

Sudden death in young athletes

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Athletes are regarded as the healthiest segment of the population by the medical and lay community and, as a result, the sudden and unexpected death of these individuals, although uncommon, raises profound attention and publicity. The lack of data on large series of sudden deaths in young athletes meant that the main causes of such events remained poorly defined. This is of particular relevance as sudden death among athletes is most prevalent in this age-group. Such data help in the identification of young athletes at risk of sudden death and would provide the rationale for pre-participation cardiovascular evaluation.

This study by Maron et al was one of the first to report on the causes of death in young competitive athletes. The series included 29 elite athletes between the ages of 13 and 30 years who engaged in a variety of sports and suffered sudden death and underwent postmortem examination. Sudden death occurred during or shortly after severe exertion on the athletic field in 22 of the 29 athletes. The athletes who constituted the study group were identified both prospectively from news media reports and retrospectively from review of two autopsy registries.

Structural heart disease was identified in 28 of the 29 (97%) athletes, and was almost certainly the cause of sudden death in 22 cases. Although the cause of sudden death in one patient without structural heart disease was unclear, a primary rhythm disturbance remained possible. A range of cardiac disorders was observed, but the most frequently encountered disorder was hypertrophic cardiomyopathy (HCM), accounting for almost half (14/29) of deaths in the series. The remaining disorders identified include anomalous origin of the left coronary artery (3 cases), atherosclerotic coronary disease (3 cases), ruptured aorta (2 cases), idiopathic concentric left ventricular hypertrophy (LVH) (5 cases), and hypoplastic coronary arteries (1 case). Cardiac disease was unrecognized during life in most of the athletes, with only two athletes being given the correct diagnosis antemortem.

The application of these data to the general athletic population is potentially problematic. The sample size is relatively small and unlikely to be a representative sampling of sudden death in athletes. Furthermore, due to the study design, unavoidable bias would influence the selection of athletes, as those with cardiac disease would be referred to the centers whose autopsy registry was reviewed.

The findings of this study raised important points regarding the evaluation of young athletes prior to participation in competitive sports. Evaluation using clinical history and examination would be insensitive for detecting the leading causes of sudden death and, even with addition of the ECG, could miss cases of HCM. The definitive diagnosis of HCM and most of the other conditions causing sudden death in athletes could only be made by echocardiography. This study provided insight into the conditions leading to sudden death in young athletes and laid the groundwork for subsequent larger studies.

1980

Jack Nicklaus wins his 5th Professional Golfers' Association (PGA) Championship;
Tatyana Kazankina of the USSR sets a new 1.5-km women's record in 3:52:47 min;
and Reinhold Messner of Italy becomes the first solo climber to scale mount Everest



An overview of randomized trials of rehabilitation with exercise after myocardial infarction

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Over one million individuals in the USA suffer myocardial infarction and over 60% survive the initial event. The beneficial effects of exercise in such individuals has been recognized for many decades and ambulation early after myocardial infarction and the implementation of exercise-based rehabilitation are widely practiced. Exercise training improves functional capacity and work efficiency, thereby decreasing the metabolic and circulatory demands of daily activities. Exercise also lowers heart rate and blood pressure, which are two major determinants of myocardial oxygen demand. These adaptations are accompanied by favorable neurohormonal and metabolic changes, such as reduction of norepinephrine and body weight, increased lean body mass, lower serum triglycerides, platelet adhesiveness, and increased high-density lipoproteins and fibrinolysis.

Prior to this overview, over 20 studies tried to define the benefits of exercise after myocardial infarction. Owing to their small size, however, they were unable to demonstrate a significant reduction in morbidity and mortality.

This overview analyzed results from over 4500 patients and included data from 22 studies with at least 1 year follow-up between 1960 and 1988. The majority of the subjects included were male and the analysis was heavily weighed towards men in the 5th and 6th decades of life. The subjects were randomized to exercise rehabilitation or the comparison groups. In 6 of the studies, the exercise rehabilitation group received only exercise programs, whereas, in the remaining studies, these individuals underwent risk factor modification in addition to exercise. The end points studied were total mortality, cardiovascular mortality, sudden death, and fatal and nonfatal myocardial infarction.

