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Some of the effects of exercise and athletics on the pre-high school child with special reference to the cardiovascular and skeletal systems

Robert E. Mockett
University of Nebraska Medical Center

SOME OF THE EFFECTS OF EXERCISE AND ATHLETICS ON THE PRE-HIGH SCHOOL CHILD WITH SPECIAL REFERENCE TO THE CARDIOVASCULAR AND SKELETAL SYSTEMS.

Robert E. Mockett

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University of Nebraska

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The doctor, parent or member of the public school system is often confronted with the question," Are athletics or severe exercise harmful to a child of pre-high school age?" This question has never been answered satisfactorily. This is probably because athletic programs are not generally included in junior high school programs or more specifically in the seventh and eighth grades. For this reason we have never been able to analyze empirical data from such a program and to formulate a sound conclusion. The practice of including junior high athletic programs within school systems is becoming more frequent but as yet it is far from universal. Our policies are still guided by those who have limited experience and who have never been able to evaluate a program in a manner which would be statistically significant. Sometimes these people conclude that athletics are acceptible for young people, or they may conclude that they are harmful to the growing child. Athletic programs have become an integral part of our senior high school programs with apparent great success as they are continually being expanded and improved. The question arises why junior high programs cannot be included.

I believe that we should not condemn a program of athletics for pre-high school youth due to the fact that it has not been done before or because of a few isolated incidents which have cast disfavor on such a program without analyzing some of the data which we have collected in man's endeavor in medicine and physiology. Although most of these studies were notdone with the idea of proving whether or not athletics or severe exercise were detrimental to the health of junior highschool students, they are certainly applicable. From the medical standpoint the controversy generally centers about the cardiovascular and skeletal systems.

For the above reason I shall try to give them the greatest emphasis though touching briefly on other systems. Also, I will discuss briefly some facts about maturity, immaturity and injuries. I will try to deal specifically with such questions with which one is often confronted when dealing with athletics in children as the following: "Does exercise damage the heart?", " Does strenuous exercise induce enlarged hearts and if so, is this harmful?", " Is there any evidence that athletes die earlier or have any more heart trouble?", "What is the most prevalent type of injury?", "Are the injuries serious and are they permanent?", "Are the injuries likely to cause disability for life?". These should be answered adequately before determining whether exercise generally and sports specifically will harm the body.

I believe that it is only right that the aforementioned question be answered for the sake of the growing child. Within many of these children is an intense desire to participate in sports. This is often denied them because of the fact that no one knows whether athletic programs will harm the growing child and few people are interested enough to find out. Often it is easier to deny all sports participation rather than determine the true answer. I believe that when millions of young people are competing in "sand lot" athletics due to the fact that the school systems will not handle the problem because they feel or are told that athletic programs are harmful to these children, then I believe that something should be done to remedy this situation. I believe that arguments against, as well as for this type of program should be backed by facts. These children are allowed to increase the incidence and seriousness of injury by

participating in their own makeshift programs. (1) For this reason I think that someone should try to determine the feasibility of a program of sports for young children in the seventh, eighth and ninth grades, so that we can either eliminate the sandlot practice and/or give an organized program to these children. We should endeavor to find out if severe or strenuous exercise is harmful to junior high school children, which part of the body it affects, and what precautions may be taken to guard against damage. Also I believe that we should determine what is the incidence, seriousness, and prognosis of injuries incurred in sports programs.

The first phase I shall try to cover will be the effect of exercise on the heart. One often hears the term "athletic" heart" in relation to people who participate in sports. Some believe that individuals who participate in sports acquire an "athletic heart" and that it is detrimental to their health.

What is an "athletic heart"? This entity is most difficult to define. The term would probably include either, several or all things as myocardial damage, cardiac dilatation, cardiac failure both at time of exercise and later in life, severe breathlessness at time of exercise, arrhthmias, and cardia hypertrophy with which would be directly associated and the result of exercise.(2)(3) What are some of the opinions regarding "athletic heart"?

Dr. H. L. Smith of the Section of Cardiology of the Mayo Clinic(2) states: "The term "athletic heart" is proof of the fact that when a term or phrase once gets into the literature it is almost impossible to get out or to cause its use to be discontinued in spite of the fact that there ever has been any positive proof of the existance of the entity which the term is presumed to represent,

and that that there has been quite conclusive proof that it does not and never did exist."

Dr. Smith (2) believes that the term might have orginated from the observation that when an individual exercises strenuously and varying degrees of dyspnea or shortness of breath develop, that people thought that this situation was homologous to cardiac failure. Sodeman (4) states: "The breathlessness experienced by the runner may not be greatly different objectively from that observed in the patient with reduced cardiac reserve after mild exertion. In the patient with heart disease, oxygen demand and lactic acid production (and accumulation) are present to the same degree after much less exercise, and not only are the stretch receptors stimulated by the increased inspiration, but also the pulmonary vascular congestion which may occur gives rise to inspiratory excitation. Both will have rapid, deep breathing with little pause between expiration and inspiration. However, the patient with heart disease may have sufficient pulmonary edema and bronchospasm to make breathing difficult. Furthermore, he may experience a feeling unknown to the runner--apprehension. It appears that the neurohumoral elements of exertional breathlessness and exertional cardiac dyspnea are similar, but the two states are quite different.".

Like Smith, Johnson (3) also believes that there is no convincing evidence that a healthy heart is damaged by strenuous er even violent exercise. It was his opinion that an individual becomes too severely fatigued to drive himself to a point where he could injure the heart. He felt that many of the concepts which are prevalent among the lay people today, resulted from the fact that thirty or forty years ago, prospective athletes were not required

to undergoe any if little physical examination to determine their fitness for strenuous competitive athletics. His opinion was that many of these instruments for detecting aberrations in the heart were not in general use at that time and for this reason many abnormalities of the heart were not perceived. Also he felt that the importance of certain types of infection was not fully appreciated in relation to their predisposition to cardiac damage. In other words, a certain number of aspiring athletes with already damaged or otherwise abnormal hearts were allowed to participate in sports which added insult to injury and therefore set the stage for cardiac breakdown later in life. The premature invalidism of death of many athletes of many years ago could probably be accounted for in this way. Participation in athletics according to Johnson(3) undoubtedly harmed their heart; but they were not healthy hearts oringally. From their condition before death and the findings at autopsy, many people probably inferred that athletics were the cause of their difficulty before death and probably contributed to their death. From these statements taken from Doctors Allen and Smith we can infer that clinically, the entity does not exist.

Is an "athletic heart" a pathological entity? Smith (2) implied that it was not. He stated," It seems reasonable (to me) to suppose that if the condition of athletic heart has existed as many years as it has been written about, some reliable pathologist would have seen and described an athletic heart, but no record exists of such a heart being found. The promiscuous use of the term "athletic heart", by certain writers, probably has had considerable influence on the introduction into the literature of some other terms which are equally inexcusable, such as "industrial heart", and "military heart", and the proof for the

existance is just as lacking as it is for athletic hearts."

