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THE POTENTIAL FOR CARDIAC SCREENING OF YOUNG ENDURANCE ATHLETES

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ABSTRACT

The practical value of a mass, "preventive" laboratory screening of athletes is reviewed briefly from a personal perspective. The total number of exercise-induced deaths in young athletes is extremely small, and in consequence difficult to prevent. Furthermore, the relationship of cardiovascular catastrophe to the commonly suggested principal cause in young adults (ventricular hypertrophy in general or cardiomyopathy in particular) is far from established. There is a very rare inherited myocardial dystrophy that predisposes to sudden death, but this is not peculiar to athletes. Indeed, those affected by this congenital disorder usually have a low physical work capacity, and are unlikely to become involved in endurance sport. As would be anticipated from Bayes theorem, in the absence of definite symptoms, the mass pre-participatory laboratory screening of endurance athletes leads to an unacceptable toll of false positive diagnoses. Detailed laboratory examinations are warranted only if there is a strong family history of early cardiac death or other significant findings in the clinical history and physical examination. The hypertrophied heart of the athlete is usually a beneficial adaptation to endurance training. An above average ventricular mass increases not only overall survival but also quality-adjusted life expectancy; it enhances the individual's physical work capacity and reduces the likelihood of dependency in old age. Certainly, isolated echocardiographic findings of an above average ventricular size should not become the grounds for prohibition of endurance sport.

Key words: sudden death, cardiomyopathy, endurance athletes, prevention, pres-screening

Sports cardiologists have for long been concerned about occasional incidents of sudden exercise-related deaths in endurance athletes such as distance cyclists, soccer players and orienteers (31, 91). Although such events are rare (probably less than 1 per 200,000 athlete-years), they often attract great publicity, because the individual is well-known and may be performing in front of a large crowd (46, 48). There has been a corresponding interest in potential methods of detecting individual competitors who might be particularly vulnerable to a cardiac catastrophe; sometimes it is also inferred that the condition should have been detected at pre-participation examination (2, 9, 96, 118).

Nevertheless, many authors have concluded that present screening techniques are largely ineffective in avoiding such incidents (13). Thus, a recent retrospective survey of 134 exercise-related fatalities from the United states concluded that only 3% of individuals who had been exposed to standard preparticipation screening were suspected of having cardiac disease, and in only 1% was the diagnosis accurate (57). Italian medical practice is in a relatively

unique position, in that since 1971, federal legislation (the "Medical Protection of Athletic Activities Act) has required that all citizens aged 12-40 years who participate in organized sport undergo an annual preventive medical evaluation; the examining physician must certify that the athlete is free of cardiovascular abnormalities that could increase the risk of sudden death unacceptably during either training or competition (72). The minimum requirements of such an examination since 1982 have included a history, physical examination, a 12-lead resting ECG and exercise and pulmonary function tests. Since 1994, there has also been a requirement for echocardiography in certain professional sports (soccer, boxing and cycling).

The purpose of the present editorial is to review the etiology of exercise-induced sudden death, and resulting implications for preventive medical practice.

Etiology of sudden, exercise-related death

Potential causes

The etiology of sudden, exercise-related death has previously been reviewed (2, 6, 21, 22, 93, 105, 118). A few incidents are due to blunt mechanical injury of the chest (55) or heat stress (90, 95). Occasionally, there is rupture of a congenital aneurysm in the Circle of Willis, or splenic rupture associated with sickle-cell disease (32). The great majority of cases reflect some underlying cardiac abnormality that causes either an arrhythmia or cardiac arrest (4, 16, 42, 104, 106, 109), but the nature of the underlying pathology is very varied, depending in part on the gender and age of the individual. Only about 10% of episodes occur in women (49, 73), reflecting lower sport participation rates, possibly less severe training demands, and a later onset of coronary vascular disease. About a half of all episodes occur in those over the age of 35 years (31, 103, 109), and in this age group, coronary atherosclerosis is almost always responsible for triggering a cardiac arrest or arrhythmia (112).

