite power lifters and bodybuilders. Eur J Appl Physiol 48: 117-126, 1982.
331. Margaria R: Pos. and neg. work performances and efficiencies in human locomotion. Environmental Effects on Work Performance. Edmonton, Can. Assoc. of Sports Sci., pp 215-228, 1972.
332. Marsden CD, Meadows JC, Merton PA: Isolated single motor units in human muscle and their rate of discharge during maximal voluntary effort. J Physiol. 217: 12-13, 1971.
333. Marsden CD, Merton PA, Morton HB, Adam JER, Hallet M: Automatic and voluntary responses to muscle stretch in man. Cerbral Motor Control in Man: Long Loop Mechanisms. JE Desmedt ed. Basel: Karger, pp 167-177, 1978.
334. Marsden CD, Merton PA, Morton HB: Anticipatroy postural responses in the human subject. J. Physiol. (Lond.) 275: 47-48P, 1977.
335. Marsden CD, Obeso JA, Rothwell JC: The function of the antagonist muscle during fast limb movements in man. J. Physiol. (Lond.) 335: 1-13, 1983.
336. Martin JP: A short essay on posture and movement. J. Neurol. Neurosurg. Psychiat. 40: 25-29, 1977.
337. Matthews PBC: Mammalian Muscle Receptors and Their Central Actions. London: Arnold, 1972.
338. Matthews PBC: Muscle spindles: Their messages and their fusimotor supply. Handbook of Physiology, Sec. 1, Vol. II, The Nervous System: Motor Control, Part 1. Bethesda: Amer Phys Soc., pp 189-228, 1981.
339. Matthews PBC: Where does Sherrington's "muscular sense" originate? Muscles, joints, corollary discharges? Ann. Rev. Neurosci. 5: 189-218, 1982.
340. McCloskey DI: Corollary discharges: motor commands and perception. Handbook of Physiology, Sec. 1, Vol. II, The Nervous System: Motor control, Part 2. Bethesda: Amer Phys Soc, pp 1415-1447, 1981.
341. McCloskey DI: Kinesthetic sensibility. Physiol. Rev. 58: 763-820, 1978.

342. McCrorey HL, Gale HH, Alpert NR: Mechanical properties of cat tenuissimus muscle. Am J Physiol, 210: 114-120, 1966.
343. McCully KK, Faulkner JA: Injury to skeletal muscle fibers of mice following lengthening contractions. J Appl Physiol 59: 119-126, 1985.
344. McDougall W: On the structure of cross-striated muscle and a suggestion as to the nature of its contraction. J Anat and Physiol. 31: 410, 1897.
344A. McQueen I: Recent Advances in the Technique of progressive resistance exercise. Brit. Med. J. 2: 328-338,1954.
345. Meigs EB: The structure of the element of cross-striated muscle and the changes of form which it undergoes during contraction. Z F Allg Physiol. 8: 81, 1908.
346. Mendell LM, Henneman E: Terminals of single Ia fibers: location, density and distribution within a pool of 300 homonymous motoneurons. J Neurophysiol. 34: 171-187, 1971.
347. Meriam, J.L. Dynamics, Wiley, New York, 1966.
348. Meriam, J.L., Statics, Wiley, New York, 1966.
349. Merton PA: Speculations on the servo control of movement. In The Spinal Cord. GE Wolstenholme, ed. London: Churchill, pp 247-255, 1953.
350. Merton PA: The silent period in a muscle of the human hand. J. Physiol. (Lond.) 115: 183-198, 1951.
351. Miller DI, East DJ: Kinematic and kinetic correlates of vertical jumping in women. Biomechanics V-B. PV Komi, ed. Baltimore: University Park Press, pp 65-72, 1976.
352. Milner-Brown HS, Stein RB, Yemm R: Changes in firing rate of human motor units during linearly changing voluntary contractions. J Physiol. 230: 359-370, 1973.
353. Milner-Brown HS, Stein RB, Yemm R: The orderly recruitment of human motor units during voluntary isometric contractions. 230: 359-370, 1973.
354. Milsum JH: Control system aspects of muscular coordination. In Medicine and Sport, Vol 6: Biomechanics II, Basel: Krager, pp 62-71, 1971.
355. Mochon S, McMahon TA: Ballistic walking. J. Biomech. 13: 49-57, 1980.
356. Mommaerts WFHM: Muscular contraction. Physiol Rev. 49: 427-508, 1969.
357. Mommaerts WFHM: Stoichiometric and dynamic implications of the participation of actin and ATP in the contraction process. Biochem Bioophys Acta 7: 477, 1951.
358. Morasso P,: Spatial control of arm movements. Exp Brain Res. 26: 59-72, 1976.
359. Morasso P: Three dimensional arm trajectories. Biol. Cybern. 48: 187-194, 1983.
360. Morecki A, Busko ZA, Fidelus K, Kedzior K, Olszewski J: Biomechanical modelling of dynamic properties of human motion. Proceed. IVth World Congress Theory Machines and Mechanisms, Newcastle upon Tyne, Sept 1975.
361. Morgan DL: Separation of active and passive components of short-range stiffness of muscle. Am J Physiol. 232: C45-C49, 1977.
362. Nashner LM, Woollacott M, Thuma G: Organization of rapid responses to postural and locomotor-like perturbations of standing man.
363. Nashner LM: Adaptations of human movement to altered environments. Trends Neurosci. 5: 358-361, 1982.
364. Nashner LM: Adapting reflexes controlling the human posture.
365. Nashner LM: Fixed patterns of rapid postural responses among leg muscles during stance. Exp. Brain Res. 30: 1-24, 1977
366. Natori R: Effects of Na and Ca ions on the excitability of isolated myofibrils. In: Molecular Biology of Muscular Contraction, Ebashi S (eds): Amsterdam, Elsevier, 1965.
367. Needham, DM: Machina Carnis. Cambridge: Cambridge University Press, 1971.
368. Nelson WL: Physical principles for economies of skilled movements. Biol. Cybern. 46: 135-d148, 1983.
369. Newell KM, Houk JC: Speed and accuracy of compensatory responses to limb disturbances. J. Exp. Psychol. 9: 58-74, 1983.
370. Newham DJ, Mills KR, Quigley BM, Edwards RHT: Pain and fatigue after concentric and eccentric muscle contractions. Clin Sci 64: 55-62, 1983.
371. Newham DJ, Clarkson PM: Repeated high-force eccentric exercise: effects on muscle pain and damage. J Appl Physiol 63: 1381-1386, 1987.

PRECEDING PAGE BLANK NOT FILMED

432. Stein RB and Milner-Brown HS: Contractile and Electrical Properties of Normal and Modified Human Motor Units: Control of Posture and Locomotion. New York: Plenum Press, pp 73-861973.

432A. Steinhause AH: Strenth of Morpurgo to Muller - A half century of Research. J. Assoc. Physiol. Mental Rehab. 9: 147-150, 1955.

433. Stephens JA, Reinking RM, Stuart DG: Tendon organs of cat medial gastrocnemius: Responses to active and passive forces as a function of muscle length. J. Neurophysiol. 38: 1217-1231, 1975.

434. Stetson RH, Bouman HD: The coordination of simple skilled movements. Arch. Neerl. Physiol.Physiol Homme Anim. 20: 177-254, 1935.

435. Stevens JC, Dickinson V, Jones NB: Mechanical properties of human skeletal muscle from in vitro studies of biopsies. Med & Biol Eng & Comput 18: 1-9, 1980.

436. Stokes M, Toung A: The contribution of reflex inhibition to arthrogenous muscle weakness. Clin Sci 67: 7-14, 1984.

437. Struppler A, Burg D, Erbel F: The unloading reflex under normal and pathological conditions in man. New Developments in Electromyography and Clinical Neurophysiology. Basel: Karger, pp 603-617, 1973.

438. Stuart DG, Enoka RM: Motor neurons, motor units and the size principle. The Clinical Neurosciences, Sec. 6, Neurobiology. RN Rosenburg, ed. New York: Churchill Livingstone, pp 471-517, 1983.

439. Sugi H: Tension changes during and after stretch in frog muscle fibres. J Physiol. 225: 237-253, 1972.

440. Szent-Gyorgyi A: Chemical Physiology of contraction in body and heart muscle. New York: Academic Press, 1953.

441. Talag TS: Residual muscular soreness as influenced by concentric, eccentric and static contractions. Res Quarterly 44: 458-469, 1973.

442. Terzuolo CA, Soechting JF, Ranish NA: Studies on the control of some simple motor tasks. V. Changes in motor output following dorsal root section in squirrel monkey. Brain Res. 70: 521-526, 1974.

443. Tesch PA, Larson L: Muscle hypertrophy in bodybuilders. Eur J Appl Physiol. 49: 301-306, 1982.

444. Thames MD, Teichholz LE, Podolsky RJ: Ionic strength and the contraction kinetics of skinned muscle fibres. J Gen Physiol. 63: 509-530, 1974.

445. Thom R: Structural stability and morphogenesis. Benjamin, Massachusetts, 1975.

446. Tidball JG, Daniel TL: Elastic energy storage in rigored skeletal muscle cells under physiological loading conditions. Amer J Physiol 250: R56-R64, 1986.

447. Tidus PM, Ianuzzo Cd: Effects of intensity and duration of muscular exercise on delated soreness and serum enzyme activities. Med Sci Sprots Exerc 15: 461-465, 1983.

448. Tomovic R, Bellman R: A systems approach to muscle control. Math Biosci 8: 265-277, 1970.

449. Travell, Janet G. and David G. Simons: Myofascial Pain and Dysfunction. Baltimore: Williams & Wilkins, 1983.

450. Triolo R, Rovinson D, Gardner E, Betz R: The eccentric strength of electrically stimulated paralyzed muscle. IEEE Transbiomed Eng. pp 651-652, 1987.

451. Vallbo A, Hagbarth K, Torebjork HE, Wallin BG: Somatosensory, proprioceptive, and sympathetic actibity in human peripheral nerves. Physiol. Rev. 59: 919-957, 1979.

452. Vallbo A: Basic patterns of muscle spindle discharge in man. Muscle Receptors and Movement. A Taylor and A Prochazka, eds. London: MacMillan, pp 263-275, 1981.

453. Vandenburgh HH, Kaufman S: In vitro skeletal muscle hypertrophy and Na pump activity. In Pette D (ed): Plasticity of Muscle. Berlin: Walter de Gruyter, pp 493-506, 1980.

454. Varga L: The relation of temperature and muscular contraction. Hung Acta Physiol 1: 1, 1946

455. Vincken MH: Control of limb stiffness. Ph.D Thesis, Rijksuniversiteit, Utrecht, 1983.

456. Vukobratovic M, Juricic D: Contribution to the synthesis of biped gait. IEEE Trans Biomed Eng BME-16, 1969.

457. Wachholder K, Altenburger H: Beitrage zur Physiolgie der willkurlichen Bewegung. Mitteilung X, Einzelbewegungen. Pflugers Arch. Physiol. 214: 642-661, 1926.

458. Wadman WJ, Denier van der Gon JJ, Derksen RJA: Muscle activation patterns for fast goal-directed arm movements. J. Human Movt. Stud. 6: 19-37, 1980.

459. Wadman WJ, Denier van der Gon JJ, Geuze RH, Mol CR: Control of fast goal-directed arm movements. J Human Movt Stud 5: 3-17, 1979.

460. Walker SM: Potentiation and hysteresis induced by stretch and subsequent release of papillary muscle of the dog. Am J Physiol 198: 519-522, 1960.

461. Wallenstein R, Eklund B: Intramuscular pressures and muscle metabolism after short term and long-term exercise. Int J Sports Med 4: 231-235, 1983.
462. Walsh GE: Physiology of the nervous system. London: Longmans, 1964.
463. Wand P, Prochackz A, Sontag K-H: Neuromuscular responses to gait perturbations in freely moving cats. Exp Brain Res. 38: 109-114, 1980.
464. Warholl MJ, Siegel AJ, Evans WJ, Silverman LM: Skeletal muscle injury and repair in marathon runners after competition. Am J Pathol 118: 331-339, 1985.
465. Waters P, Strick PL: Influence of strategy on muscle activity during ballistic movements. Brain Res. 207: 189-194, 1981.
466. Watt DGD, Stauffer EK, Taylor A, Reinking RM, Stuart DG: Analysis of muscle receptor connections by spike-triggered averaging. I. Spindle primary and tendon organ afferents. J. Neurophysiol. 39: 1375-1392, 1976.
467. Wendt IR, Gibbs CL: Energy production of rat extensor digitorum longus muscle. Amer J Physiol 224: 1081-1086, 1973.
468. Wetzel MC, Stuart DG: Activation and co-ordination of vertebrate locomotion. Mechanics and Energentics of Animal Locomotion. R Alexander and G Goldspink, eds. London: Chapman Hall, pp 115-152, 1977.
469. Wetzel MC, Stuart DG: Ensemble characteristics of cat locomotion and its neural control. Prog. Neurobiol. 7: 1-98, 1976.
470. Wilke DR: The relation between force and velocity in human muscle. J Physiol 110: 249-280, 1950.
471. Wilkie DR: Measurement of the series elastic component at various times during a single muscle twitch. J Physiol 134: 527-530, 1956.
472. Wilkie DR: The mechanical properties of muscle. Brit Med Bull 12: 177-182, 1956.
473. Williams WJ, Edwin AI: An electronic muscle simulator for demonstration and neuromuscular systems modelling. Med & Biol Eng 8: 521-524, 1970.
474. Wilson LG: William Croone's theory of muscular contraction. Notes and records of the Royal Society of London 16: 158, 1961.
475. Woledge RC: Heat production and chem. change in muscle. 39-74, 1971.
476. Woledge RC: The thermoelastic effect of change of tension in active muscle. J Physiol 155: 187-208, 1961.
477. Woods HJ: The contribution of entropy to the elastic properties of keratin, myosin and some other high polymers. J Coll Sci 1: 407, 1946.
478. Wrogemann K, Pena SDJ: Mitochondrial calcium overload: a general mechanism for cell-necrosis in muscle disease. Lancet 1: 672-674, 1976.
479. Yamada H: Strength of biological materials. Baltimore: Williams & Wilkins, 1970.
480. Yoshimura T, Tsujihata M, Satoh A, Mori M, Hasama R, Kinoshita N, Takashima H Nagataki S: Ultrastructural study of the effect of calcium ionophore A23187, on rat muscle. Acta Neuropathol (Berl) 69: 184-192, 1986.

N94-28363

52-52
207611
P. 12

Eccentric Exercise Testing and Training

Priscilla M. Clarkson, Ph.D.
Department of Exercise Science
University of Massachusetts
Amherst, Massachusetts

Introduction

Some researchers and practitioners have touted the benefits of including eccentric exercise in strength training programs. However, others have challenged its use because they believe that eccentric actions are dangerous and lead to injuries. Much of the controversy may be based on a lack of understanding of the physiology of eccentric actions. This review will present data concerning eccentric exercise in strength training, the physiological characteristics of eccentric exercise, and the possible stimulus for strength development. Also a discussion of strength needs for extended exposure to microgravity will be presented.

Not only is the use of eccentric exercise controversial, but the name itself is fraught with problems. The correct pronunciation is with a hard "c" so that the word sounds like *ekscentric* [6]. The confusion in pronunciation may have been prevented if the spelling that Asmussen used in 1953, *excentric*, had been adopted [30]. Another problem concerns the expressions used to describe eccentric exercise. Commonly used expressions are *negatives*, *eccentric contractions*, *lengthening contractions*, *resisted muscle lengthenings*, *muscle lengthening actions*, and *eccentric actions*. Some of these terms are cumbersome (i.e., resisted muscle lengthenings), one is slang (negatives), and another is an oxymoron (lengthening contractions). Only *eccentric action* is appropriate and adoption of this term has been recommended by Cavanagh [5].

Despite the controversy that surrounds eccentric exercise, it is important to note that these types of actions play an integral role in normal daily activities. Eccentric actions are used during most forms of movement; for example, in walking when the foot touches the ground and the center of mass is decelerated and in lowering objects, such as placing a bag of groceries in the car.

Training Studies

The effects of training with eccentric actions versus other types of actions has received a moderate amount of attention over the last 25 years [1, 7, 27]. Several factors need to be considered when examining these training studies. First, there is a problem in equating workloads. Eccentric actions can produce more tension than concentric or isometric actions. Thus, the workload may be set relative to the maximal tension that could be generated for each modality (i.e., concentric, eccentric, isometric), or the workload may be set at the same value for each of the modalities. Second, the method of strength assessment differs among studies. For example, some investigators measured isometric strength pre and posttraining regardless of the mode of actions used in the training exercise (i.e., eccentric, concentric, isometric), while others measured the same type of actions that were used in the training. Studies also differ on the muscle groups examined, as well as the duration and the intensity of the training regimens. However, despite the differences among studies, the results of the investigations are markedly consistent.

PRECEDING PAGE BLANK NOT FILMED

PAGE 98 INTENTIONALLY BLANK

Eccentric vs. Concentric

Mannheimer [21] investigated strength gains in 26 patients at a center for neuropsychiatric disorders. Subjects participated in a 1-month training program that consisted of either eccentric-only or concentric-only actions of the elbow flexors. The exercises were performed on a specially designed variable muscle tester with shock absorbers to provide resistance. Pre- and poststrength testing was specific to the exercise performed. Although both training regimens produced strength gain, there was no significant difference between them.

In a 6-week training program, six medical students performed a specially devised training program to include various exercises [15]. Some of these exercises were performed using concentric actions and others performed using eccentric actions. No difference in dynamic strength gain could be ascertained between the two exercise regimens.

Johnson [13] and Johnson *et al.* [14] examined strength gains that were achieved by training one limb with eccentric actions and the other with concentric actions. In the 1972 study [13], nine subjects trained for 8 weeks at 80 percent of concentric one repetition maximum (1RM). There was no significant difference in strength gain between the training regimens. In the 1976 study [14] the workloads were reset so that 80 percent 1RM was used for the concentric regimen and 120 percent 1RM (concentric 1RM) was used for the eccentric regimen. Although strength gains were found for arm curls, arm presses, knee flexions and extensions, there was no significant difference in strength gain between the exercise regimens.

Only Komi and Buskirk [19] have found that eccentric exercise produced greater strength gain than concentric exercise. In this study, three groups of subjects (n=10 per group) trained for 7 weeks on an electric dynamometer using the forearm flexor muscles. The groups performed either maximal eccentric, concentric, or isometric actions. The eccentric training regimen resulted in the largest strength gains for each of the testing modalities (i.e., concentric, isometric and eccentric). The major difference between the Komi and Buskirk [19] study and previously described studies is that Komi and Buskirk used maximal eccentric actions in the training program.

In a recent study, Jones and Rutherford [16] examined isometric strength of the knee extensors and muscle cross sectional area of the mid thigh before and after 12 weeks of eccentric exercise with one leg and concentric exercise with the other leg. The work load was set at 80 percent 1RM (concentric or eccentric) for the six subjects tested. The forces generated during the eccentric training were 45 percent higher than forces generated during the concentric training. The results showed that both eccentric and concentric training increased isometric strength (11 percent and 15 percent, respectively) but there was no significant difference between the two training regimens. There was a similar increase (5 percent) in muscle cross sectional area (determined from computerized tomography scans) for the eccentric and concentric training. Also found was a small, but significant increase in the radiological density of the muscle for both exercise regimens. This increase may result from an increased packing of the myofibrils, an increase in connective tissue, or a decrease in fat content of the muscle [16].

Eccentric vs. Isometric

Bonde Petersen [3] had subjects perform either ten-maximal isometric actions or ten-maximal eccentric actions (elbow flexors) per session for 36 sessions over 2 months. The isometric

exercise training regimen produced a small, but not significant, increase in isometric strength, and the eccentric actions had no effect on isometric strength.

The other study to compare the effects of isometric and eccentric training regimens did find strength gains and reported no difference between the training regimens [20]. Laycoe and Martiniuk [20] had two groups of 15 subjects perform either three-maximal eccentric actions or three-maximal isometric actions of the knee extensors for 6 weeks. Eccentric and isometric trained groups showed a significant increase in isometric strength (17.0 percent and 17.4 percent, respectively). The eccentric group, however, improved 41 percent in eccentric strength. Eccentric strength was not measured for the isometric trained group.

Hybrid Exercise Programs

Hakkinen and Komi [11] examined the effect of training with only concentric exercise compared with combinations of concentric and eccentric exercise. Three groups were studied: Group 1 performed concentric squatting exercise, Group 2 performed the same concentric exercise with some additional eccentric, and Group 3 performed predominantly eccentric squatting actions plus some concentric actions. Squatting ability after 12 weeks of training showed greater improvement for the groups that performed the combination exercise modalities compared with the group that performed the concentric training. There was no difference in improvement between Groups 1 and 2.

Slobodyan [29] and Pletnev [26] also examined the effectiveness of hybrid training programs in strength gain. Slobodyan studied three groups who trained with a series of standard exercises over 3 months. The groups differed in the following manner: Group 1 performed 100 percent concentric exercise; Group 2 performed 75 percent concentric exercise, 15 percent eccentric exercise, and 10 percent isometric exercise; and Group 3 performed 50 percent concentric, 30 percent eccentric, and 20 percent isometric exercise. On all tests, Group 3 showed the greatest strength gains. Pletnev [26] compared strength gains in four groups after 3 months of training: one trained isometrically, one eccentrically, one concentrically, and one used a combination of the three modalities. Overall the group performing the hybrid program demonstrated the greatest gains in strength.

Characteristics of Eccentric Exercise

Lower Oxygen Cost

In laboratory testing of eccentric and concentric muscle actions, subjects will consistently report that exercises with eccentric actions are easier to perform than exercises with concentric actions. Rasch, in his review of eccentric exercise [27], stated that when using oxygen consumption as the criterion measure, "muscles are able to produce tension three to nine times more cheaply when they are doing negative work than when they are doing positive work."

Several studies have shown that the rate of oxygen uptake by subjects who were performing concentric only exercise was greater than oxygen uptake for subjects performing eccentric exercise [27, 30]. Early studies have suggested that the lower oxygen cost of eccentric exercise was due to a smaller number of muscle fibers being recruited during eccentric actions. However, Bigland-Ritchie and Woods [2] found that motor unit recruitment could only partially explain the difference in oxygen cost between concentric and eccentric actions.

Bigland-Ritchie and Wood [2] examined integrated EMG (IEMG) of the vastus lateralis muscles and oxygen uptake during concentric and eccentric cycle ergometry exercise. The pedaling rate was set at 50 rev/min with resistances ranging from 2.5 to 15 kg. Work rates did not exceed 75 percent of the subjects' $\dot{V}O_{2max}$. When the slope of IEMG/work-rate for concentric exercise was expressed as a ratio of the slope of IEMG/work-rate for eccentric exercise the value was found to be 1.96. However, a considerably larger value (6.34) was obtained when the slope of $\dot{V}O_2$/work-rate for concentric exercise was expressed as a ratio of the slope of $\dot{V}O_2$/work-rate for eccentric exercise. These data demonstrate that only part of the lower oxygen cost during eccentric exercise could be attributed to less fiber activity. Experiments with isolated muscles that were electrically stimulated have supported this conclusion [30].

Greater Tension

It is common knowledge among those involved in strength testing and training, that individuals can lower more weight than they can lift. Thus, during an eccentric action the muscle can produce more tension than during a concentric action. The force velocity relationship maintains that as the velocity of contraction decreases, the force increases, such that during *negative* velocity contractions (i.e., eccentric actions) the force continues to increase until a certain point is reached.

Stauber in his excellent review of eccentric exercise [30] provides one explanation for the ability of muscle to produce greater tension during eccentric muscle actions. In a resting state, actin and myosin are not bound and the myosin head is in a state of activation due to the hydrolysis of ATP. The myosin head in this state is referred to as *preenergized*. During concentric exercise, myosin heads bind to actin, cross-bridges are formed, and the cycling of these cross-bridges produces tension and shortening. After each cycle of cross-bridge formation, the myosin head is detached from the actin and is recharged with another ATP. In this manner the "potential energy stored in the preenergized myosin becomes transformed into the mechanical events of cross-bridge action (tension or shortening)" [30]. During eccentric actions, the cross-bridge is forcibly pulled backward before energy can be transformed. Stauber [30] states "Each of these attachment-separation reactions produces a recorded tension (resistance to stretch) by the muscle but with no apparent energy consumption because the cross-bridge has not cycled but continues to remain in the high-energy form." To a given point, the faster the velocity during eccentric actions, the less likely a cross-bridge will cycle and the more tension can be produced. Likewise as the velocity decreases, more cross-bridges can cycle and the tension is lower.

Muscle Damage

Well known to athletes, coaches, and exercise scientists is the muscle soreness that develops 24 to 48 hours after exercises that have a large eccentric component [9]. Soreness is considered to indicate that some damage has been incurred in the exercised muscle [9]. In recent years, several studies have appeared in the literature with titles that include such phrases as *exercise-induced muscle damage*. These studies have used eccentric muscle actions as a model to study muscle damage and recovery.

Unfortunately (and undeservedly), these titles may have given eccentric exercise a *bad name*. The damage that is induced by these exercises is completely repairable in a short amount of time. In addition to being repairable, the damage also results in an adaptation in the muscle making it more resistant to damage from subsequent strenuous exercise. In other words, the muscle is designed to

deal with effects of eccentric exercise. A more comprehensive discussion of muscle damage and eccentric exercise can be found in two recent review papers [9, 30].

Friden [10] and Newham *et al.* [25] have documented ultrastructural changes in muscle biopsy samples taken from subjects who had performed strenuous eccentric exercise. Both studies noted disruption of the normal myofibrillar arrangement. In normal muscle fibers the Z lines of adjacent myofibrils are in register with one another. After eccentric exercise the Z lines appear to lose their integrity, such that the lines broaden and show a *streaming* pattern. In biopsy samples taken 6 to 7 days after eccentric exercise, the fibers were repaired and appeared essentially normal [9].

Many studies have documented an increase in the appearance of muscle proteins, particularly creatine kinase (CK), in the blood after eccentric exercise [8, 9, 23, 24]. Increased levels of these proteins in the blood has been taken as evidence of muscle fiber membrane disruption. Because CK is a large molecular weight 12 protein, the membrane must be disrupted for release to occur. Also the size of the protein necessitates its entry into the lymph (rather than capillaries) prior to entering the blood via the thoracic duct. Figure 1 shows the pattern of increase in CK activity in blood after high-force eccentric exercise of the forearm flexor muscles. Noteworthy is the 48-hour delay in the increase of CK. Other studies, where less strenuous forms of eccentric actions have been used (Figure 1), have reported a smaller increase in CK in the blood and a shorter delay, generally about 6 to 12 hours after exercise [4, 9]. This 6 to 12-hour delay may be related to the time it takes for CK to travel through the lymph before entering the blood. Presently there is no clear explanation for the long delay of 48 hours. Either there is a delayed release from the muscle or there is an inability of this large protein to enter the lymph or be transported by the lymph. High-force eccentric actions can produce more muscle swelling than less strenuous eccentric exercise (downhill running). Edema around the muscle may retard the entry of large molecular weight proteins into the lymph or retard the transport.

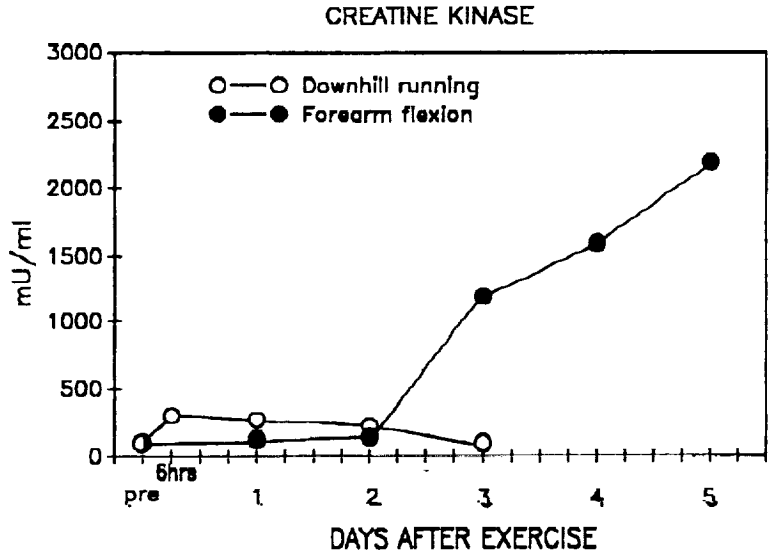


Figure 1. *Creatine kinase activity in the blood before and 5 days after a downhill running exercise (open circles) and high-force eccentric forearm flexion exercise (closed circles). Data modified from Byrnes et al. [4] and Clarkson and Tremblay [8].*

Muscle soreness develops about 24 hours after eccentric exercise, reaches peak values about 48 hours after, and gradually dissipates by 5 to 10 days after exercise (Figure 2). Some investigators have suggested that swelling and edema produce the sensation of soreness. In our studies we have noted swelling long after soreness has dissipated [9]. Soreness may also be the result of chemical irritants, such as histamine and bradykinins, generated by the damaged tissue.

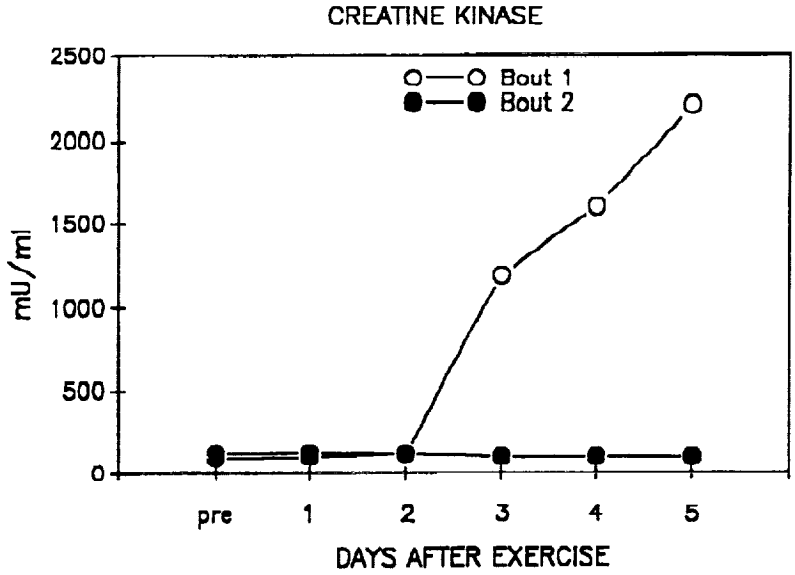


Figure 2. *Muscle soreness of the forearm flexor muscles before (pre) and 5 days after a high-force eccentric forearm flexion exercise. Data modified from Clarkson and Tremblay [8]. Bout 1 (open circles) depicts changes following 70 maximal eccentric actions with one arm. With the other arm subjects performed 24-maximal eccentric actions and 2 weeks later performed 70-maximal eccentric actions; Bout 2 (closed circles) shows data from these latter 70-maximal eccentric actions. For more detailed explanation of the study see reference 8.*

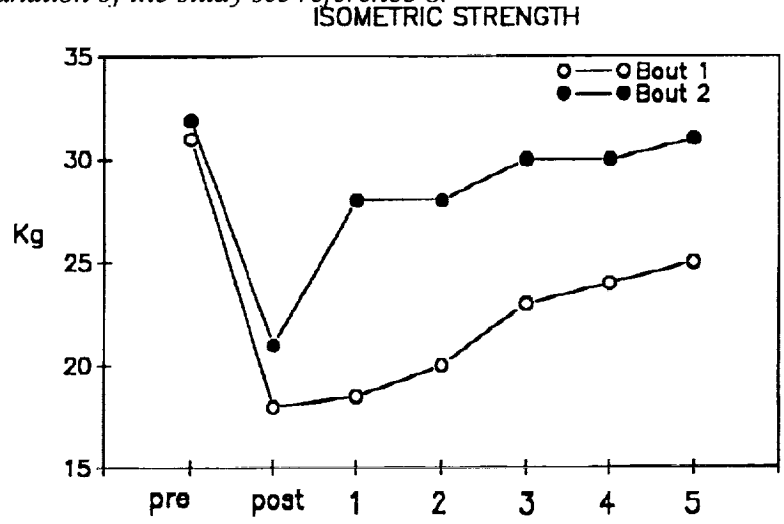


Figure 3. *Isometric strength of the forearm flexor muscles before (pre), immediately after (post), and 5 days after a high-force eccentric forearm flexion exercise. Data modified from Clarkson and Tremblay [8]. See legend for Figure 2.*

Also associated with eccentric actions is strength loss and decreased range of motion in the days following the eccentric exercise. Clarkson and Tremblay [8] showed that isometric strength was decreased by about 50 percent immediately after high-force eccentric exercise and gradually returned toward baseline (Figure 3).

Range of motion was shown to decrease immediately after the exercise, further decreased at 2 days after exercise, and then returned toward baseline [8] (Figure 4).

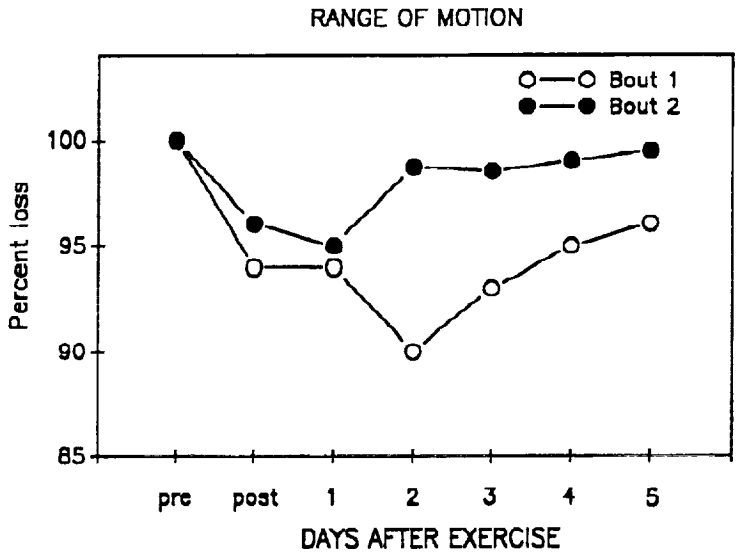


Figure 4. *Range of motion about the elbow before (pre), immediately after (post), and 5 days after a high-force eccentric forearm flexion exercise. Range of motion is expressed as a percentage of preexercise range. Data modified from Clarkson and Tremblay [8]. See legend for Figure 2.*

Within 7 to 10 days after performance of high-force eccentric exercise, muscle function is fully recovered [9]. Moreover, during the recovery process, an adaptation or training effect takes place. When subjects perform a subsequent bout of high-force eccentric exercise, they are able to perform more work [8, 9, 23]. Also, less damage is incurred from the second exercise [8, 9, 23]. In Figure 2, less soreness develops after Bout 2 compared with the first bout, strength and range of motion are restored more quickly after the second bout (Figures 3 and 4, respectively), and most noteworthy, there is no release of CK into the blood after Bout 2 (Figure 5). Thus, performance of the first eccentric exercise produced an adaptation (or rapid training effect) in the muscle such that it was more resistant to damage from a subsequent bout of strenuous exercise. This training effect can also be elicited when the first bout of eccentric exercise produces little muscle damage [8]. There is evidence to suggest that the training effect can last 6 weeks or longer [9].

It is important to note that the adaptation effect described above cannot be elicited by concentric training [28]. Sforzo and Lamb [28] investigated the effects of concentric training and eccentric training (at the same absolute workload) in two groups of subjects. After 2 weeks of training, these subjects performed a strenuous bout of exercise that incorporated concentric and eccentric actions (con-ecc). The concentric trained group developed considerable soreness after the strenuous bout of con-ecc exercise but the eccentric trained groups did not. This finding is particularly noteworthy since the absolute workloads were the same thereby making the training regimen more strenuous for the concentric trained group.

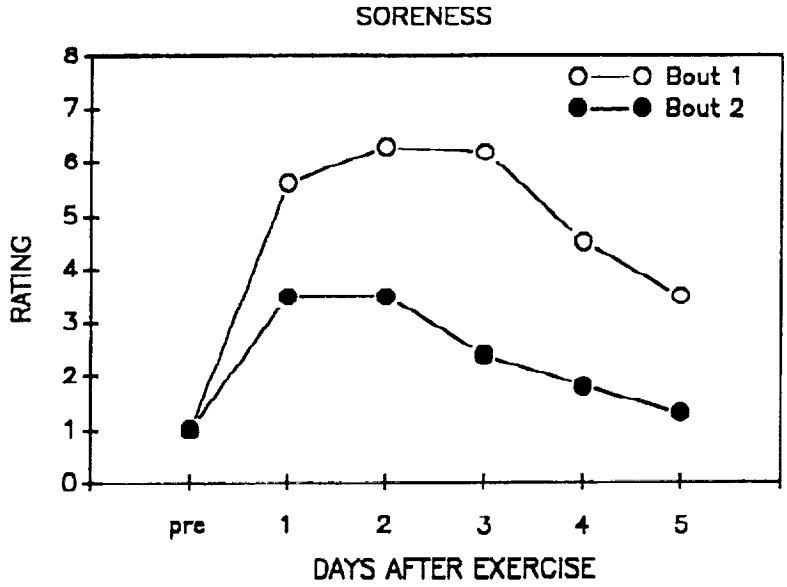


Figure 5. *Creatine kinase activity in the blood before (pre) and 5 days after a high-force eccentric forearm flexion exercise. Data modified from Clarkson and Tremblay (8). See legend for Figure 2.*

In our laboratory we have tested over 300 subjects who have performed high-force eccentric exercise and we have never had an injury occur. These subjects included a sample of persons between the ages of 60 and 70 years. Other laboratories in this country and in England have also used high-force eccentric exercise and have not had an injury (either reported in the literature or personal communication).

One physiological explanation for a lack of injury with eccentric actions is that a protective reflex serves to prevent the muscle from generating more tension than it can accommodate safely. Evidence for such a reflex is indirect. The ratio of eccentric force to concentric force is greater for isolated muscle compared with intact human muscle [30]. Stauber [30] suggested that this discrepancy can be explained as a function of central nervous system (CNS) actions in intact muscle. Also, when muscle is electrically stimulated during eccentric actions, the same high-force can be achieved as in isolated muscle [30]. Thus, the development of the full force capability during normal eccentric actions may be prevented by CNS intervention thereby protecting the muscle from injury. A similar mechanism has also been suggested for other forms of muscle actions. Stauber [30] proposes that only when these protective reflexes are fatigued will myotendinous tearing occur.

In summary, high-force eccentric exercise does produce muscle damage, but this damage is a normal event and it is completely reparable. In addition, it leads to an adaptation response. CNS control over muscle may exist to prevent serious injury. Individuals who already have a muscle or joint injury could be at risk in performing maximal high-force eccentric exercise. These individuals, however, may also be at a risk in performing high-force concentric or isometric exercise. It should also be noted that the exercise regimens used in the studies cited above are very strenuous. In many of the studies, 70-consecutive maximal eccentric actions were performed with one muscle group. From these investigations, however, there is no scientific evidence that performance of numerous, repetitive maximal eccentric actions are dangerous to healthy individuals. Moreover, there is certainly no evidence to suggest that testing of eccentric actions

where only a few trials of maximal intensity are used should be contraindicated.

Stimulus for Muscle Strength Increase

Moritani and deVries [22] have shown that during the early stages of a strength training program, the increase in strength is due to neural factors. During the later stages, after about 3 to 5 weeks of training, muscle hypertrophy is responsible for strength gains. A review of neural mechanisms in strength development can be found in a recent paper by Jones, Rutherford, and Parker [18].

The exact stimulus for muscle hypertrophy is not known. Jones, Rutherford, and Parker [18] have suggested that hormonal factors may play a role. Release of hormones such as insulin, growth hormone, and testosterone could regulate protein synthesis and degradation to promote muscle growth. Hormones may not be the primary stimulus for hypertrophy but might act in conjunction with local changes in the exercised muscles.

If factors associated with metabolic fatigue were the stimulus for hypertrophy, then one would expect that a metabolically more stressful exercise would induce greater hypertrophy and hence greater strength gains (although there is not a 1:1 relationship between strength and muscle size) than a less metabolically stressful exercise. Training programs using concentric exercise, which is metabolically more costly than eccentric exercise, have not been shown to produce greater strength gain or muscle size than training programs using eccentric exercise. Thus, a purely metabolic mechanism cannot explain the hypertrophic response.

It is tempting to suggest that factors associated with muscle damage could be the stimulus for hypertrophy. Moreover, the adaptation effect (rapid training effect) that has been described for muscle damage (see above), would be an attractive correlate to the hypertrophic response. However, no studies have shown that training using eccentric actions is any more effective in inducing strength gain or hypertrophy than concentric exercise. Thus the adaptation effect observed after eccentric-exercise-induced muscle damage seems unrelated to the stimulus for hypertrophy.
High tension levels have been considered as the stimulus for hypertrophy. However, if high tension alone were the stimulus, eccentric exercise would likely cause more hypertrophy than concentric. There is no evidence for this and, as Stauber [30] has noted, body-builders who use eccentric actions in their training have no larger muscles than weight lifters who avoid eccentric actions. Moreover, increases in cross sectional area of muscle after training with eccentric actions are not greater than increases after concentric actions that produce less tension [16].