Is there any evidence that severe exertion causes myocardial damage in a heart which is not diseased? Exertion is not listed as an etiological agent in the production of primary heart disease in the "Nomenclature and Criteria for Diagnosis of Disease of the Heart and Blood Vessels" (5) by the Criteria Committee of the New York Heart Association which is endorsed and distributed by the American Heart Association. Nor is it listed as a cause of primary myocardial damage in "Pathology" by Anderson. (6) In 5,370 autopsies which were performed at the South African Police Medico-Legal Laboratories (?) between 1934 and 1939, twenty instances of acute fatal collapse during work or in sports were found. Another forty-three cases were collected from the literature. Here, only cases in which autopsy had been performed were considered. No previously healthy individuals died from excessive exertion in any of the cases.

In all cases of death during work or sport, autopsy revealed the presence of one or more of the following diseases of the circulatory system: coronary arterial disease; inflammatory disease of the myocardium; degenerative disease of the heart muscle; rupture of heart muscle and rupture of the aorta. From this study the authors found that not one instance was encountered in which death could be regarded as due to the effect of extreme exertion on a previously healthy heart. They also noted in this study that in no fatal collapse was the individual doing anything physically more strenuous than he had been accustomed to doing previously.

Is there any evidence to support the conclusion that exercise may lead to acute cardiac dilatation? In a report by Gordon(8)

on the "Effect of Effort on the Size of the Heart", he reported a decrease in the size of the heart immediately after strenuous exercise. In a study on the effect of exercise on the heart of a group ranging in ages from 18 to 41 years who were taking part in a thirty-one mile marathon race and who were found to be fit as determined previously by chest films and ECG's, it was found that the size of the heart decreased after severe exercise in all subjects observed.(9) The diminution was seen both in the anterioposterior andlateral views of the heart. The decrease bore no apparent relationship to the increase in heart rate and it persisted after the heart rate returned to normal. It was thought that the diminution of heart size, therefore, is not comparable with that which has been shown to occur when the heart is accelerated without physical exertion. No significant abnormalities were seen to have occured in the ECG's. In th

American Marathon Race, (9) runners who had participated for as many as fifeteen years were observed. This afforded an ex-cellent opportunity to study the effect of prolonged, strenuous exercise upon the size of the heart. In this study it was found that immediately after the race, practically all of the hearts were smaller than they had been just before the race. This would tend to indicate that strenuous, prolonged effort does not cause acute dilation of the heart. Roentgenological examination made on the day following the race gave evidence of gradual return of the hearts to normal size. These runners were also observed for the effect of many years of strenuous exercise on the heart. In this study no proof of cardiac enlargement was found.

In another study in which the hearts of ricksha pullers (10) were observed for cardiac enlargement after chronic exertion, it was concluded that the hearts of 55% of the subjects were not

enlarged, but that 45% were somewhat hypertrophied. The conclusion was that this enlargement was entirely physiologic and not pathologic and th t there was no evidence that the enlargement predisposed in any way to disease.

It would seem from these observations that roentgenological evidence of acute dilatation rarely, if ever, exists after strenuous exercise.

Does the heart of the athlete hypertrophy or enlarge to a greater degree than that of individual who doesn't participate in athletics? According to Best and Taylor(11), Wright (12), and Sodeman (4), before a heart fiber will hypertrophy it must be stretched beyond its physiological limit. According to Best and Taylor (11) this does not occur in a normal heart. They explain the mechanism of cardiac hypertropy in a manner which is quite easy to understand and quite applicable here. They state: "If muscle is made to contract isometrically the tension developed during the contraction is found to be proportional to the length of the muscle. The tension which the load exerts upon the fibers just prior to their contraction is spoken of as the initial tension. The tension developed when the muscle contracts isometrically will be referred to as the developed tension which is a measure of the force of the contraction. When a resting muscle is weighted, little change in initial tension actually occurs until it is extended beyond a length corresponding to that which it possesses when in its natural position in the body, i. e. at its physiological length. Up to this point the developed tension increases with each increment in initial length. Yet, it is only when the muscle is stretched beyond its physiological length that any marked increase in initial tension occurs. The power of contraction of skeletal muscle therefore is dependent upon initial length and not

upon any stimulating effect exerted upon the muscle fibers by initial tension. The venous pressure which stretches the cardiac muscle during diastole corresponds to the weight applied to skeletal muscle. The opening of the semilunar valves against the aortic pressure represents the load against which the cardiac muscle has to contract. It will be seen that the force of ventricular contraction increases with the diastolic volume until theheart becomes over distended and a rise in diastolic pressure occurs." This is exactly what happens in exercise. Tremendous volumes of blood are returned to the heart. (11) The question then arises, is the heart capable of handling all of this blood without dilating beyond its physiological limit hence predisposing the myocardium to hypertrophy? Again this is best answered by Best and Taylor who state: "The energy set free at each contraction of the heart is a simple function of the length of fibers composing its muscular walls. Thus the heart fibers automatically gain the necessary energy to eject the greater load of blood which fills its cavities during diastole. The oxygen consumption of the heart muscle, i.e., total energy expenditure, is directly proportional to fiber length (diastolic volume). The work performed by a heart in good physiological condition bears a linear relationship to diastolic volume and consequently to oxygen consumption. When, as a result of fatigue, the condition of the heart deteriorates, its diastolic volume is much greater in proportion to the work performed, than is the case of the well-conditioned heart, i.e., the ill nourished muscle fibers in order to gain energy for the performance of a given amount of work must be stretched to a greater extent. Nevertheless, whether the condition of the heart muscle is good or bad, the relationship between oxygen consumption and diastolic volume is the same. This means, clearly, that for the

performance of a given piece of work the poorly nourished heart uses more oxygen (since it dilates more) than does a heart in good condition; or put in another way, the proportion of total energy expenditure which appears as mechanical work is lowered when the heart muscle departs from its prime physiologicalstate. " One can infer from this that as long as sufficient oxygen is supplied to the heart muscle it will continue to handle the venous return.

Best and Taylor (11) go on to say: "The enlargement (dilatation and hypertrophy) of the diseased heart is a compensatory reaction. In aortic regurgitation the heart receives blood not only from the auricle but also from the aorta as a result of the incompetence of the aortic valves. The diastolic enlargement of the ventricular cavity which is required for the accommodation of the greater blood mass also enables the heart to develop the energy necessary for the ejection of blood during systole. Cardiac dilatation, therefore, under physiological condition or in association with heart disease, is the means whereby the heart mobilizes its reserves of energy. The so-called reserve powers of the human heart, i.e., its capacity for work, resides in the extensibility of its muscle fibers, within the physiological limit. It is apparent then that the nearer the fiber during diastole approaches its maximal physiological length, the greater will be the encroachment upon the heart reserve. A well developed and efficient heart, in order to gain sufficient power to accomplish a certain amount of extra work, need dilate relatively little and is capable of discharging easily as much blood as it received. The venous pressure in consequence shows little tendency to rise and the pulse is not greatly accelerated. On the other hand, the smaller heart of the heart with a myocardium weakened by disease when given an equivalent amount of work to perform must dilate to a greater extent in order

to liberate the required energy. Indeed, an ill equipped heart may, as a result of ome extra burden, be dilated to its physiological limit and still be unable to increase its output per beat. The heart accelerates its beat as this point is approached (Bainbridge Reflex) in order to increase its output. When the full physiologic length of the fiber has been attained and the optimal pulse frequency develops, the heart has reached the limits of its powers. Blood then accumulates in the cardiac chamber, the venous pressure rises, the circulation thru the capillaries is slowed and the blood gives up a greater proportion of its oxygen load to the tissues." The heart receives less oxygen for its own consumption. Cardiac dilatation and then hypertrophy may result.(11) Why the hypertrophy occurs after such a equence of event is unknown. (11) (4)

Sodeman (4) believes that it is reasonable to assume that cardiac hypertrophy results from vigorous exercise as well as in the severely taxed heart with disease of the myocardium. In his opinion hypertrophy of the heart which results from exercise is reversible, gradually receding after termination of the exercise period. He calls such hypertrophy normal hypertrophy and believes that is has no pathological significance. Houssay(13) believes that some cardiac hypertrophy occurs in severe prolonged exercise but makes no conclusions as to its significance.