In those individuals who are under the age of 35 years, coronary artery disease still precipitates perhaps 10% of incidents, but other important causes of cardiac cattastrophe identified in one review included hypertrophic cardiomyopathy (48%), idiopathic left ventricular hypertrophy (18%), coronary artery anomalies (14%) and ruptured aorta secondary to aortic stenosis (7%) (39).

Sometimes, there is evidence of a fatal myocardial ischaemia; in young competitors, the usual cause is a previously undetected congenital anomaly such as an abnormal origin of the coronary vessels (5, 17, 30, 62, 82). Some authors have maintained that viral myocarditis is rarely a precipitant of sudden death, but others have found a history of fever (10) and/or myocarditis (14, 58, 115, 120) in 8-12% of cardiac catastrophes; a series of incidents in Scandinavian orienteers were thought to arise from a combination of infection and overtraining (27, 35). A number of recent catastrophes in long-distance cyclists have been attributed to a high red cell count, induced by doping with erythropoetin; conceivably, prolonged training at high altitude could have a similar effect. The acute or chronic administration of stimulant or narcotic drugs, particularly cocaine, has been suspected in a few incidents (23, 28, 36, 110), although this has remained difficult to prove. Other possible causes include Marfan's syndrome (15), mitral valve prolapse (29, 85), congenital abnormalities of ventricular repolarization such as a long QT syndrome, sarcoidosis, and possibly intramural (tunelled) coronary vessels.

Role of hypertrophic cardiomyopathy

In instances where no other obvious pathology can be found, blame tends to be

attributed to a myocardial ischaemia or arerhythmia, secondary to a ventricular hypertrophy that is pathological in type or extent. However, there is little relationship between ventricular hypertrophy and sudden death in the absence of an inherited dystrophy with disarray of the myocardial fibers (59). In endurance competitors, the problem thus becomes one of distinguishing between physiological hypertrophy and a congenital, dystrophic hypertrophy.

From the earliest days of sports medicine, clinicians were aware that endurance athletes such as rowers, cyclists and distance runners had unusually large hearts (70). Many physicians regarded such hypertrophy as a healthy response to prolonged exercise, but others drew a parallel with the large hearts observed in various cardiac pathologies. It was suggested that the large heart "indicates undue strain or the danger of eventual degeneration" (34), and in some instances training was restrained or even forbidden. Awareness of cardiac hypertrophy among endurance competitors increased as P-A chest radiographs began to supplement such simple clinical indices of cardiac size as thoracic percussion and detection of a prominent, displaced apical impulse or a right ventricular lift. Sports physicians quickly found that the cardiothoracic ratios of many athletes exceeded the supposed normal ceiling of 0.50 (34). The addition of lateral radiographs allowed observers to estimate the external volume at 700 ml (10-11 ml per kg of body mass) in sedentary young men, but 1000-1100 ml (14-15 $ml \bullet kg^{-1}$) in endurance athletes (79); however, it remained unclear whether the larger heart was a healthy adaptation or evidence of pathological strain.

As the quality of electrocardiograms improved, these, also, were interpreted quantitatively; left ventricular hypertrophy was diagnosed if the sum of the S wave in lead V_1 and the R wave in lead V_5 exceeded 35 mm (3.5 mV). Likewise, right ventricular hypertrophy was diagnosed if the sum of the R wave in lead V_1 and the S wave in lead V_5 exceeded 10.5 mm (1.05 mV) (98, 99). However, correlations of the radiographic and electrocardiographic findings with ultimate post-mortem evaluation remained poor. This is perhaps not surprising, since hypertrophy of the chest muscles often attenuated ECG voltages in athletes (71), and radiographs measured only the external dimensions of the heart. One might anticipate a distinction between the firm, rounded radiographic contours of a heart with physiological hypertrophy, the sagging, distended form of a failing myocardium, and the irregular contours of a heart with what is generally an asymmetric pathological hypertrophic cardiomyopathy (37). But in practice the ventricles and atria make varying contributions to the overall cardiac silhouette, depending on the axis of the heart.