The exact stimulus for muscle hypertrophy is not known. It is possible that several factors are involved in a complex interplay of events. For example, there may be a threshold force or tension level that must be achieved which would serve as the primary stimulus for hypertrophy. Then, other factors would come into play and modulate the response.

Strength Requirements for Extended Duration Space Activities, Reentry, and EVA

Exposure to microgravity environments for an extended duration results in muscle atrophy and a decline in muscle function [12]. However, little is known about how various expressions of strength are affected. Because of the lack of gravity, muscle usage will be considerably different, particularly with regard to a lack of eccentric actions. For example, in a one-g environment a person would lift up a weight by contracting the forearm flexor muscles in a concentric action and would lower the weight by having the forearm flexors perform an eccentric action. However, in microgravity, a weight would be lifted with the forearm flexors but to move the weight to the

starting position the forearm extensors would act in a concentric manner. Situations in microgravity where eccentric actions may be used, for example, would be to stop the body when it is propelled towards an immovable object.

If there is a preferential lack of eccentric actions performed in microgravity conditions, how does this impact on strength loss, atrophy, and performance—either for EVA or for reentry? It has been shown that exercise programs involving combinations of muscle action modalities are most effective in 19 producing strength gains. Conversely then, will exercises during extended periods of microgravity that do not incorporate eccentric actions be less effective in retarding atrophy and strength loss?

At present, it is difficult to recommend specific training regimens either for performance during space flight or for conditioning prior to space flight. Before such recommendations can be made, knowledge of the effects of prolonged exposure to microgravity on various modalities of strength (isometric, concentric and eccentric) are needed. Also specific performance requirements (i.e., modalities of strength, endurance) needed for EVA and reentry must be identified.

References

[1] ATHA J. Strengthening muscle. In: *Exercise and Sport Sciences Reviews*, edited by D. Miller. Philadelphia, PA: Franklin Institute, 1981; p. 1-73.

[2] BIGLAND-RITCHIE B. AND J. J. WOODS. Integrated electromyogram and oxygen uptake during positive and negative work. *J. Physiol.* 260:267-277, 1976.

[3] BONDE, PETERSEN F. Muscle training by static, concentric and eccen-t tric contractions. *Acta Physiol. Scand.* 48:406-416, 1960.

[4] BYRNES, W. C., P. M. CLARKSON, J. S. WHITE, S. S. HSIEH, P. N. FRYKMAN, AND R. J. MAUGHAN. Delayed onset muscle soreness following repeated bouts of downhill running. *J. Appl. Physiol.* 59:710-715, 1985.

[5] CAVANAGH, P. R. On "muscle action" vs. "muscle contraction." *J. Biomech.* 21:69, 1988.

[6] CHAPMAN, A. E. The mechanical properties of human muscle. In: *Exercise and Sport Sciences Reviews*, edited by R. L. Terjung. New York: MacMillian, 1985, 13:443-502.

[3] BONDE, PETERSEN F. Muscle training by static, concentric and eccen-t tric contractions. *Acta Physiol. Scand.* 48:406-416, 1960.

[4] BYRNES, W. C., P. M. CLARKSON, J. S. WHITE, S. S. HSIEH, P. N. FRYKMAN, AND R. J. MAUGHAN. Delayed onset muscle soreness following repeated bouts of downhill running. *J. Appl. Physiol.* 59:710-715, 1985.

[5] CAVANAGH, P. R. On "muscle action" vs. "muscle contraction." *J. Biomech.* 21:69, 1988.

[6] CHAPMAN, A. E. The mechanical properties of human muscle. In: *Exercise and Sport Sciences Reviews*, edited by R. L. Terjung. New York: MacMillian, 1985, 13:443-502.

[7] CLARKE, D. H. Adaptation in strength and muscular endurance resulting from exercise. *Exercise and Sport Sciences Reviews*, 1973, 1:73-102.

[8] CLARKSON, P. M. AND I. TREMBLAY. Rapid adaptation to exercise induced muscle damage. *J. Appl. Physiol.* 65:1-6, 1988.

[9] EBBELING, C. B. AND P. M. CLARKSON. Exercise-induced muscle

damage and adaptation. *Sports Med.* 7:207-234, 1989.

[10] FRIDEN, J., M. SJOSTROM, AND B. EKBLOM. Myofibrillar damage following intense eccentric exercise in man. *Int. J. Sports Med.* 4:170-176, 1983.

[11] HAKKINEN, K. AND P. V. KOMI. Effects of different combined concentric and eccentric muscle work regimens on maximal strength development. *J. Hum. Mov. Stud.* 7:33-44, 1981.

[12] HERBISON, G. J, AND J. M. TALBOT (eds.). Final report phase IV: research opportunities in muscle atrophy. *NASA Contractor Report 3796*, 1984.

[13] JOHNSON, B. L. Eccentric vs. concentric muscle training for strength development. *Med. Sci. Sports* 4:115, 1972.

[14] JOHNSON, B. L., J. W. ADAMCZYK, K. O. TENNOE, AND S. B. STROMME. A comparison of concentric and eccentric training. *Med. Sci. Sports* 8:35-38, 1976.

[15] JOHNSON, B. L. AND J. W. ADAMCZYK. A program of eccentric-concentric strength training. *Am. Corr. Ther. J.* 29:13-16, 1975.

[16] JONES, D. A. AND O. M. RUTHERFORD. Human muscle strength training: the effects of three different regimes and the nature of the resultant changes. *J. Phvsiol.* 391:1-11, 1987.

[17] JONES, D. A., D. J. NEWHAM, J. M. ROUND, AND S. E. J. TOLFREE. Experimental human muscle damage: morphological changes in relation to other indices of damage. *J. Physiol.* 375:435-448, 1986.

[18] JONES, D. A., O. M. RUTHERFORD, AND D. F. PARKER. Physiological changes in skeletal muscle as a result of strength training. *Quart. J. Exp. Physiol.* 74:233-256, 1989.

[19] KOMI, P. V. AND E. R. BUSKIRK. Effect of eccentric and concentric muscle conditioning on tension and electrical activity of human muscle. *Ergonomics* 15:417-434, 1972.

[20] LAYCOE, R. R. AND R. G. MARTINIUK. Learning and tension as factors in static strength gains produced by static and eccentric training. *Res Q* 42:299-306, 1971.

[21] MANNHEIMER, J. S. A comparison of strength gain between concentric and eccentric contractions. *Phys. Ther.* 49:1201-1207, 1969.

[22] MORITANI, T. AND H. A. DEVRIES. Neural factors versus hypertrophy in the time course of muscle strength gain. *Am. J. Phys. Med.* 82:521-524, 1979.

[23] NEWHAM, D. J., D. A. JONES, AND P. M. CLARKSON. Repeated high-force eccentric exercise: effects on muscle pain and damage. *J. Appl. Physiol.* 63:1381-1386, 1987.

[24] NEWHAM, D. J., D. A. JONES, AND R. H. T. EDWARDS. Large delayed plasma creatine kinase changes after stepping exercise. *Muscle Nerve* 6:380-385, 1983.

[25] NEWHAM, D. J., G. MCPHAIL, K. R. MILLS, AND R. H. T. EDWARDS. Ultrastructural changes after concentric

and eccentric contractions of human muscle. *J. Neurol. Sci.* 61:109-122, 1983.

[26] PLETNEV, B. A. Effectiveness of different regimens of muscle work with equivalent loads. *Theory and Practice of Physical Culture* 10:20-23, 1975.

[27] RASCH, P. J. Present status of negative (eccentric) exercise: a review. *Amer. Corr. Ther. J.* 1974; 28:77-94.

[28] SFORZO, G. A. AND D. R. LAMB. Muscle soreness after eccentric exercise: effect of early training with concentric contractions. In: *Exercise Physiology Current Selected Research, Volume 1,* edited by C. O. Dodson and J. H. Humphrey. New York, NY: AMS Press, Inc., 1985, p. 171-179.

[29] SLOBODYAN, A. P. Research on the optimal combination of various regimes of muscular work in the training of weightlifters. *Theory and Practice of Physical Culture,* 1972, 7:27-29.

[30] STAUBER, W. T. Eccentric action of muscles: physiology, injury and adaptation. In: *Exercise and Sport Sciences Reviews,* edited by K. B. Pandolf. Baltimore, MD: Williams & Wilkins, 1989, p. 157-185.

N94-28364

S3-52

207612

P-8

Exercise Detraining: Applicability to Microgravity

Edward F. Coyle, Ph.D.
Human Performance Laboratory
Department of Kinesiology and Health
The University of Texas at Austin
Austin, Texas

Reversibility of Adaptations Induced by Training

Physical training exposes the various systems of the body to potent physiologic stimuli. These stimuli induce specific adaptations that enhance an individual's tolerance for the type of exercise encountered in training. The level of adaptation and the magnitude of improvement in exercise tolerance is proportional to the potency of the physical training stimuli. Likewise, our bodies are stimulated by gravity, which promotes adaptations of both the cardiovascular and skeletal muscles. Exposure to microgravity removes normal stimuli to these systems and the body adapts to these reduced demands. In many respects the cessation of physical training in athletes and the transition from normal gravity to microgravity represent similar paradigms.

Inherent to these situations is the concept of the reversibility of the adaptations induced by training or by exposure to normal gravity. The *reversibility concept* holds that when physical training is stopped (i.e., detraining) or reduced, or a person goes from normal gravity to microgravity, the bodily systems readjust in accordance with the diminished physiologic stimuli. The focus of this chapter is on the time course of loss of the adaptations to endurance training as well as on the possibility that certain adaptations persist, to some extent, when training is stopped. Because endurance exercise training generally improves cardiovascular function and promotes metabolic adaptations within the exercising skeletal musculature, the reversibility of these specific adaptations is considered. These observations have some applicability to the transition from normal to microgravity.

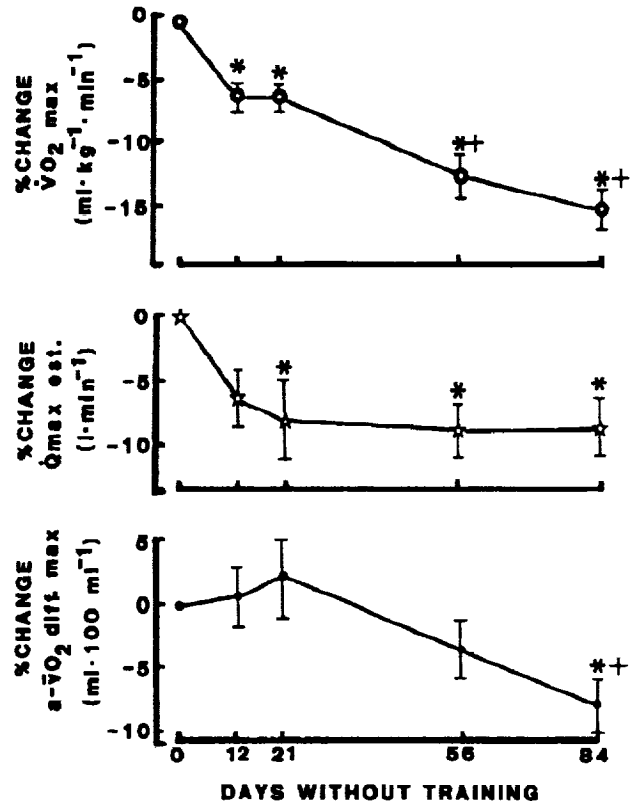
Cardiovascular Detraining

Maximal Oxygen Uptake

Endurance training induces increases in maximal oxygen uptake (i.e., $\dot{V}O_{2max}$), cardiac output, and stroke volume [1, 2]. When sedentary men participate in a 7-week, low-intensity training program (20 min/day^{-1}, 3 days/week^{-1}), $\dot{V}O_{2max}$ levels increased by 6 percent, with a return of $\dot{V}O_{2max}$ values to pretraining levels with 8 weeks of detraining [3]. Moderate endurance training increases $\dot{V}O_{2max}$ by 10 to 20 percent, yet again $\dot{V}O_{2max}$ may decline to pretraining levels when training is stopped [4-6]. Values of $\dot{V}O_{2max}$ decline rapidly during the first month inactivity, whereas a slower decline to untrained levels occurs during the second and third months of detraining [4-6]. Therefore, the available evidence suggests that the increases in $\dot{V}O_{2max}$ produced by endurance training involving exercise of low to moderate intensities and durations are totally reversed after several months of detraining.

Investigators have not yet examined and then exposed untrained individuals to several years of intense endurance training and subsequent inactivity to determine if extreme training results in a persistent elevation of $\dot{V}O_{2max}$ above untrained levels. Our present knowledge is limited to findings of studies involving already trained endurance athletes who agreed to cease training so that reversibility of their physiologic adaptations should be studied periodically [7]. Figure 1 is a

display of the time course of the decline in $\dot{V}O_{2max}$ and its components of cardiac output, and arteriovenous O_2 difference when people become sedentary after training intensely for approximately 10 years.



*Significantly lower than trained; $p<0.05$. +Significantly lower than day 21; $p<0.05$.

Figure 1. *Effects of detraining upon the percent changes in maximal oxygen uptake (i.e., $\dot{V}O_{2max}$), maximal cardiac output estimated from submaximal stroke volume and maximal heart rate (i.e., Qmax est.) and maximal arterial-venous oxygen difference (i.e., a-$\dot{V}O_{2diff \cdot max}$).*

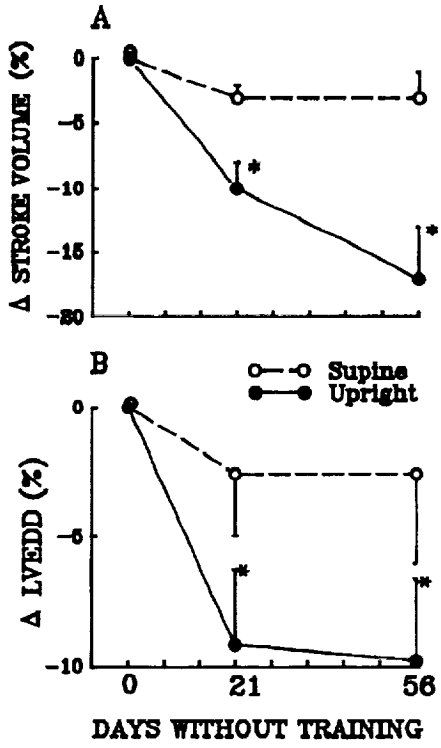
The $\dot{V}O_{2max}$ value was relatively high in trained subjects (i.e., 62 mL/min^{-1} at 0 days without training) and it declined a total of 16 percent after 84 days of detraining. A rapid decline of 7 percent occurred in the first 12 to 21 days with a further decline of 9 percent during the period from 21 to 84 days [7]. The rapid, early decline in $\dot{V}O_{2max}$ was related to a reduction in maximal stroke volume measured during exercise in the upright position. Most of the decline in stroke volume occurred during the first 12 days of inactivity. Adaptive increases in maximal heart rate compensated somewhat for this loss of stroke volume. The decline in $\dot{V}O_{2max}$ during the 21 to 84-day period was associated with a decline in maximal arteriovenous O_2 difference.

The 84-day period of detraining resulted in a stabilization of $\dot{V}O_{2max}$ and maximal stroke volume. Thus, the subjects appeared to have detrained for a sufficient length of time to display a complete readjustment of cardiovascular response in accordance with their sedentary lifestyle. Note that maximal stroke volume during upright exercise in the detraining subjects was virtually the same as that observed in people who had never engaged in endurance training. The idea that this finding does not necessarily imply a loss of heart function is subsequently discussed. Although maximal cardiac output and stroke volume declined to untrained levels, $\dot{V}O_{2max}$ levels in the detrained subjects remained 17 percent above that of untrained individuals, primarily because of an elevation of maximal arteriovenous O_2 difference. The persistent elevation of $\dot{V}O_{2max}$ values in the detrained subjects, the result of an augmented ability of the exercising musculature to extract oxygen, may be related to the observation that these subjects displayed no loss of the increased capillary density derived from the training and only a partial loss of the increase in muscle mitochondria.

Stroke Volume and Heart Size

Prolonged and intense endurance training is thought to promote an increase in heart mass, and researchers believe detraining results in a decline in heart mass [1, 8, 9]. What is not clear, however, is whether the training-induced increases in ventricular volume and myocardial wall thickness regress totally with inactivity. Athletes who become sedentary have enlarged hearts and an elevated $\dot{V}O_{2max}$ level in contrast to people who have never trained [10].

One of the most striking effects of detraining in endurance-trained individuals is the rapid decline in stroke volume. To gain information regarding the cause of this large and rapid decline, Martin *et al.* [11] measured stroke volume during exercise in trained subjects in both the upright and supine positions and again after 21 and 56 days of inactivity (Figure 2).



*Response in the upright position is significantly lower than the responses in the supine position ($p<0.05$).

Figure 2. *Percentage decline in exercise stroke volume (A) and left ventricular end diastolic diameter (LVEDD) during exercise in the upright and supine positions.*

Simultaneous measurements of the diameter of the left ventricle were obtained echocardiographically. The large decline in stroke volume during upright cycling was associated with parallel reductions in the diameter of the left ventricle at end-diastole (LVEDD). When the subjects were evaluated during exercise in the supine position, a condition that usually augments ventricular filling because of the drainage of blood from the elevated legs, reduction in LVEDD was minimal. As a result, stroke volume during exercise in the supine position was maintained within a few percent of trained levels during the 56-day detraining period.

Role of Blood Volume

Along the same lines, results of recent studies indicate that the rapid reduction with detraining of stroke volume during exercise in the upright position is related to a decline in blood volume [12]. Intense exercise training usually results in an increase in blood volume by approximately 500 mL through the expansion of plasma volume [13, 14]. This adaptation is gained after only a few bouts of exercise, and it is quickly reversed when training ceases [13, 14]. The decline in stroke volume and the increase in heart rate during submaximal exercise, which normally accompanies several weeks of detraining, can be essentially reversed, and values return to near trained levels when the blood volume of detrained men expands to a level similar to that when the subjects were trained [12].

The observation that stroke volume during exercise is maintained at near trained levels when blood volume is high suggests that the ability of the heart to fill with blood is not significantly altered by detraining. If ventricular mass does indeed decline, a thinning of the ventricular walls and not a reduction in LVEDD is probably involved [11]. Thus, the reduction in intrinsic cardiovascular function is apparently minimal after several weeks of inactivity in men who had been training intensely for several years [12]. The large reduction in stroke volume during exercise in the upright position is largely a result of reduced blood volume and not of a deterioration of heart function [12].

Detraining and Muscle Metabolism

Enzymes of Energy Metabolism

Endurance exercise training induces enzymatic adaptations in the exercising musculature that result in slower rates of glycogen utilization and lactate production and improved endurance during submaximal exercise [15]. One of the more important alterations is an increase in the activity of mitochondrial enzymes, which results in an increased ability to metabolize fuels in the presence of oxygen. Moderate endurance training (2 to 4 months duration) increases mitochondrial enzyme activity by 20 to 40 percent from untrained levels [5, 16]. When moderate training ceases, however, and the stimuli for adaptation are removed, the increases in mitochondrial activity are quickly and totally reversed. Mitochondrial activity returns to pretraining levels within 28 to 56 days after the cessation of training [5, 16].

The pattern of change in enzyme activity observed when individuals who trained intensely for 10 years stopped training for 84 days is curvilinear [17]. Mitochondrial enzyme activity in trained subjects (i.e., citrate synthase, succinate dehydrogenase, malate dehydrogenase, and ß hydroxyacyl-CoA dehydrogenase) is initially two-fold higher than those in untrained persons, declines progressively during the first 56 days of detraining, and stabilizes at levels that are 50 percent higher than the values obtained from sedentary control subjects. The half time of decline is approximately 12 days (i.e., declines on half the distance between trained and detrained in 12 days). Therefore, prolonged and intense training, in contrast to training programs that last only a few months, appears to result in only a partial loss of mitochondrial enzyme activity and thus a persistent elevation of activity above untrained levels. This elevation occurred almost entirely because of a persistent 80 percent elevation above untrained levels in the mitochondrial enzyme activity in fast-twitch muscle fibers [18].

Muscle Capillarization

Endurance training promotes increased capillarization of the exercising musculature, which theoretically both prolongs the transit time of blood flow through the muscle and reduces diffusion distances, thus improving the availability of oxygen and nutrients to the muscle while also allowing for better removal of metabolic waste products. Moderate endurance training of several months duration increases muscle capillarization by 20 to 30 percent above pretraining levels [5, 19]. Results of preliminary studies indicate that certain indices of muscle capillarization remain somewhat higher than pretraining levels 8 weeks after the cessation of moderate training [5].

More prolonged and intense training increases muscle capillary density by 40 to 50 percent from untrained levels [7, 19]. No indication exists that increases in muscle capillary density in highly trained people are reversed during 3 months of detraining [7].

Muscular Adaptations that Persist with Detraining

The detraining responses in the skeletal musculature of highly trained people who regularly engaged in intense exercise for several years apparently differ from those in individuals who have trained for only a few months. No loss of the increase in muscle capillarization occurs with the cessation of prolonged intense training, although such a loss does occur when moderate training is stopped. The cessation of moderate training results in a complete reversal of the training-induced increases in mitochondrial enzyme activity, whereas only a partial decline and therefore a persistent elevation of mitochondrial activity above untrained levels occurs with the cessation of exercise after prolonged intense endurance training [3, 5, 16, 17].

Exercise Responses of Detrained Subjects

Currently, scant evidence is available to imply that the cardiovascular or skeletal musculature adaptations derived from mild and moderate endurance training are maintained above pretraining levels with cessation of training for more than approximately 8 weeks. Therefore, a person should be stressed to the same degree during exercise of a given intensity whether untrained or after a prolonged detraining period. This hypothesis has yet to be fully evaluated, however, and one factor to consider is the possibility that people may perceive exercise to be more comfortable when they are in the detrained state having already experienced physical training, as compared to the untrained condition.

In agreement with the findings that individuals who exercise intensely on a regular basis for several years remain superior in the detrained state with respect to their muscle metabolism and intrinsic heart function (i.e., stroke volume when ventricular filling is high) compared with untrained people, it appears that these detrained people can exercise more intensely before becoming inordinately stressed. One indication of this ability is the observation that detrained persons not only possess a $\dot{V}O_{2max}$ level that is well above untrained values, but also they maintain the ability to exercise at a high percentage of $\dot{V}O_{2max}$ before lactic acid begins to accumulate in the blood [17].

Summary

Our hypothesis is that the alterations in physiological function upon transition from normal gravity to microgravity may share similar trends to the process of "detraining." When physical training ceases (i.e., detraining), the bodily systems readjust in accordance with the diminished physiologic stimuli, and many training-induced adaptations are reversed to varying extents. The available evidence to date suggests that the increases in $\dot{V}O_{2max}$ produced by endurance training of low to moderate intensities and durations are totally reversed after several months of detraining. When people detrain after several years of intense training, they display large reductions (i.e., 5 to 15 percent) in stroke volume and $\dot{V}O_{2max}$ during the first 12 to 21 days of inactivity. These declines do not indicate a deterioration of heart function, but instead are largely a result of reduced blood volume and the ability to return venous blood to the heart. The $\dot{V}O_{2max}$ of endurance athletes continues to decline during the 21 to 56 days of detraining because of reductions in maximal arteriovenous O_2 difference. These reductions are associated with a loss of mitochondrial enzyme activity within the trained musculature, which declines with a half-time of approximately 12 days. Endurance athletes, however, do not regress to levels displayed by individuals who never participated in exercise training. Levels of mitochondrial enzyme activity remain 50 percent higher

than those of sedentary subjects, skeletal muscle capillarization is maintained at high levels, and $\dot{V}O_{2max}$ and the maximal arteriovenous O_2 difference stabilize at a point that is 12 to 17 percent higher than untrained levels after 84 days of detraining.

References

[1] BLOMQVIST, C. G. AND B. SALTIN. Cardiovascular adaptations to physical training. *Ann. Rev. Physiol.* 45:169-189, 1983.

[2] ROWELL, L. B. Human cardiovascular adjustments to exercise and thermal stress. *Physiol. Rev.* 54:75-159, 1974.

[3] ORLANDER, J., K. H. KIESSLING, J. KARLSSON, AND B. EKBLOM. Low intensity training, inactivity and resumed training in sedentary men. *Acta Physiol. Scand.* 101:351-362, 1977.

[4] FOX, E. L., ET AL. Frequency and duration of interval training programs and changes in aerobic power. *J. Appl. Physiol.* 38:481-484, 1975.

[5] KLAUSEN, K., L. B. ANDERSEN, AND I. PELLE. Adaptive changes in work capacity, skeletal muscle capillarization and enzyme levels during training and detraining. *Acta Physiol. Scand.* 113:916, 1981.

[6] DRINKWATER, B. L. AND S. M. HORVATH. Detraining effects on young women. *Med. Sci. Sports Exerc.* 4:91-95, 1972.

[7] COYLE, E. F., ET AL. Time course of loss adaptations after stopping prolonged intense endurance training. *J. Appl. Physiol.* 57:1857-1864, 1984.

[8] EHSANI, A. A., J. M. HAGBERG, AND R. C. HICKSON. Rapid changes in left ventricular dimensions and mass in response to physical conditioning and deconditioning. *Am. J. Cardiol.* 42:52-56, 1978.

[9] HICKSON, R. C., G. T. HAMMONS, AND J. O. HOLLOSZY. Development and regression of exercise-induced cardiac hypertrophy in rats. *Am. J. Physiol.* 236:H268-272, 1979.

[10] SALTIN, B. AND G. G. GRIMBY. Physiological analysis of middle-aged and old former athletes: comparison with still active athletes of the same ages. *Circulation* 38:1104-1115, 1968.

[11] MARTIN, W. H., E. F. COYLE, S. A. BLOOMFIELD, AND A. A. EHSANI. Effects of physical deconditioning after intense training on left ventricular dimensions and stroke volume. *J. Am. Coll.Cardiol.* 7:982989, 1986.

[12] COYLE, E. F., M. K. HEMMERT, AND A. R. COGGAN. Effects of detraining on cardiovascular responses to exercise: role of blood volume. *J. Appl. Physiol.* 60:95-99, 1986.

[13] CONVERTINO, V. A., ET AL. Exercise training-induced hypervolemia: role of plasma albumin, renin, and vasopressin. *J. Appl. Physiol.* 48:665-669, 1980.

[14] GREEN, H. J., ET AL. Alterations in blood volume following short-term supramaximal exercise. *J. Appl. Physiol.* 56:145-149, 1984.

[15] HOLLOSZY, J. O. AND E. F. COYLE. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *J. Appl. Physiol.* 56:831-838, 1984.

[16] HENRIKSSON, J. AND J. S. REITMAN. Time course of changes in human skeletal muscle succinate dehydrogenase and cytochrome oxidase activities and maximal oxygen uptake with physical activity and inactivity. *Acta Physiol. Scand.* 99:91-97, 1977.

[17] COYLE, E. F., ET AL. Effects of detraining on responses to submaximal exercise. *J. Appl. Physiol.* 59:853-859, 1985.

[18] CHI. M. M-Y., ET AL. Effects of detraining on enzymes of energy metabolism in individual human muscle fibers. *Am. J. Physiol.* 244:C276-287, 1983.

[19] INGJER, F. Capillary supply and mitochondrial content of different skeletal muscle fiber types in untrained and endurance-trained men: a histochemical and ultrastructural study. *Eur. J. Appl. Physiol.* 40:197-209, 1979.

[20] BLOMQVIST, C. G. AND H. L. STONE. Cardiovascular adjustments to gravitational stress. In: *The Handbook of Physiology. The Cardiovascular System,* edited by J. T. Shepherd, F. M. Abboud, and S. R. Geiger. Bethesda, MD: American Physiology Society, 1982.

Omit

Section 2

Cardiovascular Fitness

N94-28365

Aerobic Fitness and Orthostatic Tolerance: Evidence Against an Association

Thomas J. Ebert, M.D., Ph.D.
Department of Anesthesiology and Physiology
Medical College of Wisconsin
and the VA Medical Center
Milwaukee, Wisconsin

54-52
207613
p-8

Introduction

When humans stand, the force of gravity (one g) promotes pooling of blood in the lower extremities. This reduces the return of venous blood to the heart and produces an *orthostatic challenge* to the cardiovascular system. Higher gravitational forces, in the head-to-foot direction, are commonly experienced by Navy and Air Force personnel and astronauts for relatively brief periods during high-speed maneuvering of sophisticated jet fighters and during launch and reentry of manned space craft.

The reduction in venous return to the heart during gravitational stress results in a lowering of cardiac output through Frank-Starling mechanisms, which can often lead to a decrease in blood pressure. If cardiac output and blood pressure are not adequately maintained, reduced perfusion of the cerebral circulation may lead to a loss of consciousness (syncope). This response in flight could be catastrophic. Thus, the high level of interest by the military and NASA in understanding gravitational stress responses and identifying physiologic situations and pharmocologic manipulations which alter individual tolerance to gravitational stress is well understood.

The two primary mechanisms whereby adequate cardiac output and blood pressure are maintained during orthostatic stress are by reflex activation of the autonomic nervous system and by contraction of skeletal muscles in the lower extremities (the *skeletal muscle pump*). During many types of orthostatic challenge, the skeletal muscles are inactive (i.e., passive head-up tilt, lower body negative pressure [LBNP]) and acceleration stress in pilots and astronauts. To lessen the pooling of blood in the lower body, flight personnel routinely employ compression trousers to *squeeze* pooled blood back to the central circulation.

The second major mechanism that is engaged to compensate for reductions in central blood volume and blood pressure is mediated through the autonomic nervous system. Baroreceptors are located in the carotid and aortic blood vessels in the arterial circulation and are located in the heart and great veins on the low pressure side of the circulation [20, 21]. These receptors respond to blood pressure and blood volume changes and initiate reflex increases in heart rate, cardiac contractility, and peripheral vascular tone.

This presentation will focus on only one side of the debate as to whether high levels of aerobic fitness have a deleterious effect on tolerance to gravitational stress. This issue was raised in the early 1970's as a result of two research publications. The first was work reported by Stegemann *et al.* [31] who studied the carotid sinus baroreflex of humans with an airtight chamber that surrounded the head and neck. The steady-state reflex changes in blood pressure that were recorded 3 minutes after application of the head and neck stimuli, were attenuated in an athletic group compared to a sedentary group of volunteers. A second report in the NASA literature by Luft *et al.* [18] indicated that five endurance-trained runners were less tolerant to LBNP than five nonrunners. These early research findings have stimulated a considerable amount of interest that

PRECEDING PAGE BLANK NOT FILMED
PAGE 100 INTENTIONALLY BLANK

has lead to a growing number of research efforts seeking an association between aerobic fitness and orthostatic tolerance in humans. I will briefly review some of the more pertinent published research information which suggests that there is no relationship between aerobic fitness and orthostatic tolerance in humans.

Cross-Sectional Studies

Prior to 1970, the general belief was that orthostatic tolerance was greater in the athletic compared to the sedentary individual. Research findings as early as the 1920's suggested that, in general, athletes made better circulatory adjustments upon standing than did nonathletes [8, 33]. Supporting data were provided several decades later by Schvartz *et al.* [28], who studied the responses of 18 healthy subjects during head-up tilt testing. Syncope occurred in four subjects. Retrospective analyses revealed that those who experienced syncope could perform fewer sit-ups and pushups compared with 14 subjects who tolerated the tilt stress. These investigators concluded that conditioning of the upper body muscles was important for maintaining orthostatic tolerance. A subsequent study by these investigators in the early 1970's, included measurements of peak oxygen uptake to better evaluate fitness levels [29]. Twenty-eight men were stressed by passive standing and the frequency of fainting was noted to be less in the men who had higher aerobic capacities than those who were less fit.

However, the early belief that cardiovascular fitness was associated with improved orthostatic tolerance was questioned as early as 1940, when investigators were unable to find differences in the orthostatic tolerance of athletes and nonathletes [1, 22]. A large number of the recently published cross-sectional studies have failed to identify a significant statistical relationship between endurance capacity and tolerance to orthostatic stress. For example, Klein *et al.* [15] reported on a group of 12 athletes who, due to many years of intense physical training (long distance running, bicycling, or ice skating), had about a 50 percent higher maximum work capacity than 12 healthy students ($\dot{V}O_{2max}$ 65 vs. 44 $mL \cdot kg^{-1} \cdot min^{-1}$). The orthostatic tolerance to both a 20-minute, 90 degree head-up tilt and centrifugal (+Gz) forces were found to be identical between groups. Similar results have been reported in cross-sectional studies on female volunteers undergoing LBNP [14].

A somewhat different research approach was undertaken by Sather and colleagues [26], who determined the tolerance of 18 men, ages 29 to 51 years, to progressive LBNP that was continued until the onset of presyncopal signs (sudden hypotension or bradycardia) or symptoms (dizziness, nausea, discomfort). Subjects were then classified into high-tolerance (n=10) or low-tolerance (n=8) groups. Retrospective analyses indicated that there were no significant differences between the two groups when age, height, weight, percent body fat, total blood volume, and $\dot{V}O_{2max}$ were compared.

Similar results have been reported for 45 healthy women who underwent graded LBNP to 50 mmHg [11]. These women had peak aerobic capacities ranging from 23 to 55 $mL \cdot kg^{-1} \cdot min^{-1}$. There were six women who developed signs and symptoms of impending syncope during LBNP, but retrospective analyses indicted that their average $\dot{V}O_{2max}$ values did not differ from those of the 39 LBNP tolerant females. These authors concluded that orthostatic tolerance was not related to aerobic capacity.

In summary, these series of cross-sectional studies on both men and women employed orthostatic stresses with a presyncopal endpoint. These investigators were unable to draw a statistical relationship between orthostatic tolerance indices and aerobic capacity as measured by $\dot{V}O_{2max}$.

Convertino, Sather, *et al.* [6] applied multiple regression statistics to their previously reported data, which had been collected from 18 subjects, to derive a prediction model for LBNP tolerance. Numerous physical and physiological variables, including age, height, weight, $\dot{V}O_{2max}$, body fat, blood volume, arterial pulse volume, thigh fluid accumulation index, thigh compliance index, heart rate, stroke volume, cardiac output, mean arterial pressure, and peripheral resistance were included in their analyses. $\dot{V}O_{2max}$ did not correlate highly or significantly with the peak level of LBNP achieved prior to onset of presyncopal symptoms. The best predictive model for LBNP tolerance was achieved by including thigh fluid accumulation index, blood volume, and thigh compliance index in the regression model. The accumulation of fluid in the thigh was a major contributor (82 percent) to the prediction model. Based upon this model, the more accumulation of fluid in the thigh, the greater the predicted tolerance to LBNP. Blood volume provided a negative contribution to the prediction equation such that individuals with larger estimated blood volumes were less tolerant to the LBNP stress. Paradoxically, it is a well-known fact that blood volume is increased in endurance-trained athletes [23].

A recent study by Ludwig and colleagues [17] employed a different statistical modeling procedure in a retrospective analysis of 25 women and 22 men who had undergone centrifuge studies $\dot{V}O_{2max}$ ranged from 18 to 50 $mL \cdot kg^{-1} \cdot min^{-1}$. Two biostatistical modeling procedures (proportional hazard and logistic discriminant function) were used to estimate the risk of *gray-out* during centrifugation. Study variables included gender, age, weight, height, percent body fat, base-line heart rate, blood pressure, $\dot{V}O_{2max}$, and plasma volume. Only age, gender, height, and plasma volume significantly contributed to the risk equation. Tall young women with large plasma volumes were at increased risk for orthostatic intolerance. Tolerance to gravitational stress was statistically independent of aerobic fitness ($\dot{V}O_{2max}$).

Longitudinal Studies

Several longitudinal studies have been carried out in which orthostatic tolerance has been carefully assessed before and after an exercise training program. Cooper *et al.* [7] determined the orthostatic tolerance of 11 subjects to +Gz forces produced by centrifugation. These subjects were randomized to two groups: six exercisers and five controls. The exercising group engaged in a daily progressive running program for 3 months, while the controls were asked to avoid vigorous exercise. At the conclusion of the 3 months, the exercising group had increased their endurance capacity by 13 percent. Despite this, there were no significant differences noted between the two groups in their ability to tolerate gravitational stresses.

In a recently published longitudinal study [16], eight young male students underwent a 10-week aerobic exercise training protocol which consisted of alternating sessions on a treadmill and cycle ergometer. This program resulted in a 21 percent increase in $\dot{V}O_{2max}$ (45.7 to 55.2 $mL \cdot kg^{-1} \cdot min^{-1}$). Subjects underwent a presyncopal-limited LBNP test before and after training which consisted of progressive, 10 mmHg increases in LBNP until one of the following criteria was met: (1) a systolic blood pressure <70 mmHg or (2) a decrease of systolic blood pressure of >25 mmHg, diastolic blood pressure of >15 mmHg or heart rate of >15 b/min in two consecutive 1-minute readings. The increases in calf circumference and heart rate and the decreases in forearm blood flow and systolic pressure produced by LBNP were not different after training. Three indices of LBNP tolerance were derived: (1) duration of exposure to LBNP, (2) magnitude of negative

pressure at onset of presyncope and, (3) cumulative stress, determined by summing the products of the duration and pressure magnitude of each LBNP level. None of these indices of tolerance were altered following the 10-week training program. These authors concluded that there was no discernible adverse effect of improved aerobic capacity on orthostatic tolerance over a 10-week training period.

There have been several other longitudinal studies on orthostatic tolerance in which exercise training programs have been employed for as short as 8 days and for as long as 6 months [5, 9, 12, 27]. None of these studies have demonstrated an adverse effect of exercise training on orthostatic tolerance. Only a few of these longitudinal studies included running as the sole type of exercise training. Various combinations of running, bicycling, and weight training have generally been employed.

There is an increasing amount of reported data that suggests that weight training may improve orthostatic tolerance. This may be related to the ability of weight training to increase the muscle mass of the lower extremities that could reduce venous compliance and thereby diminish caudal blood pooling during orthostatic stress [4]. Strength training is also known to increase plasma volume and to preserve baroreceptor reflex function [13].

To summarize these series of studies, it seems clear that in a random population there is no relationship between $\dot{V}O_{2max}$ and orthostatic tolerance. Secondly, longitudinal aerobic training programs from as little as 8 days to 6 months do not adversely influence orthostatic tolerance, despite significant increases in $\dot{V}O_{2max}$. And finally, strength training may improve orthostatic tolerance. However, there are a number of limitations of these previously reported studies that I have overlooked to reach these general conclusions. For example, in many of the reported studies, the level of orthostatic stress was quantitated by measurements of calf blood pooling. This has been done with a strain gauge or an impedance system placed on a lower extremity. These measurements are in fact quite removed from the pressure sensing receptors within the thorax that initiate autonomic reflex responses. It would seem that to best match stresses between groups, a reliable measure of central hemodynamics such as central venous pressure or left ventricular end-diastolic volume should be employed. Moreover, the fluid and electrolyte balance of research volunteers has not been well controlled (if controlled at all). It is conceivable that the endurance-trained athlete comes to the testing lab in a state of hydration that differs from that of sedentary volunteers. Thirdly, the type of exercise employed to achieve aerobic fitness has not been well controlled in the published cross-sectional and longitudinal studies. The degree of blood pooling in the legs may be significantly influenced by the amount of muscle mass that surround and restricts expansion of the venous bed [4]. The aerobically fit individual who participates in bicycling, rope jumping, and weight lifting exercise may have a larger muscle mass in their lower extremities relative to the leg muscles of the endurance-trained runner. Finally, the measurement of aerobic capacity by $\dot{V}O_{2max}$ may have inherent limitations. Up to 75 percent of $\dot{V}O_{2max}$ is determined genetically while only 15 to 25 percent may be influenced by exercise training.

In the next section of this discussion, I will focus my comments on the autonomic nervous system and its role in orthostatic tolerance. Certainly the major mediators of acute cardiovascular adjustments to gravitational stress are the arterial and low-pressure cardiopulmonary baroreceptor reflexes [20, 21]. The arterial baroreceptor reflex modulates both cardiac output and peripheral resistance to maintain blood pressure [20]. An adequate reflex response to gravitational-induced preload reduction is essential for orthostatic tolerance. We have recently examined the cardiac limb of the carotid sinus baroreflex in high-fit and sedentary subjects [2]. This was done with an airtight neck chamber that fit about the anterior two-thirds of the neck. Brief controlled pressure

pulses were administered to the chamber to reduce the transmural pressure at the carotid sinus baroreceptor, and immediate heart rate responses were assessed. Steady state responses are difficult to interpret because as the neck stimulus is maintained blood pressure increases occur. This diminishes the transmural pressure change at the carotid sinus and also alters aortic baroreceptor stretch, such that the net effect in the steady state situation is due to a combination of opposing influences. We, and others [2, 25], have been unable to demonstrate a significant difference in the immediate reflex cardio-acceleration that occurs during carotid sinus hypotension in aerobically fit individuals, when compared to sedentary volunteers.

The arterial baroreflex regulation of vascular resistance has not been adequately compared in fit and unfit humans. When high levels of LBNP are applied to reduce systemic blood pressure and unload arterial baroreceptors, reflex increases in forearm vascular tone can be assessed by plethysmography. In one study, which compared endurance-trained swimmers and nonswimmers, no significant differences in reflex forearm vasoconstrictor responses were noted [10]. However, when endurance-trained football players were studied, reflex increases in forearm vascular resistance were reported to be greater than those recorded from sedentary controls [32]. In contrast, when LBNP was applied to individuals before and after a 12-week cycle exercise program, baroreflex-mediated forearm vascular resistance increases were reported to have decreased [34]. Finally, Raven *et al.* [24] reported that increases in peripheral vascular resistance provoked by LBNP were less in high-fit compared with average-fit individuals.