When we take X-rays of the hearts of athletes what are we likely to find? From what has been said it would seem likely that we might find some slightly enlarged hearts. What is the significance of these enlargements? In a survey of the range of normal hearts in athletes, Wilce (14) found that some hearts were enlarged, some were normal and others were small. He found that out of 233 hearts, 113 were absolutely enlarged, that is, larger than the allowed normal

area range of 10 percent above average. One-hundrend-twenty were below this criteria. This author decided that some of the absolute enlargements could have been the result of exercise. However in critically evaluatin them he found that some could be explained by inheritance, disease or deficiency factors before, during or after active sport. An interesting sidelight to this author's study was the fact he found only 29 hearts out of 233 which could be called normal by the following standards: no evidence of organic disease, congenital or acquired; no abnormal variation in size or shape under accepted standards; no evidence of defiency or degenerative condition; no abnormal variation in functional efficiency; and no history of any condition which is known to predispose to heart disease or to favor it. The author found that if he eliminated size or shape from his list of criteria the he had 37 "normal hearts".* This study consisted of 233 male American athletes whose ages ranged from 16 to 80 years of age. The cases were well distributed up to the age of 50.

These authors found that only seven of the thirty-seven hearts were postulated as normal were enlarged by athletic competition, and hence would qualify as hearts enlarged by strenuous work or exercise. They found that these hearts were absolutely normal

* The 37 hearts were classed as normal because of no organic disease, or any other disease included in the army and navy list or in the criteria for cardiac diagnosis as potential heart disease factors, namely: scarlet fever, chorea, diptheria, measles, rheumatic fever, tonsillitis, influenza, typhoid fever, syphilis, gonorrhea, beri-beri or chronic focal infection. They were normal athletes, with absolutely normal function, so that athletics were postulated as the only possible influence on heart size.

clinically and functionally. Their conclusions were that these individuals had normal hearts for all practical purposes and that the hearts would permit a normal or greater than normal life expectancy. Also they concluded that generally athlete's hearts are larger than non-athletes hearts and that non-organic cardiac enlargement is probably of little clinical importance and should be considered normal. They did say, however, that the eventual result of "non-organic" enlargement is not yet definitely known but in their opinion, participation in sports should be encouraged in every way, though under alert and accurate medical supervision.

This might all be well summed up by what Best and Taylor (11) say about the permanent effects of exercise upon the heart. " The belief has been widely held that strenuous muscular effort is conducive to cardiac dilation and hypertrophy. A proportion of athletes have suffered from heart disease and an occasional race horse has died with a dilated and hypertrophied heart. Instances of this sort have been cited in support of such a view. It is now agreed, that the cardiac enlargements under these circumstances are the result of pre-existing disease and that the healthy heart cannot be dilated beyond its physiological limit, nor will a bout of strenuous exercise strain the healthy heart muscle or cause it to fail. In a healthy person the functional capacity of the heart and of the skeletal musculature are apparently so proportioned to one another that the greatest venous inflow which the muscle can provide is taken care of by the reserves of cardiac energy. The healthy heart of the marathon runner or oarsman does not hypertrophy to a pathological degree. It has been shown by X-ray examination that the diastolic diameters of the healthy heart are

materially increased during rest; that is, the heart empties more completely. It may be concluded that acute dilation or chronic dilation with hypertrophy is the result of some disease state of the heart itself. It should not be concluded, however, that muscular training exerts no effect upon the size of the heart. Mitchell(15), for example, found that the heart size of a group of undergraduates at Cambridge underwent a gradual increase over a period of a few years of athletic training. This was accompanied by a reduction in pulse rate. Lindhard (16) observed an increase of twenty percent in the resting cardiac output as a result of physical training. Others have reported more marked effects in persons indulging in very arduous types of athletics. Eyster (17), on the other hand, could demonstrate no increase in average heart size of a group of young athletes when compared with that of a group following a sedentary life. The evidence taken in review indicates that

athletic pursuits, especially of a strenuous nature, may cause moderate but definite increase in cardiac bulk. This however is purely physiological and is proportioned to, or only slightly in excess, of development of skeletal muscles. This is to say, the normal ratio of heart weight shows no change or a very moderate one.

One often hears about the irregularities associated with the heart after strenuous exercise. Many of these are without clinical significance as interpreted after ECG. (11) Cureton (18) found that many of the arrhythmias, slurring and other irregularities found in ECG's of athletes were not of significance an that the irregularities appeared to be associated with vagotonia. One should infer from this that all irregularities should be evaluated after an ECG to determine their origin and importance. Also the

ECG should govern the course to followed. Exercise should not be terminated after finding the irregularity without full evaluation.

What are the opinions of a few prominent men about the effect of exercise upon the heart? Paul White (19) said: "Physical work and exercise do not cause heart disease, though they may precipitate or aggravate symptoms and signs of heart disease already present and may temporarily exhaust the cardiovascular reserve in a healthy individual."

Smith (2) in his paper stated:" The point I want to emphasize is that strenuous physical exercise, and hard manual work do not cause, or predispose to, heart disease, that such activities do not cause abnormal cardiac hypertrophy, and that athletes do not develop early disability, and die, because of exercise in which they indulged while in school. The normal heart, and I would emphasize the work "normal", is no more likely to be injured by strenuous exercise than is any other organ or muscle in the body, and participating in athletic sports will not produce an "athletic heart", for such an entity does not exist. Of course, it is very important for every individual who participates in athletics to undergo a thorough physical examination. And anyone who has organic heart disease certainly should not participate in prolonged strenuous exercise.

Lewis (20 added to what has been said previously when he said: "Burdens imposed by physiological acts upon the normal heart, however heavy these burdens, may be said never to injure heart fibers, never to produce injurious dilatations and never to exhaust the heart's reserve."

Dublin(21) whose work on longevity of college athletes is well known stated: "Indulgence in athletics in a good many instances have deleterious effects on the heart especially if careful

selection and supervision of athletes by trained men is not available."

Cole(22) Summed up most of the above in a short statement when he said:"Our business is to look for cardiac defects and protect the defective heart. For the heart that has proved itself healthy we have no concern."