This overview demonstrated that cardiac rehabilitation programs including exercise lead to a statistically significant 20% reduction in total (odds ratio [OR] = 0.80 [0.66, 0.96]) and cardiovascular mortality (OR = 0.78 [0.63, 0.96]), which was apparent at 1 year following randomization and persisted throughout the follow-up period. It was interest-

ing to note that neither the 6 "exercise only," nor the remaining "exercise plus other interventions" studies attained traditional levels of significance. The OR for sudden death (0.63 [0.47, 0.97]) showed a large reduction, but did not reach statistically significant levels. The lack of availability of data regarding sudden death and the differing definitions of what constitutes a "sudden death" limit the interpretation of findings for this end point and more than likely shift the outcome towards null. There was a small nonsignificant increase in the risk of nonfatal myocardial reinfarctions (10.2% vs 9.5%, OR 1.09 [0.76, 1.57]). This may be due to chance or may represent either a true increase in nonfatal myocardial infarction or increased survival from myocardial infarction.

The relatively small number of "exercise only" studies, combined with the fact that they may have included formal or informal non-exercise measures, made definitive conclusions regarding the independent effects of exercise post-myocardial infarction difficult. As a result of this overview, however, other randomized large studies were carried out, which confirmed the benefits of exercise rehabilitation programs following myocardial infarction and the potential mechanisms of benefit involved.

1989

Roger Kingdom of the USA sets the 110-m hurdle record (12:92) in Zurich;
 Arturo Barrios of Mexico sets the 10-km record (27:08:23) in Berlin;
 and tests results reveal that 50 athletes tested positive for steroids during the 1988 Olympics

The athletic heart syndrome

T. P. Huston, J. C. Puffer, W. M. Rodney

N Engl J Med. 1985;313:24-32



athletic heart syndrome encompasses a constellation of cardiac findings seen in the highly trained athlete. These findings can be broadly divided into structural and electrocardiographic changes.

Chronic physical demand on the heart leading to adaptive responses can be either in the form of volume overload or pressure overload. Volume overload is encountered in isotonic activities such as endurance running, where sustained increases in cardiac output are required. In contrast, pressure overload is encountered in isometric activities such as weightlifting, where brief increases in cardiac output against huge aortic pressures are required. In practice, most athletes do not fit into a purely isotonic or isometric category, and represent a combination of the two, producing a combination of morphological adaptations.

These morphological changes may be evident soon after the commencement of training and regress with detraining. The increased left ventricular (LV) dimensions seen in isotonic athletes also lead to an increase in LV mass. The magnitude of these increases is, in most cases, modest and does not approach the extent seen in myocardial diseases. In some cases, however, the increases are of sufficient magnitude to raise the differential diagnosis of hypertrophic (HCM) or dilated (DCM) cardiomyopathy. However, certain echocardiographic features (eg, ratio of wall thickness-to-cavity size for HCM and the ejection fraction for DCM) can aid the distinction between athlete's heart and cardiomyopathy.

Most ECG abnormalities observed among athletes are a reflection of the vagotonic state and are commoner in athletes taking part in dynamic rather than static exercise. These include resting sinus bradycardia often associated with sinus arrhythmia, sinus pause, wandering atrial pacemakers, and low degrees of atrioventricular (AV) block (first-degree and Mobitz type I second-degree AV block). More advanced degrees of AV block, atrial tachyarrhythmias at rest, and atrial fibrillation (AF) are rare and cannot be assumed to be part of the physiological spectrum of athlete's heart.

Elevation of the J point associated with ST-segment elevation is common among athletes and is typically seen in the anterior precordial leads. It is associated with physical conditioning, normalizes with detraining, is considered benign and not representative of myocardial disease, and is often associated with tall T waves or T-wave inversion. The mechanism responsible for ST-segment elevation is thought to be a result of a decrease in resting sympathetic tone uncovering an inherent asymmetry of repolarization. Depression of the ST segment is very uncommon and when present is mild (-0.1 mV), with normalization on exertion. T waves are either tall and peaked or inverted with abnormalities most often in the lateral precordial leads. Tall peaked T waves are associated with ST-segment elevation, while T-wave inversion is mild and normalizes with exercise.

ECG voltage criteria for LVH are a common finding in athletes. Right ventricular hypertrophy is also evident on the ECG of athletes, but is less prevalent. Increases in voltage occur with conditioning and regress with cessation of training.

Knowledge of the athlete's heart enables the physician to reassure the athlete and avoid unnecessary further evaluation and possible inappropriate disqualification from competition.