The low-pressure baroreceptors are located at the junction of the vena cava and the right and left atria, the pulmonary veins, and within the walls of the heart. These are stretch receptors that respond to small changes in intrathoracic blood volume and initiate reflex alterations in splanchnic and muscle vascular resistance to maintain arterial pressure [21]. The integrity of these receptors in the endurance-trained athlete is not clearly known. However, there is reason to believe that neural afferent traffic from these receptors may be altered in the endurance-trained state. For example, it is well known that high levels of aerobic fitness are associated with elevations of plasma and blood volume [23]. This may lead to chronic stimulation of the low-pressure baroreceptors and may alter their discharge pattern. Moreover, ventricular mass is increased in endurance-trained athletes and this may also alter the afferent profile from baroreceptors located within the ventricle. In fact, it has been demonstrated that when the low-pressure baroreceptors of athletes are stimulated by head-out water immersion, there is an attenuated diuresis and natriuresis compared with that noted from immersed nonathletes [3]. This may be due to a blunted cardio-renal reflex or reduced cardiopulmonary baroreceptor-mediated regulation of the renin angiotensin-aldosterone system. These receptors can also be unloaded by application of low levels of LBNP to specifically reduce cardiac filling pressures without lowering systemic blood pressure. There have been several published reports in which the reflex vasoconstrictor responses of trained and untrained individuals have been compared [19, 32, 34]. However, the study populations and research techniques differ and the reported findings are at odds such that no firm conclusions can be reached at this time.

I will not have the time nor space to pursue the possible role of vasoactive hormones in the response to orthostatic challenge in trained and untrained individuals. Changes in these vasoactive hormones occur gradually during orthostatic stress and may play a role during prolonged exposures to higher gravitational forces. However, they may not have an important role in the acute orthostatic responses (first 5 to 10 minutes) in humans.

To summarize the current data on autonomic control of the cardiovascular system, it appears that arterial baroreceptor reflex regulation of cardiac function is not impaired in the endurance-trained athlete. However, it is less clear whether the reflex regulation of peripheral resistance, which may

play an important role in orthostatic tolerance, is altered in the endurance-trained athlete. Future research on this subject should be designed to control for: (l) the type of exercise that is employed to obtain the aerobically fit state, (2) the age of the research volunteers, and (3) should include specific assessment of central hemodynamics during gravitational stress. Moreover, the state of hydration of research volunteers should be carefully controlled and quantitated prior to initiating orthostatic stress testing.

In summary, the ability to withstand orthostatic stress is a function of the reflex responses of the autonomic nervous system and the degree of pooling of blood in the lower extremities and splanchnic region. The data that I have reviewed in this publication strongly suggest that orthostatic tolerance is not linearly related to aerobic capacity. Moreover, baroreflex regulation of cardiac function does not appear to be adversely influenced by high levels of aerobic capacity.

References

[1] ALLEN, S. C., C. L. TAYLOR, AND V. E. HALL. A study of orthostatic inefficiency by tilt board method. *Am. J. Physiol.* 143:11, 1945.

[2] BARNEY, J. A., T. J. EBERT, L. GROBAN, P. A. FARRELL, C. V. HUGHES, AND J. J. SMITH. Carotid baroreflex responsiveness in high-fit and sedentary young men. *J. Appl. Physiol.* 65:2190, 1988.

[3] BOENING, D., H-V. ULMER, U. MEIER, W. SKIPKA, AND J. STEGEMANN. Effects of multi-hour immersion on trained and untrained subjects: I. Renal function and plasma volume. *Aerospace Med.* 43:300, 1972.

[4] CONVERTINO, V. A., D. F. DOERR, J. F. FLORES, G. W. HOFFLER, AND P. BUCHANAN. Leg size and muscle functions associated with leg compliance. *J. Appl. Physiol.* 64:1017, 1988.

[5] CONVERTINO, V. A., L. D. MONTGOMERY, AND J. E. GREENLEAF. Cardiovascular responses during orthostasis: effect of an increase in $\dot{V}O_{2max}$. *Aviat. Space Environ. Med.* 55:702, 1984.

[6] CONVERTINO, V. A., T. M. SATHER, D. J. GOLDWATER, AND W. R. ALFORD. Aerobic fitness does not contribute to prediction of orthostatic intolerance. *Med. Sci. Sports Exerc.* 18:551, 1986.

[7] COOPER, K. H. AND S. LEVERETT, JR. Physical conditioning versus +Gz tolerance. *Aerospace Med.* 37:462, 1966.

[8] CRAMPTON, W. C. The gravity resisting ability of the circulation. Its measurement and significance (blood ptosis). *Am. J. Med. Sci.* 160:721, 1920.

[9] EPPERSON, W. L., R. R. BURTON, AND E. M. BERNAUER. The influence of differential physical conditioning regimens on simulated aerial combat maneuvering tolerance. *Aviat. Space Environ. Med.* 53:1091, 1982.

[10] FALSETTI, H. L., E. R. BURKE, AND J. TRACY. Carotid and cardiopulmonary baroreflex control of forearm vascular resistance in swimmers. *Med. Sci. Sports Exerc.* 15:183, 1983. (Abstract)

[11] FREY, M. A. B., K. L. MATHES, AND G. W. HOFFLER. Relationship between aerobic fitness and responses to lower-body negative pressure in women.

Aviat. Space Environ. Med. 58:1149, 1987.

[12] GREENLEAF, J. E., E. R. DUNN, C. NESVIG, L. C. KEIL, M. H. HARRISON, G. GEELEN, AND S. E. KRAVIK. Effect of longitudinal physical training and water immersion on orthostatic tolerance in men. *Aviat. Space Environ. Med.* 59:152, 1988.

[13] GREENLEAF, J. E., R. F. HAINES, E. M. BEMAUER, J. T. MORSE, H. SANDLER, R. ARMBRUSTER, L. SAGAN, AND W. VAN BEAUMONT. +Gz tolerance in man after 14-day bedrest periods with isometric and isotonic exercise conditioning. *Aviat. Space Environ. Med.* 46:671, 1975.

[14] HUDSON, D. L., M. L. SMITH, H. GRAITZER, AND P. B. RAVEN. Fitness-related differences in response to LBNP during sympathetic blockade with metoprolol. *Fed. Proc.* 45:643, 1986. (Abstract)

[15] KLEIN, K. E., H. BRUNER, D. JOVY, L. VOGT, AND H. M. WEGMANN. Influence of stature and physical fitness on tilt-table and acceleration tolerance. *Aerospace Med.* 40:293, 1969.

[16] LIGHTFOOT, J. T., R. P. CLAYTON, D. J. TOROK, T. W. FOURNELL, AND S. M. FORTNEY. Aerobic training does not affect lower body negative pressure tolerance. *J. Appl. Physiol.* (In press, 1989).

[17] LUDWIG, D. A., V. A. CONVERTINO, D. J. GOLDWATER, AND H. SANDLER. Logistic risk model for the unique effects of inherent aerobic capacity and +Gz tolerance before and after simulated weightlessness. *Aviat. Space Environ. Med.* 58:1057, 1987.

[18] LUFT, U. C., L. G. MYHRE, J. A. LOEPPKY, AND M. D. VENTERS. A study of factors affecting tolerance of gravitational stress simulated by lower body negative pressure. *Research Report on Specialized Physiology Studies in Support of Manned Space Flight,* NASA Contract NAS 9-14472. Albuquerque, NM: Lovelace Foundation, 1976, p. 1.

[19] MANCIA, G. AND A. L. MARK. Arterial baroreflexes in humans. In: *Handbook of Physiology, Section 2: The Cardiovascular System, Volume III: Peripheral Circulation and Organ Blood Flow, Part 2,* edited by J. T. Shepherd and F. M. Abboud. Bethesda, MD: American Physiological Society, 1983, p. 755.

[20] MARK, A. L. AND G. MANCIA. Cardiopulmonary baroreflexes in humans. In: *Handbook of Physiology, Section 2: The Cardiovascular System, Volume III: Peripheral Circulation and Organ Blood Flow, Part 2,* edited by J. T. Shepherd and F. M. Abboud. Bethesda, MD: American Physiological Society, 1983, p. 795.

[21] MAYERSON, H. S. Roentgenkymographic determination of cardiac output in syncope induced by gravity. *Am. J. Physiol.* 138:630, 1942.

[22] OSCAI, L. B., B. T. WILLIAMS, AND A. B. HERTIG. Effect of exercise on blood volume. *J. Appl. Physiol.* 24:622, 1968.

[23] RAVEN, P. B., D. ROHM-YOUNG, AND G. BLOMQVIST. Physical fitness and cardiovascular response to lower body negative pressure. *J. Appl. Physiol.* 56: 138, 1984.

[24] REILING, M. J. AND D. R. SEALS. Respiratory sinus arrhythmia and carotid baroreflex control of heart rate in endurance athletes and untrained controls. *Clin. Physiol.* 8:511, 1988.

[25] SATHER, T. M., D. J. GOLDWATER, L. D. MONTGOMERY, AND V. A. CONVERTINO. Cardiovascular dynamics associated with tolerance to lower body negative pressure. *Aviat. Space Environ. Med.* 57:413, 1986.

[26] SHVARTZ, E. Relationship between endurance and orthostatic tolerance. *J. Sports Med.* 8:75, 1968.

[27] SHVARTZ, E., A. MEROZ, A. MAGAZANIK, Y. SHOENFELD, AND Y. SHAPIRO. Exercise and heat orthostatism and the effect of heat acclimation and physical fitness. *Aviat. Space Environ. Med.* 48:836, 1977.

[28] SMITH, M. L., H. M. GRAITZER, D. L. HUDSON, AND P. B. RAVEN. Baroreflex function in endurance-and static exercise-trained men. *J. Appl. Physiol.* 64:585, 1988.

[29] STEGEMANN, J., A. BUSERT, AND D. BROCK. Influence of fitness on the blood pressure control system in man. *Aerospace Med.* 45:45, 1974.

[30] TAKESHITA, A., S. JINGU, T. IMAIZUMI, Y. KUNIHIKO, S. KOYANAGI, AND M. NAKAMURA. Augmented cardiopulmonary baroreflex control of forearm vascular resistance in young athletes. *Circ. Res.* 59:43, 1986.

[31] TURNER, A. H. The circulatory minute volume of healthy young women in reclining, sitting and standing positions. *Am. J. Physiol.* 80:605, 1927.

[32] VROMAN, N.B., HEALY, J.A., AND KERTZER, R. Cardiovascular responses to lower body negative pressure (LBNP) following endurance training. *Aviat. Space Environ. Med.* 59:330, 1988.

KAS

IN

Done

MH

N94-28366

Does Training-Induced Orthostatic Hypotension Result from Reduced Carotid Baroreflex Responsiveness?

55-52
207614
P-12

James A. Pawelczyk, Ph.D. and Peter B. Raven, Ph.D.
Department of Physiology
Texas College of Osteopathic Medicine
Fort Worth, Texas

Introduction

As manned space travel has steadily increased in duration and sophistication the answer to a simple, relevant question remains elusive. Does endurance exercise training (high intensity rhythmic activity, performed regularly for extended periods of time) alter the disposition to, or severity of, postflight orthostatic hypotension? Research results continue to provide different views; however, data are difficult to compare because of the following factors that vary between investigations:

- the type of orthostatic stress imposed (+Gz, lower body negative pressure [LBNP], head-up tilt);
- pretest perturbations used (exercise, heat exposure, head-down tilting, bed rest, water immersion, hypohydration, pharmacologically-induced diuresis;
- the length of the training program used in longitudinal investigations (days versus weeks versus months);
- the criteria used to define fitness; and
- the criteria used to define orthostatic tolerance.

Generally, research results indicate that individuals engaged in aerobic exercise activities for a period of years have been reported to have reduced orthostatic tolerance compared to untrained control subjects [9, 13, 15, 19, 22], while the results of shorter term longitudinal studies remain equivocal [5, 12]. Such conclusions suggest that chronic athletic training programs reduce orthostatic tolerance, whereas relatively brief (days to weeks) training programs do not affect orthostatic tolerance to any significant degree (increase or decrease).

Our laboratory has established a primary objective to identify the alterations in blood pressure control that contribute to training-induced orthostatic hypotension (TIOH). Although any aspect of blood pressure regulation is suspect, current research has been focused on the baroreceptor system. Reductions in carotid baroreflex responsiveness have been documented in exercise-trained rabbits [6], reportedly due to an inhibitory influence from cardiac afferent (presumably vagal) nerve fibers that is abolished with intrapericardiac denervation [7]. The purpose of this investigation was to attempt to determine if similar relationships existed in men with varied levels of fitness (using maximal aerobic power, $\dot{V}O_{2max}$, as the marker of fitness).

Methods and Procedures

Subjects

Volunteers were recruited from the Dallas-Fort Worth metropolitan area and local universities. Each prospective subject was informed of the nature and purpose of each procedure and was asked to provide written informed consent for the procedure. All procedures conformed to the Declaration of Helsinki and were approved by the Institutional Review Board at the Texas College

of Osteopathic Medicine. Screening procedures included a physical examination, resting 12-lead electrocardiogram, graded-exercise test to volitional fatigue for determination of metabolic responses to exercise, $\dot{V}O_{2max}$, and a resting echocardiogram. Subjects were classified according to $\dot{V}O_{2max}$ into one of three groups: low fit ($\dot{V}O_{2max}$ <40 mL•kg^{-1}•min^{-1}); mid fit ($\dot{V}O_{2max}$=50±2 mL•kg^{-1}•min^{-1}), and high fit ($\dot{V}O_{2max}$ >65 mL•kg^{-1}•min^{-1}). The groups of subjects reported diverse exercise histories. In the low-fit group, no subject engaged in regular exercise and all reported that their present jobs did not require any great amount of physical activity. To the contrary, all subjects in the high-fit group were trained athletes (competing in bicycle races) and trained 4 to 7 days per week for 1 to 3 hours each day, and traveled at least 150 miles (and as much as 450 miles) during a week of training. The mid-fit group encompassed elements of the low-fit and mid-fit group. Six of the eight subjects were recreational athletes, of these subjects all engaged in mixed modes of training (primarily running and weight lifting) approximately two to three times per week. The other two subjects did not engage in recreational sports, but maintained lifestyles that required some aerobic activity during the day.

Lower Body Negative Pressure (LBNP)

Venous return was reduced using graded LBNP distal to the subjects' iliac crests. Subjects were exposed to levels of LBNP at -5, -10,-15, -20, -35 and -50 torr, with each stage lasting 10 minutes. Heart rate and blood pressure were monitored continuously from the electrocardiogram and a radial artery, respectively. The final 5 minutes of the stage were reserved for assessments of carotid baroreflex responsiveness. Electromyographic activity of abdominal and quadriceps muscle groups was monitored continuously to ensure that each subject remained relaxed during the LBNP protocol, and the protocol was terminated if a subject demonstrated signs of lipothymia (derivation Greek: leipein - to fail, thymos - mind).

Central Venous Pressure

Hydrostatically-dependent peripheral venous pressure was determined on a separate day using an abbreviated version of the LBNP protocol outlined above using the *dependent arm* method of Gauer and Sieker [10]. Central venous pressure was estimated directly by zeroing the transducer to the subjects' mid-sternal line. In all experiments a prominent venous pressure wave form was observed, and venous pressure could be rapidly increased by performing a Valsalva's maneuver or passively elevating the subjects' legs.

Arterial Blood Pressure

Systolic, diastolic, mean, and pulse pressures were measured directly from a radial artery using a 1.1 mm OD, 3.2 cm Teflon catheter inserted by a collaborating anesthesiologist. Lidocaine (1 percent) was injected subcutaneously prior to catheterization to minimize discomfort. Blood pressure was transduced using a Hewlett-Packard system (Model 1280C transducer and Model 78205 amplifier) and was monitored in real time by computer. The signal to noise ratio of this system allowed us to resolve changes in pressure greater than 0.32 torr.

Carotid Baroreceptor Responsiveness

Carotid baroreflex control of heart rate and blood pressure was assessed using the rapid neck pressure and neck suction protocol outlined by Sprenkle *et al.* [20] using a malleable neck collar [8]. At least five trains with correlation coefficients between neck chamber pressure and R-R interval in excess of 0.80 were obtained during each stage of LBNP for later averaging and analysis. The neck pressure/suction system was controlled using custom software developed for a laboratory minicomputer (Digital Equipment Corporation MINC-23, Maynard, MA, with performance enhancements by Adcomp, Inc., Amesbury, MA). Beat-to-beat changes in blood pressure and heart rate were determined from computerized analysis of the radial artery pressure wave form and the ECG.

Data Analyses

Carotid baroreflex stimulus-response curves using either R-R interval or mean arterial pressure as the dependent variable were fit to a four-parameter logistic function described by Kent *et al.* [11] using the equation:

$$\text{R-R interval or mean arterial pressure } A_1 \cdot \{1+e[A_2(\text{estimated carotid sinus pressure}-A_3)]\}-1+A_4$$

where:
A_1 = the range of the response (maximum-minimum)
A_2 = a gain coefficient that is a function of carotid sinus pressure
A_3 = the carotid sinus pressure required to elicit equal pressor and depressor responses
A_4 = the minimum response of mean arterial pressure or R-R interval

Using this model, the operating, threshold, and saturation pressures for a baroreflex response can be determined. Carotid sinus pressure was estimated for each heart beat in the stimulus train as the difference of radial mean pressure and neck chamber pressure, assuming that neck chamber pressures were fully transmitted to the carotid sinuses. The gain (i.e., slope) of the carotid-cardiac and carotid-blood pressure baroreflexes was determined from the first derivative of the logistic function. Maximum gain was calculated as the gain at the neck pressure equal to parameter A_3, the centering point of the function. This point corresponds to the steepest slope of the stimulus-response curve. Mean arterial pressure or R-R interval data for the group during carotid baroreflex stimulation at each stage of LBNP were fitted to the function using nonlinear least squares regression (Statistical Analysis System, Cary, NC). Coefficients of determination (r^2) of the fitted lines ranged from 99.80 to 99.99 percent. The interaction of cardiopulmonary and carotid baroreceptors was then described by the relationship carotid baroreflex gain and central venous pressure, as central venous pressure was reduced using LBNP [14].

Results

Subjects

Characteristics of the subject groups are presented in Table 1. The groups were well matched for age, height, lean body mass, and resting blood pressure, but differed on several variables related to aerobic conditioning.

Table 1. *Physical characteristics of the subject groups*

	LF (n=8)	MF (n=8)	HF (n=8)	DIF
Age (yrs)	28.4±1.1	24.6±0.8	27.4±0.9	2
Height (cm)	179.1±2.0	179.6±2.3	181.1±1.7	
Weight (kg)	87.7±5.1	78.2±2.6	75.3±2.1	
LBM (kg)	68.0±2.8	68.7±2.1	67.3±1.6	
Resting HR (beat•min^{-1})	68.9±2.9	62.2±1.8	58.2±2.0	1,3
SBP (torr)	139.6±6.9	148.2±4.9	140.9±2.6	
DBP (torr)	70.9±4.1	69.4±2.8	72.6±2.5	
MAP (torr)	92.2±4.9	91.2±3.1	92.1±2.5	
EDD (cm)	4.8±0.1(3)	4.9±0.0(4)	5.4±0.1(6)	1,3
Blood Volume (mL•kg^{-1})	75.1±3.8	97.2±9.2	117.0±5.3	1,2
$\dot{V}O_{2max}$ (mL•kg^{-1}•min^{-1})	38.9±1.5	50.9±1.4	67.0±1.9	1,2,3

1. Significant difference between LF and HF groups, $p<0.05$.
2. Significant difference between LF and MF groups, $p<0.05$.
3. Significant difference between HF and MF groups, $p<0.05$. Values represent mean ±SEM. Numbers in parentheses=n when n≠8. HR=heart rate; SBP=systolic blood pressure; DBP = diastolic blood pressure; MAP=mean arterial pressure; EDD=end-diastolic dimension of the left ventricle; $\dot{V}O_{2max}$=maximal aerobic power.

$\dot{V}O_{2max}$ differed significantly between groups, as dictated by our screening efforts to define three distinct subject groups. Other hallmarks of aerobic conditioning (resting and maximal heart rate, heart size, and blood volume) were different between groups. As a whole, analysis of these variables suggested that the groups presented distinctly different autonomic and morphological characteristics associated with their training status.

Carotid Baroreflex Responsiveness

Results from logistic modeling of the neck pressure/suction data are summarized in Tables 2 and 3. The carotid-cardiac vagal reflex, using R-R interval as the response to a change in carotid transmural pressure, appeared to be enhanced by athletic training, since the A_1 (range) parameter was augmented in the high-fit group. As will be discussed, this conclusion may result from inappropriate representation of the data using R-R interval.

Table 2. *Effect of fitness of carotid baroreflex control of R-R interval*

	LF (n=8)	MF (n=8)	HF (n=8)	DIF
A_1	397±27	463±32	565±54	1
A_2	0.190±0.028	0.200±0.045	0.224±0.034	
A_3	106.4±2.6	104.8±2.6	93.7±2.0	
A_4	712±21	773±20	835±25	1,2,3
Threshold (torr)	93.9±3.3	89.7±3.1	80.5±4.5	
Saturation (torr)	118.9±2.4	119.9±2.7	113.0±4.5	
Operating Point (msec)	818±23	897±24	1027±32	1,2,3
Maximum Gain (msec•torr^{-1})	15.8±2.0	20.5±4.6	22.6±2.6	
Response at Threshold (msec)	797±20	871±20	954±33	
Response at Saturation (msec)	1029±24	1138±20	1280±33	1,2,3
Threshold-Saturation (msec)	232±16	267±18	326±31	1

1. Significant difference between LF and HF groups, $p<0.05$.
2. Significant difference between LF and MF groups, $p<0.05$.
3. Significant difference between HF and MF groups, $p<0.05$. Values represent mean ±SEM. A_1=maximum-minimum; A_2=gain coefficient; A_3=entering point; A_4=minimum response.

Table 3 suggests that a major difference between fitness groups lay in the ability to alter blood pressure when carotid sinus transmural pressure was changed. The range of responses was reduced significantly in the high-fit group compared to the other two groups. It is possible that this alteration may partially account for the TIOH reported in other investigations.

Table 3. *Effect of Fitness of Carotid Baroreflex Control of Blood Pressure*

	LF (n=8)	MF (n=8)	HF (n=8)	DIF
A_1	20.8±1.9	24.8±1.9	16.2±1.7	1,3
A_2	0.081±0.011	0.078±0.007	0.093±0.010	
A_3	95.9±5.0	110.9±3.7	88.8±3.1	2,3
A_4	106.6±2.5	102.4±1.7	100.3±1.7	1
Threshold (torr)	72.3±7.3	85.6±4.5	69.9±3.7	2,3
Saturation (torr)	119.6±3.7	136.1±5.6	107.8±3.6	2
Operating Point (torr)	96.5±2.6	95.8±1.7	91.4±1.8	
Maximum Gain	0.365±0.054	0.420±0.034	0.340±0.036	
Response at Threshold (torr)	102.2±2.4	97.1±1.8	96.9±1.6	
Response at Saturation (torr)	90.2±2.3	82.8±2.6	87.6±1.7	2
Threshold-Saturation (torr)	12.0±1.1	14.3±1.1	9.4±1.0	1,3

1. Significant difference between LF and HF groups, $p<0.05$.
2. Significant difference between LF and MF groups, $p<0.05$.
3. Significant difference between HF and MF groups, $p<0.05$. Values represent mean ±SEM. A_1=maximum-minimum; A_2=gain coefficient; A_3=centering point; A_4=minimum response.

Cardiopulmonary Receptor Modulation of Carotid Baroreflex Responsiveness

We hypothesized that reductions in cardiac filling pressure using LBNP would remove the inhibition of carotid baroreflex responsiveness normally exhibited by cardiopulmonary baroreceptors, leading to augmented carotid baroreflex gain.

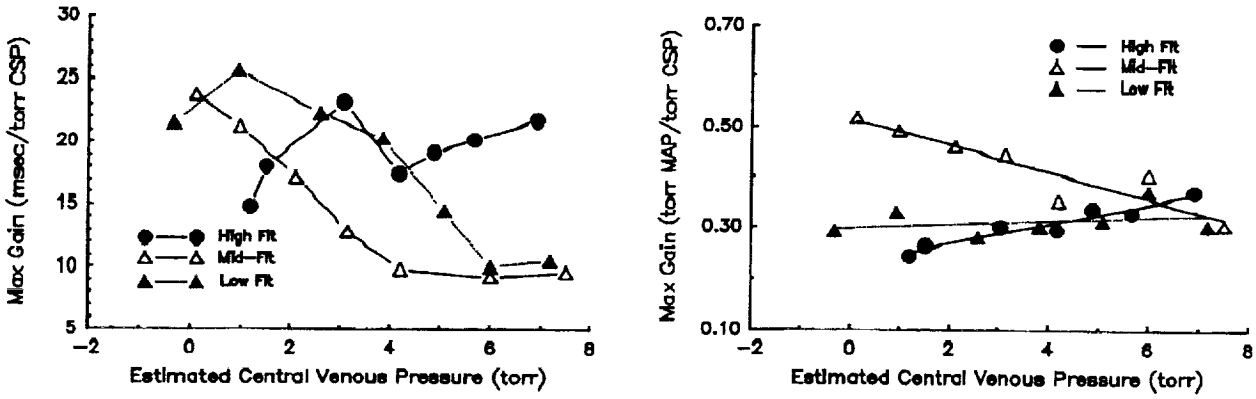


Figure 1. *Gain of the carotid-cardiac (1A) and the carotid-blood pressure (1 B), stimulus-curves when central venous pressure (CVP) was reduced using lower-body negative pressure (LBNP). In A, gain was inversely related to CVP in the LF and MF groups, Gain did not increase when CVP was reduced in the HF group. In B, gain decreased in the HF group when CVP was reduced, whereas it was unchanged in the LF group and increased in the MF group. The data suggest that an inhibitory influence from cardiopulmonary receptors on carotid baroreflex responsiveness is not removed by reducing central blood volume in the HF group. CSP=carotid sinus pressure; MAP=mean arterial pressure; LF=low fit; MF=mid-fit; HF=high fit.*

Figure 1 demonstrates that this relationship existed for the carotid-cardiac baroreflex in the low-fit and mid-fit groups, and for the carotid-blood pressure baroreflex in the mid-fit group only. Reflex gain consistently failed to increase, or actually decreased in the high-fit group during LBNP. Therefore, we were unable to eliminate cardiopulmonary receptor inhibition in the high-fit group by reducing central blood volume.

Discussion

We conducted this investigation to determine whether carotid baroreflex control of heart rate and blood pressure was different in men with varied levels of fitness (defined as $\dot{V}O_{2max}$). Furthermore, we hoped to determine whether any observed differences could be attributed to an inhibitory influence from cardiopulmonary mechanoreceptors. Changes in this regulatory system may potentially play a role in the changes in blood pressure regulation that are associated with space-flight cardiovascular deconditioning. Therefore, this discussion is divided into three sections: (1) carotid baroreflex control of cardiac chronotropism; (2) carotid baroreflex control of vascular resistance; and (3) the influence of cardiopulmonary receptors on carotid baroreflex responsiveness.

Carotid Baroreflex Control of Cardiac Chronotropism

Increases in carotid sinus distending pressure are well known to increase acetylcholine release at the sino-atrial node, hyperpolarizing pacemaker cells, and slowing the rate of phase four depolarization by reducing the permeability of *leak* membrane channels. The net result of this effect is to reduce the sino-atrial node firing rate, or increase the time required to the next depolarization. Based on this electrophysiologic point of view, some investigators have argued that the carotid-cardiac baroreflex is most properly described using the time interval between adjacent P waves of the ECG (or the analogous R-R interval) as a measure of the response to a baroreceptor stimulus.

Two points emphasize the problems with what Rowell termed, "this fallacious scheme" [16]: (1) The R-R interval scheme would indicate improved baroreflex responsiveness whenever heart rate was reduced [16]. Figure 2 illustrates that the relationship between R-R interval and heart rate is not linear; therefore, large changes in R-R interval are required to change heart rate by some increment if the initial heart rate is low. (2) The contribution of the carotid-cardiac baroreflex to maintaining blood pressure is more usefully described using heart rate rather than R-R interval, since heart rate is directly proportional to cardiac output and hence blood pressure.

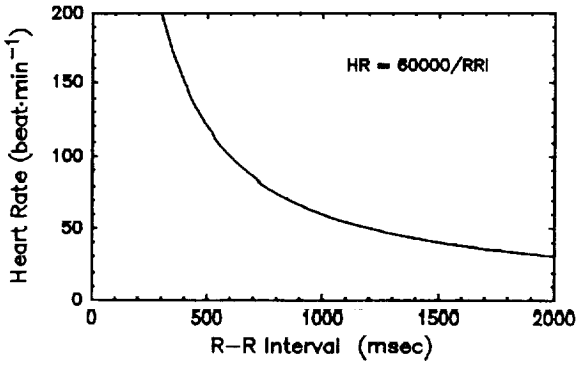


Figure 2. *Relationship between heart rate (HR) and R-R interval (RRI). The curve can be described by the relationship HR (beat•min^{-1})=60000/RRI (msec). Note that for a given change in heart rate, the change in R-R interval will be greater if the initial heart rate is low.*

Other investigators have reported that changes in R-R interval during neck suction or neck pressure are greater in endurance athletes [1]. When we expressed these data as changes in heart rate, no differences were apparent between groups other than the reduction in resting heart rate normally attributed to exercise training. Analysis of our data in this fashion revealed identical findings. Figure 3a represents modelled curves of the carotid-cardiac stimulus-response relationship for the three groups, using R-R interval as the dependent variable. The figure suggests, and statistical analysis of the parameters describing the curve (Table 2) corroborates, that the range and gain of the reflex is greater in the high-fit group. Figure 3b represents the same data using heart rate rather than R-R interval as the dependent variable. It is apparent, both visually and statistically, that no difference exists between groups other than the baseline heart rate.

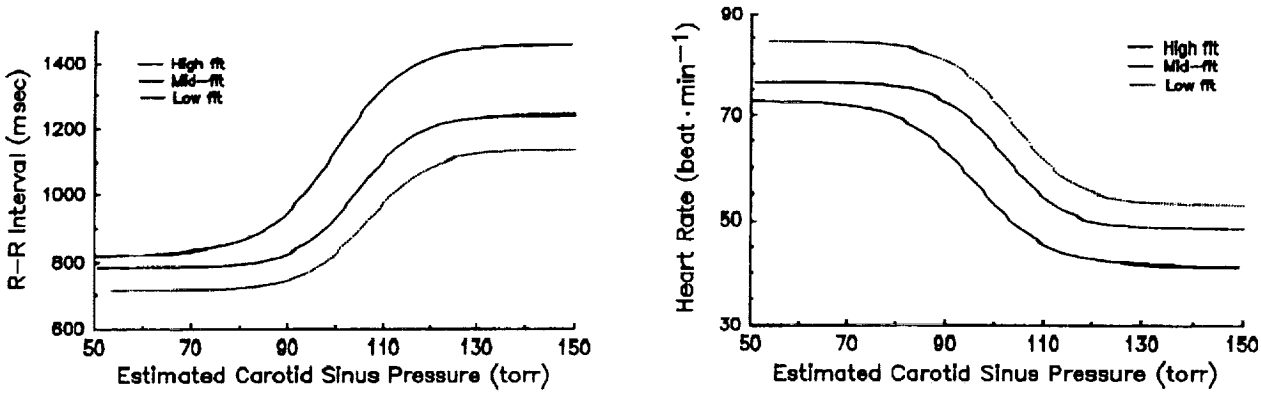


Figure 3. *Carotid-cardiac baroreflex with the response expressed as R-R interval (RRI, 3A) or heart rate (HR, 3B). A was generated using the model parameters in Table 1; B was generated from the data in A and the conversion formula in Figure 1. In A the range and slope of the curves are significantly different between groups, suggesting that the reflex response is enhanced proportionate to fitness level. In B there are no differences between groups in the slopes or ranges of the curves. Using this analysis, one would conclude that the reflex response is not different between curves, except for bradycardia proportionate to fitness level. LF=low fit; MF=mid-fit; HF=high fit.*

To summarize, although the changes in R-R interval due to neck pressure and neck suction were greatest in the high-fit group, the changes in heart rate were not different between groups. We concluded that the carotid-cardiac baroreflex plays no greater or more effective role in helping to maintain the blood pressure of endurance athletes.

Carotid Baroreflex Control of Blood Pressure

An unique aspect of this investigation was our ability to measure the blood pressure responses to rapid changes in carotid sinus distending pressure. It is likely that changes in the regulation of vascular resistance contribute more to alterations in orthostatic tolerance than do changes in the control of heart rate, since vascular resistance can be changed more widely during physiological stresses than can heart rate. For example, during exercise regional vascular resistance can decrease by as much as 20 to 30 fold, while heart rate can be altered only 3 to 4 fold [16].

While a reduction in carotid baroreflex control of arterial resistance has been well documented in endurance exercise trained rats [2, 23], only one published investigation has reported changes in carotid baroreflex control of blood pressure or vascular resistance in trained men [21]. However, the use of long pressure periods (3 minutes) that allows time for other mechanoreceptor reflexes to act and the use of full head enclosure that may affect blood pressure regulating oculomotor reflexes confound the interpretation of these data.

We sought to minimize these effects by examining the responses to *rapid* changes in pressure *applied to the neck only*. Our major finding, that changes in blood pressure to standardized changes in carotid sinus distending pressure, suggests that carotid baroreflex control of vascular resistance is less effective in endurance trained men. This finding is similar to findings reported by Stegemann *et al.* [21], measurements of muscle sympathetic nerve activity during LBNP in endurance trained men [18], and measurements of renal sympathetic nerve activity during experimental hemorrhage in exercise trained rabbits [7].

However, these data may be interpreted in an alternative fashion. If other baroreceptors (presumably those in the aortic arch) responded rapidly enough to oppose the systemic blood pressure *swing* we induced with rapid neck pressure/neck suction, then the decreased response we elicited in endurance athletes could be due to increased activity of aortic baroreceptors, rather than to reduced activity of carotid baroreceptors.

Recently Sanders *et al.* [17] suggested that aortic baroreceptors play a more dominant role in regulating muscle sympathetic nerve activity, and presumably vascular resistance, than carotid baroreceptors. Due to the anatomical location of aortic baroreceptors (i.e., relatively near the hydrostatic *indifferent* point of the body), aortic baroreceptors would sense little change in blood pressure in the transition from the supine to standing position. To the contrary, the upright posture should reduce carotid sinus blood pressure some 15 to 20 torr by changes in hydrostatic pressure alone; thus, carotid baroreceptors would seem better suited to sense a blood pressure *error signal* resulting from a postural change. Therefore, a suggestion of improved aortic baroreceptor responsiveness may be the hallmark of a baroreceptor system less able to sense changes in posture and less able to prevent orthostatic hypotension.

Influences of Cardiac Receptors on Carotid Baroreflex Responsiveness

An inhibitory influence of cardiac receptors on carotid baroreflex responsiveness has been documented in man and several animal species [3, 7, 24]. DiCarlo and Bishop [7] using rabbits were the first to demonstrate that this influence was augmented with endurance exercise training, since blockade of cardiac receptors with procaine injected into the pericardial sack restored the renal sympathetic nerve response to progressive vena cava occlusion. We hypothesized that progressive reductions in cardiac filling pressure using LBNP might alter cardiac mechanoreceptors such that carotid baroreflex responsiveness was improved.

Figures 1a and 1b suggest that the low-fit and mid-fit groups responded as hypothesized, since the gain of the heart rate or blood pressure baroreflex was inversely related to central venous pressure. However, neither the heart rate nor the blood pressure baroreflexes were improved during LBNP in the high-fit group, suggesting that inhibition was maintained despite reduced cardiac filling, and that the inhibition was not due to an augmented blood volume alone.

The nature of this inhibition remains poorly known. DiCarlo and Bishop could not remove the inhibition with reductions in cardiac filling pressure, lending support to our contention that training-induced expansions in blood volume caused increased inhibition. Perhaps the cardiac hypertrophy of endurance athletes stretched mechanoreceptive areas of the athletes' hearts in a manner not eliminated by volume changes alone. This speculative hypothesis deserves further examination.

Orthostatically-induced Lipothymia

Although the intent of this investigation was not to cause syncope, four subjects (two high-fit and two low-fit) experienced lipothymia. Considering the poor baroreflex control of blood pressure in the high-fit group, it is unlikely that similar physiological mechanisms precipitated lipothymia in the low-fit and high-fit subjects. Blomqvist [4] proposed that orthostatic tolerance could be broadly categorized according to adrenergic responsiveness and blood volume status. Based on this framework, and the data presented previously, we suggest that the high-fit subjects experienced a hypoadrenergic form of orthostatic hypotension, while the low-fit subjects

experienced a hypovolemic form of orthostatic hypotension. (As Table 1 illustrates, the blood volume of the low fit group, on a kilogram body weight basis, was more than one third less than the high-fit group).

The operational implications of these different forms of orthostatic hypotension, and the efficacy of countermeasures to ameliorate each form, are unknown. It is reasonable to hypothesize that a variety of countermeasures are available that affect orthostatic hypotension, but that specific countermeasures would act more preferentially on the hypovolemic or the hypoadrenergic variety. We suggest that orthostatic countermeasures, like exercise, should be prescribed individually. However, this approach will require future investigations to determine the sequelae of events that precipitate lipothymia.

References

[1] BARNEY, J. A., T. J. EBERT, L. GROBAN, P. A. FARRELL, VINCENT, C. HUGHES, AND J. J. SMITH. Carotid baroreflex responsiveness in high fit and sedentary young men. *J. Appl. Physiol.* 65:2190-2194, 1988.

[2] BEDFORD, T. G. AND C. M. TIPTON. Exercise training and the arterial baroreflex. *J. Appl. Physiol.* 63:1926-1932, 1987.

[3] BILLMAN, G. E., D. T. DICKEY, K. K. TEOH, AND H. L. STONE. Effects of central venous blood volume shifts on arterial baroreflex control of heart rate. *Am. J. Physiol. (Heart Circ. Physiol.)* 241:H571-575, 1981.

[4] BLOMQVIST, C. G. Orthostatic hypotension. *Hypertension* 8:722-731, 1986.

[5] CONVERTINO, V. A., L. D. MONTGOMERY, AND J. E. GREENLEAF. Cardiovascular responses during orthostasis: effect of an increase in $\dot{V}O_{2max}$. *Aviat. Space. Environ. Med.* 55:702-708, 1984.

[6] DICARLO, S. E. AND V. S. BISHOP. Exercise training attenuates baroreflex regulation of renal nerve activity in rabbits. *Am. J. Physiol. (Heart Circ. Physiol.)* 255:H974-979, 1988.

[7] DICARLO, S. E. AND V. S. BISHOP. Exercise training augments cardiac receptor inhibition of baroreflex function in conscious rabbits. *FASEB J.* 2:A717, 1988.

[8] ECKBERG, D. L., M. S. CAVANAUGH, AND A. L. MARK. A simplified neck suction device for activation of carotid baroreceptors. *J. Lab. Clin. Med.* 85:167-173, 1975.

[9] FALSETTI, H. L., E. R. BURKE, AND J. TRACY. Carotid and cardiopulmonary baroreflex control of forearm vascular resistance in swimmers. *Med. Sci. Sports Exerc.* 15:183, 1983. (Abstract)

[10] GAUER, O. H. AND H. O. SIEKER. The continuous recording of central venous pressure changes from an arm vein. *Circ. Res.* 4:74-78,1956.

[11] KENT, B. B., J. W. DRANE, B. BLUMENSTEIN, AND J. W. MANNING. A mathematical model to assess changes in the baroreceptor reflex. *Cardiol.* 57:295-310, 1972.

[12] LIGHFFOOT, J. T., R. P. CLAYTOR, D. J. TOROK, T. W. JOURNELL, AND S. M. FORTNEY. Ten weeks of

aerobic training do not affect lower body negative pressure responses. *J. Appl. Phvsiol.* 67:894-901, 1989.

[13] MANGSETH, G. R. AND E. M. BERNAUER. Cardiovascular response to tilt in endurance trained subjects exhibiting syncopal reactions. *Med. Sci. Sports Exerc.* 12:140, 1980. (Abstract)

[14] PAWELCZYK, J. A. AND P. B. RAVEN. Reductions in central venous pressure improve carotid baroreflex responses in conscious men. *Am. J. Phvsiol. (Heart Circ. Physiol.).* (In press)

[15] RAVEN, P. B., D. ROHM-YOUNG, AND C. G. BLOMQVIST. Physical fitness and cardiovascular response to lower body negative pressure. *J. Appl. Physiol.: Respirat. Environ. Exercise Phvsiol.* 56:138-144, 1984.

[16] ROWELL, L. B. *Human Circulation: Regulation during Physical Stress.* Oxford: Oxford University Press, 1986.

[17] SANDERS, J. S., D. W. FERGUSON, AND A. L. MARK. Arterial baroreflex control of sympathetic nerve activity during elevation of blood pressure in normal man: dominance of aortic baroreflexes. *Circ.* 77:279-288,1988.