As has been shown, no evidence exists from a physiological or clinical point of view that severe or strenuous exercise has any detrimental effects on the normal heart. What other aspects must be considered in determining whether young people can safely participate in athletics? One often hears that children in the junior high school age are too immature to participate in strenuous sports. The reson often given is that the severe effort required in sports injures them. I wonder what part of the body is injured? As I have indicated earlier in this paper most reputable sources do not feel that the heart is injured. If the organ which is most essential to exercise is not harmed I wonder what organ or system is injured. Next I begin to doubt the conclusion that these young individuals cannot participate in sports without harm incurred by the exercise itself.

Wright (12) and Mitchell and Nelson (23) indicate that the period of most growth in children is in the ages of 12 to 15 years. This means that for most children that the first year in high school is during one year of the most rapid growth. Most of our educators and physicians according to one report (27) think that athletics in the tenth grade are perfectly acceptable. Some even think that a ninth grade program of sports is acceptable.(27) Some schools are installing programs for ninth grade pupils. This means that strenuous sports are approved in one to two years of the period of most rapid growth. A fourteen year old individual in the ninth

grade which is six or seven years before complete maturity (12)(15) is allowed to participate in any athletic activity in which he is judged to be fit. Is it any less injurious to participate in athletics before complete maturity or in the last year of the most rapid growth than it is from ten or eleven years of age when the interest first develops. Again when we speak of physical immaturity we are confronted with a relative term which means little.

What do we mean by immature? Is the immature organism any less likely to respond favorably to the activity of sports than the mature organism? Or is it possible that the immature organism may adapt itself better to exercise than the mature organism in regards to physical response? Maturity in Webster's Collegiate Dictionary (24) is defined as: "Brought by natural process to completeness of growth and development; fully grown, ripe." This seems to me to be a relatively meaningless term, when one has to become specific as to the degree of maturity which decides when one age group may safely participate in strenuous activities and another group may not. The sensible approach to this problem would seem to be to evaluate the child as to his ability to participate in strenuous exercise, and not to arbitrarily state that this age group cannot participate because its members are too immature. This term strike me as being similar to "athletic heart". I think that it is interesting to note the comparison of the responses of the bodies of young people as against those of adults or mature individuals.

What happens to the blood pressure in young people as compared to the older mature person. During muscular exercise or at the moment of its contemplation in the adult, the pressure begins to rise and reaches a height of 180 millimeters of Mercury to 200 millimeters of Mercury. (11) The diastolic pressure usually

shows a less pronounced rise. (11) It usually reaches 100 to 110 millimeters of Mercu . (11) Immediately after exercise, the pressure drops momentarily to normal or even slightly below. (11) It then mounts rapidly to a previous high level, and then gradually declines again, and in a healthy person reaches the normal within from one to four and one-half minutes. (11) How does this compare to what happens in children.

Shock (25) reported that in 100 children whose ages ranged from thirteen to eighteen years and who were observed for six to twelve months, that after climbing five flights of stairs t' t their maximum systolic blood pressure averaged 160 millimeters of Mercury in the thirteen year old boys and rose to an average of 175 millimeters of Mercury in the seventeen and one-half year old boys. Some evidence was found that the systolic pressure response to exercise take place more rapidly in younger children than in older children. Diastolic pressure dropped less with each succeeding year. A severe drop in diastolic pressure to a very low level after exercise was often seen and was thought to be related to physiological immaturity as indicated by growth rate, ossification of epiphyses, and secondary sex characteristics than to chronological age. It was concluded from this that adult vessels (arteriols) relaxed less than children's.

From this comparison it would seem that nothing extraordinary occurs in children while exercising in regard to the blood pressure. The level attained in children is certainly not excessive and is lower than in the adult. One could infer from this that young adults have no difficulty from elevations of this level so why should a child. One might ask but what about the chronic effects of exercise upon the resting blood pressure in children.

In a study of the effects of basketball on junior high boys by Bowyer (26), he found that the blood pressure was lower and the pulse a little higher at rest at the end of the season as compared to that found at a pre-season exam. The blood pressure at rest at the pre-season examination averaged 130/79 and at the postseason examination averaged about 114/76. This would indicate that the blood pressure had dropped slightly after chronic exercise. One could interpret this as an effect of "training". How do adults respond to chronic exercise or "training"?

Wyman (28), Herxheimer (29), Ewig (31), and Ackerman (32) found that in a majority of cases a considerable reduction in the resting systolic blood pressure resulted from strenuous "training. Also Herxheimer (30) found a drop immediately after exercise which averaged about 116 to 106 millimeters of Mercury though this was not observed until after the strenuous daily practice of the training period gave way to a more moderate daily exertion as training was rounded out.

Both Ackermann (32) and Herxheimer (29)(30) found this decrease less noticeable in youthful athletes or in the first year of training. In comparing trained and untrained athletes Dawson (34) found: "(a) the systolic pressure rises more rapidly and much higher than is the case of the untrained individual; (b) the pulse pressure is enormously increased." Fabre(35) reported similar findings and emphasized rapidly falling systolic and more slowly falling diastolic, shortly after the beginning of work, as marking the untrained.

It would appear from these observations that no significant changes in blood pressure occur in children as compared with adults.

What happens to the pulse in the child as compared to the adult in strenuous exercise during and directly after the exercise. Browyer (26) in studying the effects of basketball on junior high school boys found that in a game in which the boys were not pressed that all pulses were back to their pregame levels twenty minutes after the game. In a close game, won in the last few seconds, the heart rates of two players returned to normal in five minutes but in the remaining players, they did not return to normal for twenty minutes. It was noted that of the boys whose pulses were normal in five minutes had been removed on fouls in the third quarter and the other had played only two quarters. These observations were continued throughout the season in the home team as well as in four visiting teams. These authors concluded that it was the extra quarter of play which was responsible for the elevated pulse which remained for twenty minutes. From this it could be concluded that the homeostasis of the organism is altered for a somewhat longer period and that the compensatory mechanisms have not brought the organism back to the finely balanced state of normal homeostasis. Morse, Schultz and Cassels(37) found the elevation of heart rate after moderate exercise, which consisted of walking 15 minutes at 3.5 miles per hour, was higher above the resting level and its deceleration after the walk was faster in boys twelve to fourteen years than in both younger and older boys. In severe exercise which consisted in running to exhaustion at a speed of six to seven miles per hour on a treadmill set at an 8.6 per cent grad they found that the elevation of the heart rate above the resting level was higher in older boys, beginning at 13 years. The group observed consisted of 110 boys between the ages of ten and seventeen. The only conclusion that might be drawn from this would be that younger people compensate faster +

than older ones. That their compensatory mechanisms are more able to supply the debts which are incurred during depletion with exercise. However Houssay (13) does not agree with this. He states that the hear rate increases with the intensity of the effort made and that with the same intensity of exercise, a greater increase in heart rate is seen in young subjects than in older ones. However, he uled as a frame of reference a chart by Dill (39) which showed that the heart rate of subjects ranging from six to forty-two yeara after walking at a rate of 5.6 kilometers per hour on 8.6 per cent grad. Certainly this is not to the point of exhaustion so the observation by Morse, Schultze and Cassel may be true.