1985

Germany's Boris Becker, aged only 17 years, beats Kevin Curren for Wimbledon tennis title; French cyclist Bernard Hinault wins his fifth Tour de France; and Britain's Steve Cram breaks both the 1.5-km (3:29:67 min) and 1-mile (3:46:31 min) world records



The incidence of primary cardiac arrest during vigorous exercise

D. S. Siscovick, N. S. Weiss, R. H. Fletcher, T. Lasky

N Engl J Med. 1984;311:874-877

Several epidemiological studies have suggested that regular habitual exercise is associated with decreased cardiovascular morbidity and mortality and a reduction in the risk of sudden cardiac death. Conversely, the relationship between vigorous exercise and sudden death has long been recognized and been a subject of debate. Overall, however, the net effect of habitual vigorous exercise is believed to be favorable.

Previous studies have not looked at the risks of sudden death with vigorous exercise along with the benefits of habitual exercise training in the same population, making explicit conclusions regarding habitual activity difficult.

This community-based study was carried out to determine whether the risk of sudden death is increased during vigorous exercise and the extent to which it detracts from the potential benefit of habitual vigorous exercise. By assessing the two components of the effect of vigorous exercise in the same population it was possible to put the risks and benefits into perspective.

One hundred and thirty-three sudden cardiac deaths in males between the ages of 25 and 75 years with no history of cardiac disease formed the study cohort. The subjects' wives were interviewed to ascertain the pattern of leisure-time activity over the previous year, the circumstances surrounding the sudden deaths, and the presence of potential risk factors for sudden death.

These findings suggest that the risk of sudden cardiac death is transiently increased during vigorous exercise. The increase in risk was particularly large for men with low levels of habitual activity. Among men who engaged in low levels of habitual activity, the risk during exercise was particularly large as compared with the risk at other times (relative risk [RR] 56). In contrast, among men with the highest level of habitual activity, the risk during exercise was increased to a much lesser extent (RR 5).

The study supports the clinical impression that unusual exercise may be associated with greater risk and, even among men who are habitually active, the risk of sudden death increases with exercise. Furthermore, the data also support the view that habitual participation in exercise is associated with an overall reduction in the risk of sudden cardiac death.

1984

French new wave film director François Truffaut dies of cancer, aged 52 years;
Steffi Graf plays her first professional tennis match;
and Steve Jones of Britain runs the Chicago Marathon in a world record time of 2:08:05 h

Triggering of acute myocardial infarction by heavy physical exertion. Protection against triggering by regular exertion. Determinants of Myocardial Infarction Onset Study Investigators

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The American Heart Association (AHA) recommends increasing physical activity as an important means of reducing the risk of myocardial infarction (MI). A sedentary lifestyle has consistently been shown to increase the risk of coronary artery disease. However, it is well recognized that heavy physical exertion sometimes leads to MI. Studies have showed that in about 5% of patients with a heart attack such physical activity preceded symptoms. Heavy physical exertion, therefore, appears to be a double-edged sword, both triggering and preventing MI.

This multicenter, interview-based study used a case-cross-over design to quantify the relative risk of MI after heavy exertion as compared with periods of lighter exertion or no exertion, and its potential modification by habitual physical activity in 1228 patients with confirmed acute MI. In the interview, data were obtained on the timing of the MI, the estimated usual frequency of physical exertion during the previous year, and the intensity and timing of heavy exercise and other potential triggering factors in the 26 hours prior to the onset of the MI (hazard period). The degree of physical exertion was quantified on a scale from 1 to 8 metabolic equivalents (METs). Patients were considered to have engaged in heavy exertion if they reported a peak exertion level estimated to be 6 METs or more during the period of interest.

Heavy physical exertion was associated with a transient risk of MI in the subsequent hour that was 5.9 times higher than the risk during periods of lighter or no exertion. The relative risk varied greatly depending on the usual frequency of heavy exercise carried out by the patient. It was only 2.4 among those reporting regular physical exertion, but 107 among those who were habitually sedentary. Recall bias is unlikely to confound these results, as the patients were unaware of the 1-hour hazard period and because the case-crossover design eliminated potential confounding factors that differed among patients.

The findings of this study generally agreed with other studies, which quoted the relative risk of sudden death from

cardiac causes to be between 5 and 100 during periods of heavy exertion. Furthermore, other studies have also shown that the risk of sudden cardiac death during heavy exertion is decreased with habitual exercise.