[18] SEALS, D. R., L. A. CLAYTON-BARE, AND M. J. REILING. Sympathetic responses to excitatory stimuli in endurance athletes and untrained subjects. *Physioloqist* 31:A103, 1988.

[19] SMITH, M. L. AND P. B. RAVEN. Cardiovascular responses to lower body negative pressure in endurance and static exercise-trained men. *Med. Sci. Sports Exerc.* 18:545-550, 1986.

[20] SPRENKLE, J. M., D. L. ECKBERG, R. L. GOBLE, J. J. SCHELHORN, AND H. C. HALLIDAY. Device for rapid quantification of human carotid baroreceptor-cardiac reflex responses. *J. Appl. Physiol.* 60:727-732, 1986.

[21] STEGEMANN, J., A. BUSERT, AND D. BROCK. Influence of fitness on the blood pressure control system in man. *Aerospace Med.* 45:45-48, 1974.

[22] StEGEMANN, J., U. MEIER, W. SKIPKA, W. HARTLIEB, B. HEMMER, AND U. TIBES. Effects of a multi-hour immersion with intermittent exercise on urinary excretion and tilt-table tolerance in athletes and nonathletes. *Aviat. Space Environ. Med.* 46:26-29, 1975.

[23] TIPTON, C. M., R. D. MATTHES, AND T. G. BEDFORD. Influence of training on the blood pressure changes during lower body negative pressure in rats. *Med. Sci. Sports Exerc.* 14:81-90, 1982.

[24] VICTOR, R. G. AND A. L. MARK. Interaction of cardiopulmonary and carotid baroreflex control of vascular resistance in humans. *J. Clin. Invest.* 76:1592-1598, 1986.

Acknowledgement

The authors gratefully acknowledge the clinical assistance of Steven Stern, D.O. and Kevin Wilson, D.O. We also thank Glen H. J. Stevens for his technical assistance and the subjects for their cheerful cooperation. This study was supported, in part, by AOA #86-11-203, NIH #HL34397 and NIH #HL43202. James A. Pawelczyk's doctoral research was supported by NIH #T32-HL07652.

N94-28367

Cardiac Output and Cardiac Contractility by Impedance Cardiography during Exercise of Runners

56-52
207615
P-9

W. G. Kubicek, BSEE, Ph.D. and R. A. Tracy, Ph.D.
Department of Physical Medicine and Rehabilitation
University of Minnesota Medical School
Minneapolis, Minnesota

Historical Perspective

Most of the solid state electronic engineering of the system now generally known as the Minnesota Impedance Cardiograph was performed with the support of a 5-year contract, NAS9-4500, with the NASA Lyndon B. Johnson Space Center, Houston, Texas. This contract ran from 1965 to 1970, W. G. Kubicek, Principal Investigator. In addition to the engineering design and development of the hardware (Figure 1), the contract called for testing on both animals and human subjects. This project also provided funds to construct twenty impedance cardiographs and place them in selected research and clinical facilities for further evaluation. This, then, led to the First Symposium on Impedance Cardiography, held at the NASA Lyndon B. Johnson Space Center, Houston, Texas, June 2 to June 4, 1969. Twenty-four excellent papers were presented.

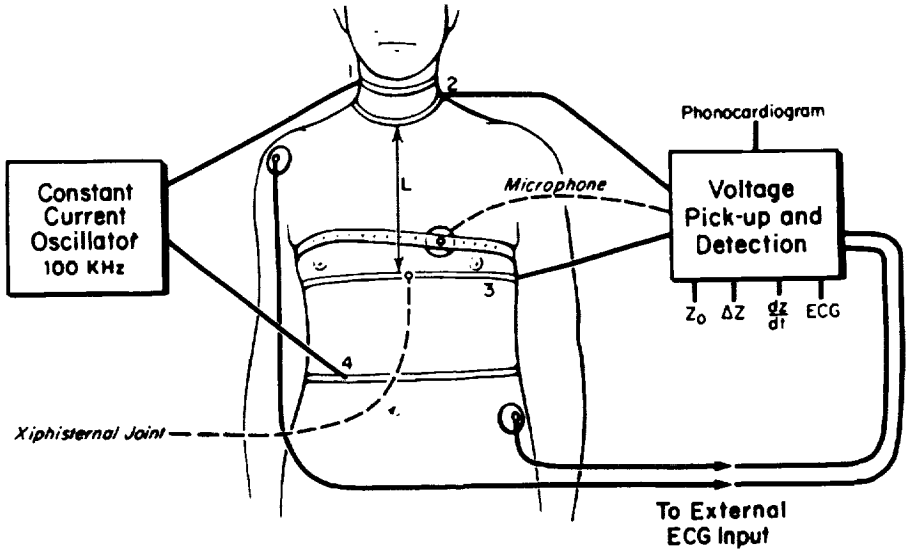


Figure 1. *A schematic diagram of the main elements of the Minnesota Impedance Cardiograph. From left to right, the constant current oscillator at 100 kHz, 4 ma (RMS). L=distance between electrodes 2 and 3 in cm, Z_o=longitudinal thoracic impedance between electrodes 2 and 3 in ohms, ΔZ=magnitude of impedance change during cardiac cycle in ohms, dZ/dt=first time derivative of ΔZ in ohms per second, ECG=electrocardiogram and finally, a phonocardiogram output.*

A separate contract was negotiated by the NASA Center with Space Labs, Inc., Van Nuys, California, to construct six miniaturized impedance units as shown in Figures 2 and 3. Testing of one of the systems showed fairly good function (Figure 4). However, a satisfactory calibration system was not incorporated into the design, making use of the device very difficult. One of these units was used during space flight by Dr. William Thornton (NASA).

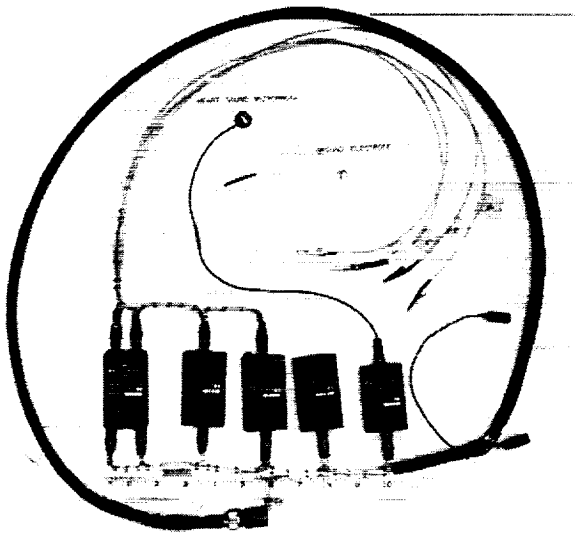


Figure 2. *A photograph of one of the six miniaturized impedance cardiographs, patterned after the circuitry shown in Figure 1. A satisfactory calibration circuit was not incorporated into these units. These units were constructed by Space Labs, Inc., Van Nuys, California, about 1968.*

Figure 3. *A photograph of the unit in Figure 2 worn in a "cartridge belt" suspension.*

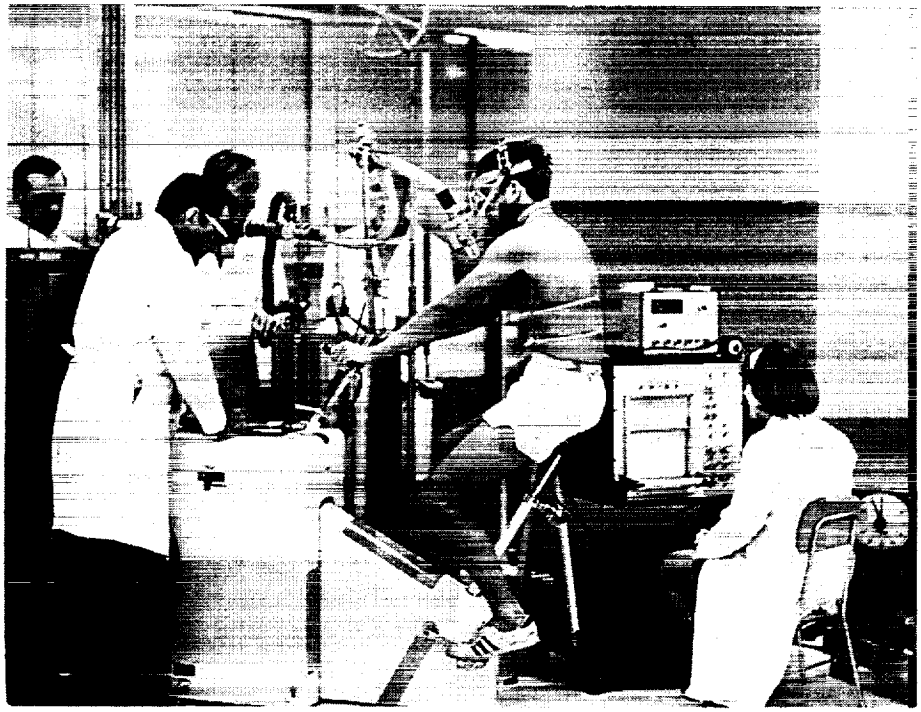


Figure 4. *A photograph of the laboratory arrangement for bicycle ergometer exercise of six middle distance runners (4,000 meters) . From left to right, Dr. R. A. Tracy, a runner in place on the bicycle ergometer with a face mask attachment for measuring oxygen consumption rate, and the strip chart recorder with the impedance cardiograph mounted on top with the technician ready to start the recorder, when needed.*

An excellent comprehensive review by Miles and Gotshall [1] on the subject, "Impedance Cardiography: Noninvasive Assessment of Human Central Hemodynamics at Rest and During Exercise," is most appropriate for this conference.

Scientific Observations of Importance during Exposure to Microgravity

Two major scientific achievements were made during our NASA contract. First, the derivation of a stroke volume formula and second, the use of the first time derivative (dZ/dt) of thoracic impedance change (ΔZ) during cardiac cycle to refine the stroke volume formula as widely used today [2].

Later experiments on anesthetized dogs revealed that peak (dZ/dt) occurs simultaneously with peak ascending aortic blood flow and that peak (dZ/dt) is directly proportional to peak aortic flow [3].

An example of a strip chart recording of (dZ/dt), phonocardiogram and ECG on a normal individual is shown in Figure 5. In addition to use in computing stroke volume and cardiac output, this recording to determine several systolic time intervals and contractility indices. It would appear that these parameters would be of great importance if there is deterioration in cardiac mechanical function during prolonged space flight.

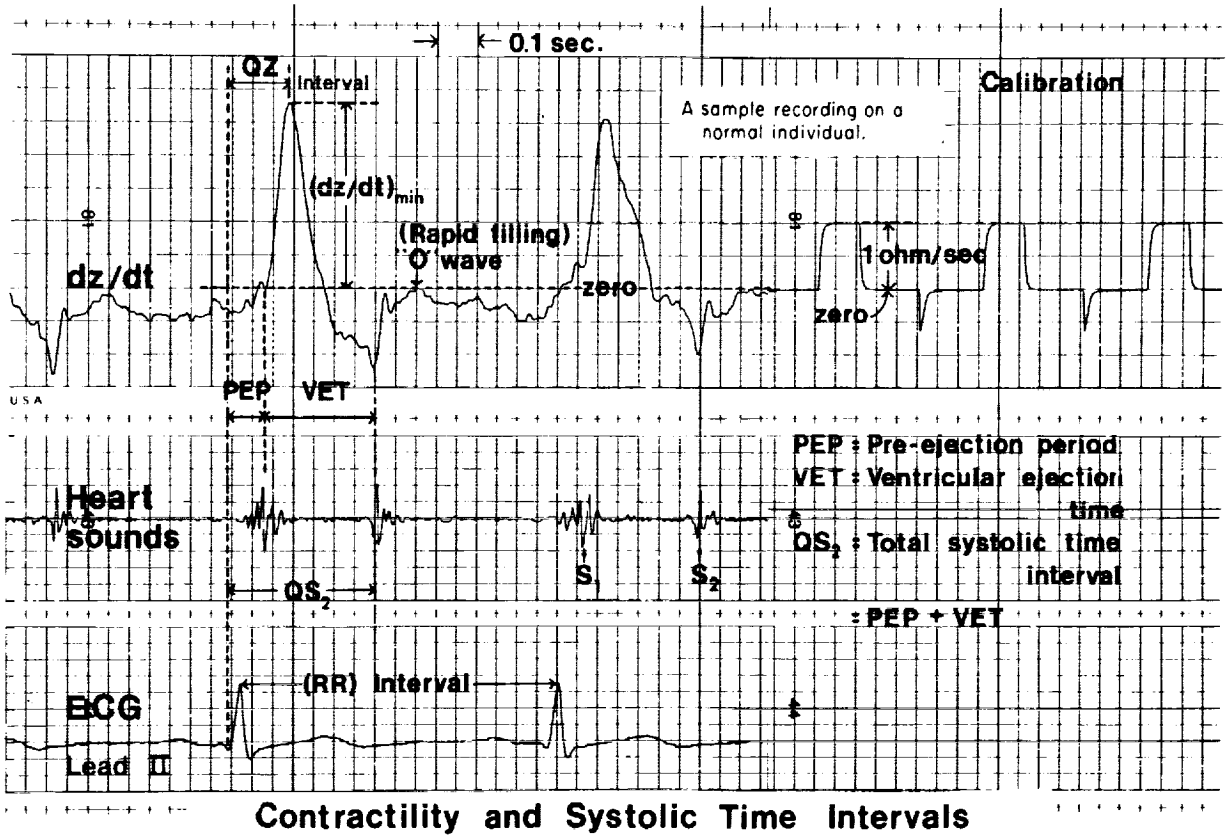


Figure 5. *An example of a strip chart recording on a normal individual at rest. From left to right, (Q-Z) interval in seconds=time from Q wave to peak dZ/dt. PEP preejection period in seconds, VET=ventricular ejection time in seconds, QS_2=total systolic time interval in seconds, $(dZ/dt)_{min}$ in ohms per second, "O" wave=rapid ventricular filling wave, (RR)=ime interval between two heart beats in seconds. This recording illustrates the type of recording used to compute or measure the various parameters shown in Figures 6 to 10.*

It is generally accepted that when a normal individual changes from a standing position to supine (at one g) cardiac output increases due to an augmented venous return from the lower parts of the body. Table 1 illustrates the change in several parameters obtained by impedance cardiography when normal individuals changed from standing to supine. Of special importance here is the decrease in Z_0 in the supine position. This indicates an increase in fluid in the pulmonary vascular bed.

Table 1. *Standing and supine data from 19 (8 female, 11 male) normal individuals, aged 21 to 54 years (Mean ±SD)*

	Standing	Supine (1 to 2 min)
SV	67 ±17	123 ±30
HR	79 ±13	64 ±12
CO	5.3 ±1.4	7.9 ±2.0
CI	2.9 ±0.8	4.3 ±1.2
dZdt	2.4 ±0.7	2.6 ± 0.8
ET	230 ± 21	306 ±25
PEP	117 ±14	88 ±10
Q-Z	157 ±15	142 ±12
HI	15 ±5.5	19 ± 6.5
Z_o	29 ±3.7	26 ± 3.5
Z_o/L	1.0 ±0.17	0.9 ±0.15

SV=ventricular stroke volume (CC), HR=heart rate in beats per minute, CO=cardiac output in liters per minute, CI=cardiac index, CO/body surface area (M2)=CO/M2, dZ/dt=first time derivative of thoracic impedance change (ΔZ) during cardiac cycle in ohms/second, $(dZ/dt)_{min}$ (Figure 5), ET=ventricular ejection time in milliseconds, (VET Figure 5), PEP=preejection period in milliseconds (Figure 5), Q-Z=time interval between ECG Q wave and peak dZ/dt in milliseconds (Figure 5), HI=Heather index (dZ/dt)/(Q-Z) in ohms/sec/sec, Z_o thoracic impedance (ohms) between electrodes 2 and 3 (Figure 1), Z_o/L=ohms/cm.

$$SV=\rho L^2/Z^2o\ T(dZ/dt)_{min}$$

where ρ=150 ohm cm, T=ejection time (see references 2 and 3), CO=SV•heart rate/1000=liters per minute

It would be reasonable to predict that immediately after reaching microgravity, the cardiovascular response would be similar to a ground-based individual changing from standing to supine. Z_o

Table 1 over an extended time, any adaptation to microgravity should be apparent.

If there is any tendency toward congestive heart failure during prolonged space flight, these relatively simple measurements could be of great importance.

Response to Exercise of Well Conditioned Runners

An exercise study was performed on six well-conditioned middle distance (4000 meters) runners. The primary objective was to determine if a special 6-weeks training program would improve their overall cardiac function. A perusal of the results presented graphically in Figures 6 to 10 indicates that the special training program had no effect on cardiac performance.

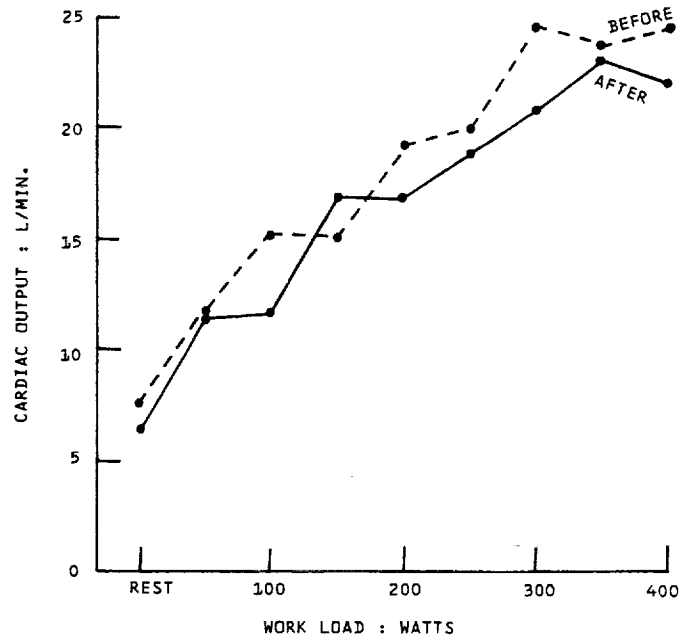


Figure 6. *Relationship between mean cardiac output and bicycle ergometer work load for six middle distance runners before and after 6 weeks of special training.*

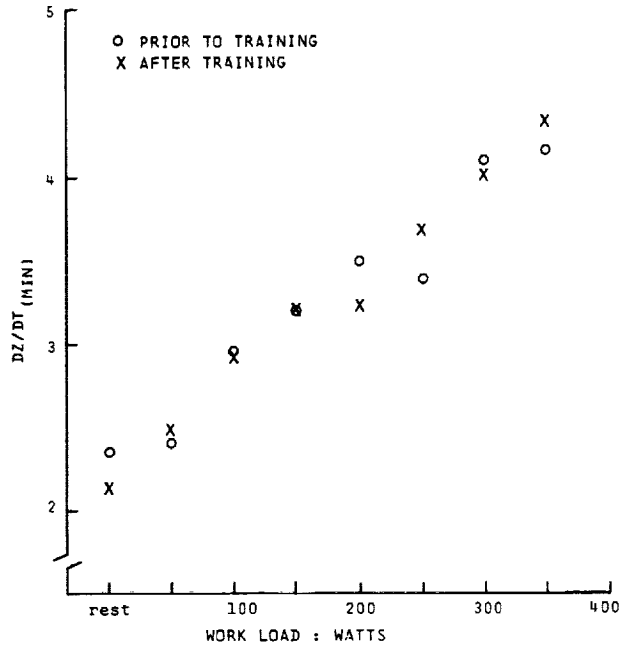


Figure 7. *A graph showing the relationship between mean dz/dt values and increasing work loads on the bicycle ergometer for six middle distance runners before and after 6 weeks of special training.*

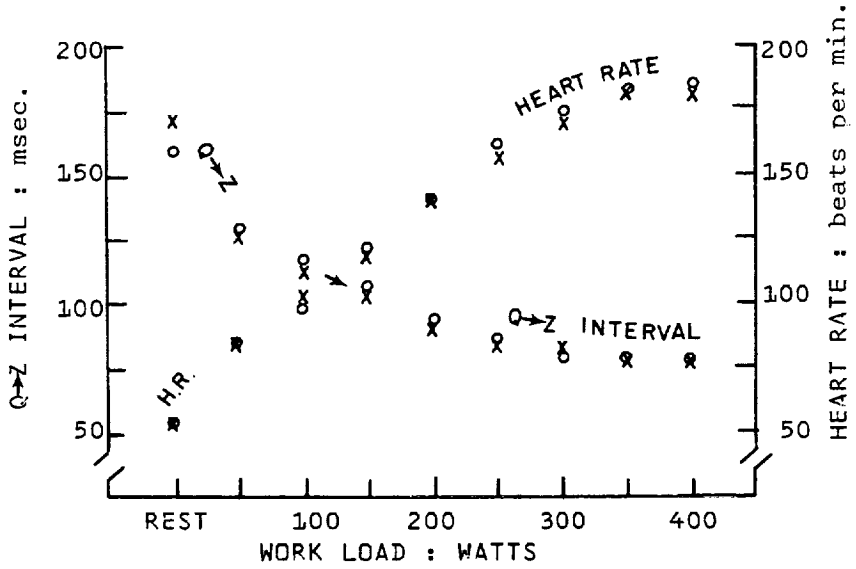


Figure 8. *The relationship between the mean heart rate values and mean Q-Z interval values and increasing work loads on the bicycle ergometer for six middle distance runners before and after 6 weeks of special training.*

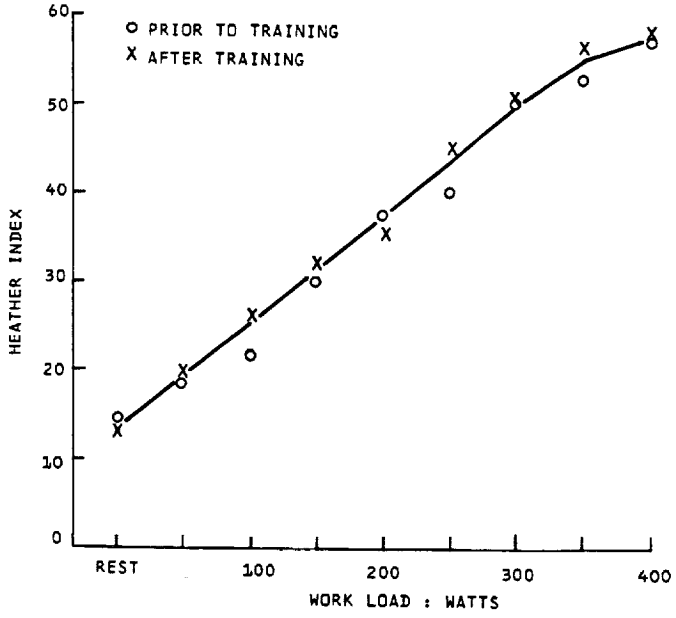


Figure 9. *A graph illustrating the relationship between the mean Heather Index values from rest to maximal bicycle ergometer exercise for six middle distance runners before and after 6 weeks of special training.*

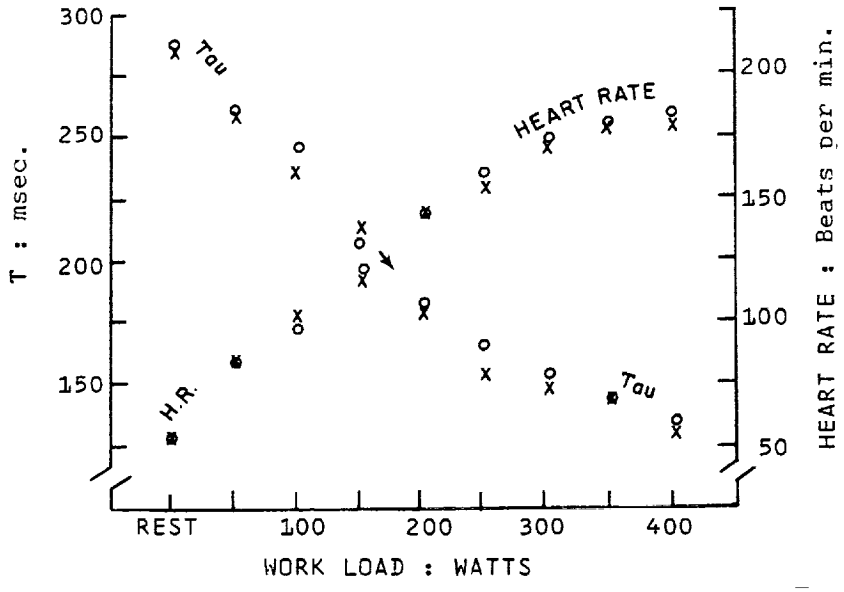


Figure 10. *A graph showing the relationship between mean T (ejection time in milliseconds) values and mean heart rate values and increasing work loads on the bicycle ergometer for six middle distance runners before and after 6 weeks of special training.*

These data are presented here as an illustration of how impedance cardiography could be used in relation to space flight and microgravity. Careful and repeated ground-based testing of the response to appropriate exercise could be carried out to establish a pattern of response prior to space flight. These baseline data could then be used for comparison, either for pre- and postflight testing for short-duration flights or for onboard testing during prolonged space flight.

Summary

Impedance cardiography is here to stay. About 500 published articles, utilizing impedance cardiography, are available. It is safe, convenient, noninvasive, and cost effective. It is the only proven method available to NASA to monitor the various parameters, described here, related to the intimate physiology of cardiac dynamics. In addition, changes in thoracic fluid volume can be followed. If desired, peripheral circulation can be studied by impedance.

Cardiac output is the historic standard for measuring cardiac mechanical function. However, changes in parameters such as the Heather contractility index (HI) may provide an early warning of impending serious myocardial dysfunction before any changes in cardiac output occur.

References

[1] MILES, D. S. AND R. W. GOTSHALL. Impedance Cardiography: Noninvasive Assessment of Human Central Hemodynamics at Rest and During Exercise. In: *Exercise and Sport Sciences Reviews*, edited by K. B. Pandolf. Baltimore, MD: Williams & Wilkens, 1989, p. 231-263.

[2] KUBICEK, W. G., J. M. KARNEGIS, R. P. PATTERSON, D. A. WITSOE, AND Evaluation of an Impedance Cardiac Output System. *Aerospace Medicine* 37:1208-1212, 1966.

[3] KUBICEK, W. G., F. J. KOTTKE, M. U. RAMOS, R. P. PATTERSON, D. A. WITSOE, J. W. LABREE, W. REMOLE, T. E. LAYMAN, H. SCHOENING, AND J. T. GARAMELLA. The Minnesota impedance cardiograph-theory and applications. *Biomedical Engineering* 9:410-416, 1974.

Acknowledgment

The material shown in Figures 4, 6, 7, 8, 9, 10, is part of an unpublished Ph.D. thesis study carried out in our laboratory by Robert Tracy (1971).

omit

Section 3

Skeletal Countermeasures

PRECEDING PAGE BLANK NOT FILMED

N94-28368

Weightlessness and The Human Skeleton: A New Perspective

S7-52
207616
P-16

Michael F. Holick, Ph.D., M.D.
Director, General Clinical Research Center
Boston University School of Medicine
Boston, Massachusetts

Introduction

It is now clear after more than two decades of space exploration that one of the major short- and long-term effects of microgravity on the human body is the loss of bone. The purpose of this presentation will be to review the data regarding the impact of microgravity and bed rest on calcium and bone metabolism. I will take the position in this Socratic debate that the effect of microgravity on bone metabolism can either be reversed or be mitigated.

As we begin to contemplate long-duration space flight, habitation of Space Station Freedom and the Moon, one of the issues that will need to be addressed is whether humans need to maintain a skeleton that has been adapted for the one-g force on Earth? Clearly in the foreseeable future a healthy and structurally sound skeleton will be required for astronauts to shuttle back and forth from Earth to the Moon, Space Station, and Mars. Based on most available data from bed-rest studies and the short- and long-duration microgravity experiences by astronauts and cosmonauts, bone loss is a fact of life in this environment. With the rapid advances in our understanding of bone physiology is it now possible to contemplate measures that can prevent or mitigate microgravity-induced bone loss. Will the new therapeutic approaches for enhancing bone mineralization be useful for preventing significant bone loss during long-term space flight? Are there other approaches such as exercise and electrical stimulation that can be used to mitigate the impact of microgravity on the skeleton? A recent study that evaluated the effect of microgravity on bone modeling in developing chick embryos may perhaps provide a new perspective about the impact of microgravity on bone metabolism.

Human Bone Disease: Historical Perspective and Concerns for the Future

Bone disease has afflicted humans almost since the beginning of time. As the industrial revolution took hold in Northern Europe, rickets became an epidemic. This devastating bone disease was eventually eradicated as significant health problem because of the appreciation of the beneficial effect of sunlight in producing vitamin D in the skin [6]. During the past two decades osteoporosis has become a significant health issue affecting more than 20 million elderly Americans and costing $6 billion dollars that by the year 2020 we will begin the colonization of the Moon and travel to Mars. This exciting new era in space exploration could result in a new bone disease caused by microgravity. If we are unable to prevent or reverse microgravity-induced bone loss, this negative effect on the skeleton could severely limit human aspirations to explore and colonize the universe.

As we initiate a program for long-term human habitation in a microgravity environment on board Space Station Freedom and the Moon there remains concern about the acute and chronic loss of bone mineral. Acutely the rapid loss of bone mineral in combination with microgravity-induced hemodynamic changes could significantly increase the risk of kidney stones and soft tissue calcification. Chronic unrelenting bone loss could compromise its structural integrity to such a degree that when astronauts are exposed to the G-forces upon reentry into Earth's gravitational field, micro- and macrofractures of the skeletal network could occur. In addition, since the entire skeleton is unloaded in microgravity, it is possible that its three-dimensional structure is

PRECEDING PAGE BLANK NOT FILMED PAGE 152 INTENTIONALLY BLANK

reorganized to such a degree that when astronauts return to Earth and initiate routine activity in a one-g environment that micro- and macrofractures of the skeleton could occur as the body realigns the framework of the skeleton to adapt to the forces put upon it by Earth's gravity.

The Effect of Bed Rest and Microgravity on Bone Metabolism

The model that is most often used to evaluate the potential impact of microgravity on bone and calcium metabolism is to unload the skeleton of healthy subjects by putting them at strict bed rest [4, 9, 14, 20]. Schneider and McDonald [14] and Arnaud, Schneider, and Morey-Holton [1] measured intestinal calcium transport 4 weeks before and 20 weeks during strict bed rest. They showed the efficiency of intestinal calcium absorption gradually declined during the first 4 weeks of bed rest and plateaued to about 50 percent of the original level during that last 16 weeks (Figure 1).

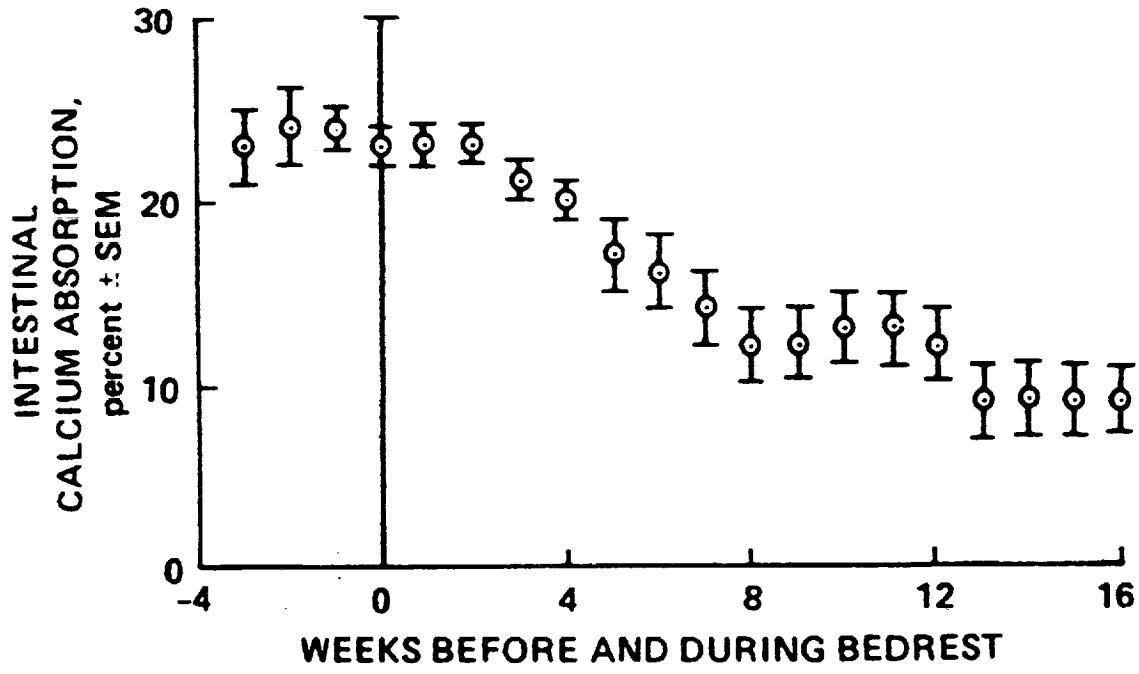


Figure 1. *The intestinal absorption of calcium estimated from balance data in 19 to 47 healthy subjects immobilized by bed rest for 16 weeks. Absorption was determined in subjects consuming 1000 mg daily (reproduced with permission).*

An increase in the mobilization of calcium from the skeleton may transiently increase the circulating ionized calcium concentrations which in turn caused a decrease in parathyroid hormone (PTH) secretion [1, 16]. A decrease in PTH secretion would result in the renal wasting of calcium causing an increased urinary output of calcium (Figure 2). The decrease in intestinal calcium absorption, that was most likely caused by a decrease in the PTH mediated metabolism of 25-hydroxyvitamin D to 1,25-dihydroxyvitamin D, resulted in an increase loss of calcium in the feces (Figure 2). This caused a negative calcium balance of approximately 200 mg/day during the last 16 weeks of bed rest (Figure 2).

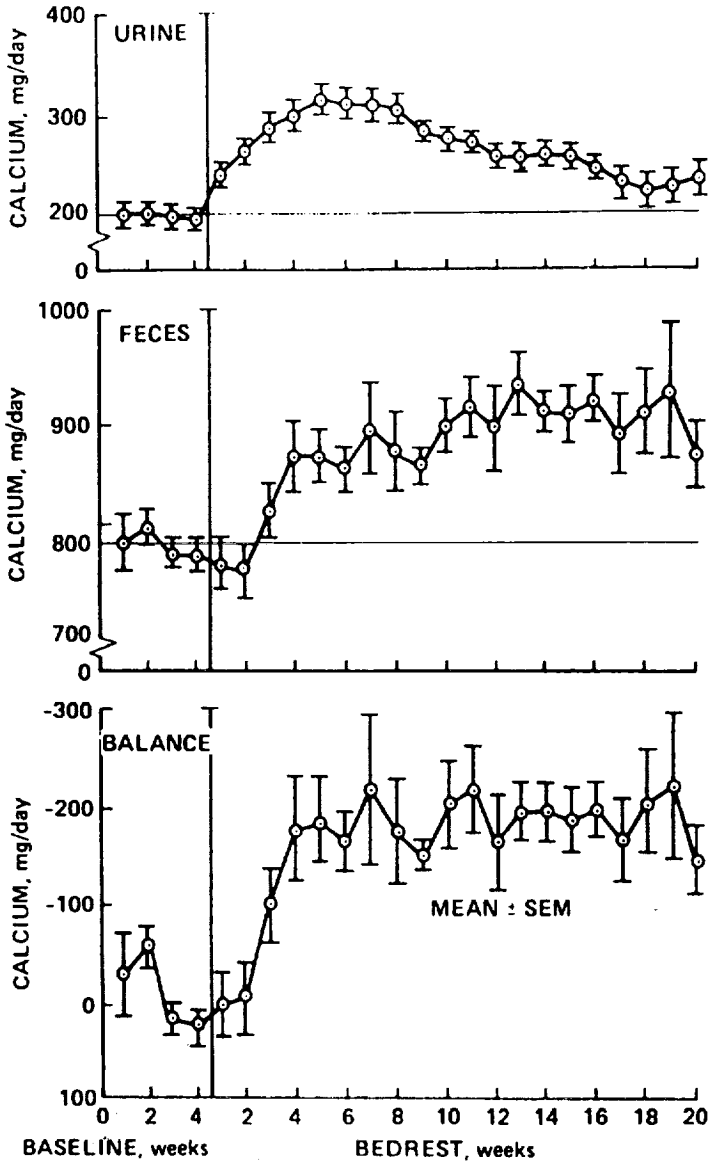


Figure 2. *The excretion of calcium in the urine and feces, in milligrams per day, in 12 to 32 healthy subjects immobilized by bed rest for 20 weeks. Dietary calcium was kept constant in these subjects at 1000 mg/d. Fecal calcium was corrected for time by polyethylene glycol 4000 administered three times daily in a dose 500 mg. The lowest panel shows the results of the balance study estimated from the intake and excretion of calcium in the urine and feces (reproduced with permission).*

Urinary hydroxyproline concentrations were increased providing evidence that the increase loss of calcium in the urine was partly due to an increase turn-over of the bone (Figure 3).

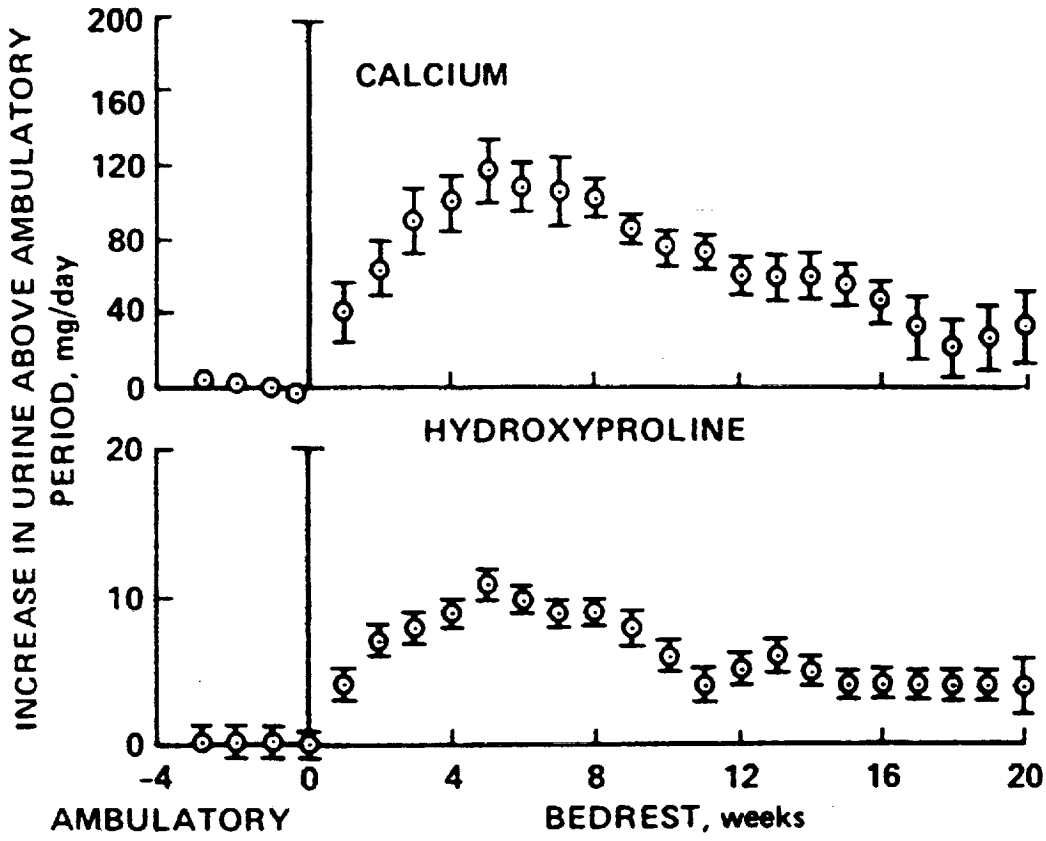


Figure 3. *The daily urinary excretion of hydroxyproline and calcium in the urine. Excretion is shown as the percentage of increase from the ambulatory control period of 4 weeks in 14 to 40 normal subjects during 20 weeks of bed rest (reproduced with permission).*

Bone density measurements of the calcaneous from the bed-rest subjects showed a marked decline in bone mineral density. A significant decrease in the calcaneal bone density was evident by 5 to 8 weeks and continued to decline at about the same rate for the next 20 weeks. Approximately 30 percent of the bone mineral density was lost by the end of the study (Figure 4).