Of what is this cardiac acceleration the result? Houssay (13) remarks that it is the result of several factors acting upon the heart. The initial increase just before exercise is probably due to a psychic effect on the vagal tone. With the onset of exercise the greater venous return increases, the distention of the right auricle and it reflexly increases the heart rate. Also he considers increase in body temperature, sympathetic nerve impulses, adrenalin and other minor factors to effect the rate. This indicates that all of theae different factors are interpreted by the body integrated and in the normal organism that the different compensating mechanisms are stimulated or depressed in order to maintain homeostasis of the organism. What we observe are the compensatory mechanisms maintaining homeostasis in action. This would indicate that the increase in heart rate in these circumstances has nothing to do with harmful effects on the organism. This is further substantiated by the decrease in heart rate after training.(36)(13) This would indicate that the body is better able to compensate and therefore the cardiac output need not be so great.

One might wonder if the severe exertional dyspnea is harmful and an indication of strain upon the organism. Certainly it shows a taxation of the compensatory mechanisms of the organism but probably is not harful. (11) With severe exertion a shortness of breath, rapid respiratory rate, and deep breathing develop. (11,12, 13) This is the result of several factors. The carbon dioxide tension rises, the oxygen tension falls, and the pH decreases. (13) These factors stimulate the respiratory center and peripheral chemoreceptors and cause an increase in the rate of respiration and increase in depth of respiration. (13). The fact that the respiratory rate increases rapidly from the very beginning of exercise, before metabolic products can accumulate in the blood and act on the respiratory center, has been attributed to reflex stimulation of this center. (13) These reflexes have their origin in the active muscles and joints, (40) in the lungs, in the carotid or aortic bodies, and in the great veins and auricles, or are due to substances produced by the muscles acting on chemoreceptors. (13) To sum up, nerve impulses arising in the active limbs and the lungs, or irradiated from the cortex to the respiratory center, play an important, but not exclusive, part in the hyperpnea of exertion. The increase in carbon dioxide and the decrease in pH in the blood, the accumulation of lactic acid, the changes in the circulation occuring in the respiratory center, impulses arising in the carotid and aortic bodies and even the increase in body temperature, add their effects to those mentioned above and contribute to produce hyperpnea. (40) One could conclude from this that with exertion that much work is done, hence much energy consumed, metabolism increased, metabolic products increased, oxygen consumption greatly increased and to keep the organism going the compensatory mechanisms swing into action. During the first few minutes of exercise, while the oxygen consumption

rises, the oxygen absorbed is less than that needed to oxidize the metabolic products of muscular contraction. (13) This is due to the delay in the adaptation of the respiratory, and especially the circulatory function to the greaterdemand of oxygen by the tissue. (13) Nevertheless, the muscle continues to contract, in great part by means of the energy set free by anaerobic chemical reaction. (13) The relative lack of oxygen prevents a complete resynthesis of lactic acid produced into glycogen. (13) This acid accumulates in the muscle and passes out into the blood; therefore, lactacidemia increases. (13) During the steady state, a balance is established between the need of oxygen and the speed at which it is delivered to the muscles and used up by them. The amount of lactic acid accumulated neither increases nor diminishes. (13) During recovery, the rate of oxygen consumption should fall suddenly to the resting level. but it does not because the lactic acid which is accumulated during the first states and which is not removed during the steady state must be disposed during the period of recovery. (13) The organism has contracted an "oxygen debt" the payment of which is postponed until the xercise has ended. By " oxygen debt" is meant, the amount of oxygen, in liters, necessary for the removal of metabolic products accumulated while the supply of oxygen is below the needs of the organism. This debt is measured by the amount of oxygen consumed above the basal rate between the end of exercise and the time when the oxygen consumption has fallen to the resting level. (13) Before the breathing mechanism returns to normal the oxygen debt must be paid. In violent exercise the oxygen-consumption curve is different. It first increases to a maximum, which is higher than that of the steady state of moderate exercise, but nevertheless does not

completely satisfy the oxygen requirements of the organism. The oxygen debt is much greater and the recovery period more prolonged. (11,13)

The exercise an individual can perform is limited by oxygen supply in one of two ways. In the first place, there is a maximum rate at which oxygen can be absorbed, transported, and used, determined by the efficiency of the respiratory and circulatory adaptations. (13) This maximum is usually 4 liters per minute but in exceptional cases higher figures up to 5.35 liters have been recorded. (13) This maximum rate puts a limit to the speed of exercise. In the second place, there is a limit to the oxygen debt that the organism can contract to be paid during the period of recovery. (13) This puts a limit to the time during which violent exercise can be performed. (13)

When oxygen demand is greater than the supply, lactacidemia increases six to twelve times the normal level. (13) The lactacidemia level represents an equilibrium between the production and removal of lactic acid. One source of lactic acid is its formation in the blood by glycolysis, which increases in alkalosis and also when the blood passes through the lungs. (13) Another and more important source is muscular contraction, during which glycogen is first broken down to lactic acid and then again resynthesized to glycogen when energy is provided by oxidation. (13)

When there is a strong or sustained muscular contraction, the insufficiency of the oxygen supply causes the accumulation of lactic acid, which diffuses into the blood stream. (13) The lactic acid formed by glycolysis or in the course of muscular contraction can be excreted in the urine or sweat, or utilized by the heart, or reconverted by the muscle, into glycogen, but the greater part of that which has passed into the blood is taken up by the liver and

converted into glycogen.(13) Oxygen is needed for this synthesis. (13)

When the formation of lactic acid does not exceed the capacity of the organism to remove it, as in the steady state of moderate exercise lactacidemia does not increase. (13) When the effort is greater, during the initial phase, while the oxygen supply is below the demand, lactic acid will accumulate in the blood. (13) It will remain at a fairly constant high level if a steady state is established. (13) Later, during the period of recovery, lactacidemia will fall rapidly to the resting level. When the exercise is such that even the maximum possible absorption of oxygen falls short of the requirements, there is no steady state; lactic acid continues to accumulate until the exercise must be discontinued because of fatigue.(13) In this case a maximum oxygen debt has been contracted, and there is a prolonged period of recovery. This can be altered by training.

The lasting effects of repeated exercise shows increase in the working capacity of the organism. (13) The object of training is to acquire this increased capacity by increasing: (a) strength; (b) resistance (sustaining an effort); (c) accuracy and sureness of movements.(13) The increase in strength is obtained by great muscular development (hypertrophy and increase in motor units and perhaps by physical and chemical adaptation: larger stores of glycogen and phosphocreatine, increased ability to dispose of lactic acid, greater activity of oxidation-reduction mechanisms, etc.(13)

Increase in resistance is conditioned by the capacity to respond to the greater oxygen demand created by exercise. (13) Training results in hypertrophy of the heart and increase in blood volume. (13) There is a diminished cardiac minute volume at rest, and an increase in the vital capacity of the lung, so that in exercise the minute

volume and pulmonary ventilation can increase to a greater extent than in untrained subjects. (13) The alkaly reserve also increases, and thus larger amounts of lactic acid can accumulate in the blood. (13) Trained subjects have a lower resting pulse rate than the untrained, because of an increase in vagal tone. (13)

A more specific result of training is sureness and accuracy in the performance of movements. (13) This is due in great part to improved neuromuscular coordination. The muscles respond more rapidly, and there is more accurate adjustment in the reciprocal contraction and relaxation of synergical and antagonistic muscles.(13) For this reason, to obtain better results in a particular type of exercise, e.g. rowing, it is necessary to practice this type of exercise besides undergoing the general training. The more accurate coordination of the different movements results in economy of effort, as only the appropriate muscles are contracted and only in the necessary measure; thus fatigue is delayed. (13) The recovery period is also much shorter in trained subjects than in untrained ones. (13)

How do children respond to strenuous exercise? In Morse, Schultz and Cassel' report (37) on boys between ten and seventeen running to exhaustion they found that in the first one-half minute of the run, during the period of adjustment, oxygen consumption accelerated at a faster rate in younger boys. During the first two minutes of the run, the acceleration of respiratory rate and lung ventilation relative to body weight was slower in older boys but was accompanied by relatively greater utilization of oxygen of the inspired air. At exhaustion, oxygen consumption showed minimum values at thirteen years then increased to a maximum at seventeen years; elevation of blood lactate concentration was at a minimum at twelve years,

increased progressively from twelve to seventeen years; respiratory rates decreased with age from ten to seventeen years, while the ratio of tidal air to vital capacity reached higher levels beginning at twelve years.