Diagnostic contributions of echocardiographic and magnetic resonance imaging

The advent of wide-angle two-dimensional echocardiography (50, 66) and magnetic resonance imaging (13, 63) increased interest in cardiac enlargement as a possible and diagnosable cause of sudden, exercise-related death (3, 68, 86). Nevertheless, over-enthusiastic use of the echocardiogram led to further unwarranted diagnosis of supposed abnormalities, and critics termed hypertrophic myocardiopathy an "ultrasound-specific" disease (84).

The new techniques allowed the cardiologist to measure the thicknesses of the ventricular walls and interventricular septum, as well as the internal dimensions of the ventricular chambers. Important differences were noted between endurance and resistance athletes; in the former, thick ventricular walls seemed an adaptation to vo-

lume loading, but in the latter thickening of the free walls and interventricular septum was not accompanied by any increase in left-ventricular end-diastolic diameter (19, 66, 97, 107). In principle, the distinction between physiological and pathological hypertrophy "depends largely on the judgment whether the magnitude of the left ventricular wall thickness exceeds that expected as a result of athletic training alone" (74). But in practice, the diagnostic potential of echocardiography has not always been realized, because the limits of resolution, typically 2 mm for an individual dimension (75) are similar to the differences which distinguish the athletic from the pathologic heart. Furthermore, the choice of dividing line between normal and abnormal remains contentious. The originally specified ceiling for physiological hypertrophy was a posterior wall diastolic thickness of 11 mm, but it was quickly discovered that 60 percent of endurance athletes exceeded this limit. Pellicia et al. (74) opted for an upper limit of 13 mm; however, 7 percent of rowers, canoeists and cyclists in their series had a wall thickness greater than 13 mm, and it is hard to believe that all of these individuals had a pathological enlargement of the heart. In fact, all of the individuals thus identified had enlarged left ventricular end-diastolic dimensions, in the range 55 to 63 mm, a useful point of contrast with pathological hypertrophy (where the ventricular cavity is usually either normal or smaller than normal). Other investigators have found it necessary to choose still higher normal limits for wall thickness: 16 mm (89, 101, 117), 18 mm (108) or even 19 mm (78). The overall size of the competitor further complicates diagnosis. Large competitors have a substantial advantage in endurance sports like rowing, and such individuals necessarily have large hearts with thick ventricular walls (43, 97, 108). Plainly, it is difficult to take seriously limits of ventricular size that have varied so widely in the space of four years. The most recently proposed ceilings leave little or no margin to distinguish physiological hypertrophy from the 17-18 mm wall thickness typical of pathological pressure (aortic stenosis, hypertension) or volume (aortic or mitral regurgitation) overload (19). Echocardiography enthusiasts argue that current standards offer a small margin to distinguish some cases of hypertrophic cardiomyopathy; this abnormality is commonly associated with a ventricular thickness of 20 mm or more, although even such values fall within the limits of resolution of an 18 or 19 mm normal reading. Moreover, some patients with hypertrophic cardiomyopathy have readings as low as 13-15 mm (54).

Endurance competitors also show another criterion that has been advanced for the diagnosis of pathological hypertrophy, a thickening of the interventricular septum (24). Some cardiologists have diagnosed a pathological cardiac enlargement if the ratio of inter-ventricular septum to left posterior ventricular wall thickness exceeds 1.3: 1 (26). However, ratios can rise as high as 2.0 in athletes (61, 83, 89), and such ratios do not, in themselves, appear to be either dangerous or pathologic (26). Other cardiologists have argued that the ratio of septal thickness to left-ventricular end-systolic or end-diastolic diameter is the most useful diagnostic feature of the echocardiogram. The upper limit of normality for the septal thickness/end-systolic diameter ratio has been set somewhat arbitrarily at 0.48 (3 SD above normal values (26)); this choice is debatable, given that some cardiorespiratory characteristics of international endurance competitors are four or more SD above the population average.