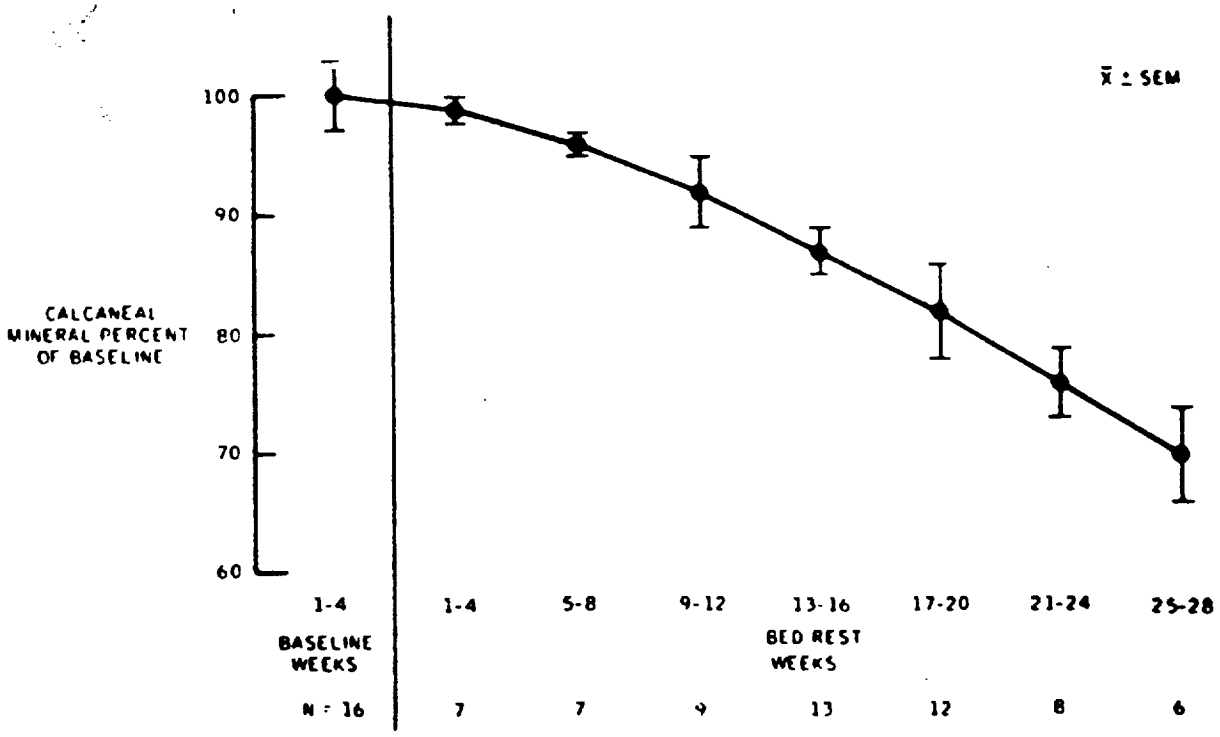


Figure 4. *Photon absorptionometry of the calcaneous. Percent decrease from ambulatory values during untreated bed rest (reproduced with permission).*

The results of these bed-rest studies have mirrored the observations seen in astronauts and cosmonauts exposed to microgravity. Rambaut, Leach, and Whedon [11] observed significant increases in urinary calcium excretion in Skylab 4 astronauts (Figure 5).

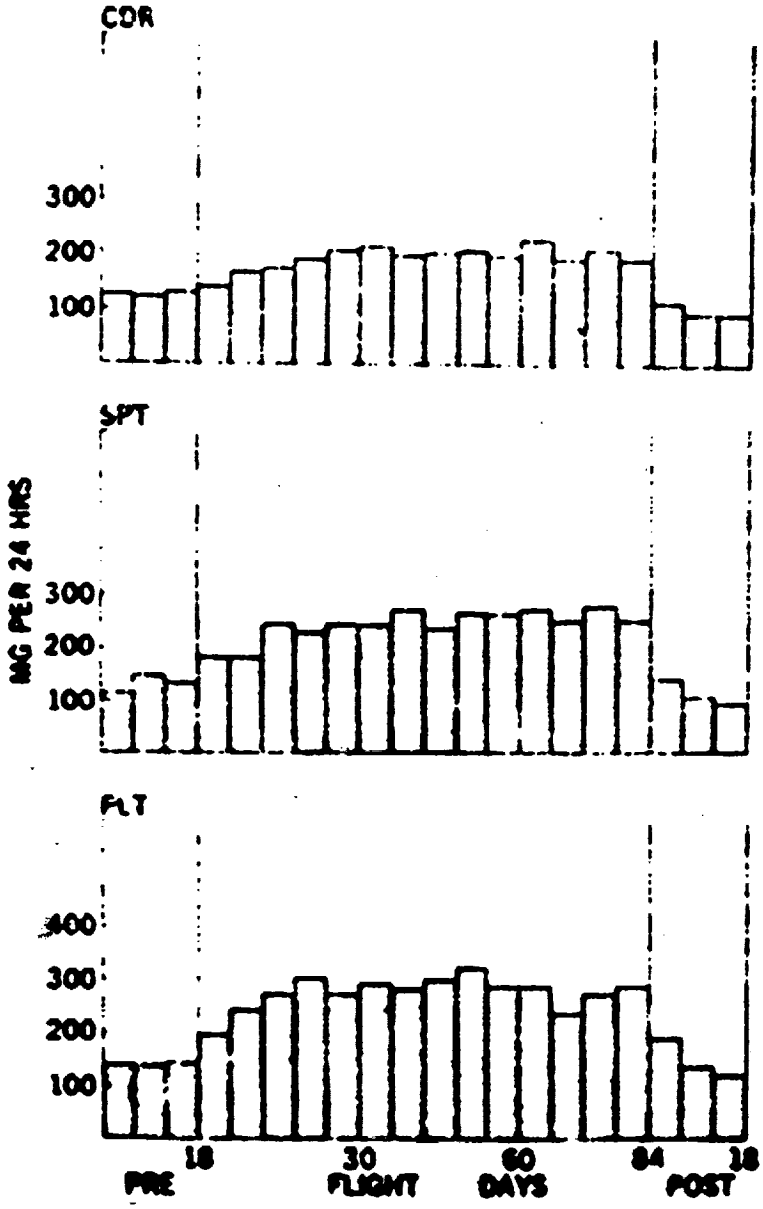


Figure 5. *Urinary calcium in Skylab 4 astronauts (reproduced with permission).*

They also found a significant decrease in calcaneous bone density during the 84-day mission on board Skylab 4 (Figure 6).

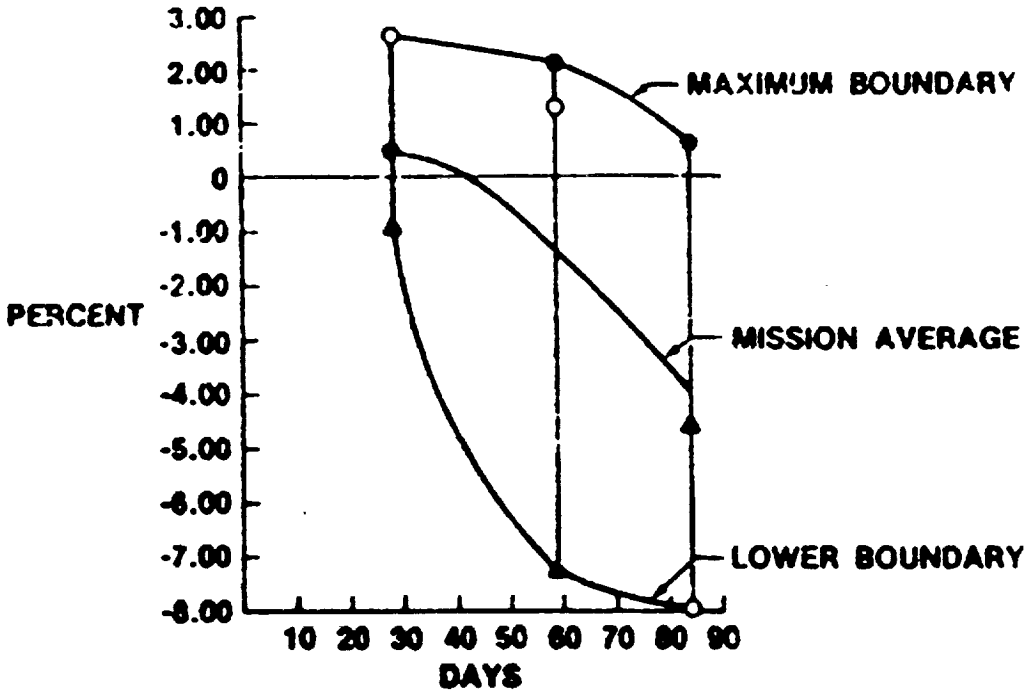


Figure 6. *Calcaneal mineral loss as a function of Skylab flight duration (reproduced with permission).*

Parfitt [10] analyzed the calcaneal mineral bone density in astronauts on board Skylabs 2, 3 and 4 and found a significant decrease of about 4 percent in the longest flights of 59 and 84 days. By contrast no significant changes were noted in the cortical bone density of the radius and ulna in the same astronauts. The Skylab astronauts experienced a negative calcium balance of about 200 to 300 mg/day. Parfitt's analysis of the Skylab 4 astronauts' calcium metabolism data also revealed that they lost about 300 mg of calcium a day that resulted in a total body loss of about 18 g (with a range of 10 to 22 g). Most of the loss was not due to a decrease in intestinal absorption of calcium; it was caused by an increase in urinary excretion of calcium. Parfitt concluded that two-thirds of the total calcium loss came from trabecular bone and one third came from cortical bone. Similar observations have been made in cosmonauts. In Table 1 all of the cosmonauts had significant decrease in the bone density of after 140 days in microgravity [17].

Table 1. *Percentage change in mineral content of calcaneous in cosmonauts*

Crew Member	Flight Duration (days)	Postflight Testing Day	Entire Flight	Monthly Average
CDR	140	5	-3.0	-0.64
FLE	140	4	-19.8	-4.24
CDR	175	5	-9.8	-1.69
FLE	175	5	-3.0	-0.51
CDR	184	14	-9.6	-1.56
FLE	184	14	-4.4	-0.71
CDR	75	4	-0.9	-0.36
FLE	75	4	-3.2	-1.31

Physiologic Function of the Skeleton

In light of the observations just presented there is the possibility that a significant amount of skeletal mass will be lost during long-term space travel. One of the questions that is worthwhile asking is, "Do we need a skeleton?" Is it possible that we will ultimately evolve in microgravity into protoplastic-thinking beings that do not require a skeleton? Some of the functions of the skeleton are obvious. It protects our soft vital organs, provides attachment for our musculature and permits us to be mobile in our terrestrial environment. A more subtle but equally important reason for the evolution of a calcified skeleton is to provide a storage depot for calcium. As vertebrates evolved in the fertile oceans they were bathed in calcium. When they left this environment for terra firma they were confronted with a calcium deprived environment. They needed to develop mechanisms by which they could efficiently utilize, assimilate, and store the limited supplies of calcium that were in their environment. Vertebrates also developed elaborate mechanisms to call upon these reserves at times of need.

Calcium plays an essential role in neurotransmission and neuromuscular function. In addition, cytosolic calcium concentrations are carefully regulated and are responsible for a multitude of cellular functions. Calcium also acts as an important second messenger to the cell. Therefore, I believe that there is a need to maintain our skeletal structure if for no other reason than to act as a storage site for calcium so that the body can call on this depot to maintain intra- and extracellular calcium concentrations within physiologically acceptable limits.

Dynamics of Bone Formation and Bone Resorption: Strategies for Maintaining Bone Mineral Density in a Microgravity Environment

When bone formation rates are greater than bone degradation rates there is an increase in bone mass whereas, when bone formation rates are less than bone degradation rates there is a decrease in bone mass. Osteoporosis is a state whereby the bone matrix along with the bone mineral is lost. Osteomalacia on the other hand occurs when there is loss of bone mineral with the maintenance of the matrix [8]. Although it is well documented that microgravity causes a decrease in bone mineral content it is not known whether the bone mineral matrix is lost to the same degree causing osteoporosis or whether the matrix is partially or fully maintained causing osteomalacia. If the matrix is maintained in a microgravity environment then there is an opportunity to develop therapeutic strategies to replenish the demineralized matrix. If, on the other hand, microgravity induced bone demineralization causes the loss of the staging network (matrix) and calcium

hydroxyapitite (mineral) that results in the destruction of the bone trabeculae, then it will be very difficult if not impossible to restore the very fine trabecular network. Dempster *et al.* [3] have demonstrated this in an exquisite fashion with scanning electromicroscopy. Figure 7a shows the trabecular pattern from a healthy adult male skeleton while Figure 7b shows a marked loss in the thickness and number of trabeculae as well as gaps between the trabeculae in a woman with osteoporosis and multiple compression fractures of her spine. It would be very difficult to induce bone cells to bridge these gaps.

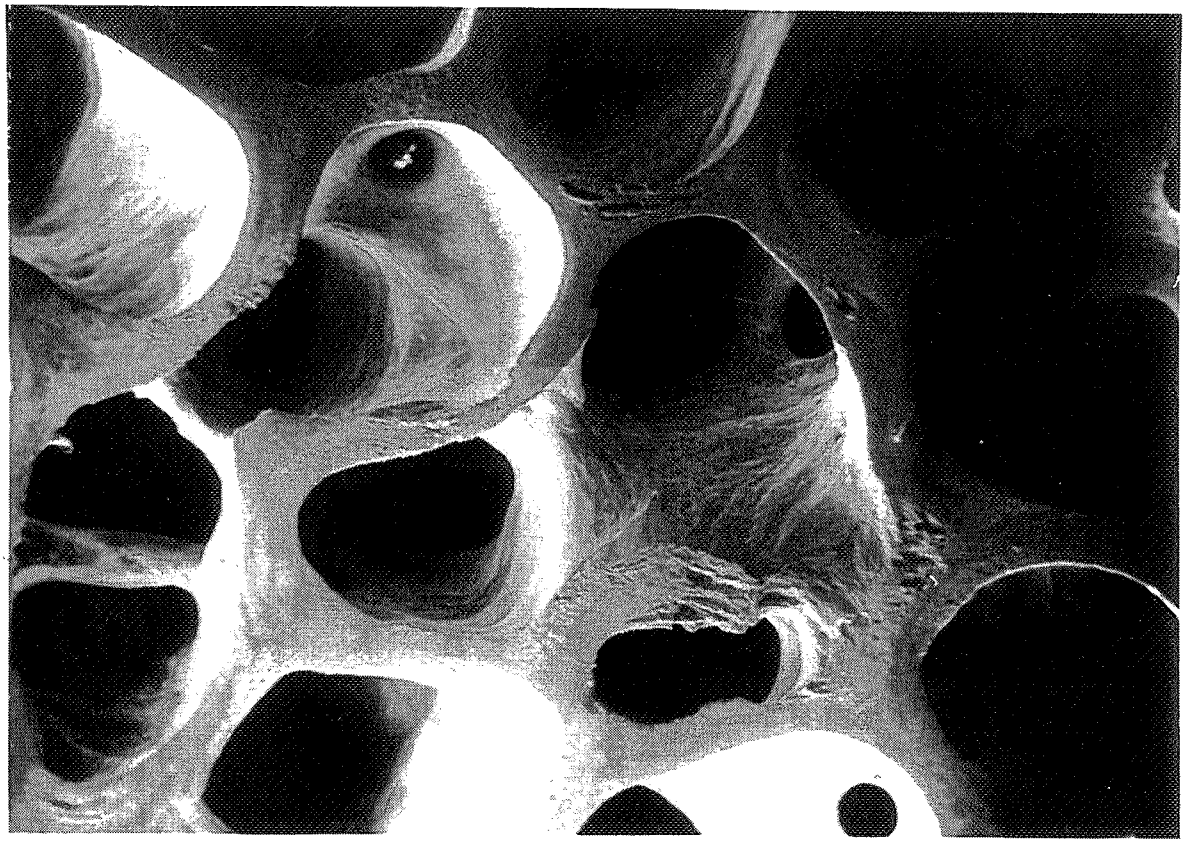


Figure 7a. *Low power scanning electron micrograph of an iliac crest biopsy from a 44-year-old normal male (reproduced with permission).*

Figure 7b. *Low power scanning electron micrograph of a iliac crest biopsy from a 47-year-old osteoporotic female with multiple vertebral compression fractures (reproduced with permission).*

It is known from prolonged bed-rest studies that once the subjects are restored to their normal activity they are capable of restoring their bone mineral density in the os calcis to prebed-rest levels [4]. In Skylab studies follow-up bone density measurements at 3 months showed that the deficit had been repaired in only one astronaut, 50 percent recovery in one, and no recovery in the third who had measured calcaneal loss during space flight [13]. A 5-year follow-up of the mineral content in the os calcis of nine crew members from the Skylab program when compared to eight of their alternates who served as controls also showed a statistically significant loss compared to the control group [18]. The authors cautioned, however, that there are so many potential causes for the observed differences that more studies needed to be done. It is not known whether this restoration would also occur in astronauts and cosmonauts who have experienced microgravity for extended periods such as a 3-year mission to Mars. Furthermore, even if the bone mineral content is restored we do not know whether the three dimensional structure of the bone could be restored to its original condition.

Can Pharmacologic Intervention Regulate Bone Remodeling?

Can you maintain and/or restore bone mineral density while in microgravity? Schneider and MacDonald [14] evaluated a variety of therapies including lower body negative pressure, exercise, static compression, calcitonin, disphosphonate, phosphate, and calcium plus phosphate, and concluded that they were unable to prevent negative calcium balance during bed rest (Figures 8 and 9).

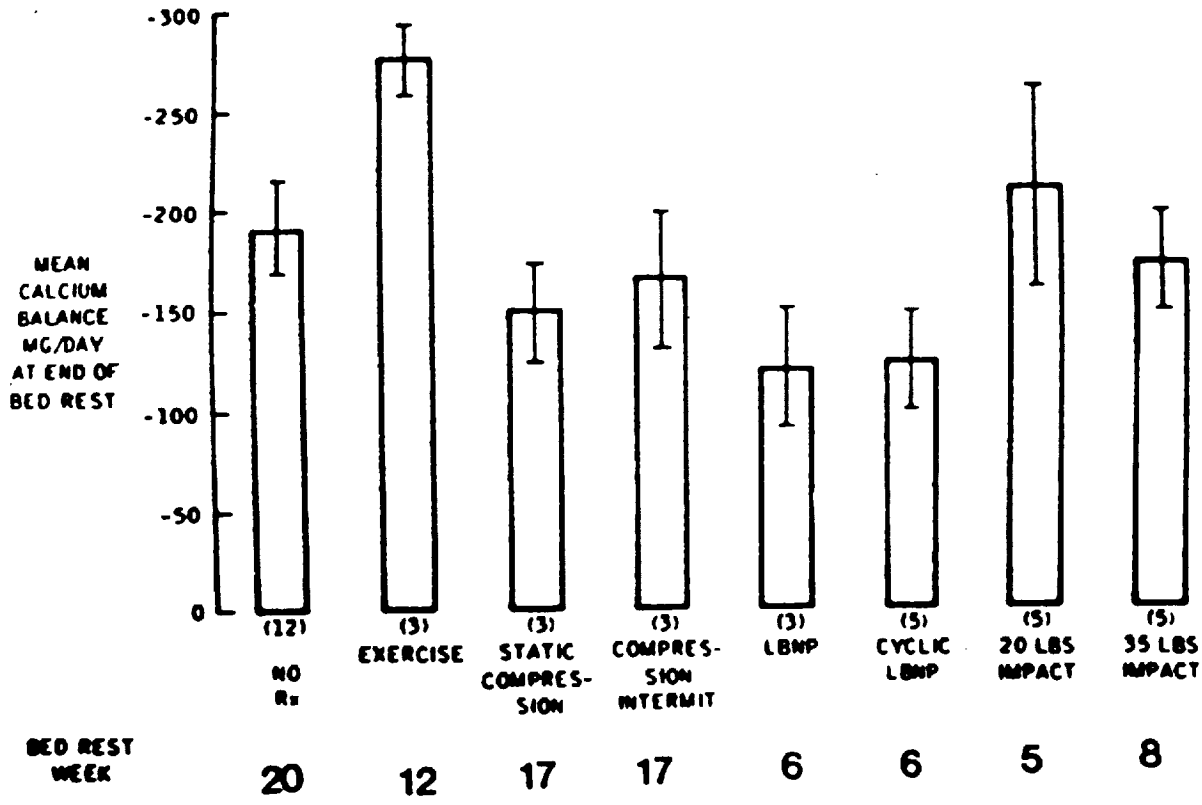


Figure 8. *Mean calcium balance of the final week of study during untreated bed rest and in eight physical interventions tried during bed rest (lower body negative pressure [LBNP]) (reproduced with permission).*

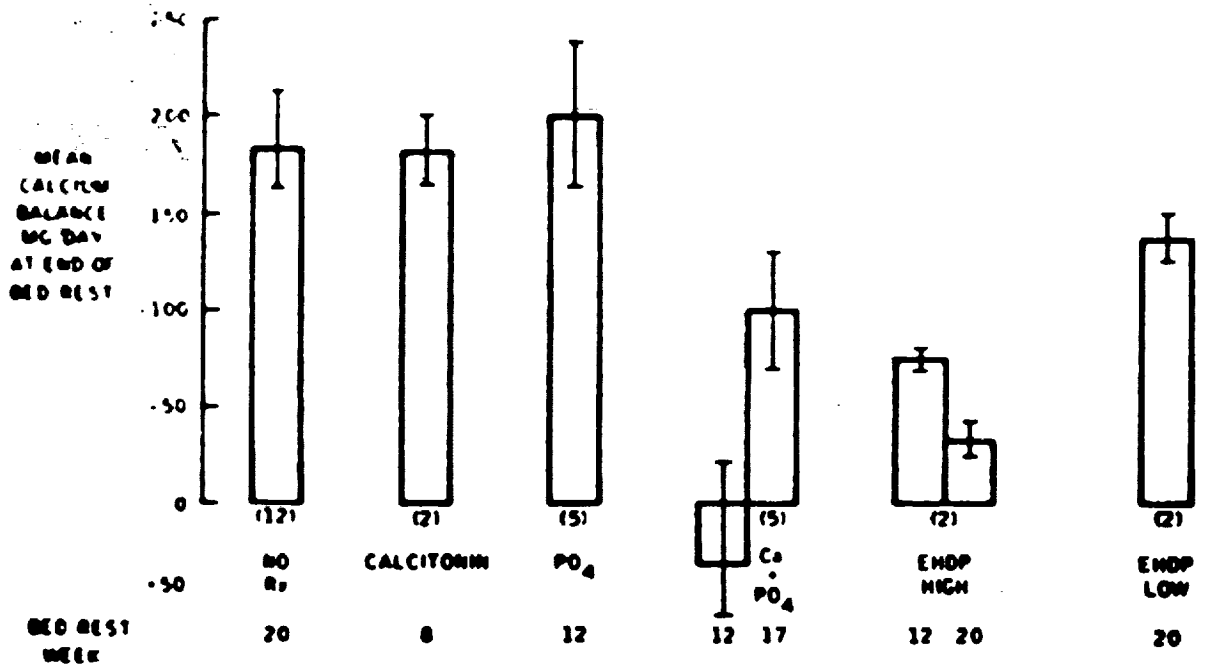


Figure 9. *Mean calcium balance during the final week of study during untreated bed rest and in four biochemical therapies tried during bed rest (etidronate [EHDP]) (reproduced with permission).*

There is mounting evidence that both $1,25(OH)_2D$ and parathyroid hormone not only enhance bone calcium mobilization but also act as anabolic hormones to maintain bone remodeling activities. Mature osteoclasts do not possess receptors for either $1,25(OH)_2D$ or PTH. Instead it is now believed that both of these hormones act on osteoblasts to induce cytokines that regulate osteoclastic activity [6]. Additionally $1,25(OH)_2D$ and PTH may mobilize stem cells to become mature osteoclasts. Other factors that may play a role in the bone remodeling process include TGF-beta and other bone growth factors. In some studies calcitonin has been shown to increase bone mineral density, but in general it is believed to be an agent to inhibit the bone mobilization and has little effect on the bone remodeling process. Of great interest is the possibility of activating then depressing the bone remodeling activity. This has led to a new cyclic therapy called ADFR (activation, depression, free period, repeat). Although preliminary results suggests that PTH followed by $1,25(OH)_2D_3$ or phosphate followed by $1,25(OH)_2D_3$ may significantly increase spinal bone density [15], Schneider and MacDonald [14] have reported that during bed rest calcitonin, phosphate, or diphosphonate therapy did not significantly reverse the negative calcium balance although some improvement was found in the group receiving high amounts of diphosphonate. Recently Judge *et al.* [7] evaluated bone biopsies from four male volunteers after 17 weeks of bed rest and found a mismatch between bone resorption and bone formation, whereby bone resorption continued or was increased while bone formation was decreased. Arnaud *et al.* [2] evaluated bone biopsies from volunteers after a 2-week period of bed rest. After double tetracycline labeling, it was found that bone formation rates significantly diminished in six

subjects, was unchanged in one, and higher in one subject who was a swimmer (personal communication).

Can Bone Cells Adapt to Microgravity?

Little is known about the impact of microgravity on bone cell activity. Can bone cells adapt to a microgravity environment? Are bone cells addicted to gravity and therefore require gravity to maintain normal bone remodeling activity? An insight into these questions has recently become available as a result of a study on board STS-59. Sixteen fertilized chicken eggs at gestational ages of 2 and 9 days were housed in a incubator and flown on board the Space Shuttle Discovery. Control eggs of the same gestational ages were maintained in a similar incubator and exposed to the vibrations and jostling to simulate liftoff and reentry. After the 5-day flight one-half of the eggs from both groups were immediately sacrificed. The 2-day old embryos that were flight flown all stopped developing and died within 24 hours, whereas all of the 2-day old control embryos survived. The flight flown 9-day old embryos all developed similar to the control group. One-half of the flight flown and control embryos had their bones evaluated. Preliminary results suggest that the bone modeling process in the flight flown animals was no different from the earth controls [19]. These results suggest that microgravity does not alter the bone mineralization process. Therefore, it is likely that bone cells become adapted to gravity. Once exposed to microgravity they decrease their activity. Thus, if we could understand what the signal or signals are that microgravity provides to bone cells to limit their activity, it may be possible to reverse this action. Alternatively it may be that if humans are born in a microgravity environment that their bone cells would develop normally and a normal skeleton would evolve. As we contemplate exploring new solar systems it may be that there will be a need for microgravity born astronauts to complete such missions.

Conclusion

The likely physiologic events in calcium and bone metabolism that occur in a microgravity environment are as follows:

1. The unloading of the G-forces on the skeleton induce or inhibit a signal(s) that causes a depression in bone formation and an acceleration in bone demineralization that results in an increase in circulating ionized calcium concentrations.

2. The small rise in ionized calcium concentrations is recognized by the parathyroid glands, which in turn decrease their secretion of parathyroid hormone.

3. The decrease in parathyroid secretion causes an increase renal loss of calcium into the urine.

4. A decrease in PTH secretion also results in the depression of the metabolism of 25-hydroxyvitamin D (25-OH-D) to 1,25-dihydroxyvitamin D (1,25$[OH]_2$D) [5, 6].

The decrease in production of 1,25$(OH)_2$D results in a decrease in the efficiency of intestinal calcium transport (Figure 10). This cycle ultimately results in the continued wasting of the bone mineral.

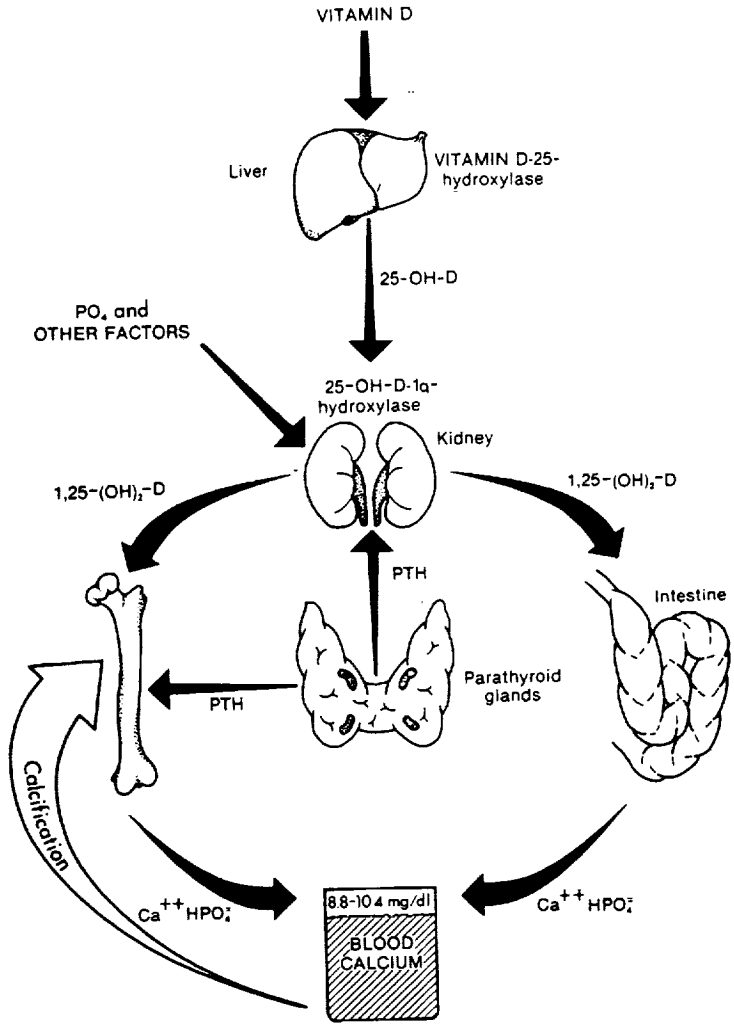


Figure 10. *Schematic representation of the hormonal control loop for vitamin D metabolism and function. A reduction in the serum calcium below approximately 8.8 mg/mL prompts a proportional increase in the secretion of parathyroid hormone, which enhances the mobilization of calcium stores from bone. Parathyroid hormone also promotes the synthesis of 1,25(OH)2D in the kidney, which in turn stimulates the mobilization of calcium from bone and its absorption from the intestine (reproduced with permission).*

We do not know whether microgravity in enhancing the mobilization of calcium from bone also causes the loss of bone matrix. If the matrix is maintained there is a good possibility that astronauts can stimulate bone mineralization in a microgravity environment during prolonged space travel. If, however, gravity adapted bone cells are further stimulated during astronaut training to become more dependent on gravitational forces then the acute loss of Earth's G-force could accelerate the mobilization of calcium from the bones of astronauts. A detraining period of the skeleton may be an alternative approach to limiting the total amount of calcium lost from bone during exposure to microgravity.

References

[1] ARNAUD, S. B, V. S. SCHNEIDER, AND E. MOREY-HOLTON. Effects of inactivity on bone and calcium metabolism. In: *Inactivity: Physiological Effects,* edited by H. Sandler and J. Vernikos. London: Academic Press, Inc., 1986, p. 49-76.

[2] ARNAUD, S. B., D. J. SHERRARD, N. MALONEY, R. T. WHALEN, AND P. FUNG. Reduced bone formation is measurable in the iliac crest of normal men after 7 days in a bed rest model of weightlessness. *J. Bone Min. Res.* 4:s233, 1989. (Abstract)

[3] DEMPSTER, D. W., E. SHANE, W. HORBERT, AND R. LINDSAY. A simple method for correlative light and scanning electron microscopy of human iliac crest bone biopsies: qualitative observations in normal and osteoporotic subjects. *J. Bone Min. Res.* 1:15-21, 1986.

[4] DONALDSON, C. L, S. B. HULLEY, J. M. VOGEL, R. S. HATTNER, J. H. BAYERS, AND D. E. MCMILLIAN. Effect of prolonged bed rest on bone mineral. *Metabolism* 19:1071-1084, 1970.

[5] HOLICK, M. F. Vitamin D and the kidney. *Kid. Internatl.* 32:912-929, 1987.

[6] HOLICK, M. F. Vitamin D: biosynthesis, metabolism, and mode of action. In: *Endocrinology, Volume 2,* edited by L. J. DeGroot. New York: Grune & Stratton, 1989, p. 902-926.

[7] JUDGE, D., V. S. SCHNEIDER, AND A. LEBLANC. The histomorphometric changes in disuse osteoporosis. *J. Bone Min. Res.* 4:S238, 1989. (Abstract)

[8] KRANE, S. M. AND M. F. HOLICK. Metabolic bone disease. In: *Harrison's Principles of Internal Medicine, 11th Edition,* edited by E. Braunwald, K. H. Isslebacher, R. G. Petersdorf, J. S. Wilson, J. B. Martin, and A. S. Fauci. New York: McGraw-Hill, 1986, p. 1889-1900.

[9] LEBLANC, A., V. S. SCHNEIDER, J. KREBS, H. EVANS, S. JHINGRAN, AND P. JOHNSON. Spinal bone mineral after 5 weeks of bed rest. *Calcif. Tissue Int.* 41:259-261, 1987.

[10] PARFITT, A. M. Bone effects of space flight: analysis by quantum concept of bone remodeling. *Acta Astronautica* 8:1083-1090, 1981.

[11] RAMBAUT, P. C., C. S. LEACH, G. D. WHEDON. A study of metabolic balance in crewmembers of Skylab 4. *Acta Astronautica* 6:1313-1322, 1979.

[12] RAMBAUT, P. C. AND R. S. JOHNSTON. Prolonged weightlessness and calcium loss in man. *Acta Astronautica* 6:11131122, 1979.

[13] SCHNEIDER, V. S., A. LEBLANC, AND P. RAMBAUT. Bone and mineral metabolism. In: *Space Physiology and Medicine,* edited by A. Nicogossian, C. Huntoon, and S. Pool. Philadelphia: Lee & Febiger, 1989, p. 214-221.

[14] SCHNEIDER, V. S. AND J. MCDONALD. Skeletal calcium homeostasis and countermeasures to prevent disuse osteoporosis. *Calcif. Tissue Int.* 36:S151-S154, 1984.

[15] SLOVIK, D. M., D. I. ROSENTHAL, S. H. DOPPELT, J. T. POTTS, M. A. DALY, J. A. CAMPBELL, AND R. M. NEER. Restoration of spinal bone in

human parathyroid hormone (1-34 and 1,25-dihydroxyvitamin D. *Res.* 1:377-381, 1986.

[16] STEWARD, A. F., M. ADLER, C. M. BYERS, G. V. SEGRE, AND A. E. BROADUS. *N. Engl. J. Med.* 306:1136-1140, 1982.

[17] STUPAKOV, G. P., V. S. KAZEYKIN, A. P. KOZLOVSKIY, AND W. KOROLEY. Evaluation of changes in human axial skeletal bone structures during long-term space flights. *Kosm. Biol. Med.* 18:33-37, 1984.

[18] TILTON, F. E., J. J. C. DEGIOANNI, AND V. S. SCHNEIDER. Long-term follow-up of Skylab bone demineralization. *Aviat. Space, Environ. Med.* 51:1209-1213, 1980.

[19] VELLINGER, J., M. DEUSER, M. CASTONGUAY, H. KAVOOKJIAN, D. FERMIN, R. KERSCHMANN, S. HESTER, R. HULLINGER, S. HUNTER, AND M. F. HOLICK. Effect of weightlessness on bone development and mineralization in chick embryos on board the Space Shuttle Discovery. *J. Bone Min. Res.* 4:s409, 1989. (Abstract)

[20] VICO, L., D. CHAPPARD, C. ALEXANDRE, S. PALLE, P. MINAIRE, G. RIFFAT, B. MORUKOV, S. RAKHMANOV, AND V. S. OGANOV. Quantitative histological study of the bone mass and cellular activities after a 120-day period of decubitus. Test of preventive protocols. *Ann. Biol. Clin.* (Paris) 45:145-151, 1987.

N94-28369

Irreversibility of Advanced Osteoporosis - Limited Role for Pharmacologic Intervention

58-52
207617
P. 6

A. M. Parfitt, M.B., B. Chir.
Bone and Mineral Research Laboratory
Henry Ford Hospital
Detroit, Michigan

Introduction

Osteoporosis is of medical interest only because it increases bone fragility and risk of fracture, and except for relief of symptoms, preventing fracture is the only purpose of intervention. To prevent the first fracture, adequate bone density must be accumulated and conserved, but to prevent subsequent fracture, bone density must be augmented so that the supportive function of the skeleton can be restored [3]. Almost 50 years after the recognition of postmenopausal osteoporosis as a clinical entity [1], not one of the many treatments that have been used has been demonstrated to be efficacious in reducing subsequent fracture risk. My purpose is not to recite this chronicle of disappointment, but to account for it in terms of bone biology, to consider some possible exceptions, and to reiterate the importance of preventing damage to the skeleton rather than belatedly attempting its repair.

Bone Remodeling and Bone Loss or Gain

The essence of bone remodeling in the adult human is the focal excavation and repair of microscopic cavities on bone surfaces. The outcome of each remodeling episode depends upon the balance between resorption depth, which is an expression of the collective work of a team of osteoclasts, and wall thickness, which is an expression of the collective work of a team of osteoblasts. It is an empirical observation that for most of adult life wall thickness on the endosteal envelope is less than resorption depth, so that each remodeling episode results in a small loss of bone [22]. There is only a brief window of time during which the osteoblasts needed for complete repair must be recruited and assembled [26], and when each has exhausted its limited capacity for matrix synthesis, the remodeling transaction is completed, and its outcome, like that of a completed financial transaction, is irrevocable. It is in this sense that the loss of bone intrinsic to the remodeling process on the endosteal envelope is irreversible.

Because of the delay between resorption and formation, there is a temporary mineral deficit associated with bone remodeling, comprising bone matrix not yet formed and bone matrix formed but not yet fully mineralized [16]. The magnitude of the deficit is proportional to the rate of bone turnover, and averages about 2 percent of the total for the whole body, and about 10 percent in regions of relatively high turnover such as the spine [23]. An increase in the frequency of remodeling activation and consequent increase in bone turnover will be accompanied by a corresponding increase in the reversible deficit and reduction in skeletal mineral content. Conversely, a reduction in remodeling activation will be accompanied by a reduction in the reversible deficit and a corresponding increase in skeletal mineral content. The reversibility of bone loss resulting from increased turnover depends upon restoring bone turnover to its previous level, that is the basis for almost all instances of an increase in bone density as an effect of treatment in an adequately controlled study [23], or as a consequence of curing the underlying

disease [28]. Bone gain by this mechanism generally continues only for about 6 months, and in most patients will not restore bone density to normal.

Reversible bone loss depends upon the number of *uncompleted* remodeling transactions in progress at a particular time, whereas irreversible bone loss depends upon the number of *completed* remodeling transactions that have accumulated over some period of time. Both types of bone loss occur in hyperparathyroidism. After successful surgical treatment of primary hyperparathyroidism, the increase in cortical porosity is reversed but the rise in bone density continues only for about 6 months and amounts only to about 10 percent of the total deficit [16, 20]. Cortical thinning, due to the cumulative effect of increased resorption depth on the endocortical surface in conjunction with increased frequency of remodeling activation, is unaffected. Even the large increase in bone mineral that results from the treatment of osteomalacia has no effect on the cortical thinning that is due to the associated secondary hyperparathyroidism [17]. Both types of bone loss also occur in estrogen deficiency, accounting for the different effects on bone density of estrogen replacement, depending on its timing [10]. If begun before bone turnover has increased, bone density remains stable with neither loss nor gain. If begun 2 years later, when bone turnover is still high, there is an initial increase in bone density until the expected plateau is attained. Finally, if begun 5 years later, when turnover has returned to normal, further loss is prevented but there is no initial increase.

Other Possible Mechanisms of Bone Gain

During growth, cancellous bone is made by endochondral ossification and cortical bone mainly by subperiosteal apposition. If osteoporosis occurs during the growth period, and its cause is either amenable to treatment (as in Cushings syndrome), or subject to spontaneous disappearance (as in idiopathic juvenile osteoporosis), the remaining potential for growth may allow restitution of an almost normal skeleton [18], but this is no longer possible in adults. Endochondral ossification necessarily stops after epiphysial closure, but periosteal apposition continues slowly throughout life and may lead to a net gain of bone in subjects older than 90 years, in whom endosteal loss has become very slow [5]. Periosteal gain can be amplified by an increase in frequency of remodeling activation for any reason, but the gain is insufficient to offset increased endosteal loss [15]. Periosteal gain is amplified by exercise without endosteal loss [12], but the net effect is of only modest magnitude. Physiologically excessive strain in response to biomechanical loading [21] and prostaglandin administration [4] may both lead to rapid gain of laminar or plexiform bone at the periosteum, but this would be cosmetically unacceptable, and if generalized, could lead to compression of nerves as they run through cranial or spinal foramina.

Prevention of fractures is likely to require reversal of the normally inexorable loss of bone from the endosteal envelope, for which it is both a necessary and a sufficient condition that wall thickness exceeds resorption depth [22]. Sodium fluoride apparently bypasses the normal remodeling sequence, with direct transformation of quiescent to forming surfaces, but the bone initially formed is structurally abnormal, and in controlled trials vertebral fracture rate has not been reduced [8]. Furthermore, axial bone gain can be offset by appendicular bone loss. Coherence therapy has the potential for adding bone if osteoclast recruitment can be increased and the new osteoclasts constrained to erode shallower than normal cavities that can be overfilled by normally functioning osteoblasts, but none of the assumptions underlying this concept have yet been validated [25]. Cyclic administration of human parathyroid hormone in conjunction with calcitriol has led to impressive gains in spinal bone mineral in a few patients with idiopathic osteoporosis [31], but the

cellular mechanism is unknown, and there have been no measurements of fracture rate and no controlled trials with this form of treatment.