This study demonstrates protection against the triggering of MI with regular exertion and provides further evidence encouraging regular physical activity, as recommended by the AHA. Such a program most likely lowers the overall risk of MI, since it may lower the baseline risk, and also decrease the relative risk that an episode of heavy physical exertion will trigger an MI.

The mechanism involved in the triggering of MI is thought to be the disruption of a vulnerable coronary plaque in response to hemodynamic stresses associated with strenuous exercise. Thereafter, hemostatic and vasoconstrictive forces determine whether the resultant thrombus becomes occlusive. The protective effect of regular exercise, as suggested in this study, was postulated to be due to a reduction in number and the stabilization of the coronary plaque and this has been supported by more recent studies. Further studies in this area may lead to further clarification of some of the uncertainties regarding the beneficial effects of physical exertion and lead to new forms of prevention.

1993

Danny Blanchflower, the North Ireland
soccer legend, dies at 67;

Germany beats Australia in Düsseldorf
(4-1) to win the 82nd Davis Cup;
and Eduardo Frei is elected President of Chile



The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes

A. Pelliccia, B. J. Maron, A. Spataro, M. A. Proschan, P. Spirito

N Engl J Med. 1991;324:295-301

Long-term regular physical training leads to structural adaptations, eg, increased left ventricular (LV) wall thickness, LV end-diastolic cavity diameter, and LV mass, characterizing the “athlete’s heart.” The increase in LV wall thickness is generally modest, but, in rare cases, the increase is significant and raises the differential diagnosis of hypertrophic cardiomyopathy (HCM). This is crucial as HCM is the commonest cause of sudden death in young athletes. The distinction between physiological athlete’s heart and HCM is dependent largely on the assessment of whether the magnitude of LV hypertrophy (LVH) is in excess of that expected in response to athletic training alone. Furthermore, although this distinction can usually be made on the basis of either the ECG or echocardiogram, the ECG changes in some cases of HCM can be equivocal or nondiagnostic and therefore require echocardiographic differentiation. Prior to this study, however, the upper limits of physiological hypertrophy remained unknown due to the fact that previous echocardiographic studies had focused on small groups of athletes.

This landmark study defined the upper limits of LVH as a result of athletic training by assessing 947 elite athletes free of cardiovascular disease during intensive training. The athletes had a mean age of 22 years (range 13-49 years) and represented a wide range of disciplines including both predominantly isotonic and isometric activities.

The cardiac dimensions of the athletes were characteristic of highly trained individuals. The LV cavity size ranged from 40 to 66 mm and exceeded the upper limit of normal (54 mm) in 38% of the study population, with a small, but significant, proportion (4%) exceeding 60 mm. The interventricular septum thickness ranged from 6 to 16 mm and the posterior wall thickness ranged from 6 to 13 mm.

Only 16 athletes (1.7%) had an LV wall thickness compatible with HCM (≥ 13 mm). All were male and either rowers, canoeists, or cyclists. No female athlete had an LV wall thickness >11 mm. All 16 male athletes had an enlarged LV cavity (diameter >54 mm), and normal systolic function and left atrial size.

There was an independent significant association between wall thickness and age, gender, body size, and type of sport (rowing, canoeing, and cycling). Diastolic function was normal in all athletes with LVH, in contrast to HCM, where there is generally a degree of diastolic abnormality. The ECG was normal in 9 of 16 athletes with LV wall thickness of ≥ 13 mm. The other 7 had minor abnormalities, such as voltage criteria for LVH, mild T-wave inversion, and first-degree heart block. No athlete had ECG changes typically seen in HCM such as deep T-wave inversion, pathological q waves, ST-segment depression, and marked left axis deviation.

This study provided important insight into the differentiation of athlete’s heart from HCM. As no athlete had an LV wall thickness >16 mm, it can be deduced that LVH >16 mm very likely represents pathological LVH such as HCM. Furthermore, an LV wall thickness ≥ 13 mm was very uncommon and was seen in the context an enlarged LV and only in certain sports. This finding itself may be enough to distinguish athlete’s heart from HCM, since most HCM patients have a normal or small LV cavity. No female athlete had an LV wall thickness >11 mm, and this suggests that athletic training virtually never leads to LVH compatible with a diagnosis of HCM. Finally, as the athletes in this study were almost entirely white, caution should be exercised in the evaluation of athletes of different ethnicity based on these findings.