One possible method of distinguishing between physiological and pathological hypertrophy may be to collect data over

several weeks or months of reduced training. Once training ceases, the heart of the endurance competitor regresses towards "normal" sedentary values over the course of several months (11, 53, 69), although athletes may not be prepared to tolerate such a long remission of training. A purely pathological enlargement of the heart is likely to remain unchanged in response to such a tactic, although it is less clear what will happen in a person with hypertrophic cardiomypathy who has previously been engaged in rigorous training.

Perhaps the most important method of differentiating a physiological from a pathological enlargement of the heart remains functional. If the cardiac enlargement is pathological, the cardiac ejection fraction is poor and the patient shows a limited effort tolerance (107), but if the hypertrophy is a physiological response to endurance training, both ejection fraction and peak power output are large. Other features of a pathological enlargement include abnormalities of conduction and cardiac rhythm (51, 77), mitral regurgitation (89), limited ventricular compliance and poor diastolic filling (9) and hypotension (53, 59).

Prevalence of hypertrophic cardiomyopathy in endurance athletes

It is frequently stated that hypertrophic cardiomyopathy is the "commonest cause of death" in young athletes (2, 3, 56, 68, 100, 108), causing from 35% (57) to 48% (39) of all incidents in young adults. However, such statements must be viewed in the perspective of the minuscule number of exercise-related deaths: around a hundred incidents per year in young US athletes (118), and one death per 11 million hours of physical activity in Finnish men aged 20-39 years (111).

A retrospective study of 215,413 marathon runners disclosed four exercise-related deaths; three of the four fatalities were in runners aged 32-58 years, and all of these had evidence of coronary atherosclerotic disease. There was only one young runner in this series, aged 19 years, and in his case post-mortem examination disclosed an anomalous coronary artery (57). Likewise, a computerized search of the US literature (6) covering a fifty-year period revealed only four incidents where an exercise-related death was thought due to hypertrophic cardiomyopathy. A ten-year retrospective study accumulated reports on 158 sudden exercise-related deaths in US athletes under 35 years of age (57). The cardiovascular system was responsible for 134 of the 158 incidents. The diagnostic criteria were relatively weak: a hypertrophied and non-dilated ventricle, and one other supporting clinical or morphological feature. Nevertheless, only a third of the group were said to be "probable" or "definite" cases of hypertrophic cardiomyopathy. Even accepting that all of the "probable" or "definite" diagnoses were correct, hypertrophic cardiomyopathy would have caused only 4-5 exercise-related deaths per year in the US. Other studies have used even less satisfactory diagnostic criteria; for example, Van Camp et al. (108) classified as "probable hypertrophic cardiomyopathy" all deaths where the cardiac mass exceeded 400 g in the absence of other systemic, valvular or cardiac disorders. But despite the broad inclusiveness of the diagnostic net, the total incidence among US athletes was apparently only about 5 cases per year. The most recent study of 33,735 athletes suggested that the total prevalence of hypertrophic cardiomyopathy, including asymptomatic cases, was 0.07% (7); given the specificity of any conceivable battery of screening tests, it seems likely that almost all of the 0.07% were false positive diagnoses, leaving an infinitessmally small true prevalence of the condition. Plainly, a recent statement of prevalence (1 in 500 athletes (2)) is a gross overestimate.

The view that hypertrophic cardiomyopathy is a "common" cause of exercise-related death can probably be traced to an early paper by Maron et al. (56). A four-year retrospective study summarized case histories on 29 US athletes who had died between the ages of 13 and 30 years. Nineteen of the 29 individuals were said to have hypertrophic cardiomyopathy, although the septal/free wall ratio met the minimum diagnostic criterion of 1.3/1.0 in only 8 of the 19 cases. It is unlikely that all of these eight individuals had hypertrophic cardiomyopathy; indeed, in the absence of a documented family history and histopathology, a substantial fraction of these cases could have had a physiological rather than a pathological hypertrophy. Thus, the whole hypothesis that hypertrophic cardiomyopathy is a common cause of exercise-related death seems to have been founded on two possible cases per year across the United States. Moreover, the majority of reports concerning hypertrophic cardiomyopathy have come only from two reference laboratories (100); the doctrine of widespread prevalence arises, at least in part, from a repeated description of the supposed 19 cases of Maron et al. (56) in 25 or more publications.