Architectural Aspects or Bone Loss and Gain—the Necessity for Prevention

Rapid postmenopausal loss of cancellous bone is a reflection of increased resorption depth leading to perforation of trabecular plates, disruption of structural connectivity, and disproportionate reduction in compressive strength of the vertebral bodies [21]. The complete removal of a structural element confers another dimension of irreversibility, since there is no longer a surface on which the osteoblasts can build. Even the largest attainable increase in thickness of the structural elements that remain will fail to restore normal connectivity, and so will fail to restore normal bone strength [19]. It is probably for this reason that the increase in bone density produced by sodium fluoride does not reduce the risk of fracture. It is conceivable that induction of osseous metaplasia in the marrow, for example by the local instillation of bone morphogenetic protein [32], might allow large discontinuities to be bridged, but uncontrolled proliferation of woven bone could displace hematopoietic tissue, curing osteoporosis at the expense of causing aplastic anemia.

Many patients with postmenopausal osteoporosis have defects in the activation of bone remodeling and in the recruitment and function of osteoblasts that may prevent adaptation to the initial bone loss and contribute to bone fragility by compromising the detection and repair of fatigue microdamage [24]. The sequence of rapid osteoclast-mediated bone loss and decreased connectivity, followed by slow osteoblast-mediated bone loss and accumulation of bone of increased age and liability to fatigue damage, occurs also in the osteoporoses of corticosteroid excess, traumatic osteodystrophy and immobilization, and in some patients with intestinal malabsorption and hepatobiliary disease [22]. But, even if the osteoblast dysfunction in these disorders were of known cause and fully correctable by treatment, by the time that the first spontaneous vertebral fracture had occurred, restoration of normal cancellous bone architecture would still be impossible.

Prevention of estrogen-dependent bone loss by estrogen replacement therapy reduces the occurrence of measurable vertebral deformations by at least 90 percent [9], so that the abnormalities in bone remodeling described earlier will only rarely be of clinical significance unless the initial osteoclast-mediated loss of bone is allowed to occur. These considerations establish the cardinal importance of early intervention, long before the first fracture. For women, who should not or will not take estrogen replacement therapy or who are unable to tolerate it, the discovery of safe and effective substitutes, and for patients at risk for other forms of osteoporosis, the discovery of new preventive measures, should become the most important focus of osteoporosis research. For the immediate future, unless they are still losing cortical bone faster than normal, patients with established spinal osteoporosis will continue to benefit more from advice than from medication.

Bone Loss and Microgravity

Application of these concepts to the skeletal problems of space flight is unclear because correspondence between the effects of microgravity and any terrestrial model for microgravity has not been demonstrated. The available densitometric and metabolic balance data from Skylab are consistent with the known effects of prolonged bed rest [17], but for neither microgravity nor bed rest have the bone remodeling mechanisms been adequately studied. The reversibility of bed-rest induced bone loss [6] and the increase in biochemical markers of bone remodeling [11] indicate that more frequent activation of bone remodeling and consequent increase in cell team recruitment is the most likely principal mechanism [22], but the continuous decline in bone density for more than 6 months [30] suggests an additional effect on differentiated cells. In immobilized monkeys,

there is an increase in depth as well as extent of osteoclastic resorption [13] and in the rat, microgravity leads to a profound depression of osteoblast function [14]. Limited data are consistent with similar effects of bed rest in human subjects [7, 11, 33].

A temporary enhancement of the activity of existing osteoclasts or a temporary interruption or retardation of the activity of existing osteoblasts are not inconsistent with complete reversibility, but a persistent increase in the number of osteoclasts or decrease in the number of osteoblasts recruited for each team would be more likely to cause an irreversible bone deficit [22]. To address these uncertainties, persons returning from space flight of progressively longer duration must undergo both sequential bone densitometry and bone biopsy after tetracycline labeling. This advice has been disregarded since it was first given more than 10 years ago [17], but only when the pathogenesis of microgravity related bone loss is better understood can preventive measures be derived and the role of exercise be determined.

References

[1] ALBRIGHT, F., F. BLOOMBERG, AND P. H. SMITH. Postmenopausal osteoporosis. *Trans. Assoc. Am. Phys.* 55:298-305, 1940.

[2] ARNAUD, S. B., D. J. SHERRARD, N. MALONEY, R. T. WHALEN, AND P. FUNG. Reduced bone formation is measureable in the iliac crest of normal men after 7 days in a bedrest model of weightlessness. *J. Bone Min. Res.* 4(1):S233, 1989.

[3] ERIKSEN, E. F., S. F. HODGSON, AND B. L. RIGGS. Treatment of osteoporosis with sodium fluoride. In: *Osteoporosis: Etiology, Diagnosis and Management,* edited by B. L. Riggs and L. J. Melton. New York: Raven Press, 1988, p. 415-432.

[4] HIGH, W. B. Effects of orally administered prostaglandin E-2 on cortical bone turnover in adult dogs: a histomorphometric study. *Bone* 8:363-373, 1988.

[5] HUI, S. L., P. S. WISKE, J. A. NORTON, AND C. C. JOHNSTON, JR. A prospective study of change in bone mass with age in postmenopausal women. *J. Chron. Dis.* 35:715725, 1982.

[6] HULLEY, S. B., J. M. VOGEL, C. L. DONALDSON, AND J. H. BAYERS. The effect of supplemental oral phosphate on the bone mineral changes during prolonged bed rest. *J. Clin. Invest.* 50:2506-2518, 1971.

[7] JUDGE, D. M., V. SCHNEIDER, AND A. LEBLANC. Disuse osteoporosis. *J. Bone Min. Res.* 4(1):S238, 1989.

[8] KLEEREKOPER, M., E. PETERSON, E. PHILLIPS, D. NELSON, B. TILLEY, AND A. M. PARFITT. Continuous sodium fluoride therapy does not reduce vertebral fracture rate in postmenopausal osteoporosis. *J. Bone Min. Res.* 4(Sl):S376, 1989.

[9] LINDSAY, R., D. M. HART, C. FORREST, AND C. BAIRD. Prevention of spinal osteoporosis in Oophorectomized women. *Lancet* ii:1151-1154, 1980.

[10] LINDSAY, R. Sex steroids in the pathogenesis and prevention of osteoporosis. In: *Osteoporosis: Etiology, Diagnosis and Management,* edited by B. L. Riggs, and J. L. Melton. New York: Raven Press, 1988, p. 333-358.

[11] LUEKEN, S., S. ARNAUD, A. K. TAYLOR, K. H. W. LAU, V.

PERKEL, AND D. J. BAYLING. Bedrest causes an acute and progressive increase in skeletal resorption in normal men: indirect evidence that this is mediated by osteocalcin. *J. Bone Min. Res.* 4(1):S308.

[12] MARTIN, A. D., D. A. BAILEY, J. B. LEICESTER, AND I. GULKA. Bone and muscle relationships in the forearms of lifetime tennis players. In: *International Symposium on Osteoporosis,* edited by C. Christiansen, J. S. Johansen, and B. J. Riis. Denmark: Viborg, 1987, p. 599-602.

[13] MATHEWS, C. H. E., S. P. ASWANI, AND A. M. PARFITT. Hypo-gravitational effects of hypodynamcis on bone cell function and the dynamics of bone remodeling. In: A 14-day Ground-Based Hypokinesia Study in Nonhuman Primates - A Compilation of Results. *NASA Technical Memorandum 81268,* April 1981.

[14] MOREY, E. R. AND D. J. BAYLINK. Inhibition of bone formation during space flight. *Science* 1138-1141, 1978.

[15] PARFITT, A. M. Richmond Smith as a clinical investigator: his work on adult periosteal bone expansion, and on nutritional and endocrine aspects of osteoporosis, in the light of current concepts. *HFH Med. J.* 28:95-107, 1980.

[16] PARFITT, A. M. The morphologic basis of bone mineral measurements. Transient and steady state effects of treatment in osteoporosis. Editorial, *Mineral Electrolyte Metab.* 4:273-287, 1980.

[17] PARFITT, A. M. Bone effects of space flight: analysis by quantum concept of bone remodeling. *Acta Astronautica* 8:1083-1090, 1981.

[18] PARFITT, A. M. AND H. DUNCAN. Metabolic bone disease affecting the spine. In: *The Spine, 2nd Edition,* edited by R. Rothman and F. Simeone. Philadelphia: W. B. Saunders, 1982, p. 775-905.

[19] PARFITT, A. M. Remodeling and microstructure of bone: relation to prevention of age related fracture. In: *Second International Conference on Osteoporosis: Social and Clinical Aspects,* edited by A. Vagenakis, P. Soucacos, A. Avramides, G. Serge, and L. Deftos. Milano: Masson Italia Editori SIA, 1986, p. 197-209.

[20] PARFITT, A. M. Primary and secondary hyperparathyroidism. In: *Current Concepts of Bone Fragility,* edited by H. Uhthoff and Z. F. G. Jaworski. Berlin Heidelberg: SpringerVerlag, 1986:279-285.

[21] PARFITT, A. M. Trabecular bone architecture in the pathogenesis and prevention of fracture. *Am. J. Med.* 82(SIB):68-72, 1987.

[22] PARFITT, A. M. Bone remodeling: relationship to the amount and structure of bone and the pathogenesis and prevention of fractures. In:

Management, edited by B. L. Riggs and L. J. Melton. New York: Raven Press, 1988, p. 45-93.

[23] PARFITT, A. M. The composition, structure and remodeling of bone: a basis for the interpretation of bone mineral measurements. In: *Bone Mineral Measurements by Photon Absorptiometry: Methodological Problems,* edited by J. Dequeker, P. Geusens, and H. W. Wahner. Leuven: Leuven University Press, 1988, p. 9-28.

[24] PARFITT, A. M. Surface specific bone remodeling in health and disease. In: *Clinical Disorders of Bone and Mineral Metabolism,* edited by M. Kleerekoper and S. Krane. New York: Mary Ann Liebert Publishers, Inc., 1989, p. 7-14.

[25] PARFITT, A. M. ADFR, or coherence therapy, for osteoporosis. .In: *Proceedings of the Nineteenth Steenbock Symposium, Osteoporosis,* edited by H. G. Deluca, et al. New York: Elsevier Science Publishing Co., 1989 (in press).

[26] PARFITT, A. M. Bone forming cells in clinical disorders. In: *Bone: A Treatise, Osteocyte*, edited by B. K. Hall. 1989 (in press).

[27] PARFITT, A. M., D. S. RAO, J. STANCIU, A. R. VILLANUEVA, M. KLEEREKOPER, AND B. FRAME. Irreversible bone loss in osteomalacia: comparison of radial photon absorptiometry with iliac bone histomorphometry during treatment. *J. Clin. Invest.* 76:2430-2412, 1985.

[28] POCOCK, N. A., J. A. EISMAN, C. R. DUNSTAN, R. A. EVARLS, D. H. THOMAS, AND N. L. HUG. Recovery from steroid-induced osteoporosis. *Ann. Intern. Med.* 107:319-323, 1987.

[29] RUBIN, C. T. AND L. E. LANYON. Regulation of bone mass by mechanical strain magnitude. *Calcif. Tissue Int.* 37:411-417, 1985.

[30] SCHNEIDER, V. S. AND J. MCDONALD. Skeletal calcium homeostasis and countermeasures to prevent disue osteoporosis. *Calcif. Tissue Int.*

[31] SLOVIK, D. M., D. I. ROSENTHAL, S. H. DOPPELT, J. T. POTTS, M. A. DALY, J. S. CAMPBELL, AND R. M. NEER. Restoration of spinal bone in osteoporotic men by treatment with human parathyroid hormone (1-34) and 1,25-dihydroxyvitamin D. *J. Bone Min. Res.* 1:377-388, 1986.

[32] URIST, M. R., R. J. DELANGE, AND G. A. M. FINERMAN. Bone cell differentiation and growth factors: induced activity of chondro-osteogenetic DNA. *Science* 220:680-686, 1983.

[33] VICO, L., D. CHAPPARD, C. ALEXANDRE, S. PALLE, P. MINAIRE, G. RIFFAT, B. MORUKOV,
Effects of a 120-day period of bed rest on bone mass and bone cell activities in man: attempts at countermeasure. *Bone and Mineral* 2:383-394, 1987.

N94-28370

Exercise and Osteoporosis: Methodological and Practical Considerations

Jon E. Block, Ph.D., Anne L. Friedlander, Peter Steiger, Ph.D.,
and Harry K. Genant, M.D.
Department of Radiology
University of California at San Francisco
San Francisco, California

59-52
207618
p. 6

Introduction

Physical activity may have important implications for enhancing bone density prior to the initiation of space flight, for preserving bone density during zero gravity, and for rehabilitating the skeleton upon return to Earth. Nevertheless, the beneficial effects of exercise upon the skeleton have not been proven by controlled trials and no consensus exists regarding the type, duration, and intensity of exercise necessary to make significant alterations to the skeleton. The following sections review our current understanding of exercise and osteoporosis, examine some of the methodological shortcomings of these investigations, and make research recommendations for future clinical trials.

Cross-sectional Studies: Exercisers versus Comparison Subjects

The majority of studies that have compared individuals participating in habitual physical activity with less active comparison subjects have found greater bone density in the former [1, 2]. The finding of higher bone density values among athletes does not appear to be site or exercise specific. For example, in two classic studies Jones *et al.* [3] and Huddleston *et al.* [4] observed unilaterally greater bone density in the playing arms of lifetime tennis athletes when compared with their nondominant arms. However, a number of additional studies [5, 6, 7] have found similarly higher levels of peripheral bone density regardless of the type of activity. Athletes also appear to have greater spinal bone density whether engaged in activities that are weight-bearing [8, 9, 10] or nonweight-bearing [10, 11]. Recent studies likewise found greater hip bone density among athletes [10, 12]. While the bulk of these types of studies shows greater levels of bone density in athletes, not all do [2, 13] and their cross-sectional nature suggests that selection bias may be another reasonable explanation for these apparently positive findings.

Cross-sectional Studies: Fitness Predictors of Bone Density

In an attempt to explain the variability in bone density seen in normal populations as well as athletes, a number of investigators have examined the predictive ability of objective measures of physical fitness.
are some of the variables that have been assessed and related to standard determinations of bone density.

Pocock *et al.* [14] and Chow *et al.* [15] observed that aerobic capacity was an independent predictor of both spine, hip, and total body bone density. In the study by Pocock and colleagues [14], aerobic capacity, in fact, was found to explain a greater amount of the variability in bone density than either age or parameters of body mass. The findings of these first studies led some investigators to conclude that participation in any activity that significantly increases aerobic capacity will have a salubrious effect on bone as well. However, several subsequent studies have failed to confirm these earlier findings. Bevier *et al.* [16] found that aerobic capacity was not correlated with radial or spinal bone density in older women; however, in older men the relationship was significant. A more recent study by Pocock and coworkers [17] showed that

muscular strength had greater predictive value than aerobic capacity at the hip in women over the entire age range.

Block and colleagues [10] did not observe a significant relationship between aerobic capacity and bone density in either the trabecular portion of the spine or the hip in young men engaged in rigorous aerobic or anaerobic physical activity. Lastly, Kirk *et al.* [18] observed a strongly positive ($r=0.509$, $p<0.03$) relationship between aerobic capacity and spinal trabecular bone density in sedentary women and female runners who were premenopausal; this relationship did not hold up among postmenopausal women. One explanation for these equivocal findings is that activity, per se, may have some impact on bone density but the mechanism may not be one where the predominant factor is aerobic capacity. Athletic individuals have generally greater bone density and generally greater aerobic capacity, but whether the two are uniquely related remains uncertain.

Investigations that have examined the role of muscular strength in predicting the variability in bone density have consistently, although not without exception, found that this fitness parameter is an important contributor. Sinaki and colleagues [19], Bevier *et al.* [16], and Pocock *et al.* [17] all observed a moderately strong relationship between bone density at several sites and muscular strength as assessed by varying methods. While Block *et al.* [10] did not find a significant correlation between spinal trabecular bone density and back strength, they did observe a robust relationship between the cross-sectional area of the paraspinous musculature and the density of both the trabecular and integral portions of the thoracolumbar spine.

Several other parameters of physical fitness have likewise been found to be associated with bone density including daily energy expenditure and determinations of body composition. Kanders and colleagues [20] showed a moderately strong correlation ($r=0.41$, $p<0.005$) between average daily energy expenditure as expressed in kilocalories and vertebral integral bone density. Results have been somewhat mixed when body composition studies have been examined as possible predictors of bone density. Pocock *et al.* [17] observed a positive association between body mass index and femoral bone density in addition to it being a strong, independent predictor. However, Stillman and coworkers [21] observed that while females with a history of physical activity had greater bone density, they were significantly leaner.

Prospective Studies

The limited number of prospective studies of the effects of various types of exercise programs on bone density have yielded conflicting findings [1]. The majority of studies failed to show that exercise of short duration can significantly enhance radial compact bone density [22, 23, 24, 25]; although in several of these studies, the nonexercising group showed significant losses over the study period [25, 24]. Report of a long-term trial by Smith and colleagues [26] found favorable results of a 4-year exercise program on radial bone density in women over a large age range. Additionally, an earlier study by Smith and coworkers [27] reported a significant increase in radial bone density among nursing home residents participating in a 3-year fitness regimen.

Prospective studies examining the benefits of exercise at the spine have been few in number, although several controlled trials are currently underway. Krolner *et al.* [25] observed an insignificant increase in spinal integral bone density among postmenopausal women engaged in a physical fitness program; however, the posttreatment differences between exercisers and less active control subjects was significant. Dalsky and colleagues [28], on the other hand, found a significant rise in spinal integral bone density following 9 months of running exercise in postmenopausal women, but could not identify a significant difference between intervention and control women over this duration.

In a review of the literature, Block *et al.* [1, 2] suggested that the disparate findings from prospective studies may, in part, be due to serious methodological shortcomings. While investigations of prospective design provide the only means of establishing valid relationships between exercise and bone density accretion, good methodological rigor has not been adhered to in most of the clinical trials that have been undertaken. Randomization is the best way to avoid many of the biases that can lead to invalid results. Unfortunately, only one published study has utilized a randomized design [29]; this investigation showed no effect of a generalized walking program on radial bone density among a large population of postmenopausal women. Lack of randomization in all other prospective trials renders their results suspect.

Several additional methodological flaws are evident in prospective investigations of exercise and bone density and may have lead to invalid conclusions. No study accounted for extraneous events and influences that took place between baseline and final bone density measurements (e.g., drug and medication usage, dietary changes, additional fitness programs), no estimations were made of the sample size or power necessary to detect "real" changes in bone density, questionnaire instruments used to categorize subjects lacked appropriate validation, and no attempt was made to identify characteristics of dropouts or to adjust statistically for the significant attrition that occurred in several of the studies.

Certainly the lack of methodological rigor, coupled with the somewhat mixed results from the published prospective trials to date, make it difficult to confidently recommend exercise as an effective agent for building or maintaining bone density. Gains observed after initiating exercise have not been nearly as impressive as those anticipated by extrapolating from the cross-sectional studies of athletes and nonexercisers. Additionally, because no consensus has been reached on the type, duration, or intensity of exercise necessary to make significant bone density gains, no clinical prescription can be currently recommended. Lastly, physiological and practical considerations must be given equal consideration when designing exercise programs. Recent research using animal and in vitro models suggests that certain biomechanical actions may have the greatest benefit for the skeleton. However, whether these theories can be translated into practical fitness program remains to be tested. From a public health standpoint, an exercise program that provides only a small benefit to the skeleton but can be adhered to by a large cross-section of the general population has greater ramifications than a very specific regimen, with perhaps potentially large skeletal benefits, that can only be adhered to by a select few.

Current Research Priorities and Conclusions

Exercise strategies have tremendous potential for affecting bone density and reducing the likelihood of osteoporosis over a large age range compared to other possible interventions. For example, fitness programs might be utilized in young adults to enhance the level of peak bone density (i.e., primary prevention). Additionally, either by itself or in combination with some other intervention, exercise may be effective in reducing the rapid loss of bone that occurs with the cessation of ovarian function at the time of menopause (i.e., secondary prevention). Lastly, fitness programs might be used among the elderly population to modify risk factors for falls and fractures without specific increases occurring necessarily in bone density (i.e., tertiary prevention).
Randomized, prospective trials need to be undertaken in a number of different age and sex populations, examining the effects of different types and durations of exercise on bone density and other factors associated with fracture. These rigorously designed studies potentially will rectify the methodological shortcomings inherent in previous prospective trials. We believe immediate research attention should be focused in two distinct areas: enhancement of peak bone density and possible detraining effects on the skeleton. It is thought that the level of peak bone density is a

support this hypothesis. However, Melton *et al.* [30] have projected from population-based data at the Mayo Clinic that any alterations made to bone density among younger adults will have significantly greater effects at reducing the risk of fracture than equivalent changes made among older adults. In fact, their projections would indicate that even therapeutic interventions that have large effects on the skeleton in the elderly will do little to reduce the risk of hip fracture. Regarding the focus on detraining issues, one prospective study [28] suggests that any gains in bone density made as a result of fitness training will be lost following the cessation of exercise. This concept should take a research priority because of its particular relevance to long-term space flight. Controlled trials should be undertaken to explore the specific detraining effects as a function of age, sex, type and duration of exercise, as well as an examination of possible maintenance programs that might be incorporated if bone density can be enhanced preflight.

REFERENCES

[1] BLOCK, J. E., R. SMITH, D. BLACK, AND H. K. GENANT. Does exercise prevent osteoporosis? *JAMA* 257:3115-3117, 1987.

[2] BLOCK, J. E., R. SMITH A. L. FRIEDLANDER, AND H. K. GENANT. Prevention of osteoporosis with exercise: a review with emphasis on methodology. *Med. Hypotheses*, 1989. (In Press)

[3] JONES, H. H., PRIEST, J. D., W. C. HAYES, ET AL.: Humeral hypertrophy in response to exercise. *J. Bone Joint Surg.* 59A:204-208, 1977.

[4] HUDDLESTON, A. L., D. ROCKWELL, D. N. KULUND, AND R. B. HARRISON. Bone mass in lifetime tennis athletes. *JAMA* 244(10):1107-1109, 1980.

[5] JACOBSON, P. C., W. BEAVER, S. A. GRUBB, ET AL. Bone density in women: college athletes and older athletic women. *J. Orthop. Res.* 1984.

[6] ALOIA, J. F., A. VASWANI, H. ATKINS, I. ZANZI, K. ELLIS, AND S. H. COHN. Radiographic morphometry and osteopenia in spinal osteoporosis. *J. Nucl. Med.* 18:425-431, 1977.

[7] DALEN, N. AND K. E. OLSSON. Bone mineral content and physical activity. *Acta Orthop. Scand.* 45:170-174, 1974.

[8] BLOCK, J. E., H. K. GENANT, AND D. BLACK. Greater vertebral bone mineral mass in exercising young men. *West J. Med.* 145:39-42, 1986.

[9] LANE, N. E., D. BLOCK, H. H. JONES, ET AL. Long-distance running, bone density, and osteoarthritis. *JAMA* 255(9):1147-1151, 1986.

[10] BLOCK, J. E., A. L. FRIEDLANDER, G. A. BROOKS, P. STEIGER, H. A. STUBBS, AND H. K. GENANT. Determinants of bone density among athletes engaged in weight-bearing and non-weight bearing activity. *J. Appl. Physiol.* 67(3):1100-1105, 1989.

[11] ORWOLL, E. S., J. FERAR, S., K. OVIATT, K. HUNTINGTON, AND M. R. MCCLUNG. Swimming exercise and bone mass. edited by C. Christiansen, J. S. Johansen, and B. J. Riis. Proceedings of the International Symposium on Osteoporosis, September 27 - October 2, 1987. Copenhagen, Denmark: Osteopress ApS 1987, Volume 1, p. 494-498.

[12] COLLETTI, L. A., J. EDWARDS, L. GORDON, J. SHARY, AND N. H. BELL. The effects of muscle-building exercise on bone mineral density of the radius, spine, and hip in young men. *Calcif. Tissue Int.* 45:12-14, 1989.

[13] BREWER, V., B. M. MEYER, M. S. KEELE, S. J. UPTON, AND R. D. HAGAN. Role of exercise in prevention of involutional bone loss. *Med. Sci. Sports Exer.* 15(6):445-449, 1983.

[14] POCOCK, N. A., J. A. EISMAN, M. D. YEATES, ET AL. Physical fitness is a major determinant of femoral neck and lumbar spine bone density. J. Clin. Invest. 78:618-621, 1986.

[15] CHOW, R. K., J. E. HARRISON, C. F. BROWN, ET AL. Physical fitness effect on bone mass in postmenopausal women. *Arch. Phys. Med. Rehabil.* 67:231-234, 1986.

[16] BEVIER, W. C., R. A. WISWELL, G. PYKA, K. C. KOZAK, K. M. NEWHALL, AND R. MARCUS. Relationship of body composition, muscle strength, and aerobic capacity to bone mineral density in older men and women. *J. Bone Min. Res.* 4(3):421-432, 1989.

[17] POCOCK, N., J. EISMAN, T. GWINN, P. SAMBROOK, P. KELLY, J> FREUND, AND M. YEATES. Muscle strength, physical fitness, and weight but not age predict femoral neck bone mass. J. *Bone Min. Res.* 4(3):441-448, 1989.

[18] KIRK, S., C. F. SHARP, N. ELBAUM, D. B. ENDRES, S. M. SIMONS, J. G. MOHLER, AND R. K. RUDE. Effect of long-distance running on bone mass in women. *Resear.* 4(4):515-522, 1989.

[19] SINAKI, M., M. C. MCPHEE, S. F. HODGSON, ET AL. Relationship between bone mineral density of spine and strength of back extensors in healthy postmenopausal women. *Mayo Clin. Proc.* 61:116-122, 1986.

[20] KANDERS, B., D. W. DEMPSTER, AND R. LINDSAY. Interaction of calcium nutrition and physical activity on bone mass in young women. *J. Bone Min. Resear.* 3(2):145-149, 1988.

[21] STILLMAN, R. J., T. G. LOHMAN, M. H. SLAUGHTER, AND B. H. MASSEY. Physical activity and bone mineral content in women aged 30 to 85 years. *Med. Sci. Sports Exer.* 18(5):576-580, 1986.

[22] ALOIA, J. F., S. H. COHN, J. A. OSTUNI, ET AL. Prevention of involutional bone loss by exercise. *Ann. Intern. Med.* 89:356-358, 1978.

[23] SIMKIN, A., J. AYALON, AND I. LEICHTER. Increased trabecular bone density due to bone-loading exercises in postmenopausal osteoporotic women. *Calcif. Tissue Int.* 40:59-63, 1987.

[24] WHITE, M. K., R. B. MARTIN, R. A. YEATER, ET AL. The effects of exercise on the bones of postmenopausal women. *Int. Orthop.* 7:209-214, 1984.

[25] KROLNER, B., B. TOFT, S. P. NIELSEN, ET AL. Physical exercise as prophylaxis against involutional vertebral bone loss: a controlled trial. Clin. Sci. 64:541-546, 1983.

[26] SMITH, E. L., C. GILLIGAN, M. MCADAM, C. P. ENSIGN, AND P. E. SMITH. Deterring bone loss by exercise intervention in premenopausal and postmenopausal women. *Calcif. Tissue Int.* 44:312-321, 1989.

[27] SMITH, E. L. , W. REDDAN, AND P. E. SMITH. Physical activity and calcium modalities for bone mineral increase in aged women. *Med. Sci. Sports Exer.* 13:60-64, 1981.

[28] DALSKY, G. P., K. S. STOCKE, A. A. EHSANI, ET AL. Weight-bearing exercise training and lumbar bone mineral content in postmenopausal women. *Ann. Intern. Med.* 108:824-828, 1988.

[29] SANDLER, R. B., J. A. CAULEY, D. L. HOM, D. SASHIN, AND A. M. KRISKA. The effects of walking on the cross-sectional dimensions of the radius in postmenopausal women. *Calcif. Tissue Int.* 41:65-69, 1987.

[30] MELTON, L. J., S. H. KAN, H. W. WAHNER, AND B. L. RIGGS. Lifetime fracture risk: an approach to hip fracture risk assessment based on bone mineral density and age. *J. Clin. Epidemiol.* 41(10):985-994, 1988.

omit

Section 4

Electrical Stimulation in Exercise Training

N94-28371

510-52
207619
p. 9

Electrical Stimulation in Exercise Training

Walter Kroll, Ph.D.
Department of Exercise Science
University of Massachusetts
Amherst, Massachusetts

Introduction

Electrical stimulation has a long history of use in medicine dating back to 46 A.D. when the Roman physician Largus found the electrical discharge of torpedo fishes useful in the treatment of pain produced by headache and gout. A rival Greek physician, Dioscorides, discounted the value of the torpedo fish for headache relief but did recommend its use in the treatment of hemorrhoids. In 1745, the Leyden jar and various sized electrostatic generators were used to treat angina pectoris, epilepsy, hemiplegia, kidney stones, and sciatica. Benjamin Franklin used an electrical device to treat successfully a young woman suffering from convulsive fits. In the late 1800's battery powered hydroelectric baths were used to treat chronic inflammation of the uterus while electrified athletic supporters were advertised for the treatment of male problems.

Fortunately, such an amusing early history of the simple beginnings of electrical stimulation did not prevent eventual development of a variety of useful therapeutic and rehabilitative applications of electrical stimulation. Over the centuries electrical stimulation has survived as a modality in the treatment of various medical disorders with its primary application being in the rehabilitation area. Recently, a surge of new interest in electrical stimulation has been kindled by the work of a Russian sport scientist who reported remarkable muscle strength and endurance improvements in elite athletes. Yakov Kots reported his research on electric stimulation and strength improvements in 1977 at a Canadian-Soviet Exchange Symposium held at Concordia University in Montreal. Since then an explosion of new studies has been seen in both sport science [9] and in medicine [13].

Based upon the reported works of Kots and the present surge of new investigations, one could be misled as to the origin of electrical stimulation as a technique to increase muscle strength. As a matter of fact, electric stimulation has been used as a technique to improve muscle strength for over a century. Bigelow [3] reported in 1894, for example, the use of electrical stimulation on a young man for the purpose of increasing muscle strength. Employing a rapidly alternating sinusoidal induced current and a dynamometer for strength testing, Bigelow reported that the total lifting capacity of a patient increased from 4328 pounds to 4639 pounds after only 25 minutes of stimulation. In 1965, Massey *et al.* [11] reported on the use of an Isotron electrical stimulator that emitted a high frequency current. Interestingly enough, the frequencies used by Massey *et al.* and the frequencies used by Bigelow in 1894 were in the same range of frequencies reported by Kots a being the most effective in strength development. It would seem the Russian secret of high frequency electrical stimulation for strength development, then, is not a modern development at all.

Strength Improvement by Electrical Stimulation

Kots claimed that electrical stimulation could produce a stronger muscular contraction than that possible via a maximal volitional effort. Such a claim attracted great attention in the exercise science community where optimal strength development is believed to be produced by overload training; that is, the muscle contraction must exceed some critical tension level for strength improvement to occur. If electrical stimulation could indeed produce a supernormal maximal

PRECEDING PAGE BLANK NOT FILMED C-3 PAGE 182 INTENTIONALLY BLANK

contraction and activate more muscle fibers than a volitional contraction, it could constitute a super strength training technique. And, indeed, Kots presented data that 20 10-minute electrical-stimulation sessions increased strength by 40 percent in athletes and also resulted in a decrease of subcutaneous fat under the stimulated area. Such results were clearly in the realm of spectacular and researchers hurried to study the new super training technique.

But, alas, the almost unbelievable claims made for electrical stimulation as a super training technique were not reproduced by other researchers. As Nelson and Currier [13] noted in their review of electrical stimulation as a strength development technique, peak torque developed by electrical stimulation of muscle was consistently less than that produced by a maximal voluntary contraction. If electrical stimulation was combined with a voluntary contraction, the torque developed was less than that produced by the voluntary contraction alone. Although Nelson and Currier did not identify the cause of the latter effect, it would be described by Russian neurophysiologists as the well known Wedenski inhibition that occurs when two high-intensity stimuli occur simultaneously. Certainly the notion of a supermaximal contraction and an enhanced overload training effect simply was not seen.

An earlier review by Kramer and Mendryk [9] made note of the fact that different kinds of electrical stimulators were being employed with different wave forms, pulse frequencies, and maximum current capabilities. Many of the electrical stimulators lacked conformity with Kots' specifications for a sine wave pulse, high-frequency pulse rate to produce an anesthetic effect allowing high current flow for maximum activation of muscle tissue, number of stimulations administered, and single stimulation duration and rest interval. Specific details about the methodology employed were not provided in many of the electrical-stimulation strength studies. It might also be added that several electrical-stimulation strength studies employed incorrect statistical analysis techniques making their reported results subject to question. Notwithstanding such acknowledged limitations to any review of research findings, Kramer and Mendryk drew the only conclusion possible: "...the available literature does not support claims made for the Russian technique of electrical stimulation."

Concern about the proper application of the Russian technique a well a use of a proper electrical stimulator is justified before any final disposition can be valid of Kots' claims for electrical stimulation. It is interesting to note, however, that Kots was involved in a study in which he personally applied the electrical stimulation using his own stimulator [18]. The short-term effects over seven sessions with proper protocols and proper stimulator showed that isokinetic knee extension strength declined 13.6 and 17.6 percent in three female volleyball players and 16.7 and 3.1 percent in seven male football players at 24 deg/s and 126 deg/s, respectively. Only the difference at 24 deg/s for males was statistically significant although it must be noted that the small sample sizes of three and seven constitutes a serious deficiency for any statistical contrasts. Significant atrophy of fast twitch muscle fiber area occurred in the males. Thus, the Kots regimen not only failed to increase strength, it also failed to maintain strength in trained athletes. Both of these failures refute specific claims Kots had made for the Russian electrical-stimulation technique. The results of the Kots' involved study fail to provide any challenge to the conclusion that Russian claims of electrical stimulation as a super training technique are dubious.

Tonic Sensory Function of Electrical Stimulation

It would seem clear that the utilization of electrical stimulation as a means to increase strength is an interior substitute for simple voluntary muscle contraction. As the Russian M. A. Cherepakhin [5] noted back in 1977, electrical stimulation combined with physical exercise—isometric or dynamic—was no better than the physical exercise alone. And the same is true for deconditioned subjects (i.e., voluntary exercise is better than electrical stimulation for disuse atrophy). However, electrical stimulation might serve a function as a deconditioning preventive when used as tonic sensory protocol rather than as a strength training protocol.

Boris B. Yegerov [20] believed electrical stimulation might be used to combat some weightlessness effects not confined to muscle strength loss. Noting that low-voltage impulses could reduce vestibular vegetative reactions he stated that "...skeletal muscle under electrostimulation becomes the source of a strong stream of afferent impulses to the central nervous system. We are entitled to assume that the periodic repetition of such stimuli may counteract the vegetative reaction which takes place under conditions of weightlessness."

Rehabilitation specialists know that the speed with which muscle atrophy occurs is so rapid that a neural factor must be operative [10, 4, 17]. And it is also known that simple isometric contractions in a casted limb significantly deters muscle atrophy [15]. But it has also been shown that electrical stimulation, whether in combination with isometric contractions or alone, has a significant effect upon disuse atrophy [16, 6]. Perhaps the most instructive study might be that conducted by Gould *et al.* [8] who compared isometric contraction, control, and electrically stimulated groups of ten subjects each who wore long-leg casts from groin to toes for 2 weeks. The electrically stimulated group suffered significantly less thigh muscle volume loss than the other groups, retained its calf volume (loss of 0.98 percent, nonsignificant) and actually increased in ankle dorsiflexion strength. These results may be related to the beneficial effects electrical stimulation has been shown to have upon muscle protein synthesis and quadriceps atrophy due to immobilization [7].

Thus, high intensity electrical stimulation may not be essential to prevent a significant portion of disuse atrophy. There is even some limited evidence that electrical stimulation used as a tonic sensory protocol could maintain performance quality of some kinesthetic sense tasks [2]. In our own work using patterned electrical stimulation on stroke paralysis, thrice weekly sessions have produced return of somesthetic sense in hemiplegic limbs as well as return of significant movement capability in paralysis lasting up to 23 years. It would seem, therefore, that the investigation of electrical stimulation as a tonic sensory protocol has been neglected in disuse atrophy studies and may warrant closer scrutiny.

References

[1] ANDERS, F. F. AND S. ULLA. Transient decrease in number of motor units after immobilization in man. *J. Neurol. Neurosurg. Psychiat.* 41:924-929, 1978.

[2] BENNETT, K. AND A. DICKINSON. The effects of low intensity electrical current during immobilization on proprioceptive function. *Med. Sci. Sports Ex.* 4:119, 1982.

[3] BIGELOW, H. R. *An International System of Electro-Therapeutics.* London: F. J. Rebman, 1894.

[4] BUCHTHAL, F. AND A. MADSEN. Synchronous activity in normal and atrophic muscle. *Electoenceph. Clin. Neurophysiol.* 2:425-444, 1950.

[5] CHEREPAKHIN, M. A., YE. KAKURIN, I. IL'INA-KAKUYEVA, AND G. T. FEDERENKO. Evaluation of effectiveness of muscular electrostimulation for the prevention of disorders related to prolonged restriction of motor activity in man. *Kosm. Biol. Med.* 2:64-68, 1977.

[6] ERIKSSON, E. AND T. HAGGMARK. Comparison of isometric muscle training and electrical stimulation supplementing isometric muscle training in the recovery after major knee ligament surgery. *Amer. J. Sports Med.* 7:169-171, 1979.

[7] GIBSON, J. N. A., K. SMITH, AND M. J. RENNIE. Prevention of disuse muscle atrophy by means of electrical stimulation: maintenance of protein synthesis.

[8] GOULD, N., D. DONNERMEYER, M. POPE, AND ASHIKAGA. Transcutaneous muscle stimulation as method to retard disuse atrophy. *Clin. Orthop.* 164:215-220, 1982.

[9] KRAMER, J. F. AND S. W. MENDRYK. Electrical stimulation as a strength improveme[illegible] review. *J. Orthop.* [illegible] 4:91-98, 1982.

[10] LIPPMAN, R. K. experimental study of *Surg. Gynec. and Ob* 1928.

[11] MASSEY, B. H., R. C J. SHARKEY, T. COM C. OTTOT. Effects of electrical stimulation o strength of skeletal mus *Med. Phys. Fit.* 5:136-14

[12] MCNEAL, DONALD R. [illegible] of electrical stimulation. In: *Functional Electrical Stimulation*, edited by F. Terry Hambrecht and James B. Reswick. New York: Dekker, 1977, p.3-35.

[13] NELSON, ROGER M. AND DEAN P. CURRIER (eds.). *Clinical Electrotherapy*. Norwalk, CT: Appleton & Lange, 1987.

[14] PATEL, A. N., Z. A. RAZAK, AND D. K. DASTUR. Disuse atrophy of human skeletal muscle. *Arch. Neurol.* 20:413-421, 1969.

[15] ROZIER, C. K., J. D. ELDER, AND M. BROWN. Prevention of atrophy by isometric exercise of a casted leg. *J. Sports Med.* 19:191-194, 1979.

[16] STANISH, W. D., G. A. VALIANT, A. BONEN, AND A. N. BELCASTRO. The effects of immobilization and of electrical stimulation on muscle glycogen and myofibrillar ATPase. *Can. J. Appl. Spt. Sci.* 7:267-271, 1982.

[17] STOBOY, H. AND G. FRIEDEBOLD. Changes of electrical activity, of actual circumference of thigh during contraction and of strength-endurance product during isometric training in functional and organic muscle atrophy. *Med. Sport* 8:492-496, 1973.

DIANE, ALBERT W. ARCEL LAVOIE, W. D YAKOV M. KOTS. Hz sinusoidal current on ength of the quadriceps *orts Med.* 26:60-66,

NICE M. AND H. ER (eds.). Tonic ry systems. *Annals* . 290, 1977.

Soviet space M.D. by electricity for ...ssness. *Med. Trib.* 9:2, 1968.

[21] WOLF, E., A. MAGORA, AND B. GONEN. Disuse atrophy of the quadriceps muscle. *EMG* 11:479-489, 1971.

N94-28372

The Value of Electrical Stimulation as an Exercise Training Modality

Dean P. Currier, Ph.D., J. Michael Ray, M.D., John Nyland, M.S.Ed.,
and Tim Noteboom
Division of Physical Therapy
University of Kentucky Medical Center
Lexington, Kentucky

511-52
207620

Introduction

Voluntary exercise is the traditional way of improving performance of the human body in both the healthy and unhealthy states. Physiological responses to voluntary exercise are well documented. It benefits the functions of bone, joints, connective tissue, and muscle.

In recent years, research has shown that neuromuscular electrical stimulation (NMES) simulates voluntary exercise in many ways. Generically, NMES can perform three major functions: (1) suppression of pain, (2) improve healing of soft tissues, and (3) produce muscle contractions. Low frequency NMES may gate or disrupt the sensory input to the central nervous system which results in masking or control of pain. At the same time NMES may contribute to the activation of endorphins, serotonin, vasoactive intestinal polypeptides, and ACTH which control pain and may even cause improved athletic performances [1]. Soft tissue conditions such as wounds and inflammations have responded very favorably to NMES. NMES of various amplitudes can induce muscle contractions ranging from weak to intense levels.