1991

Fu Mingxia of China, aged only 12, wins a World Swimming Championships gold medal; the New York Giants win SuperBowl XXV, defeating the Buffalo Bills 20-19; and Monica Seles beats Jana Novotna (5:7 6:3 6:1) to win the Australian Women’s Tennis championship

Screening for hypertrophic cardiomyopathy in young athletes

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N Engl J Med. 1998;339:364-369

Most sudden deaths (SD) in athletes are due to cardiovascular disease, with nearly half of SDs in young athletes being due to hypertrophic cardiomyopathy (HCM). This paper reports on the findings of a pre-participation program, which prospectively evaluated over 33 000 athletes in the Veneto region of Italy between 1979 and 1996, on the basis of clinical and family history, 12-lead ECG, limited exercise testing, and additional tests in case of positive findings at initial evaluation.

A total of 269 SDs were reported in people below the age of 35 years: 49 were athletes (1.6 per 100 000/year) and 220 were nonathletes (0.75 per 100 000/year), giving a relative risk of SD in athletes vs nonathletes of 2.1 ($P>0.001$).

In 40 of the 49 athletes, SD occurred either during (35 cases) or immediately after (5 cases) sporting activity, 14 athletes had previously reported palpitations, syncope, or both; 16 had had ECG or rhythm/conduction abnormalities. The most common cause of SD in the athletes was arrhythmogenic right ventricular cardiomyopathy (ARVC) (11 cases; 22.4%), atherosclerotic coronary artery disease (CAD) (9 cases; 18.4%), and anomalous origin of a coronary artery (6 cases; 12.2%). ARVC ($P=0.008$) and anomalous origin of a coronary artery ($P<0.001$) were associated with SD significantly more often among athletes than nonathletes. HCM caused only 1 SD (2%) among athletes, vs 16 SDs in nonathletes (7.3%). None of the nonathletes who died suddenly from HCM had been screened prior to death.

During pre-participation screening, the most frequent cardiovascular conditions leading to disqualification were rhythm and conduction abnormalities (38.3%), hypertension (27.1%), and valve disease (21.4%). Of the 33 735 athletes screened, 3016 (9%) were referred for echocardiography, and HCM was identified in 22 young athletes (0.07%). During a mean follow-up of 8.2 ± 5 years, none of the 22 athletes disqualified due to HCM died.

In this study, SD in young athletes was related to the expected underlying cardiac disorders, but the prevalence of

each disorder differed substantially from previous studies. Prior studies, mainly from the USA, have consistently found HCM to be the leading cause of SD in young athletes (40% to 50% of cases). In this Italian study, HCM caused only 1 SD among the athletes, but caused SD in young nonathletes with a similar frequency to reports from the USA. Furthermore, a high prevalence of ARVC and premature CAD was noted in both groups. The low prevalence of HCM among young athletes suffering SD was most likely the result of the long-standing pre-participation screening in practice in Italy. This is supported by the similar prevalence of HCM among the nonathletes in this study and studies from USA.

Of the young athletes screened, altogether 22 (0.07%) were identified with HCM and disqualified. Other studies suggest a prevalence of 0.2% for HCM in the general population. The prevalence of 0.07% is reasonably similar, seeing that the screening was based mainly on ECG, while US studies are based on echocardiography. Furthermore, using clinical evaluation and ECG made it possible to target echocardiography to only 10% of the screened population, resulting in considerable cost saving. This adds strength to the argument that a screening program based largely on ECG is effective at picking up HCM among young athletes.

1998

An IRA bomb explodes in the town of Omagh, killing 27 people; South Africa defeats the Australian Wallabies (16-12) to win the rugby tri-nations championship; and Finnish F1 racing driver Mika Häkkinen wins the German Grand Prix at Hockenheim on his way to securing the 1998 drivers' championship



Investigation of the physiological basis for increased exercise threshold for angina pectoris after physical conditioning

D. N. Sim, W. A. Neill

J Clin Invest. 1974;54:763-770

In patients with coronary artery disease, physical conditioning can increase the exercise threshold for angina. Physical conditioning results in alterations of exercise hemodynamics reflected in a lower heart rate and systolic blood pressure at any given workload. The decrease in these determinants of myocardial oxygen consumption after conditioning suggests that the increased tolerance for exertion in patients with a limited myocardial oxygen supply might be due to a lower myocardial oxygen requirement at a given level of exertion. The other possibility is that of increased myocardial oxygen supply. This study investigated the physiological basis for the increased exercise threshold for angina and looked to define the effect of conditioning on myocardial oxygen supply.