The foregoing comments in no way deny the existence of rare inherited malformations of the ventricular wall, based on molecular abnormalities in the genes encoding synthesis of the beta-myosin heavy chain, cardiac troponin T and troponin-I, alpha-tropomyosin and myosin-binding protein C of the sarcomeres (18, 38, 45, 81, 88, 113). Although the left ventricle is more frequently affected by such abnormalities, there have also been reports of right ventricular lesions (8). A few of the athletes who die while exercising may be affected by such a pathology. However, the diagnosis of ventricular cardiomyopathy requires a clear family history, reinforced by such evidence as cardiac symptoms on exertion, abnormal patterns of left ventricular filling (53), and failure of the cardiac enlargement to regress out of season (51), preferably accompanied by carefully blinded histopathology and/or demonstration of the characteristic gene structure. Moreover, there is no particular reason why this particular disorder should affect endurance athletes; indeed, myocardial dysfunction seems likely to preclude exceptional performance and participation in major competition.

Potential for prevention in older athletes

As in sedentary subjects, exercise electrocardiography offers the primary basis for the detection of potentially fatal coronary vascular lesions. Often, a vulnerable myocardium is revealed by substantial, downward sloping depression of the ST segment of the electrocardiogram, or the onset of malignant abnormalities of cardiac rhythm. The fraction of the population demonstrating an abnormal exercise electrocardiogram at any given age seems rather similar in athletes and in sedentary individuals (33); except in the oldest competitors, the prevalence of disease is very low, so as would be predicted from Bayes theorem and the specificity of the exercise electrocardiogram (no more than 85%), the proportion of false positive diagnoses actually exceeds the proportion of true positive results (92). This severely limits the value of an abnormal electrocardiogram in preventive sports cardiology.

Precision in the diagnosis of a dangerous limitation of coronary blood flow could be increased if additional tests (scintigraphy and/or angiography) were to be performed on all individuals showing a "positive" electrocardiogram, but the added costs and dangers to the test candidate make this approach unwarranted in any

programme for the mass screening of athletes. It seems likely that those who will succumb to an exercise-induced cardiac catastrophe while they are young enough to be involved in vigorous endurance competition will, in general, have gross coronary narrowing. The optimal approach is thus to increase the specificity of the electrocardiogram at the expense of its sensitivity by requiring a gross ST depression (>2-3 mm) or a major abnormality of cardiac rhythm for a positive diagnosis; the number of individuals requiring further tests such as scintigraphy and angiography is then appropriately limited, and much unwarranted anxiety and iatrogenic disability is avoided. However, to the present author's knowledge, there has as yet been no randomized test examining the value of such an approach in reducing the incidence of exercise-induced incidents; further, the number of subjects required to make such a definitive test is such that it remains unlikely to be undertaken in the near future. The approach suggested seems logical, and it may save lives, but as yet it is not evidence-based medical practice.

Potential for prevention in younger athletes

Family history

Perhaps the clearest evidence of hypertrophic cardiomyopathy, congenital repolarization anomalies and a gross inherited abnormality of lipid metabolism may all come from a history of sudden, exercise-related death in relatives at a young age; 35-50% of affected families with hypertrophic cardiomyopathy have a history of premature deaths (15).

Clinical examination

An abnormal origin of the left coronary artery may cause intermittent anginal pain, dyspnoea, and syncopal attacks, particularly if the vessel can become compressed between the aorta and the pulmonary artery.

A recent history of fever and/or viral infection is a clear warning for temporary avoidance of intense training or competition; the Swedish orienteering team halted an epidemic of sudden deaths when greater care was taken to reduce training during viral infections.