Shock(25) found that after exercise (running up five flights of stairs in a group of one-hundred children from ages of thirteen to eighteen over a six to twelve month period) the oxygen uptake attained increased with increasing age. The fall in oxygen uptake during the first minute after exercise was also greater in older than in younger children. However the rate of recovery of oxygen uptake during the later stage of recovery was slower in older than in ypunger children.

What influence has age, physique and muscular development on physical fitness? In an article by Cullumbine, Bibile, Wikramanayake and Watson (41) in which they observed seven-thousand Ceylonese subjects ranging in age from ten on up with the majority in the age group of ten to twenty years they concluded after using the Harvard Step Test for assessment of the ability of the people to perform moderate muscular exercise as measured by an index based on the duration of exercise and the heart rate during recovery that the ability is constant for boys in the preadolescent years, declines with the onset of adolescence, shows a further significant decrease at the age of seventeen years, then rises steadily to a new maximum between the ages of twenty-one to twenty-five years and thereafter declines slowly again.

Espenschade (42) found that older, taller and heavier children are stronger and in general more proficient in activities than are younger, shorter and lighter children. MacCurdy (43) found evidence concerning the relationship between strength of boys and physical

growth which indicated that physical growth is gradual up to the age of twelve and then is very rapid. The maximum growth is reached at approximately eighteen years. McCloy (44) agreed with this generally when he found that the most rapid increase in strength of boys occurs between thirteen and sixteen years. He found only a slight increase after the age of seventeen years. In relation of strength to puberty imoek (49) found that the postpubescent at thirteen was stronger than the prepubescent at fifeteen. And that the acceleration of strength lagged behind the spurt in height and was most marked in the first year after the postpubescent stage. Espenschade (42) also found that body growth and athletic achievement runs parallel, that there is no significant correlation between performance and physical proportion at any one age. Also the motor performance is related to age, weight and height during the elementary and junior high school years but shows slight correlation with body build. Physiological maturity evidently influences the rate of increase but the nature and extent of this influence has not been determined. Espenschade (42) concluded that motor performance of boys is positively and significantly related to all measures of maturity, chronological, anatomical and physiological.
When chronological age is held constant, relationships between motor performances and anatomical or physiological maturity are lower but remain significantly positive in some events. The relationships between gross motor performances and physical measures of boys between the ages of thirteen and seventeen years are strongly influenced by the range of maturity and variability of physical growth rate, and that relationships would be lower in a group more homogeneous in these factors.

Jokl and Cluver (46) when testing South African children, noted that puberty caused a retardation in the rate of progress of the ability of boys to run 100 yards and stopped the progress in the ability of girls to run 100 yards. They concluded that the capacity to perform everexercise decreases with advancing age in both sexes from about the age of fourteen onward, with a temporary increase at seventeen and possibly at twenty years. They also found that the endurance of boys increases steadily between the ages of five and twenty years, adolescence retarding, but not interrupting, the progress. Speed of movement, muscular strength and the ability to sustain moderate exercise to fatigue do increase with age to a maximum in early manhood or womenhood. Similarly, strength and exhaustion indices increase with increasing developmental levels and are positively and significantly correlated with developmental levels.

What is the incidence of injury and how serious are the injuries incurred in teenage ports? Injuries are ever present in any type of athletics, just as they are in every day life. It is interesting to see the frequency of the injuries incurred in sports, their relative probability and their prognosis. In the sports which are most generally included in programs in the high schools and colleges about the country (most frequently football, basketball, track, wrestling, baseball, softball, tennis, swimming and physical education (47) the most common cause of injury is football. (48, 49) This may vary from area to area but it seemed to be a fair indication of the relative incidence of injury of one sport compared to another. The next most injury prone sport according to one study (49) was basketball. However it was only one-fifth as dangerous from an injury incidence standpoint as football. (49) In analizing this data Hibbert (48) found that in observing 47,000

athletes participating in sports, one in five would expect to be injured. In two studies (48; 49) covering 60,000 athletes competing in all sports, the incidence of fracture was about three per cent. This indicates that a person being injured has about a 15-20 per cent chance of having a fractured bone. In a study by Kramer (49) in which the injuries in basketball were one-fifth as great as in football, the most common injuries in basketball were dental injuries, sprains and fractures. Of the 105 injuries in basket ball, 81 were in the above group. (49) Twenty-five were fractures. Seven of these fractures were of the nose. I believe the fracture group is the most important consideration in this group of injuries. In this study seventeen received fractures which could be considered of significance. This seems to be a rather small umber. This is only one set of figures but represents a trend in several other studies. (50, 51, 48) From this I would conclude that the sport which incurs most of the injuries is the one which should demand the most attention. This is football.

What is the incidence of injury in football? At Princeton in a ten year period a member of the Princeton Football Team had sixty-nine changes in one-hundred of being injured. This study covered about 780 men. Players averaged 5.2 days away from practice. One in twenty-five was injured severely enough that he had to give up the sport for the season and one in one-hundred had to give up the sport entirely. An injury here constituted a day lost from practice. In 11,500 enrolled in athletic accident benefit plan at Topeka, Kansas, (49) 691 sustained injuries requiring the services of physicians and dentists for treatment. Five-hundred-forty-five of these were from football or eighty per cent of the injuries. In a study by Hibbert (48) of high school football injuries during 1948-1949 covering four states and 47,00 athletes,

eighty per cent of th injuries were from football. At Northwestern University (53) in a two year period in 195land 1952, one-hundred-twenty injuries were seen. Here seventy players were engaged in 225 practice sessions and games which accounted for 15,750 player-days. In the Boston High School System from 1930 to 1939, excluding 1933, (1) 614 injuries occured among 7053 participants. In a report made by Gallagher (51 on athletic injuries among adolescents he reported 869 juries among 1082 participants. Five-hundred-sixty of these were from football. At Belmont, Massachusetts, (1) a group of former football players who had sons ranging in age from 8 to 14 years of age organized their sons, 46 in number, into a team and supervised their play throughout the season, during which thirteen games were played. Careful investigation showed that aside from a few minor contusions and occasional traumatic epistaxis no injuries occurred. All except the last report would indicate that football has a rather high incidence of injury. This makes a rather serious sounding situation and at first one wonders what sort of mayheim this game is. However, if one analyzes the type of injury other facts are made obvious.