NMES seems to have made its greatest gains in rehabilitation where directed muscle contractions may improve joint ranges of motion [2]; correct joint contractures that result from shortening muscles [3]; control abnormal movements through facilitating recruitment or excitation into the alpha motoneuron in orthopedically, neurologically, or healthy subjects with intense sensory, kinesthetic, and proprioceptive information [4]; provide a conservative approach to management of spasticity in neurological patients [5]; by stimulation of the antagonist muscle to a spastic muscle [6], stimulation of the agonist muscle [7], and sensory habituation [8]; serve as an orthotic substitute to conventional bracing used with stroke patients in lieu of dorsiflexor muscles in preventing steppage gait and for shoulder muscles to maintain glenohumeral alignment to prevent subluxation [5]; and of course NMES is used in maintaining or improving the performance or torque producing capability of muscle [9-11]. NMES in exercise training is our major concern in this presentation.

Muscle Strengthening by Neuromuscular Electrical Stimulation

Maintaining or improving the performance or torque producing capability of muscle is our major concern. Although resistance voluntary exercise has been the traditional method of improving muscle performance (strengthen or increase torque), NMES has also been demonstrated to strengthen muscle [9-11]. The object in strengthening muscle with NMES is to induce very intense contractions with little or no discomfort. Pulse charge and frequency are stimulus characteristics that need to be considered to meet the objective of strong contractions with little or no discomfort. Pulse charge is comprised of pulse duration and amplitude. The optimal pulse duration that feels comfortable ranges from 0.02 to 0.2 msec, while safe amplitudes range from 0 to 100 mA. Because electrical pulses are very short lasting, they are comfortable and their shape does not seem to be a critical factor if symmetrical biphasic (rectangular or sine). Intense muscle contractions that produce the greatest torque are induced at pulse frequencies between 50 and 90

per second. Subject variation exists and therefore no definite single type of stimulator, other than line powered, or set of stimulus characteristics are associated with the least discomfort. Level of contraction for muscle strengthening in any program has been reported to be 50 to 80 percent of maximum voluntary contraction (MVC) effort. [9-11]. Strength gains are directly related to the training amplitude of NMES. Strength training programs using NMES have usually employed ten contractions per session ranging from 10 to 40 sessions.

The difference between success and failure using NMES for maintaining or augmenting muscle mass and muscle torque during periods of joint immobilization or minimum exercise may be related to the selection of specific stimulus characteristics. Experience at the University of Kentucky confirms that. We hypothesize that a pulse or burst frequency and charge sufficient to induce fused tetanic contractions equivalent to 50 percent of isometric MVC delivered for ten contractions per session, three sessions per week for 6 weeks will improve muscle performance. Our hypothesis should also be appropriate for work with astronauts and problems that they encounter in zero-g conditions.

Our recent pilot study that assessed the ability of electrically induced contractions for preventing muscle wasting and weakness during the first 6 weeks after reconstructive surgery for anterior cruciate ligaments should serve as support for using electrically induced muscle contractions.

Method

Patient Data

Sixteen patients were treated sequentially after surgery. Patients numbered 1 through 3 served as controls (n=3) and did not receive electrically induced contractions because of inaccessibility of treatments as outpatients at out-reach facilities. Patients numbered 4 through 10 received conventional NMES only (n=7), and patients 11 through 16 received the simultaneous combination of NMES and magnetic stimulation (n=6).

All patients were operated on with the same arthroscopic assisted reconstruction technique of the anterior cruciate ligament using the middle third of the patellar tendon. An orthotic device maintained the postoperative limb in full extension to 5 degrees of flexion. All patients in this study received physical therapy which progressed with each succeeding week.

Measurements

Torque measurements on patients 4 through 16 were made preoperatively to determine amplitude of NMES for producing torque equivalent to 50 percent of isometric MVC. Torque was determined by a Biodex dynamometer system. Starting with the ninth patient the protocol was altered to accommodate comparisons between pre- and postoperative torque scores at 6 weeks, and use of magnetic stimulation (a new mode of clinical therapy).

Thigh girths were measured on all patients in this pilot study at 12.24, 20.32, and 25.40 cm (6, 8, and 10 in) proximal to the superior patellar pole using a plastic tape measure.

Patients receiving the combined NMES and magnetic stimulation were asked to rate each mode of induced stimulation with 10 cm visual analog scale (VAS).

Treatment

Patients 4 through 10 were treated with NMES only in the conventional manner. Patients 11 through 16 were treated by the combined NMES and magnetic stimulation (MES). A modified Cadwell MES-10 magnetic stimulator (used for peripheral and cerebral nerve conduction studies) with a 26 cm copper coil (designed for the thigh) was used in the study, and delivered a peak amplitude of 1.5 tesla (15000 gauss) along the coil edges with a single cosine pulse form at 60 pulses per second. From previous experience this magnetic field is sufficient to produce torque ranging from 8 to 82 percent of MVC of healthy subjects' quadriceps femoris muscle [12]. However, because of the subject variation in torque response to the peak amplitude of the magnetic field, we decided to use magnetic field augmentation by combined magnetoelectric technique reported by Bickford and co-workers [13]. This technique applies the conventional NMES simultaneously with the magnetic stimulation to elicit contractions sufficient to produce 50 or more percent of MVC. The magnetic field was superimposed and applied at peak amplitude simultaneously with NMES for 10 seconds per contraction. A 10-second muscle contraction time was used with the magnetoelectric stimulation rather than the previous 15 seconds with conventional NMES only because of the coil heating (120 to 150 °F, 48.9 to 65.6 °C).

Results

The two NMES patients measured for torque at pre- and postoperative increased their knee torque by a mean 24.7 percent during 6 weeks of treatment, while those receiving MES showed a mean 15.1 percent decrease. The mean torque loss among patients not in the study measured in our clinic is 50 percent of preoperative MVC.

The mean percent of decreased girth 6 weeks after knee surgery for controls was 8.3 percent, 0.5 percent for patients receiving NMES only, and 0.0 percent for patients having MES.

The induced stimulation duration per treatment session varied from 0.04 hours/day (150 seconds) for NMES only patients, to 0.03 hours/day (100 seconds) for MES patients. All patients receiving induced stimulation were treated under the supervision of a physical therapist.

Patients receiving MES perceived differences between the NMES given during the hospital stay (three sessions) and MES given during outpatient visits (15 sessions). The mean rating on the VAS for NMES patients was 7.1 cm and that for MES patients was 3.7 cm.

Discussion

Patients from the NMES only treatment increased muscle torque during the 6 weeks postoperative. Until the success of a few patients receiving NMES was demonstrated, postoperative torque measurements at 6 weeks were believed to result in deleterious forces to the graft [14]. No torque for the controls is available because of postoperative restrictions at the time of their rehabilitation. Patients receiving MES lost torque as a group, although three of six patients contributed to the loss. The torque loss in MES patients may have been attributed to the 10-second contraction duration or 33 percent less induced contraction time than NMES patients, and the full knee

extension position used in treatment of MES patients. The torque was tested with 10 degrees of knee flexion, thus contributing to loss of torque-angle specificity.

Findings on girth measurements among our patients were within expected limits based on previous experiences. Patients receiving NMES averaged far less muscle wasting than that reported by others [15-17], while our MES patients displayed no muscle wasting 6 weeks after having major knee surgery. The induced stimulation times of our patients were considerably less than reported by others, yet our results merit attention when compared with other studies ranging from 1.5 [16] to 17.7 [15] hours per day. The difference in results obtained in our pilot study and that of others may be attributed to our aggressive stimulation regimen consisting of line powered units capable of delivering ten-fused tetanic contractions that were equivalent to our greater than 50 percent of MVC.

Another important finding in our study was the patient tolerance for intense induced muscle contractions when using the magnetoelectric method of treatment. The patients' ratings on the VAS showed that the MES method is more acceptable to their comfort than that of the conventional NMES.

Conclusion

The data of this pilot study support the conclusion that both conventional NMES and the augmented method of combining NMES with magnetic stimulation are capable of preventing muscle wasting and reducing muscle weakness during the first 6 weeks after major knee ligament surgery. The MES method is tolerated better by patients than the conventional NMES. Data are encouraging to date for the MES method but our goal is the development and improved clinical application of magnetic stimulation to replace the conventional NMES.

References

[1] KAADA, B. Improvement of physical performance by transcutaneous nerve stimulation in athletes. *Acupuncture Electrotherap. Res. Int. J.* 9:165-180, 1984.

[2] BAKER, L. L., C. YEH, D. WILSON, ET AL. Electrical stimulation of wrist and fingers for hemiplegic patients. *Phys. Ther.* 59:1495, 1977.

[3] PECKHAM, P. H., J. T. MORTIMER, AND E. B. MARSOLAIS. Alteration in the force and fatigue ability of skeletal muscle in quadriplegic humans following exercise induced by chronic electrical stimulation. *Clin. Orthop.* 114:326, 1976.

[4] FUHRER, M. J. Dishabituation of flexor withdrawal activity mediated by functionally transected human spinal cord. *Brain Res.* 63:93, 1973.

[5] BAKER, L. L. Clinical uses of neuromuscular electrical stimulation. In: *Clinical Electrotherapy,* edited by R. M. Nelson and D. P. Currier. Norwalk, CT: Appelton & Lange, 1987, p. 121-126.

[6] LEVINE, M. G., M. KNOTT, AND H. KABAT. Relaxation of spasticity by electrical stimulation of antagonist muscles. *Arch. Phys. Med.* 33:668, 1952.

[7] BOWMAN, B. AND T. BAJD. Influence of electrical stimulation on skeletal muscle spasticity. In: *Proc Internat Sympos External Control Human Extrem,* Belgrade, Yugaslav

Comm Electronic Automation, 1981, p. 561-576.

[8] DIMITRIJEVIC, M. R. AND P. W. NATHAN. Studies of spasticity in man. 4. Changes in flexion reflex with repetitive cutaneous stimulation in spinal man. *Brain* 93:743, 1970.

[9] CURRIER, D. P. AND R. MANN. Muscle strength development by electrical stimulation in healthy individuals. *Phys. Ther.* 63:915-921, 1983.

[10] WALMSLEY, R. P., G. LETTS, AND J. VOOYS. A comparison of torque generated by knee extension with a maximal voluntary muscle contraction vis-a-vis electrical stimulation. *J. Orthop. Sports Phys. Ther.* 6:10-17, 1984.

[11] SELKOWITZ, D. M. Impovement in isometric strength of the quadriceps femoris muscle after training with electrical stimulation. *Phys. Ther.*

[12] CURRIER, D. P., R. M. KELLOGG, AND A. NITZ. Pulsed magnetic stimulation: effects upon quadriceps torque and blood flow. *Phys. Ther.* 69:395, 1989. (Abstract)

[13] BICKFORD, R. G., M. GUIDI, P. FORTESQUE, ET AL. Magnetic stimulation of human peripheral nerve and brain: response enhancement by combined magnetoelectric technique. *Neurosurg.* 20:110-116, 1987.

[14] KAIN, C. C, J. A. MCCARTHY, S. ARMS, ET AL. An in vivo analysis of the effect of transcutaneous electrical stimulation on the quadriceps and hamstrings on anterior cruciate ligament deformation. *Am. J. Sport Med.* 16:147-152, 1988.

[15] GOULD, N., D. DONNERMEYER, G. G. GAMMON, ET AL. Transcutaneous muscle stimulation to retard disuse atrophy after open meniscectomy. *Clin. Orthop.* 178:190-197, 1983.

[16] ARVIDSSON, I., H. ARVIDSSON, E. ERIKSSON, ET AL. Prevention of quadriceps wasting after immobilization: an evaluation of the effect of electrical stimulation. *Orthopedics* 9:1519-1528, 1986.

[17] WIGERSTAD-LOSSING, I., G. GRIMBY, T. JONSSON, ET AL. Effects of electrical muscle stimulation combined with voluntary contractions after knee ligament surgery. *Med. Sci. Sports Exerc.* 20:93-98, 1988.

OMIT

Section 5

Biomechanics of Microgravity

PRECEDING PAGE BLANK NOT FILMED

N94-28373

The Use Of Biomechanics in the Study of Movement in Microgravity

R. J. Gregor, Ph.D., J. P. Broker, B.S., and M. M. Ryan, B.S.
Human Biomechanics Laboratory, Department of Kinesiology
University of California, Los Angeles
Los Angeles, California

S12-52
207621
P-7

Introduction

As biomechanists interested in the adaptability of the human body to microgravity conditions, it appears that our job is not only to make sure that the astronauts can function adequately in space but also that they can function upon their return to Earth. This is especially significant since many of the projects now being designed at NASA concern themselves with humans performing for up to 3 years in microgravity. While the Extended Duration Orbiter flights may last 30 to 60 days, future flights to Mars using current propulsion technology may last from 2 to 3 years. It is for this range of time that the adaptation process must be studied.

Specifically, biomechanists interested in space travel realize that human performance capabilities will change as a result of exposure to microgravity. The role of the biomechanist then is to first understand the nature of the changes realized by the body. These changes include adaptation by the musculoskeletal system, the nervous system, cardiorespiratory system, and the cardiovascular system. As biomechanists, it is also our role to take part in the development of countermeasure programs that involve some form of regular exercise. Exercise countermeasure programs should include a variety of modalities with full knowledge of the loads imposed on the body by these modalities. Any exercise programs that are to be conducted by the astronauts during space travel must consider the fact that the musculoskeletal and neuromuscular systems degrade as a function of flight duration. Additionally, we must understand that the central nervous system modifies its output in the control of the human body during space flight and most importantly, we must prepare the astronauts for their return to one g.

The science of biomechanics is interrelated with many other scientific disciplines that must integrate their objectives to develop exercise programs to counter the effects of microgravity (Figure 1).

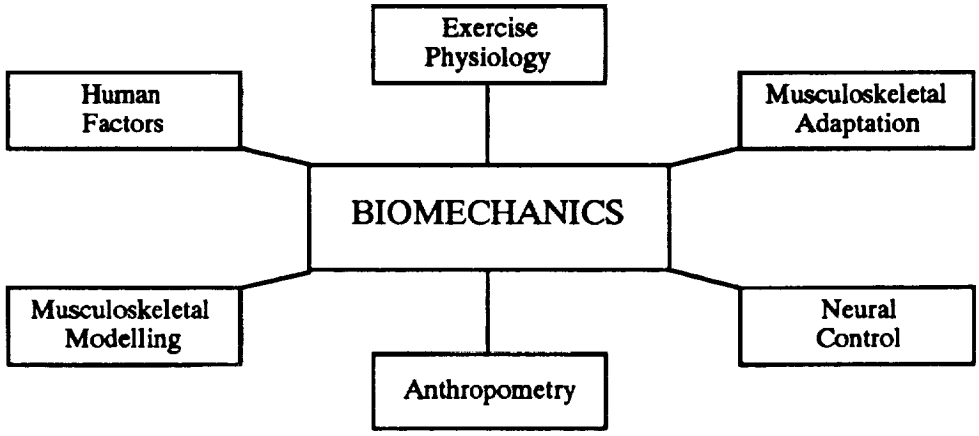


Figure 1. *Interaction between biomechanics and other scientific disciplines in the study of movement in microgravity.*

PRECEDING PAGE BLANK NOT FILMED
PAGE 194 INTENTIONALLY BLANK

anthropometry, human factors engineering, and musculoskeletal modeling. Interrelationships with musculoskeletal adaptation include the understanding that the musculoskeletal system degrades as a function of space-flight duration. The adaptation of bone to space flight, as documented by Zernicke *et al.* [9], includes skeletal degeneration and the observation of a negative calcium balance. Weight-bearing bones appear to be more sensitive to the effects of microgravity with mechanical strength and stiffness reported to be markedly reduced as a result of exposure to microgravity. In addition, there are certain geometric alterations in bone that are related to their mechanical properties and there is an ongoing debate regarding in-flight vs. ground-based models in the study of bone loss.

Regarding the adaptation of skeletal muscle to microgravity, we must first review the various factors that affect the muscle's ability to produce force. These factors include: (l) velocity of contraction, (2) the type of contraction, (3) muscle length, (4) muscle architecture, (5) muscle fiber type, and (6) the activation history of the muscle. As a result of several research projects, it appears that antigravity extensors are most affected by exposure to microgravity [3]. Force production in lower extremity extensors such as the vasti muscles, tricep surae muscles, and gluteus maximus are markedly lower. It appears that lower extremity flexors, such as the hamstrings and tibialis anterior, and upper extremity flexors and extensors are less affected. Ground-based hindlimb suspension models and evidence from research on animals during space flight indicate that slow muscle atrophies more than fast muscle, and that slow motor units may increase their speed of shortening [3]. These latter two observations are consistent with general observations made using disuse models in which slow fibers appear to be more affected than fast, and that slow fibers increase their contraction velocity. In summary, the musculoskeletal system appears to be markedly affected as a result of exposure to microgravity.

With the interface of biomechanics and neural control, our attention focuses on sensorimotor integration. In review, we know there are certain muscle fiber types that are recruited in certain ways as a function of the Size Principle [6] and that any electrical signal seen from the muscle (i.e., the electromyogram) typically precedes the initiation of force production in that same muscle [7]. Consequently, there is an orderly process to the recruitment of certain muscle types and there is a delay between the excitation of that muscle and the actual mechanical output observed as muscle force. These output parameters appear tightly coupled to sensory input and the various feedback systems employed for motor control. Regarding motor control, the losses observed to date have been in motor coordination, sensorimotor integration, and losses in the vestibulo-ocular system. These result in disorientation in the astronauts, loss of balance and stability, and impairment of sensory motor integration that may subsequently affect their ability to control the vehicle (i.e., Space Shuttle). Theories on central motor programs apply to this particular area, and use of biofeedback in understanding the impairment experienced by the astronauts and, subsequently, in training them to overcome these impairments, involves the coordination and integration of biomechanical principles and neural control.

The integration of biomechanics and anthropometry focuses in the field of human factors engineering. If, indeed, skeletal muscle and bone mass losses do approach 10 to 20 percent, one must consider the effect of these tissue losses on actual body segment parameters (i.e., mass and moments of inertia of the body segments involved). Any changes in these body segment parameters have an ultimate consequence on musculoskeletal modeling in which the mass and mass moment parameters are required for input to equations of motion. There has been a great deal of work done on work-space design in the Space Shuttle environment as well as in space suit design (i.e., reach envelops) the mechanical interface of the human with the vehicle, and tools designed to enhance work output. Biomechanics play a significant role here since one must first understand

the requirements imposed on the astronauts and the need to develop specific technology so that the astronauts can carry out the various jobs required of them in microgravity.

The final link with biomechanics involves the field of musculoskeletal modeling. Musculoskeletal modeling includes: (1) principles in inverse dynamics, (2) the use of optimization algorithms, and (3) the use of forward solutions methods in which force may be directly predicted from electromyography (EMG). These various types of modeling procedures are used in movement analysis in one g and can, of course, be applied in movement in microgravity if proper mechanical conditions are taken into account (i.e., loss of gravitational acceleration). The ultimate goal of individuals in biomechanics as they interface with scientist's musculoskeletal modeling, of course, is to develop predictive models of human kinetics and dynamics in microgravity [2].

It seems, then, that the role of biomechanics in the study of human performance in microgravity involves interface with a variety of other scientific disciplines. It is the integration of the techniques used in these various disciplines that must be coordinated in the study of human movement in microgravity. We must first realize the effects of microgravity on the musculoskeletal, neuromuscular, cardiovascular, and cardiorespiratory systems. Our objective is to then develop countermeasure techniques that will enable the astronauts to perform in microgravity and return to the one-g environment. The use of predictive models to understand human performance in microgravity requires knowledge of the degrading system losses (i.e., the effects of the microgravity on bone and muscle) as well as knowledge of the loads imposed on the human body exercising in microgravity. While biomechanics will play a role in, first, monitoring these forces and describing the loads imposed during various tasks in microgravity, any countermeasure program using principles in biomechanics must include information on the magnitude of the losses in the musculoskeletal system. Any exercise program designed to counter the effects of microgravity must: (1) employ principles in biomechanics to study the magnitude of loss in the musculoskeletal system, (2) understand the nature of the loads imposed on the body, and (3) recognize the need to monitor the system losses and develop the potential for one learning to accommodate and adapt to the effects of microgravity.

Certainly, locomotion (i.e., walking) imposes the highest loads on the lower extremity extensors that are the muscles most affected by microgravity. It is conceded that the musculoskeletal system will degrade. Results from the most recent experiments indicate this loss will proceed despite the use of exercise programs. Consequently, the development of countermeasures to the effects of microgravity must include knowledge of these losses and must be flexible enough to change exercise regimens in light of these losses for the health and safety of the astronauts.

The procedures that I am going to describe in the remainder of this manuscript are procedures, in principle, that can be applied to any exercise regimen used in microgravity. While I will use cycling as an example, principles employed here could easily be used for activities such as running, walking, rowing, or jumping. The first principle concerns the identification of loads imposed on the human body by the environment. Identification of the orientation and magnitude of ground reaction forces or any external reaction force in the environment is requisite to our understanding of exercise as a potential countermeasure to microgravity. In walking, we would want to understand the orientation and magnitude of the ground reaction force as one proceeds through the stance phase of locomotion. During cycling, we would want to understand the orientation and magnitude of the pedal reaction force on both the right and left lower extremities. It is clear in both cases that the orientation and magnitude of this force changes as one proceeds through the stance phase of locomotion as well as through the power phase of cycling (from top dead center to bottom dead center in the pedaling cycle). Cycling offers a more constrained environment in which the trajectory of the lower extremities is more repeatable than during

locomotion and can be controlled with respect to the magnitude of the external forces imposed. The cycling task need not include a bicycle but may include any seated position in which the astronaut will sit and pedal any circular coupling system similar to a bicycle chain drive system. This cyclic alternation of the right and left lower extremity can be controlled with respect to range of motion, speed and load imposed. One can envision a series of isometric contractions at various crank angles or a continuous motion in which the velocity of the crank is held constant and the load varied during the cycling pattern. Certainly, in this controlled environment, the forces imposed on the lower extremity are, in general, considerably lower than those imposed during locomotion, but at some point in an exercise regimen involving cycling, walking, and rowing, these lower forces may be desirable.

When using any exercise regimen in microgravity, one must know how the human system performs at one g. Information related to the cycling task shows that the pedal reaction force varies from a minimum value at top dead center to a maximum at approximately 100 degrees of the pedaling cycle [4]. The magnitude of this pedal reaction force then declines as one proceeds down to bottom dead center. The magnitude of the joint reaction forces, while much lower than those during walking, do change as a function of the pedal reaction force. Additionally, the muscle moment patterns indicate there is a large hip extensor moment during the power phase that approaches zero as the cyclist proceeds through recovery (i.e.,
cycle). The knee moment appears to be extensor during the early part of the power phase and then changes to a flexor moment in the second quadrant ranging from 90 to 180 degrees of the pedaling cycle. This flexor moment continues, although of lower magnitude, through early recovery. It then switches to an extensor moment as the cyclist proceeds through the fourth quadrant up to top, dead center. The ankle shows a continuous plantar flexor moment that is almost a mirror image of the resultant pedal reaction force.

It appears that the tricep surae muscles are predominantly responsible for this particular muscle moment at the ankle. Using optimization algorithms designed to calculate the magnitude and direction of the muscle moments at hip, knee, and ankle in microgravity, we can see that the muscle moment actually increases in magnitude and is similar in direction as one proceeds from one g to microgravity (Figure 2).

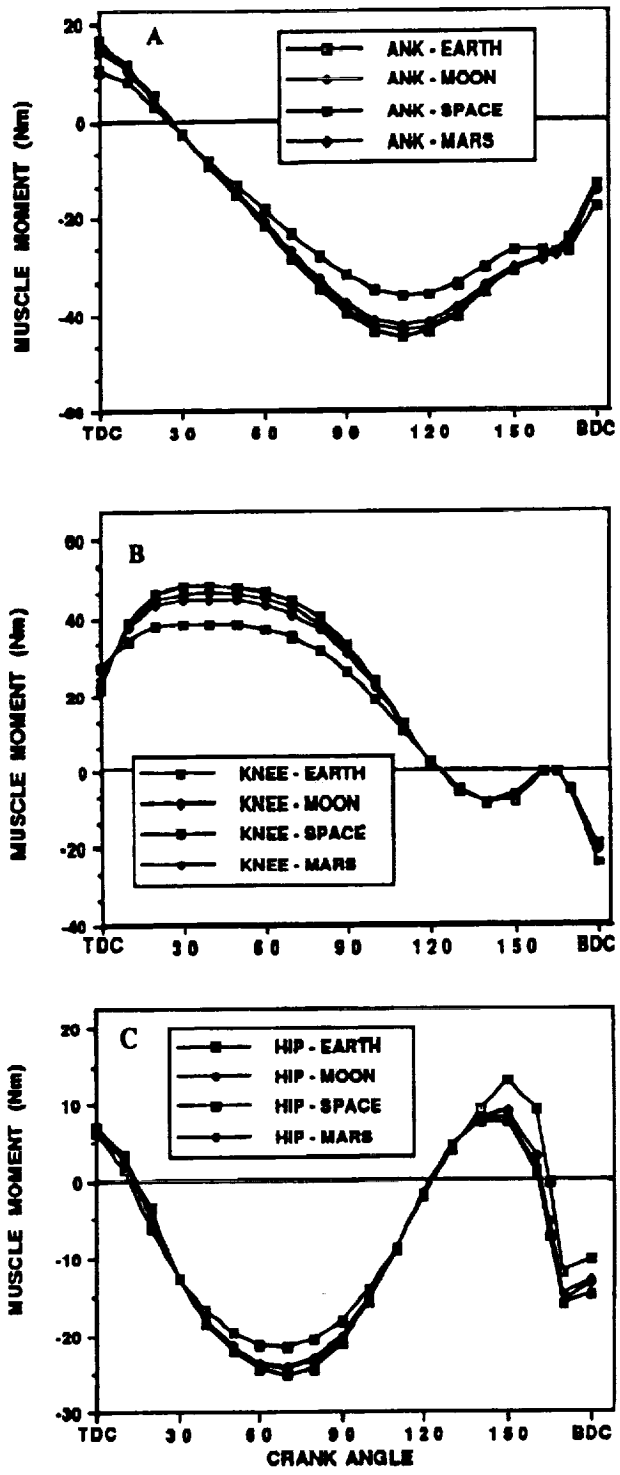


Figure 2. *Muscle moments during the power phase of cycling (0 to 180 degrees) at the ankle (A), knee (B) and hip (C) simulated using the gravitational fields of the Earth (one g), Moon (0.16 g), Mars (0.38 g) and in space (zero g).*

when they move from ground-based models to microgravity above the surface of the Earth. Since the muscle moment increases as one moves from one g to microgravity, further information is needed as to the exact role played by the muscles during the cycling task in an exercise regimen in microgravity.

The motor program of lower extremity musculature indicates that the single joint extensors are relatively immutable during the cycling task [8]. That is, from subject to subject, the single joint, hip extensors, knee extensors, and ankle extensors show a relatively constant activation pattern during the pedaling cycle. It is currently felt that the single joint extensors provide the power to the lower extremity and, subsequently, to the bicycle. This is quite similar to the loads imposed on the lower extremity extensors during the walking cycle in which the ground reaction force during stance imposes heavy loads on single joint hip, knee and ankle extensors. Cycling, to some degree, is similar. Since the single joint, lower extremity extensors are most affected by microgravity, one might ask the question as to which exercise is most useful in attenuating this loss of musculature and force production capability. While walking may stress the lower extremity extensors more than cycling, the loads imposed on the lower extremity also affect other tissues (i.e., bone). If these tissues respond differently to the various loads, then I feel we must be ready to show some flexibility in a countermeasure program so that loads imposed on the lower extremity can be sustained by the degrading muscle and bone. One might further ask the question, if the lower extremity extensors are immutable during cycling in one g, does this hold true in microgravity? Certainly, the flexors are less affected by microgravity, and the rider may shift to the flexors and pull up on the crank since gravity assisted propulsion no longer exists.

Exercise regimens, such as walking and cycling, can also be used to study musculoskeletal mechanics. The use of cycling in The Human Biomechanics Laboratory at UCLA has been extensive in the study of the human musculoskeletal system. We have current information that shows the actual force production of the tricep surae complex during the cycling task in response to three separate power outputs [5]. The information gained on parameters such as muscle force, muscle EMG patterns, muscle length changes, velocity of muscle action, and joint moments as well as joint moment arms, is useful input to predictive models of the musculoskeletal system. In our laboratory, the cycling task has been used to study lower extremity function and can be used to study musculoskeletal function in microgravity. With knowledge of losses of muscle and bone, one can, from the information that we have recorded, develop predictive models for performance in microgravity. For example, using the cycling task, we can develop an optimization algorithm to predict function in the lower extremity. This algorithm can be modified by changing inputs as a result of changes in tissue from exposure to microgravity. The changes in input parameters to this model, with knowledge of requirements of imposed external loads, can prove useful in our understanding of human performance in microgravity. The development of such a model may, of course, be extrapolated to other tasks such as walking and rowing in which external loads are imposed on the body and tissue changes are similar.

One final use of biomechanics concerns its interface with neural control and motor learning. We have developed some techniques in our laboratory in which selected aspects of the cycling task can be used as feedback to the subject to enhance their performance [1]. This feedback is critical for updating the individual regarding the status of their motor output. Any task, whether it be rowing, walking, jumping, cycling, or possibly just a single joint movement, can employ feedback techniques to update the astronauts on their motor function capabilities. This type of information could be very useful in monitoring the capabilities of the astronauts as a result of extended duration orbits. We have studied the effects of feedback during single as well as continuous motor tasks, and more information is being collected regarding the way in which the human body processes

information during a continuous task. The nature of this feedback can enhance performance and be used as a monitoring device for astronaut function in microgravity. One could envision that knowledge of the forces required for an emergency egress, once the Shuttle lands on Earth and returns the astronauts to a one-g environment, could serve as an acceptable baseline for continued function in microgravity. These loads would be the lowest acceptable limits tolerated by the degrading musculoskeletal system that would determine whether the flight continued. If we could continuously update the astronauts through some form of exercise regimen and use some motor tasks to provide information about their status, then we can best serve the safety and the continued progress of the crew.

In summary, biomechanics can be employed in the study of human performance in microgravity. Principles in biomechanics include integration with a variety of other fields to first identify the magnitude of change in the human body as a result of prolonged exposure to microgravity as well as in the design of flexible and adaptable exercise regimens that could be used as countermeasures to the effects of microgravity. Principles in biomechanics as they relate to motor control can give astronauts necessary feedback regarding the status of their motor function and can serve as a diagnostic tool to indicate to the astronaut when they are reaching the lowest acceptable limits for emergency egress upon return to one g. While cycling was the example used in this particular discussion, certainly the principles employed in understanding this task and the possible use of this task as an exercise countermeasure, can be applied to any other type of exercise regimen whether it be locomotion, jumping, rowing, or single joint, flexion/extension actions.

References

[1] BROKER, J. P. AND R. J. GREGOR. extrinsic feedback and the learning of cycling kinetic patterns. press)

[2] DONAHUE, T. M. (ed.). *A Strategy for Space Biology and Medical Science.* Washington, DC: National Academy Press, 1987.

[3] EDGERTON, V. R.AND R. R. ROY. *Adaptation of skeletal muscle to spaceflight.* (In review)

[4] GREGOR, R. J., P. R. CAVANAGH, AND M. LA FORTUNE. Knee flexor moments during propulsion in cycling: a creative solution to Lombard's Paradox. *J. Biomech.* 18:307-316, 1985.

[5] GREGOR, R. J., P. V. KOMI, AND R. C. BROWNING. A comparison of the triceps surae and residual muscle moments at the ankle during cycling. *J. Biomech.* (In review)

[6] HENNEMAN, E., G. SEMJEN, AND D. O. CARPENTER. Functional significance of cell size in spinal motoneurons. *J. Neurophysiol.* 28:560-580, 1965.

[7] NORMAN, R. W. AND P. V. KOMI. Electromechanical delay in skeletal muscle under normal movement conditions. *Acta Physiol. Scand.* 106:241-248, 1979.

[8] RYAN, M. M., R. J. GREGOR, AND J. A. HODGSON. Neural patterning of lower extremity muscles during cycling at a constant load. *J. Biomech.* 21:854, 1988.

[9] ZERNICKE, R. F., A. C. VAILAS, AND G. J. SALEM. Biomechanical response of bone to weightlessness. In: *Exercise and Sports Science Reviews,* edited by K. B. Pandolf. Baltimore, MD: Williams & Wilkins, 1990, p. 167-192.

OMIT

Section 6

Psychophysiology of Exercise

PRECEDING PAGE BLANK NOT FILMED

PAGE 90 INTENTIONALLY BLANK

N94-28374

Psychophysiology in Microgravity and the Role of Exercise

J. M. Shaw, M.A. and A. C. Hackney, Ph.D.
Exercise Physiology Laboratory
Department of Physical Education, Exercise and Sports Science
College of Arts and Sciences
University of North Carolina
Chapel Hill, North Carolina

S13-52
207622
P-9

Introduction

The Space Transportation-Shuttle (STS) Program has greatly expanded our capabilities in space by allowing for missions to be flown more frequently, less expensively, and to encompass a greater range of goals than ever before. However, the scope of the United State's role and involvement in space is currently at the edge of a new and exciting era. The National Aeronautics and Space Administration (NASA) has plans for placing an orbiting space station (Space Station Freedom) into operation before the year 2000 [18]. Space Station Freedom promises to redefine the extent of our involvement in space even further than the STS program.

Space Station crewmembers will be expected to spend extended periods of time (~30 to 180 days) in space exposed to an extremely diverse and adverse environment (e.g., the major adversity being the chronic microgravity condition) [18]. Consequently, the detrimental effects of exposure to the microgravity environment is of primary importance to the biomedical community responsible for the health and well-being of the crewmembers.

Space flight and microgravity exposure presents a unique set of stressors for the crewmember; weightlessness, danger, isolation/confinement, irregular work-rest cycles, separation from family/friends, and mission/ground crew interrelationships [7]. A great deal is beginning to be known about the physiological changes associated with microgravity exposure, however, limited objective psychological findings exist. Examination of this latter area will become of critical concern as NASA prepares to place crewmembers on the longer space missions that will be required on Space Station Freedom [5]. Psychological factors, such as interpersonal relations will become increasingly important issues, especially as crews become more heterogeneous in the way of experience, professional background, and assigned duties.

In an attempt to minimize the detrimental physiological effects of prolonged space flight and microgravity exposure, the United States and Russian space agencies have taken steps to implement various countermeasure programs. One of the principle countermeasures used by both nations is exercise during space flight. The purpose of this paper is to present a brief overview of the major research findings examining the psychophysiological changes associated with microgravity exposure, and to address the potential role of exercise as a countermeasure in affecting these psychophysiological changes.

Psychophysiology is concerned with the mind/body interaction. It can be viewed as a general systems approach to human behavior that integrates findings from different disciplines [4, 9, 11, 18]. In discussing the psychophysiology of microgravity exposure, several important factors complicate and hinder attempts to address the nature and scope of this issue. These include:

• Sample size. To date only 250 astronauts/cosmonauts have flown in space.

202
203
PAGE 204 INTENTIONALLY BLANK

- Extensive use of countermeasures. Prophylactic and therapeutic use of countermeasures has undoubtedly masked some of the direct effects attributable to microgravity.

- Limited research focus. Until recently, biomedical research has focused on physiological rather than psychology.

- Limited accessibility to data. Most of the space missions involving prolonged exposure to microgravity have been conducted by Russia. Therefore, findings have been available to United States scientists on a limited basis.

- Limited capabilities for scientific observation. Biomedical observations have been restricted by the operational constraints imposed on most space missions, and also by the time spent in space.

After reviewing the literature it becomes apparent that Russia is the leader in the area of psychological assessment and evaluation of crews during space flight. This is for two basic reasons: (1) it has been a major point of the Russian's to include a professional "Psychological Support Group" as an essential component of their mission control unit, and (2) the Russian's have allowed behavioral scientists to take an active, integrated role in the research focus of their missions [5, 20]. Furthermore, Russia has logged a much greater number of sustained man-hours in space than the United States (see Table 1). This has allowed Russia to study the psycho-physiological effects of prolonged exposure to microgravity and space flight extensively [16]. As noted however, access to this substantial data base that Russia has amassed on human function during prolonged manned space flight has been limited. Hopefully, with the changing political climate, more of the Russian information will become available and be incorporated into the United States scientific community.

Table 1. *U.S. and Russian Space Program Summary*

U.S. Program	Days in Space	Russian Program	Days in Space
Mercury	≤1	Vostok	≤2
Gemini	4-8	Voskhod	1-2
Apollo	6-13	Soyuz	1-185
Skylab	28-84	Salyut	16-237
Apollo-Soyuz	9		
STS	2-7		

Psychological Findings

There is little objective psychological data on the effects of prolonged microgravity exposure. However, the limited reports available suggest that the psychological consequences of exposure to space flight and the microgravity environment can be classified into affective, behavioral, and cognitive responses. Although presented separately, these responses are closely related and in most cases not independent of each other.

Affective

Anecdotal information from space missions of the United States and Russia indicates that the affective states of crewmembers become dramatically altered in space. Specifically, increased levels of anxiety, boredom, irritability, hostility, and anger have been reported in astronauts and

cosmonauts [18]. These affective states seem to be linked to mission length, as their frequency and intensity increases during longer intervals in space [16, 20].

These affective changes can and have presented potential compromises to the successful outcome of missions. This is illustrated especially by the incidences of elevated levels of hostility and anger that have been frequently reported in prolonged space missions [1, 7, 20]. On several Russian missions, cosmonauts note in their diaries that interpersonal hostility begins to develop about 30 days into the mission and grows continually worse. This has lead to withdrawal from one another and a minimizing of interaction. The increasing hostility, however, has not been experienced amongst crewmembers alone. Hostility has also developed between space crews and ground-control crews. A frequently cited episode occurred on Skylab 4 where mission scientists and ground control disagreed on work schedules. This led to increased tensions between the groups which resulted in a work slow-down in space. Considerable measures were taken in order to bring about an agreeable adjustment in schedules so mission operations could continue (an open, frank "bull session" between the crew and ground control occurred) [2, 7]. Furthermore, on several of the extended Russian missions (>100 days), the cosmonauts have even reported feeling relieved when communications with ground control were interrupted and even desired at times to disrupt communications themselves [2, 7].

Behavioral

Many of the behavioral changes found coincide with the altered affective states noted above. Those commonly reported lethargy and fatigue, decreased motivation, and inappropriate psychosocial interaction [1, 2, 7, 14]. Additionally, psychosomatic symptoms and sleep disturbances have occurred in some crewmembers [18]. These last two changes are of particular interest.

There is the now famous psychosomatic incident involving Russian cosmonaut Valeri Ryumin during the 175-day Soyuz 32 mission. Ryumin was constantly afraid during the first half of the mission that he would get a toothache while in space. During the latter part of the mission, while asleep, he dreamed he had a toothache. When he awoke his tooth actually did hurt [1]. Similar complaints and incidents have been noted on other missions [1, 18, 20].

Historically, sleep disturbances have been a common complaint on most space missions [6, 16]. Throughout the Gemini, Apollo, Skylab, and STS programs, crewmembers have reported difficulty in establishing appropriate sleep/wake cycles. Some of this disruption may have been due to mission operations, comfort limitations, design problems, and physiological accommodations to microgravity. However, Russian reports would also suggest psychological factors play a role. The frequency of cosmonaut's reporting sleep problems is greatest during early and late phases of a mission, when crew anxiety levels are likely heightened due to the demanding events at hand [18, 20]. Currently, there is no evidence that these sleep changes significantly impair performance [18]. However, one-g based sleep deprivation studies have substantially

shown reductions in psychological and physiological performance as chronic fatigue develops [3, 18].

Cognitive

Perception seems to be affected on a transient basis with microgravity exposure. Illusory sensations have been noted upon experiencing weightlessness, such as overturning or inversion of the body and movement of objects in the visual field [2]. The genesis of such sensations is not fully understood. Possible explanations for the origin of these phenomena lie in vestibular system dysfunctions, space sickness, or psychological manifestations. Whether it is any one of these possibilities or several acting in a synergistic fashion remains to be determined.

An interesting event occasionally reported by crewmembers has been labeled *time compression* [2, 18]. The phenomenon involves an altered sense of time and is manifested in a perceived slippage between performance and scheduled time-lines [2]. It probably evolves from excessive mental work-load, information overload, and cognitive processing involving inferences, judgment and decision-making [2, 13, 18].

These cognitive disruptions, while slight in nature, increase the mental effort demanded of an individual performing a task in space. The accumulative effect of this increased demand could have negative consequences during extended space flight.

Physiological Findings

There are numerous physiological changes associated with exposure to microgravity and space flight, which are summarized in Table 2 [21]. Many of these physiological changes warrant significant discussion; however, within the scope of the present paper only those changes of primary interest to psychophysiology will be addressed.

Table 2.

Facial puffiness	Vestibular difficulties
Altered posture	Decreased body mass
Decreased bone density	Decreased blood volume
Decreased red cell mass	Decreased plasma volume
Orthostatic intolerance	Cardiac deconditioning
Decreased leg volume	Variable reflex times
Decreased urinary ADH	Decreased plasma osmolarity
Increased angiotensin I	Increased cortisol
Increased urinary aldosterone	Increased growth hormone
Increased catecholamines	Increased serum enzymes
Decreased submaximal exercise capacity	
Decreased strength of different muscle groups	

Neuroendocrine - Metabolite

Throughout both the United States' and the Russian space programs, biochemical markers of stress have been assessed in crewmembers. Elevations in urinary catecholamines and cortisol as well as plasma ACTH, growth hormone, cortisol, catecholamines, and aldosterone have all been reported [8]. Additionally, elevations in select serum enzymes (creatine phosphokinase, lactic acid dehydrogenase, and gammaglutamyl transpeptidase), also indicative of stress responses, have occurred. The changes in these stress markers has been extremely variable and the data have been compromised by technical difficulties and/or mission constraints. Additionally, it is known that a general population exhibits a great deal of interindividual variation in the biochemical responses to stress and the astronauts-cosmonauts are no exceptions [18].