A history of exertional dyspnoea, anginal chest pain, fatigue and syncope is common in hypertrophic cardiomyopathy, although the severity of such symptoms correlates better with the severity of the condition in adults than in younger individuals (25). Careful physical examination may also disclose cardiac enlargement that is disproportionate to physical work capacity, and there may be a characteristic systolic murmur along the upper left sternal border; the latter increases on moving from the supine to a standing position, and is further exacerbated by a Valsalva manouevre or the inhalation of amyl nitrite (15). Unfortunately, many of the affected athletes have the non-obstructed form of cardiomyopathy, where the murmur is very soft or absent (44, 57, 116).

Marfan's syndrome can frequently be detected from the characteristic physique: long limbs, with an arm span greater than standing height, often a pectus excavatum or carinatum, joint hypermobility, scoliosis and myopia. Clinical examination also discloses aortic and mitral regurgitation.

Careful auscultation may disclose the mid-systolic click and late systolic murmur of mitral valve prolapse.

Some forms of repolarization anomalies may give a history of syncope.

Special investigations

An exercise electrocardiogram should reveal substantial ST segmental depression in individuals with abnormal coronary arterial anatomy, and suspicion may also be

raised at routine echocardiography (17, 30). A poorly perfused area of myocardium can be detected non-invasively by scintigraphy. The precise orientation of the vessels can be determined by selective coronary arteriography.

If the family history and clinical examination point to a diagnosis of hypertrophic cardiomyopathy, special investigations may be warranted. The electrocardiogram, typically with a picture of left ventricular hypertrophy, is often abnormal (7, 40, 119), but is not very helpful in reaching a specific diagnosis because of the physiological changes associated with a normal ventricular hypertrophy; if electrocardiographic screening is adopted, as many as 25% of athletes examined are subjected to further costly investigations (47). Echocardiography and/or magnetic resonance imaging may reveal asymmetric and/or gross thickening of the ventricular wall and/or septum; unfortunately, left ventricular hypertrophy may be absent or mild in adolescent athletes with hypertrophic cardiomyopathy. Doppler measurements may also demonstrate increased flow velocities in the obstructed outflow tract. The diagnosis can be confirmed by catheterization of the left ventricle, with the demonstration of substantial pressure gradients between the ventricular cavity and the outflow tract. A combination of genetic heterogeneity and expensive, time-consuming techniques as yet preclude genetic screening of populations for hypertrophic cardiomyopathy (18, 45, 88, 102, 113).

In Marfan's syndrome, radiographs may disclose mediastinal enlargement from aortic enlargement, and this can be confirmed by transoesophageal echocardiography, magnetic resonance imaging or computed tomography.

Echocardiography can readily demonstrate prolapse and redundant mitral valve leaflets and Doppler studies can assess the extent of regurgitation.

Right ventricular dysplasia is best detected by magnetic resonance imaging; unfortunately, this remains expensive, and is not universally available (60, 80).

Abnormalities of repolarization are revealed by electrocardiography; typically, there is a prolonged QT interval and large T waves.

Implications for health policy

Although death on the sports field attracts much attention, the critical issue from the viewpoint of health policy is the impact of prolonged endurance training on the individual's overall survival prospects and quality of life. In terms of survival, epidemiological data show that the life expectancy of endurance athletes compares favorably with that of the general population, even in the absence of any special medical examination. A retrospective examination of 2613 athletes who had represented Finland in either the Olympic Games, the World or the European championships between 1920 and 1965 demonstrated that endurance athletes survived to an average age of 75.6 years, compared with 69.9 years in a sample of 1712 sedentary adults, and 71.5 years for those who had been Finish champions in power sports (87). Thus, participation in endurance competition was associated with a lengthened rather than a shortened lifespan. Not all of the apparent six-year advantage can be attributed to endurance training, since personal lifestyle, particularly cigarette consumption, is likely to have differed between the endurance athletes and the general population. On the other hand, the true impact of endurance training may have been larger than suggested by the mortality statistics, since it is uncertain whether all of the athletes continued training in the long interval between competition and their average age at death.