Of 7,090 injuries occurring at Harvard University (50) in the years 1932 through 1952 Thorndike showed that only 15 per cent were serious injuries. He classified an injury as any injury causing an athlete to miss one practice session. He defined a serious injury as a fracture , dislocation or internal injury. The rest came unde the heading of minor injuries and were mainly sprains, strains, contusions, lacerations, abrasions and minor infections.

Of 545 injuries reported by Kramer (49) due to football, twenty-

six per cent were dental injuries. Forty per cent of the injuries were reported as dislocations and fractures. However, this is not as serious as it sounds for about one-fourth of these were fractures of the nose. Contusions and strains were not included under injuries in this study. No internal injuries were reported in this group.

In the Boston High School System (1) from 1930 to 1939 excluding 1933, among 7053 participants in football there were 614 injuries. Of these 170 were major injuries which included fractures, dislocations, concussions, and contusions of the kidney. Major injuries constituted twenty-eight per cent of the total number of injuries. Fracture was the most common major injury with the nose contributing about twenty-five per cent of the fractures. At Princeton (52, from 1933 to 1942 there were 928 injuries. About ten per cent were of the major type, either fractures, dislocations or internal injuries. The rest were of the minor type. At Phillips Academy, Andover, Massachusetts, (51) the incidence of major injury, which was defined here as any injury causing ten missed practices, was found about twenty-five per cent of the time. Hibbert (48) tabulating the football injuries over a four state area found serious injuries (fractures and dislocations) contributing about twenty-one per cent of all of the injuries.

From this it can be seen that serious injuries, defined usually as fractures, dislocations and internal injuries, have an incidence ranging from ten to forty per cent. It is noted that the incidence in high school seems to run a somewhat higher than in the colleges and in the younger groups. This is probably because both the younger and older groups got more attention. Also in high school an injury has to be reasonably severe before a doctor is seen, for the teams

usually do not have a doctor at the practice and the boy often has to see a doctor on his own. As in the case of Kramer's Study (49), only those injuries in which compensation was paid were records made, so necessarily the serious injuries were high.

In considering the serious injuries, what were the most prevalent types of serious injury? We will define a serious injury as one in which either a fracture, dislocation or internal injury resulted. In Kramer's study (49) fracture of the nose was the most common, followed closely by the ribs, less frequently the clavicle, hand and tibia or fibula. These four made up about eighty per cent of the fractures in this group. Also thirty dislocations were reported. They were distributed about most of the joints. A report by Hibbert (48) showed results similar to those of Kramer. He found the most frequent fracture to be of the clavicle followed by the leg, finger, forearm and nose. He found the most prevalent dislocation to be of the shoulder followed by the knee, elbow and finger. At Phillips Academy (51) whose program was for boys between thirteen and eighteen, the most common fracture was that of the fingers followed closely by the nose and the somewhat less frequently the metacarpels, foot, toes, and clavicle. In this series fractures of the humerous, radius, ulna, femur, tibia or fibula were rare. It would seem from these groups, which at best are only a limited sampling, that fracture of the large bones is not too common. The most common dislocation in this group was that of the humerous followed by the elbow and finger. Dislocation other than the shoulder were rare.

One wonders what is the outcome of these injuries. Thorndike (50), who is chief surgeon to the department of athletics of Harvard University and affiliated with their medical school, was of the

opinion that in a well organized college athletic program, where the coaching and medical supervision were well coordinated, there was little likelihood that the athlete could leave college with some disabling injury which was the result of the sport in which he participated.

Apparently in his experience of over twenty years he regarded contusions, lacerations, abrasions, inflammation and infections of such little import in creating permanent disability that he failed to even mention them. Sprains he concluded rarely ever resulted in a complication or disability. In regard to long-bone fractures he found that almost always they healed well and in good alignment. However, he did state that some of the fractures into joint surface or through growing epiphyses did result in disability. In his report he had three skull fractures on which he failed to comment on the outcome, one ruptured kidney which was treated promptly and successfully by surgical intervention; and two cases of ruptured spleen which were recognized and successfully operated upon.

In McPhee's (52) review of football injuries at Princeton University he had eight men or one per cent of the squad which were injured severely enough that they had to drop the sport permanently. Three were knee injuries, three were back injuries and two were head injuries. According to him, only three carried any residual and of these three only one could be directly attributed to football. The other two were questionable and were probably the result of another injury received somewhere else. Thirty-one cases, however, at one time or another did, cause discontinuence of football for the season. Twelve were sprained knees, sixteen were fractures, one was a contused kidney, another a sprained back, one was a severe laceration and contusion. All of these fully recovered.

In regard to the specific type of injuries, McPhee (52) thought that bruises or contusions usually cleared up without any trouble, but occasionally calcium was deposited in the hematoma and that this prolonged the healing. This occured seven times in his series. All but one subsided without difficulty and it had to be removed surgically. Eighty-six knees were sprained in this series. Surgical intervention was needed to restore stability in eight cases and one was amputated. In all only three were dropped permanently from the squad. The ankle was sprained sixty-seven times in this series; however, in no instance did it result in cessation of competition. All of the strain and fracture cases made complete recoveries. In sixty concussion cases all cleared without any residual. In twenty dislocations of which seven were of the shoulder only two required surgical intervention.

These reports from two colleges are not completely applicable to the junior high school student because of the difference in age. However, this is about all one can do for information. Rarely do any institutions other than universities have such extensive data on the injury and its outcome. High schools are attempting to give better medical coverage but as yet only the major or more troublesome injuries are seen and/or reported. A high school student rarely, if ever, reports a sprained finger or contusion. Reporting of an abrasion is almost never seen. With such a few junior high school programs and probably no more medical or statistical coverage than the high schools, and probably at times even less, fewer facts about junior high school injuries are known. The only data we have is from some of the boarding schools and usually this includes all of the boys in the school. Usually their ages range from twelve to eighteen years. Obviously we are not observing directly the group in which we are interested. We

can only infer from the older age groups what may happen to the younger age groups. For this reason I feel that we can only truthfully say we don't know what will happen to these children concerning injuries when they compete or participate in athletics in a junior high school program. Though the tudy by Hibbert (48) indicated that in the ages sixteen to eighteen the sixteen year old youth was more prone to injury, I don't believe that one could say without question that the incidence of injury for a child fifteen would be that much greater than the incidence of injury for a sixteen year old youth as compared to a seventeen year old. Or that the incidence of injury would increase proportionately to diminishing youth of a group of junior high school children. Hibbert (48) thought that age and experience usually parallel each other and that the first year of participation would probably have the greatest indidence of injury. As the child becomes more proficient and better coordinated he will be better able to take care of himself.

The fracture appears to be the major injury of the greatest incidence. The rest are rather rare. (48,49, 50,51,52) If one is justified in inferring from the older age groups that fracture would have the greatest incidence in a junior high school youth participating in athletics then it might be interesting to see if they are any more serious in children than in their elders.