Eight patients (all men) with angiographically confirmed coronary artery disease, normal left ventricular function, and exertional angina completed 11 to 15 weeks of endurance exercise conditioning. Angina threshold was determined using upright bicycle exercise and atrial pacing. Supine resting measurements of brachial artery and left ventricular pressure, paired arterial and coronary venous blood sampling for oxygen, pH and lactate analyses, and coronary blood flow were made and the measurements were repeated at subangina and angina threshold following pacing.

Resting heart rate and the heart rate \times systolic blood pressure product (RPP) at the same level of work were lower after conditioning ($P < 0.02$). The exercise angina threshold, as determined by bicycle exercise testing, was higher after conditioning in all the patients as reflected by the work level reached ($P < 0.05$) and the duration of exercise ($P < 0.005$). Similarly, the systolic blood pressure and RPP at the onset of angina were higher after conditioning. The angina threshold determined by atrial pacing, however, was not increased by exercise conditioning, with the heart rate, systolic brachial and left ventricular blood pressure, and RPP virtually the same before and after conditioning. Various indirect indices of myocardial oxygen consumption demonstrated a rise with bicycle exercise after conditioning, but remained unchanged with pacing after conditioning.

The effects of exercise conditioning on measures of myocardial oxygen supply were not significant. Atrial pacing did not significantly increase coronary blood flow or myocardial oxygen consumption. There was no change in coronary arteriovenous oxygen gradient after conditioning, in contrast to other previous studies.

The increase in exertional threshold angina, and the decrease in resting heart rate and in heart rate at the same levels of workload indicate that the patients did experience a conditioning effect. The data suggest that exercise conditioning did not change myocardial oxygen supply, at least during angina induced by atrial pacing. However, the data also point to a difference between exercise and pacing-induced tachycardia. Indirect indices of myocardial oxygen consumption at the angina threshold after conditioning were higher for exercise, but not for pacing. This suggests that exercise conditioning exerts some effect pertaining specifically to exercise and does not carry over to a different stress such as pacing-induced tachycardia. The study did indicate that the increase in exercise capacity of angina patients after exercise conditioning appeared to be due to a functional adaptation in either delivery or utilization of oxygen by the myocardium, rather than an alteration of the coronary arteries, as confirmed by coronary angiography. This study was unable to ascertain the mechanisms involved, as critical measurements during exercise would need to be obtained directly for valid comparisons.

1974

Jimmy Connors and Billie Jean King win the
US Open single tennis crowns;
US President Gerald Ford pardons former
President Richard Nixon of all federal crimes;
and a military coup in Ethiopia overthrows
Emperor Haile Selassie

Physical activity and the incidence of coronary heart disease

K. E. Powell, P. D. Thompson, C. J. Caspersen, J. S. Kendrick

Annu Rev Public Health. 1987;8:253-287

Nowadays, the beneficial effects of regular exercise on cardiovascular health are well established, with physicians prescribing regular exercise, as well as statins, aspirin, and angiotensin-converting enzyme (ACE) inhibitors, to individuals deemed to be at risk of coronary heart disease (CHD). Exercise rehabilitation programs form a routine part of secondary prevention for CHD. The primary preventative role of exercise in CHD had been the subject of many studies in the second half of the twentieth century. The general message from most was a lower risk of CHD in physically active individuals. This review formed a systematic analysis of all these studies and provided an assessment of the quality of each one.

Forty-three studies met the selection criteria where the incidence of cases could be separated from prevalent cases and where it was possible to estimate incidence rates, relative risk, odds ratios, mortality ratios, or where a regression analysis had been done. The particular area of interest was data comparing the risk of CHD between inactive and active persons. In terms of design, 36 were prospective cohort studies, 3 were retrospective mortality studies, and 4 were observational case-control studies. The majority of the studies provided information primarily about North American and European working-age men.