The most consistent and significant elevations in these neuroendocrine hormones and enzymes have typically occurred before and after flights while in-flight values have been relatively stable [8, 18]. The time course of the changes would seem to correspond to heightened levels of anxiety reported by crewmembers at these critical points in missions. These findings also suggests that some degree of stress accommodation may be occurring during the missions (i.e., in flight).

Cardiovascular

Typically, in one-g experimentation, the monitoring of cardiovascular vital signs provides useful information concerning the psychophysiological status of an individual [3]. It is questionable whether this may be the case in space as many factors induce cardiovascular changes which compromise the interpretation of the data (e.g., gravitational changes). Historically, there have been consistent findings of elevations in resting heart rates, and in some incidences blood pressure, observed before, during, and after missions [6, 15, 16]. Other than anticipatory rises pre- and postflight, however, these changes do not seem to be reflecting vigorous stress responses. Furthermore, in-flight changes, if any, are likely due to the cardiovascular adjustments accompanying exposure to the weightless environment (e.g., cephalic fluid shift) [15, 16].

Circadian Disruptions

The psychophysiological aspects of the circadian cycle are of particular importance to space flight. As noted earlier, sleep disturbances are frequently reported during space flight. This alteration of the sleep/wakefulness cycle can disrupt many physiological systems which are rhythmic in nature (e.g., endocrine) [13, 18]. Evidence indicates circadian disruptions can lead to a *desynchronization* of the body's biological clock and play a role in the development of some of the affective and behavioral problems discussed earlier (e.g., mood shifts, lethargy, or fatigue) [2, 13, 18]. Our understanding of the underlying mechanisms of circadian rhythms is still quite limited, but environmental cues seem to play an important role in the process. In space, environmental cues are extremely limited, therefore the level of desynchronization becomes exacerbated. Mission length may also play a factor in the extent of desynchronization. Several cosmonauts from the extended Soyuz missions (>175 days) report a greater number of sleep disruptions during the latter half of their extended missions [20].

The Role of Exercise

Traditionally, the role of exercise as a countermeasure in space has been entirely physiological in nature, however, an alternative role may exist. That is, the use of exercise as a psychological countermeasure to challenge the negative psychophysiological changes associated with space flight and microgravity exposure.

Physiological Countermeasure

It is well established that during prolonged space flight, significant detrimental changes in certain physiological functions of the human body take place [16, 21]. In particular, biomedical research indicates that muscle atrophy, cardiovascular deconditioning, and bone demineralization have occurred in Russian and American crewmembers [15, 16, 21]. These changes appear to be the physiological consequences of exposing the body to microgravity and a reduction in the typical level of Earth-bound activity. The extent of these detrimental adaptations seems to be a function of space-flight duration.

Both the United States and Russia, in an attempt to minimize these detrimental adaptations to space, have implemented in-flight exercise programs. The extent and nature of the exercise programs have varied tremendously from nation to nation as well as within space programs. To date, the work by biomedical scientists suggests that the use of exercise as a physiological countermeasure seems most promising [16]. However, this research is still in its infancy and many questions remain unresolved. To address this issue, NASA has established the Exercise Countermeasures Project (ECP) working group. The ECP has been given the following charge: ...implementing a preventive health care program for flight crews that will: (1) offset the physiological and operational effects of adaptation to microgravity; (2) ensure effective functional return to Earth; and (3) increase the rate of postflight readaptation.

Over the next few years the ECP working group will attempt to fulfill these objectives, and should therefore provide valuable information to the scientific community as to the effectiveness of exercise as a physiological countermeasure.

Psychological Countermeasure

The use of exercise as a psychological countermeasure has been examined in several one-g studies, but apparently not so in space. The Earth-bound studies suggest that vigorous exercise is associated with positive, beneficial psychological changes to participants. Both affective and behavioral changes have been reported [4, 10, 11, 12, 17].

Specifically, an increased sensation of well-being and positive mood shift occurs after acute bouts of exercise and the effects seem to persist for 2 to 5 hours (10). These subjective states have been objectively quantified with psychophysiological data. For example, Profile of Mood States (POMS) scores show reduced levels of state anxiety and depression following exercise. Concomitantly, lowered heart rates, blood pressure, and catecholamines have been observed [10, 11, 17]. Exposure to chronic exercise (i.e., training programs) has produced similar effects. Participants in such training programs have persistent reductions in state anxiety and depression, as well as increases in self-esteem [10]. Also, it is well established that exercise training produces

accommodations in the neuroendocrine system result in reduced physiological *stress responses* (e.g., catecholamine, heart rate, blood pressure responses) to external challenges [4, 17].

Potentially, these positive psychological effects of exercise have important implications for space flight. Exercise could become an additional *tool* for the behavioral scientist to use in working with crewmembers. Specifically, exercise could be used as one mechanism for inducing positive affective states in crewmembers, and/or play a role in behavioral coping strategies during the prolonged space missions [19]. This potential is based upon the assumption that the positive psychological effects of exercise found in a one-g environment are applicable to microgravity, since no space based research exist. The validity of such an assumption is uncertain, but warrants the attention of the scientific community and presents a unique opportunity for future research.

Recommendations, Future Concerns, and Conclusions

As NASA enters this new and exciting era of space exploration, it is vital that a firm commitment to continued physiological research exist. However, it is equally important that an expanded role be given to the psychological research community. The United States has lagged far behind Russia in this area. The first steps in this direction seem to have been taken with the establishment of the Biobehavioral Research Group at the NASA Johnson Space Center. In light of the information that is becoming available, it seems only logical that NASA's future studies examining man's capacity and ability in space, should be of a more integrated nature and take on a psychophysiological approach.

In the focus of exercise as a countermeasure to microgravity exposure, some key issues that need to be addressed in the future are: (1) does exercise in space induce the same positive psychological changes as found in one-g studies; (2) what type or mode of exercise will be the most effective in producing positive psychophysiological responses in space; and (3) what is the optimal exercise prescription in space? This last issue is especially important as an inappropriate exercise prescription can lead to an undertrained or overtrained states in the crewmember. Undertraining is associated with physical deconditioning (e.g., decreased cardiovascular and muscular function) which could led to an augmentation of the detrimental physiological effects of microgravity exposure. Conversely, overtraining induces some severe psychological and physiological changes (see Table 3) which could tremendously exacerbate the psychophysiological perturbations of microgravity exposure [4].

Table 3. *Psychophysiological Changes Found in Overtrained Individuals*

Apathy	Muscle soreness
Lethargy	Sleep loss
Appetite loss	Mood changes
Weight loss	Increased depressing
Lymphadenopathy	Increased anxiety
Gastrointestinal disturbances	Increased fatigue

Regardless, both conditions (under and overtraining) are inappropriate training stimulus and are representative of extremes which should be avoided.

In conclusion, some of the existing research suggests exercise may be an effective countermeasure for dealing with some of the psychophysiological responses to space flight and microgravity exposure. Many questions, however, remain to be answered, as well as identified, which presents

exciting new avenues of research for biomedical scientists to pursue in the future.

References

[1] CHAIKIN, A. The loneliness of the long-distance astronaut. *Discover* 2:20-31, 1983.

[2] CHRISTENSON, J. M. AND J. M. TALBOT. A review of the psychological aspects of space flight. *Aviat. Space Environ. Med.* 57:203-212, 1986.

[3] GUYTON, A. C. *Textbook of Medical Physiology.* Philadelphia, PA: W. B. Saunders Co., 1981.

[4] HACKNEY, A. C. Overtraining and exercise motivation: a research prospectus. *NASA/ASEE Technical Report, NGT44001800,* 1989.

[5] HARRISON, A. A. On the resistance to the involvement of personality, social, and organizational psychologists in the U.S. space program. *J. Social Behavior and Personality.*

[6] JOHNSTON, R. S. AND L. F. DIETLEIN (eds.). *Biomedical Results from Skylab, NASA SP-377,* 1977.

[7] KANAS, N. Psychological and interpersonal issues in space. *Am. J. Psychiatry* 144:703-709, 1987.

[8] LEACH, C. S., S. I. ALTCHULER, AND N. M. CINTRON-TREVION. The endocrine and metabolic responses to space flight. *Med. Sci. Sports Exerc.* 15:432-440, 1983.

[9] MILLS, F. J. The endocrinology of stress. *Aviat. Space Environ. Med.* 56:642-650, 1985.

[10] MORGAN, W. P. Affective beneficence of vigorous physical activity. *Med. Sci. Sports Exerc.* 17:94-100, 1985.

[11] MORGAN, W. P. Psychogenic factors and exercise metabolism: a review. *Med. Sci. Sports Exerc.* 17:309-316, 1985.

[12] MORGAN, W. P., D. R. BROWN, J. S. RAGLIN, P. J. O'CONNOR, AND K. A. ELLICKSON. Psychological monitoring of overtraining and staleness. *Brit. J. Sports Med.* 21:107-114, 1987.

[13] MOUNTCASTLE, V. B. (ed.). *Medical physiology.* St. Louis, MO: C. V. Mosby, 1974.

[14] NICHOLAS, J. M. Small groups in orbit: group interaction and crew performance on space station. *Aviat. Space Environ. Med.* 58:1009-1013, 1987.

[15] NICOGOSSIAN, A. E. *The Apollo-Soyuz test project: medical report.* Washington, DC: NASA Scientific and Technical Information Office, 1977.

[16] NICOGOSSIAN, A. E., J. F. PARKER, AND V. GARSHNEK. Space vehicles for manned programs. In: *Space Physiology and Medicine, 2nd Edition,* edited by A. E. Nicogossian, C. L. Huntoon, and S. L. Pool. Philadelphia, PA: Lea & Febiger, 1982, p. 77-103.

[17] RANSFORD, C. P. A role for amines in the antidepressant effect of exercise: a review. *Med. Sci. Sports Exerc.* 14:1-10, 1982.

[18] SANTY, P. The journey out and in: psychiatry and space exploration. *Am. J. Psychiatry* 140:519-527, 1983.

[19] SANTY, P. Psychiatric components of a health maintenance facility (HMF) on Space Station. *Aviat. Space Environ.*

Med. 58:1219-1224, 1987.

[20] TIMACHEFF, N. One year in space. *Literaturnaya Gazeta*, January:1-16, 1989.

[21] TIPTON, C. M. Preface to weightlessness and the 1980's. *Med. Sci. Sports Exerc.* 15:408-409, 1983.

Footnote

Ms. J. M. Shaw is a research assistant and Dr. A. C. Hackney is an assistant professor at the Exercise Physiology Laboratory in the Department of Physical Education, Exercise and Sports Science at the University of North Carolina.

OMIT
TO
END

Section 7

Summary of Socratic Debates and Presentations on the Use of Physiological Countermeasures

PRECEDING PAGE BLANK NOT FILMED

SUMMARY

This is a summary of presentations and conclusions from Socratic debates made at the September 26-28, 1989 workshop dealing with exercise as a countermeasure for physiological adaptations to microgravity. This workshop was sponsored by the Exercise Physiology Laboratory at the NASA Johnson Space Center. This review includes the conclusions from the Socratic debates on the issues of the use of electric stimulation, the relationship between fitness and orthostatic tolerance, the role of eccentric exercise in testing and training, and the reversibility of microgravity induce osteoporosis. The impact of biomechanics on the development of exercise countermeasures, the role of psychophysiology and exercise in microgravity, the use of exercise detraining as a model of microgravity, practical considerations for exercise and osteoporosis, and the practicality of exercise impedance cardiography also were presented for discussion.

Eccentrics in Exercise

The use of eccentric exercise for the prevention of degradation of muscle performance, was discussed by Drs. Clarkson and Ariel. Dr. Clarkson presented arguments in support of the hypothesis and Dr. Ariel presented the opposing viewpoint.

PRO: Priscilla Clarkson, Ph.D.

Dr. Clarkson presented a brief review of the literature on testing and training with eccentric contractions. Eccentric contractions were defined from physiological and performance based assessments.

- Training that involved eccentric contractions (wholly or in part of the training regimen) showed significantly larger improvements in strength than those programs that did not included this type of contraction.
- Dr. Clarkson reviewed some theories for the increase in muscle strength that accompanies exercise training.

CON: Gideon Ariel, Ph.D.

Dr. Ariel presented the argument that there are no net differences between eccentric and concentric exercise training programs.

- Dr. Ariel suggested that as the neuromuscular process of a contraction during eccentric and concentric actions are identical, the muscular performance outcomes also are identical.
- Based on the similarity of the changes in performance for both concentric and eccentric actions and the greater risk of muscle injury during eccentric contractions, Dr. Ariel suggested that training only be prescribed using concentric actions.

Electric Stimulation in Exercise Training

The use of electrical stimulation for the prevention of degradation of muscle performance was discussed by Drs. Currier and Kroll. Dr. Currier presented arguments in support of the hypothesis and Dr. Kroll presented the opposing viewpoint.

PRECEDING PAGE BLANK NOT FILMED

PAGE 216 INTENTIONALLY BLANK

PRO: Dean Currier, Ph.D.

Dr. Currier presented results from a research study comparing the effectiveness of the electric stimulation to controlled conditions.

- Neuromuscular electrical stimulation (NMES) and NMES augmented with magnetic electric stimulation were "...capable of preventing muscle wasting and reducing muscle weakness..." in patients recovering from surgery.
- The patients had a better tolerance for magnetic electrical than the NMES therapy.

CON: Walter Kroll, Ph.D.

Dr. Kroll presented a brief review of the literature on the use of electrical stimulation. His conclusions are summarized below.

- Electric stimulation as a strength trainer for healthy subjects has been shown to have equivocal results and maybe "...an inferior substitute for simple voluntary contraction."
- Electric stimulation at lower frequencies is proposed as a way to help maintain tonic sensory function.

Bone and Microgravity

The reversibility of microgravity induced osteoporosis by pharmacological intervention was discussed by Drs. Holick and Parfitt. Dr. Holick presented the arguments that were in support of the hypothesis and Dr. Parfitt presented the opposing viewpoint.

PRO: Michael Holick, M.D.

Dr. Holick presented a review of literature supporting the hypothesis that "...the effect of microgravity on bone metabolism can either be reversed or mitigated." The question of the maintenance of the viability of the bone matrix was discussed.

- Dr. Holick suggested that "If the matrix is maintained there is good possibility that astronauts can stimulate bone mineralization... during prolonged space travel."
- Dr. Holick also cautioned that if "...gravity adapted bone cells are further stimulated during astronaut training to become more dependent on gravitational forces then the acute loss of Earth's g-force could accelerate the mobilization of calcium from the bones of astronauts. "

CON: Michael Parfitt, M.D.

Dr. Parfitt stated that his purpose was "...not to recite this chronicle of disappointment, but to account for it in terms of bone biology, to consider some possible exceptions, and to reiterate the importance of preventing damage to the skeleton, rather than belatedly attempting its repair."

- Mechanisms of bone gain were discussed. One mechanism dealing with the reversing of bone loss was based on an increased bone turnover. Dr. Parfitt suggested that bone gains by this pathway continues for approximately 6 months and generally does not restore bone density.
- Dr. Parfitt presented the argument that complete removal of structural elements (perforation of trabecular plates) can confer "...another dimension of irreversibility, since there is no longer a surface on which the osteoblasts can build."

Orthostatic Tolerance and Exercise

Endurance training has been suggested as a degradation factor of orthostatic tolerance. Endurance training was generally characterized by $\dot{V}O_{2max}$. The association between fitness and orthostatic tolerance was discussed by Drs. Ebert and Pawelczyk. Dr. Ebert presented arguments in support of the hypothesis and Dr. Pawelczyk presented the opposing viewpoint.

PRO: Thomas Ebert, Ph.D.

Dr. Ebert presented a review of the literature on the relationship between endurance trained individuals and their degree of orthostatic tolerance. He had three main conclusions.

- The arterial baroreflex is not impaired in endurance trained subjects .
- The relationship between the reflex control of peripheral resistance and endurance training is less clear and warrants further study .
- Orthostatic tolerance is not linearly related to aerobic capacity $\dot{V}O_{2max}$.

CON: James Pawelczyk, Ph.D.

Dr. Pawelczyk presented results from a research study that compared the carotid baroreflex response to lower body negative pressure among three groups of subjects classified by fitness levels ($\dot{V}O_{2max}$). The authors of the paper examined the hypothesis that higher "...fitness ($\dot{V}O_{2max}$) results in a form of orthostatic hypotension..." and that this was due to changes in carotid baroreflex responsiveness. The authors concluded:

- The results show "...that there is no improvement in carotid baroreflex in endurance-trained men."
- They also reported that high fit "...men were less able to regulate blood pressure when carotid transmural pressure changed."

Indirect Cardiac Output Monitoring during Exercise

William G. Kubicek, Ph.D.

Dr. Kubicek presented a brief historical perspective and the possible use of impedance cardiography as a tool during space flight for the evaluation of resting and exercise cardiac outputs and contractility. He also stated that "It is the only proven method available to NASA to monitor the various..." cardiac parameters.

Biomechanics of Microgravity

Robert Gregor, Ph.D.

Dr. Gregor suggested that the "...exercise countermeasures program should include a variety of modalities with full knowledge of the loads imposed on the body by the modalities." The assessment of these loads in normal and microgravity environments should be assessed through biomechanical techniques. Dr. Gregor also recommended that the biomechanical principles related to motor control may provide the astronaut with a "...tool to indicate when they are reaching the lowest acceptable limits..." of operationally based performance.

Psychophysiology in Microgravity

Anthony Hackney, Ph.D.

Dr. Hackney presented the psychophysiological impact of microgravity and suggested a tentative role for exercise as an affector for the stress related problems. Dr. Hackney suggested three key issues that should be addressed by NASA: "...(1) does exercise in space induce the same positive psychological changes as found in one-g studies, (2) what type or mode of exercise will be the most effective in producing positive psychophysiological responses in space; and (3) what is the optimal exercise prescription in space?"

Detraining Application to Microgravity

Edward Coyle, Ph.D.

Dr. Coyle put forth the hypothesis "...that the alterations in physiological function upon transition from normal gravity to microgravity may share similar trends to the process of 'detraining'." His research paper compares changes in $\dot{V}O_{2max}$ heart rate, cardiac output, stroke volume, and histochemical alterations in muscle after subjects detrained for up to 84 days.

Osteoporosis and Exercise

Jon Block, Ph.D.

Dr. Block reviewed the relationship between exercise and osteoporosis and suggested a role for exercise over a large age range. Exercise programs for young adults to help increase bone density. Exercise for women approaching menopause to reduce the rapid loss of bone and exercise for the older subject to modify risk factors for falls and fractures without directly affecting bone density. Dr. Block suggested that the effects of detraining from ongoing fitness programs to evaluate the effect on bone "...should take a research priority because of its particular relevance to long-term space flight."

Section 8

Free Communications

BIOMECHANICS

BONE

PRECEDING PAGE BLANK NOT FILMED

PAGE 222 INTENTIONALLY BLANK

LOCOMOTION IN REDUCED GRAVITY : AN OVERVIEW OF 50 YEARS OF RESEARCH

B.L. Davis & P. R. Cavanagh, Center for Locomotion Studies, Penn State University, University Park, PA. 16802, U.S.A

A major goal of research in the area of space physiology is to understand the many facets of musculoskeletal adaptation that occur once an astronaut is weightless. Early research concentrated on determining whether or not a person could maneuver in one-sixth gravity, how much oxygen was required while performing simple tasks and whether or not artificial gravity levels were necessary in a rotating space station. More recently other facets of locomotion have become important foci of research - such as bone stresses and strains during activities of daily living in space as well as optimal modes of exercise during extended missions.

It has long been accepted that to study these issues some earth-bound methods of simulating zero gravity are required. The reasons for this stem from both the cost of performing experiments on board spacecraft and the infrequent nature of orbital missions. The result has been a plethora of methods which are not widely known, to first negate the effects of gravity and second, to reintroduce "gravity-like" forces spanning the range from 0 to 1 g. Techniques in the former category include the use of aircraft, water immersion, cable suspension and elevators. Methods of achieving "gravity-like" conditions have included the use of centrifuges, modified flight paths in the case of aircraft, magnets (to increase traction between the feet and ground), and inclined walkways that allow a component of the gravity vector to be effective.

Although many of these techniques pre-dated some major advances in the field of gait analysis, some very interesting data were collected while subjects walked, loped, bounded and ran in these most unusual circumstances. Information regarding ground reaction forces, EMG profiles, joint angles and oxygen consumption rates was gathered and analyzed to obtain a deeper understanding of reduced-gravity locomotion. Other aspects that are crucial to understanding the mechanics of this process include the questions of traction, balance and impact forces. These will be reviewed with an aim to providing a consolidated picture of 50 years of research

COUNTERING SPACE ADAPTATION WITH PATTERNS OF EXERCISE FORCE, VELOCITY, POWER, ENERGY AND G's

J.A. Mastropaolo, Trisphere Institute of Sports Medicine, Huntington Beach, CA 92647

The purpose was to devise a system to analyze the elements of exercise in 1 G in order to counter space adaptation. As a pilot study, an optical encoder was used to transduce the movements of a stack of weights. The error from the encoder was -0.16%. The error in the timing circuit was -0.02%. A cubic was fitted locally to the data points by least squares with a computer program dubbed CUBIC. A test program, TESTX, according to an equation (yielding sinusoidal force, parabolic power), generated times and displacements, gave these to CUBIC, then compared CUBIC's derived elements with those calculated mathematically from the equation. For 50 determinations at 20 ms intervals, the mean difference (SD) for force was 0.066 (1.687) N, velocity 0.000 (0.001) $m \cdot s^{-1}$, power 0.029 (0.923) W, energy 0.024 (0.037) J and G's 0.000 (0.006). For each element, an array of weights lifted provided a family of curves or patterns. The hypothesis was that if the element patterns were simulated in space, then those functions would remain intact to return to 1 G. The methods seemed adequate to test the hypothesis.

NORMALIZED BONE MASS AND STRENGTH IN EXERCISING OOPHORECTOMIZED AND SHAM OPERATED RATS. E.L. Hume, D.C. Sutton, M.N. Perlmutter, J.H. Zoller, M.T. Madsen, E.R. Myers+, W.C. Hayes+, M.M. Jaweed. Thomas Jefferson University, Philadelphia, PA and +Harvard Medical School, Boston, MA.

Our aim is to describe the response of bone mass and strength in normal and oophorectomized(OX) rats from running, swimming, and weightlifting. Sprague Dawley rats exercised 5 days/week for 12 weeks. Mechanical testing and single photon absorptiometry(SPA) data was analyzed by two way ANOVA followed by Fisher least significant difference.

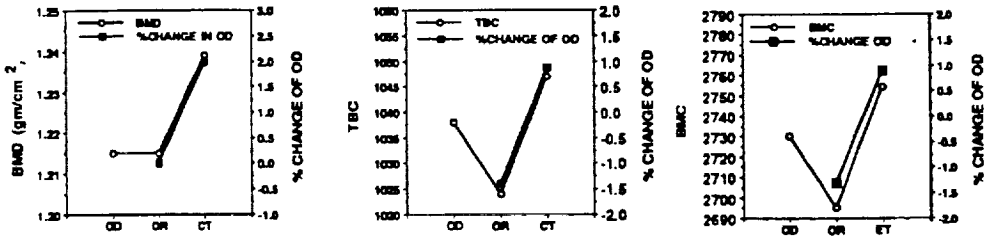
ANOVAs:		p exercise	p oophorectomy	p interaction
HIP:	ULT FORCE	.002	<.001*	NS
	SPA	NS	<.001	NS
FEMUR:	ULT FORCE	.001	.037	NS
	SPA	.023	NS	NS
LUMBAR 6:	ULT FORCE	.029	<.001	NS
BODY WEIGHT		<.001	<.001*	.003

* sham parameter > oophorectomy except

Conclusions: 1)Exercise increases bone strength similarly with and without ovarian function. 2)Swimming and lifting were greater than controls. 3)Exercise blunts but does not reverse the adverse effects of OX. 4)The effect of increased body weight load on the hip strength in OX seems to overpower any adverse effect of OX on mineral loss in the hip. (Supported in part by an OREF research grant #88-475).

BONE DENSITY AND NUTRITIONAL CHANGES OF A COMPETITIVE POWER LIFTER FOLLOWING DECONDITIONING . THEN CONDITIONING OVER A FIVE MONTH PERIOD, A CASE STUDY. K.T. Kear, R.C. Hamdy, K. Whalen. East Tennessee State University & Veterans Admin. College of Medicine, Johnson City, TN 37614.* (presently at) NASA, Johnson Space Center, Exercise Countermeasures Project, Houston TX, 77258

Intro: It is currently believed that bone remodels in response to the forces that it receives. This case study investigated changes that occurred to bone mineral density (BMD) and bone mineral content (BMC) when heavy weight training was abruptly discontinued followed by a period of rest then reconditioning. Total body scans were measured by a DP-4, Lunar Dual Photon Absorptiometer at the onset of detraining (OD) during onset of retraining (OR) and at the end of the continuous training period (CT). Both detraining and then training lasted 2.5 months. Dietary calcium (Ca), magnesium (Mg), phosphate (PO4) and protein (P) were analyzed from 5 months of 7 day dietary records, totalling (77 days). The BMC, BMD and dietary intake responses are shown below:



From the beginning of OD to the end of the continuous training ET period, BMD increased (+1.98%), BMC increased (+.88%) while Ca decreased (-49%), Mg decreased (-44%), PO4 decreased (-42%) and protein decreased (-40.%). In conclusion: It appears that BMD may be enhanced in a 2.5 month period, if the bone receives adequate forces as produced during power weight training. Dietary changes in all observed indices were similar and reduced over the same period but were adequate to support bone remodelling.

CARDIOVASCULAR

MUSCLE

PRECEDING PAGE BLANK NOT FILMED

PAGE 228 INTENTIONALLY BLANK

THE EFFECT OF EXERCISE TRAINING AND PLASMA VOLUME EXPANSION ON RESTING CARDIAC DIMENSIONS. **S. J. Montain, J.D. Beltz, and E.F. Coyle. Human Performance Laboratory, Department of Kinesiology, The University of Texas, Austin, TX 78712**

The purpose of this investigation was to measure resting cardiac dimensions before, after 10 weeks of endurance training and following 10 weeks of resumed inactivity under conditions of normal and expanded blood volume.(PVX). Seven subjects participated in the study. Plasma volume was expanded by intra-venous infusion using 500 ml of 6% Dextran™. Cardiac dimensions were measured in the supine position using M-mode echocardiography. Left ventricular end-diastolic dimension (LVEDD) tended to increase with training and decrease towards the untrained baseline with detraining. However, the changes in LVEDD were not significant ($P > 0.05$). PVX increased LVEDD 1.1-1.9 mm ($P < 0.05$) independent of training. In addition, PVX tended to decrease posterior and septal wall thickness ($P < 0.10$ and $P < 0.06$; respectively). The effect of training and PVX on LVEDD are summarized below (mean±sd):

	LVEDD (mm)		
	Untrained	Trained	Detrained
Normal	49.9±1.8	50.5±1.7	50.1±1.8
PVX	51.0±2.6*	52.2±1.9*	52.0±2.5*

The data suggest that training may alter resting cardiac dimensions. Furthermore, the results provide evidence that PVX increases LVEDD to similar levels in the untrained, trained and detrained condtion.

Supported by the American Heart Assoc.- Texas Affiliate

SEASONAL VARIATION IN PLASMA VOLUME CHANGES DURING SUBMAXIMAL EXERCISE AT 20°C AND 35°C J.W. Agnew and D.A. DuBose US Army Research Institute of Environmental Medicine Natick, MA.

Hypovolemia has been a consistent response of exposure to the microgravity encountered during space flight. Factors which predispose astronauts to a state of hypohydration are still not clearly understood. This present study has examined plasma volume changes during cycling exercise at 40% VO_{max} in two different ambient conditions in the Spring and Summer. Seven male subjects (X age=28.7 SD 3.7, X VO_{max}=45.94 $mL.kg^{-1}$ SD 4.49) exercised in an environmental chamber maintained at 20°C, RH 40% and 35°C, RH 90% on two separate occasions separated by at least one week in the Spring and again in the Summer. At each condition the subjects exercised twice for 45 min, resting for 15 min in between. Blood samples were collected at 20 min prior to entering the chamber, at 20 min and 45 min during each of the two exercise periods, and finally at 60 min after leaving the chamber. Hematocrit and hemoglobin measurements were used to determine the positive (+) or negative (-) % change in plasma volume (%PV). As expected the -%PV was significantly greater while exercising at Ta=35°C than while exercising at Ta=20°C (P<.003) during both seasons. While there was no significant overall seasonal response in %PV, the key finding with these preliminary data was that the -%PV during the Summer test at Ta=20°C was significantly less (P<.05) during all sampling periods, with the exception of the initial 30 min of exercise, when compared to the Ta=35°C test in the Summer. We also observed a significantly less -%PV (P<.05) in the majority of sampling periods of the Ta=20°C test compared to both Spring conditions. These data suggest that a greater heat tolerance in the Summer months, possibly due to seasonal temperature change and/or activity level variation, mediate either an elevated transvascular colloid osmotic pressure or increased interstitial fluid pressure while exercising in a cool environment. This adaptive response is not sufficient, however, in preventing fluid filtration into the extravascular space while exercising in a hot, humid environment.

RELATIONSHIP BETWEEN INTEGRATED ELECTROMYOGRAPHY (IEMG) AND ISOMETRIC FORCE OUTPUT OF EXTENSOR CARPI RADIALIS LONGUS (ECRL) MUSCLE IN NORMAL FEMALE SUBJECTS.

M.A. Jaweed, G.J. Herbison and J.F. Buchta. Department of Rehabilitation Medicine, Thomas Jefferson University, Philadelphia, PA 19107

It is generally assumed that IEMG output is directly related to isometric force during voluntary contractions. However, the existence of this relationship is questionable during muscle fatigue and recovery. Fourteen 22-28 year old, female volunteer health professionals were tested once weekly for a period of 4 weeks for the EMG output of the ECRL with the Nicolet Viking during voluntary contractions. The pre-trained subjects performed single, 5-second isometric contraction to generate 2, 4, 6 or 8 Kg-F on a Penny and Giles force transducer, followed by a 100% maximum voluntary contraction (MVC). The inter-subject variability, as determined by percent coefficient of variation (%CV) was 33.1, 64.1, 59.8 and 50.0 for 2, 4, 6 and 8 Kilogram Force output, respectively. The mean force and its IEMG for the subject population was 9.6 ± 1.4 (% CV=14.6) Kg-F and 407.1 ± 160.4 (%CV=39.4) microvolt respectively. In 75% of the subjects, the IEMG remained the same or declined between 8 Kg-F and the MVC of the subjects. It is proposed that IEMG can not be correlated with force output beyond 80% MVC. (Supported in part by a NASA Grant NAG9-133)

TITLE: INTRASUBJECT VARIABILITY IN FORCE PRODUCTION OF THE RIGHT QUADRACEPS MUSCLE DURING ECCENTRIC ISOKINETIC LOADING AT 30 DEGREES PER SECOND.

AUTHORS: A.H. Dubin, M.D., D. Diamant, B.S., G.J.Herbison, M.D. Thomas Jefferson Univ. Hospital

The purpose of this study was to determine the intrasubject coefficient of variation during eccentric isokinectic loading at 30 degrees per second. Six untrained healthy subjects between the ages of 21 and 28 were enrolled in the study. The maximal peak torque of three trials per day for eight days was used in the determination of the intrasubject coefficient of variation. The coefficient of variation for days one through three and days four through eight were compared to determine a possible learning effect. The results of the study showed that the average peak torques for the six subjects ranged from 626 Newtons to 990 Newtons There was no difference in the coefficient of variation comparing days one through three with days four through eight. (10% & 10% respectively). In conclusion, the average intrasubject coefficient of variation of the right quadraceps during eccentric isokinetic loading at 30 degrees per second was 10% for days one through three and four through eight respectively. Secondly the results of this study suggest that there is no learning effect when comparing days one through three to days four through eight. These results have important implications in the use of eccentric isokinetic evaluation of exercise detraining and its application to space flight.

BODY COMPOSITION

ENERGY METABOLISM

PRECEDING PAGE BLANK NOT FILMED

PAGE 334 INTENTIONALLY BLANK

RESEARCH ISSUES IN ESTIMATING BODY DENSITY: PROBLEMS WITH BIOELECTRICAL IMPEDANCE.

A previous paper (Jackson 1984) outlined the problems and procedures of estimating body composition from anthropometric methods. The purpose of this paper is to extend the generalizability and validity issues raised in the paper and apply them to bioelectrical impedance methods of measuring body composition. Published bioelectrical impedance equations with functions of estimating fat-free weight and body density were applied to the data of 82 women and 52 men. Hydrostatically measured body density was the dependent variable of this analysis. The cross-validation correlations between impedance estimated and hydrostatically determined body density were low, below 0.76, which was similar to what was obtained with body mass index. Multiple regression analysis was used to examine the independent relationship of the radio of $Height^2$/resistance in combination with body mass index. The $Height^2$/resistance was combined in a multiple regression equation with body mass index, the $Height^2$/resistance regression weight was low and near zero while the body mass index regression weights were over three times larger. This showed that height and weight and not bioelectrical impedance were the major predictors of body density.

Jackson, A.S. Research design and analysis of data procedures for predicting body density. *Medicine and Science in Sports and Exercise*. 16:616-620, 1984.

SLOWER KINETICS OF OXYGEN UPTAKE IN SUPINE THAN IN UPRIGHT EXERCISE. R.L. Hughson, G.D. Swanson, C. Borkhoff, H. Xing, and G.C. Butler, Univ. of Waterloo, Waterloo, Canada, and Univ. Colorado HSC, Denver, CO.

Cycling in the supine posture (S) has been used as an analog of the space environment to simulate the removal of the normal head to foot gravitational vector. 8 healthy men completed exercise tests in both the upright (U) and S postures. An incremental ramp exercise test was used to measure VO_2 peak. The values were greater in U (3798±181 ml/min) than S (3439±149 ml/min, $P<0.05$). Step changes in work rate from 25 W to 105 W to 25 W were used to assess the kinetics of the VO_2 response. Both for the step-on and step-off change in work rate, the total lag times for the U tests were faster than for S (31.7±1.3 vs. 42.0±3.1 s; 34.7±1.1 vs.38.9±1.4 s). The effect of this slower response in S was a significantly greater oxygen deficit (531±120 ml O_2) than U (403±56 ml). Kinetics of CO_2 production (VCO_2) were not significantly different between U and S (55.2±3.8 vs. 66.3±5.2 s). But VCO_2 kinetics were slower than those of VO_2. Removal of the normal gravitational vector alters the cardiorespiratory response at the onset of exercise. Because many activities are nonsteady state, the slower kinetics of VO_2 and the greater oxygen deficit in S implies increased reliance on high energy phosphate stores and anaerobic glycolysis for energy production. The present results will be discussed in the context of observations made during the Skylab missions.

ENERGY METABOLISM

MUSCLE

PRECEDING PAGE BLANK NOT FILMED

PAGE 238 INTENTIONALLY BLANK

CHARACTERIZING BREATH-BY-BREATH PSEUDORANDOM BINARY SEQUENCE (PRBS) GAS EXCHANGE AS A SUBMAXIMAL INDEX OF EXERCISE DETRAINING, G. Swanson, R. May, B. Dietrich & R. Hughson, Univ. of Colo. HSC, Denver, CO, & Univ. of Waterloo, Waterloo, Canada.

Dynamic submaximal exercise tests yield estimates of gas exchange kinetic parameters and offer an alternative to other non-invasive methods of assessing exercise detraining, such as maximal oxygen uptake tests. The PRBS input, in which the work-rate is switched between two levels in a random fashion, is scheduled to be used on the D-2 mission with Spacelab for assessing exercise detraining. However, the present methods of Fourier data analysis are contaminated with a large breath-to-breath (B-B) variation because B-B gas exchange is simply determined by the volume of gas inspired minus the volume of gas expired (*in-out algorithm*) without compensation for B-B changes in pulmonary stores. In contrast, we present a method that combines a Fourier series expansion with an alveolar extraction gas exchange algorithm that by design minimizes the B-B variation. This *extraction algorithm* yields estimates of the Fourier frequency response coefficients as well as estimates of effective lung volume and effective pulmonary blood flow and their standard errors. The procedure is demonstrated using the ground based data from 8 subjects. The *in-out algorithm* is unable to distinguish among subject's high frequency response characteristics. In contrast, using the *extraction algorithm* we are able to show differences among 3 of the 8 subjects. This suggests the *extraction algorithm* has more utility for detecting differences between subjects and between pre and post flight conditions for the same subject. Furthermore, it yields estimates of effective lung volume and blood flow.

TITLE: ELECTRICAL STIMULATION: POTENTIAL APPLICATION TO EXERCISE DETRAINING.

AUTHORS: R. Noble, M.D., G. Herbison, M.D., W. Rollei, B.S., A. Shah, B.S.
Thomas Jefferson University Hospital

A muscle contraction of 20% of maximum voluntary contraction (MVC) reportedly prevents loss of strength of trained muscle. Detraining may be prevented by muscle contractions elicited by electrical stimulation (ES) if a contraction equal to 20% of MVC can be obtained and the associated discomfort is tolerable. PURPOSE: To determine the force of contraction obtained by ES and if tolerance to ES developes. METHOD: The force of contraction of the right quadriceps of 6 subjects was measured during one session under the following conditions: 1. Voluntary Contraction (VC) 2. ES 3. VC & ES 4. Ipsilateral VC & contralateral ES to the quadriceps. In a second experiment, 6 different subjects underwent ES to the quadriceps on 5 consecutive days. Force of contraction obtained by ES was measured daily. RESULTS: The force of contraction obtained by ES was 14% of MVC (CV = 140 %) in the first experiment. In the second experiment, the force of contraction was 17% of MVC (CV = 106 %) on Day 1 and 38% of MVC (CV = 70%) on day 5, an increase of 216% (CV = 76%). CONCLUSION: The force of contraction obtained by ES is greater than 20% of MVC and tolerance to the pain of ES developes over time.

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

Public reporting burden for this collection of information is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of Information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to Washington Headquarters Services, Directorate for information Operations and Reports, 1215 Jefferson Davis Highway, Suite 1204, Arlington, VA 22202-4302, and to the Office of Management and Budget, Paperwork Reduction Project (0704-0188), Washington , DC 20503.

1. AGENCY USE ONLY (Leave Blank)	2. REPORT DATE	3. REPORT TYPE AND DATES COVERED
	Feb/94	Conference Publication

4. TITLE AND SUBTITLE
Workshop on Countering Space Adaptation with Exercise: Current Issues

5. FUNDING NUMBERS

6. AUTHOR(S)
Bernard A. Harris, Jr. and Steven F. Siconolfi, editors

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)

Medical Sciences Division
Lyndon B. Johnson Space Center
Houston, TX 77058

8. PERFORMING ORGANIZATION REPORT NUMBERS
S-755

9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)

National Aeronautics and Space Administration
Washington, DC 20546-0001

10. SPONSORING/MONITORING AGENCY REPORT NUMBER
CP-3252

11. SUPPLEMENTARY NOTES

12a. DISTRIBUTION/AVAILABILITY STATEMENT
Unclassified/Unlimited
Available from NASA Center for AeroSpace Information
800 Elkridge Landing Road
Linthicum Heights, MD 21090
(301) 621-0390 Subject Category: 52

12b. DISTRIBUTION CODE

13. ABSTRACT *(Maximum 200 words)*

Exercise countermeasures have been a part of long-duration space flight for both the U.S. and Russian programs. In this regard, the Exercise Countermeasures Project (ECP) sponsored a workshop entitled, "Countering Space Adaptation with Exercise: Current Issues." The workshop proceedings represent an update to the problems associated with living and working in space and the possible impact exercise would have on helping reduce risk. The meeting provided a forum for NASA scientists, engineers, and technicians to discuss and debate contemporary issues in exercise science and medicine as they relate to manned space flight with outside investigators. These outside investigators are experts who have performed investigations relating to exercise and its effect on the deconditioning process before, during, and after space flight or its analog. This meeting also afforded an opportunity to introduce the current status of the ECP science investigations and inflight hardware and software development. In addition, techniques for physiological monitoring and the development of various microgravity countermeasures were discussed. This document is a collection of the papers presented, abstracts submitted, and Socratic debates held at the NASA Johnson space Center.

14. SUBJECT TERMS
Weightlessness, Biodynamics, Orthostatic tolerance, Space flight stress, Cardiovascular system, Exercise physiology, Osteoporosis, Deconditioning

15. NUMBER OF PAGES
245

16. PRICE CODE

17. SECURITY CLASSIFICATION OF REPORT	18. SECURITY CLASSIFICATION OF THIS PAGE	19. SECURITY CLASSIFICATION OF ABSTRACT	20. LIMITATION OF ABSTRACT
Unclassified	Unclassified	Unclassified	Unclassified/Unlimited