Watson-Jones (54, states that a green stick fracture is often seen in children. This would indicate that their bones are more elastic than those of their elders. It may be that less force must be applied to a young bone to create a fracture than in an older individual though I found no evidence to support this and hence might have more fractures. However, one must take into

consideration that in sports young people so not move as fast, hit as hard or play as rough. They may hit or fall as hard as their elders, in proportion to their weight and size, but in actual units of force I don't believe that they do.

Once a fracture occurs the child heals more quickly than the adult. (54) This is even apparent in children which are four or five months old as compared to ones which are fifeteen years old. (54) There is probably some small difference between twelve years and twenty-one years though very small. (54) In fractures of long bones slight loss of apposition of fragments may be of no significance or much less in children than in adults. (54) In long bones the younger child the better the chance of remoulding and disappearance of any deformity. (54)

The epiphyseal separation is often the suject of conversation when speaking of junior high school athletics. Many of the articles written in opposition to teen age athletics mention the extreme seriousness of epiphyseal separations and their associated growth disturbances. The school systems of the country have so few junior high school athletic programs that little if any statistical evidence is presented to indicate the incidence of this fracture in children. For this reason I believe that the only way we may determine what the result would be is to run a series. However this is very difficult. Key and Conwell state that epiphyseal separations are most frequent in the lower end of the tibia, lower end of the radium and upper end of the humerous but nothing is said about the incidence. (55)

Due to the fact that all of the epiphyses have united by the twenty-fifth year and that most of them have united before the age of twenty, epiphyseal separation practically does occur after twenty years of age and are very rare after 15 years of age. (55) Most

cases occur between the ages of six and twelve at a time when the epiphyses are rather large and epiphyseal cartilage is relatively soft, this being the period of active growth. (55). Key and Conwell (55) are of the opinion that when epiphyseal separation does occur that growth disturbances are rare though these growth disturbances may result in very serious consequences and require operative treatment.

It would seem to me if we can infer that the statistics in the two surveys of high school athletics covering 60,000 participants (48,49) are applicable to junior high school student then the incidence of injury which might lead to permanent disability is very small. In the two series (48,49) the incidence of all injuries as compared to those participating ran about 15-20 per cent. The incidence of fracture of those injured was about twenty percent. Out of the total group this mounted to about three per cent of those participating. As was shown earlier about eighty per cent of all of the injuries were from football. If this were applicable to all cases then we would expect that the incidence of fracture in other sports to run around one-half of one percent. This would mean that in sports other than football, if these figure indicate a true trend, that fractures in sports other than football occur once per year for each two hundred athletes and that five out of every two-hundred football players a fracture will occur in a season. In about 2000 fractures in three groups (48, 49, 51) in which two groups were high school age and the third were of an age range of thirteen to eighteen the most common fractures (about two-thirds to three quarters) were of the nose, fingers and hand, clavicle, ribs and lower leg, tibia and/or fibula. It would seem that here that the only fractures of very great importance would be of the lower leg. These lower leg fractures had the

lowest incidence of all of the fractures of this group. All of this would indicate to me that the incidence of serious injury is extremely low. The incidence of internal injury was not reported in Kramer's and Hibbert's (48,49) series of 60,000 cases though it ran about four percent in Thorndikes series (50) in college. Dislocations in the 60,000 cases ran about four per cent of all of the injuries. This seems extremely low when compared with the total number participating. And as indicated by Burnett when he compared the incidence of injuries of sandlot football game in Boston to the injuries in the Boston High School System, the sandlot games ran much higher by evidence of the boys missing school and being seen at e the admitting room of a Boston hospital. He found that the number of injuries sustained in organized play contrasted sharply with the number of injuries received by the boys who played on the street, in vacant lots and backyards, in parks and other similar places. He found that these boys when injured were usually unable to receive medical care on the spot and as a result found aid at the nearest hospital. According to the records(1) the incidence rose sharply in early September until early December. It was customary to treat from twenty-five to fifty players in the accident ward of the Boston City Hospital for football injuries alone in this period. On Monday morning during this period an average of thirty-five to fifty players were treated in the outpatient department. Although a great many had no fractures, a number needed X-ray examinations to rule it out. The Headmaster of a Boston High School states (1) that he was constantly having absentees on Monday mornings or receiving requests from boys for permission to go to their physicians. Or more often, they wanted to go to a hospital to seek relief from injuries incurred

over the weekend particularly on Sundays. He added that these boys played in scrub amesand were not connected with any organized group.

A Boston coach (L) said, "We had no injuries during the season except for a few muscle bruises and according to the report of our truant officer, no member of the squad was absent from school on account of any injury received from football. We did, however, have seven fractures and dislocations from boys playing sandlot or backyard football. These boys, however, were not members of the high school squad." This indicates no statistical evidence of the injuries incurred in sandlot play but it does express the opinion that it is more common in such activities than in organized sport.

After reviewing these figures I would have to conclude that the incidence of injury in sandlot games is higher than that of organized athletics. The incidence of serious injury is extremely low in the organized sport programs reviewed and those injuries which resulted in permanent disability were very rare. From these figures it was appparent that the incidence of serious injury even in football was very low though four times as great as the rest of the sports. This is certainly no indication of what might happen if our junior high school children were allowed to participate in athletics but it gives us some medical basis for formulating an opinion as to the advisability of such activity.

Summary and Conclusions:

Evidence is presented that exercise, no matter how severe, causes no harm to the heart unimpaired by congenital defects or organic disease. Reports, though not in complete agreement, suggest that chronic exercise results in slight cardiac hypertrophy. Most sources believe that this is absolutely of no consequence and is a normal physiologic response which may aid the organism.

Observations of respiration, pulse and Blood pressure revealed that the exercising child did not respond much differently than the adult. Nor did the small deviations in the response to exercise reveal anything which could be considered significant. Some studies showed that the youngster who had not reached puberty was better able to tolerate strenuous exercise than the one who had passed puberty though endurance increased with age.

Studies reveal that the compensatory mechanisms of increased blood pressure, rapid pulse, increased rate and depth of respiration, etc. remained in action longer in youth just as they do in the untrained adult. In youth these compensatory mechanisms subside after exercise more quickly after training. However some changes were noted which could have been directly attributed to youth though they were of no consequence. The evidence presented suggested that all of the manifestations of exercise are compensatory in nature, thattheir duration was a function of the organisms ability to return to a homeostatic state and probably of no significance other than as a measure of the ability of the organism to return to homeostasis.

Review of the incidenceof injury in athletics revealed an incidence of about twenty percent of all of those participating.

About eighty per c nt of the total injuries were found to be the result of football. Most of the injuries in all sports were minor injuries. Major injuries which were defined as fractures, dislocations or internal injuries contributed a very small percentage of the total number of injuries. The fracture was the most common injury in this group. It was about eight times as frequent as the dislocation in the statistics reviewed. Very few ruptured spleens, contused kidneys and similar internal injuries were reported. Fractures were seen i about three per cent of all of those participating in two studies covering about 60,000 athletes. Complications with th se fractures were rare.

Some of the effects of exercise and athletics upon the pre-high school child have been considered. Some have been observed and other have been inferred. In my opinion I can see no contraindication for athletics in normal children from the aspects considered. It is true that a few would be injured. However some would be injured at the same period of time if they were not allowed to participat in athletics. I believe that if a normal child desires to participat in sports that no medical contraindication to unlimited participation exists."

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