The existing literature reviewed supports the notion that there is an inverse association between physical activity and CHD. No study reported a significant direct association between physical activity and the incidence of CHD. Approximately two thirds of the studies reported either a significant association or a graded response or both. The repeated observation in different settings and populations is evidence that physical inactivity is a fundamental cause of CHD. Interestingly, methodologically superior studies were more likely to report an inverse association, suggesting that less carefully conducted studies have obscured the association. The relative risk of CHD associated with inactivity was about 2.0, with better studies tending to report higher relative risks. Furthermore, even well-accepted risk factors such as hypertension, hypercholesterolemia, and

smoking had relative risks only marginally greater than inactivity. Most studies adequately demonstrated that the activity level predated the onset of CHD, therefore implying a cause-and-effect relationship.

Potential confounding factors such as age, sex, smoking status, serum cholesterol, and blood pressure were considered by many of the studies, and the inverse association between inactivity and CHD was just as likely to be present in these studies as those that made no adjustments. This suggests that physical activity exerts a protective effect on CHD that is independent of these other risk factors.

The inverse association between physical activity and CHD incidence is consistently observed, appropriately sequenced, biologically graded, and plausible. The risk of inactivity seems to be similar in magnitude to that of conventional CHD risk factors. Given the increasing sedentary lifestyle of the Western world, these findings have important public health implications and should encourage regular physical activity in the population at risk.

1987

Kapil Dev takes his 300th Test wicket. At the age of 28, he is the youngest bowler to achieve this milestone; Edwin Moses, the US 400-m hurdler, is defeated by Danny Harris, bringing to an end his 122-race winning streak stretching back over ten years; and Ajax wins the 27th European Football Cup



Exertion and acute coronary artery injury

A. Black, M. M. Black, G. Gensini

Angiology. 1975;26:759-783

Our understanding of the pathogenesis of the acute coronary syndromes has progressed significantly over the last three decades. The respective roles played by the atherosclerotic plaque, the inflammatory cascade, and the coagulation systems are better defined. It is also widely accepted that regular exercise is beneficial to cardiovascular health. However, it is also well known that unusual or strenuous physical exertion or emotional stress is associated with acute myocardial infarction and sudden death.

The authors of this study, more than 25 years ago, supported the hypothesis that exertion can be a very definite precipitating factor of acute coronary occlusion and that the relationship of exertion to acute coronary insult, myocardial infarction, and sudden death is very much significant. They went on to present 12 clinical cases and three angiographic cases where acute coronary artery injury occurred during or immediately after strenuous exertion and/or severe emotional stress. In all the cases, the clinical picture, including symptoms and ECG findings, correlated with pathological findings consisting of coronary artery occlusion on a background of a "vulnerable" plaque.

The mechanism suggested involves the initial "cracking" of the plaque, which can put the coronary artery into spasm. If spasm is severe and prolonged enough, it can lead to myocardial infarction and even ventricular fibrillation and cardiac arrest, and if short-lived results in angina. The initial injury is of such nature that at some later interval further injury in the nature of extension of the crack or fissure occurs and produces an acute occlusion. This interval is labeled as the preocclusion latent period and can be as short as a few seconds or as long as weeks or months, supporting the idea that the preocclusion state is not related to gradual vessel occlusion by thrombosis, but rather that of a cracked plaque that finally elevates or ruptures.

Coronary arteries are unique in that they are subjected to regular vigorous movements even at resting heart rates. Over 80% of coronary artery occlusions are found within the epicardial course of the vessel and not in branches within

the myocardium. The movements of the coronary arteries include pulsation, shortening and lengthening, twisting, snapping (acute bending), and flow currents. These movements are substantially increased with physical exercise or any adrenergic drive requiring an increased cardiac output and can lead to the fracturing of a vulnerable plaque. The pathological changes within the plaque will vary depending on whether the plaque simply heals and becomes stable, is complicated by acute inflammatory processes including edema and hemorrhage, or becomes vulnerable or fragments and ruptures with discharge of its contents downstream.

The authors conclude that the above concept of the "crack in the plaque" accounts for the sudden appearance of clinical coronary artery disease during or shortly after physical or emotional exertion. It could also account for the exacerbation of symptoms or sudden death occurring in individuals with asymptomatic coronary artery disease. Our current improved understanding of the pathogenesis of acute coronary syndromes supports the authors' conclusions.

1975

US golfer Tiger Woods is born on
30th December 1975 in Cypress, California;
Raul Ramirez wins the Davis Cup for Mexico
by defeating Jimmy Connors in the final match;
and the Indonesian army occupies East Timor