Because independence is prolonged by endurance training, the athlete's quality of

life is also enhanced relative to that of a sedentary person. Physicians have worried excessively about the one in ten million chance that endurance sport might cause sudden death, while neglecting the major public health problem presented by the millions of sedentary senior citizens who require full-time geriatric care. Health policy must be assessed not simply in terms of survival, but also in terms of quality-adjusted life expectancy (94). And in such terms, the large, hypertrophied heart of the endurance athlete is an important advantage.

Need for detailed pre-screening of competitors

International competition places heavy physical demands on the heart, accompanied by a massive release of stress hormones, and this may reveal a minor cardiac abnormality that would not have been apparent in a sedentary young adult. Nevertheless, it is plain from the foregoing discussion that the risk of any type of cardiac death during competition is extremely small, and current evidence suggests that endurance training reduces rather than increases the overall risk of sudden death. Many sports physicians still feel than an exercise-related death is a reflection on the quality of care that they have given, and they continue to cherish hopes that some miraculous laboratory test will enable them to detect vulnerable individuals and warn them against major competition. However, a consideration of Bayes theorem shows that any mass screening process is doomed to failure, given the very low prevalence of pathological findings. Study of 33,735 Italian athletes over seven years led to the identification of 22 athletes suspected of hypertrophic cardiomyopathy (7), these individuals were disqualified from participation in sport, and all survived for an average follow-up of 8.2 years. However, it is unclear how many of these were false positive diagnoses, and there is no proof that those identified would not have survived equally well if they had continued sports participation. Likewise, in the U.S., less than 1% of those who received preparticipation screening received an accurate diagnosis (57). Moreover, the fact of participation in physically "dangerous" sports implies some acceptance of risk, and indeed this risk may even contribute to enjoyment of the activity.

Some 60% of U.S. states now have a locally approved standard history and examination for preparticipation screening, but often there is no clear indication of the level of competence required for such an examination (20), and too often there is reliance on costly and ineffective technology at the expense of an adequate history and clinical examination (76).

The recent position statement of the American Heart Association (2) seems ambivalent about the need for mass echocardiographic examination of athletes. Although pointing to the major problems of high screening costs and false positive diagnoses, it still states that "This viewpoint, however, is not intended to actively discourage all efforts at population screening". Nevertheless, it has been estimated that it would be necessary to screen 200,000 athletes to detect one competitor under the age of 35 years who would die during exercise (12). The best practical advice for the young athlete seems to avoid any pre-participation cardiac screening beyond a standard clinical examination focussed on family history, illicit drug use, syncope, chest pain, dizziness, fatigue, palpitations, high blood pressure, and heart murmurs (1). An echocardiographic study of 280 Harvard University athletes found 14% with mitral valve prolapse, and 11% with ventricular septal hypertrophy; however, the estimated cost of the investigation was $100,000, and finally it was concluded that the "ab-

normalities" were either due to physiological hypertyrophy or of no great practical significance (41). The routine performance of echocardiography and other sophisticated laboratory examinations is likely to do little more than precipitate a chain of costly invasive procedures, with attendant cardiac phobias, unnecessary medical costs (at least US $600 per athlete), premature cessation of sport involvement, and loss of life insurance coverage (15). Further, although there do not as yet seem any clear legal precedents (64, 65), "expert" endorsement of ineffective screening tools carries the danger of exposing honest physicians who reject such tactics to costly claims for "negligence". A recent report (57) noted that most of the young athletes who died while exercising had been screened, but cardiovascular disease had been diagnosed in only 3% of those examined, and the diagnosis had been correct in less than 1% of the sample! Specifically, only 1 of 48 suspected cases of hypertrophic cardiomyopathy had been diagnosed prior to death! Although some investigators have advocated less expensive, short-form echocardiographic screening (67, 114), this seems doomed to an even lower success rate.

Finally, in the event that a cardiovascular diagnosis is clearly established, it is important to recognise that this should not necessarily lead to disqualification from further participation in sport. Often, the prognosis will remain better if activity is continued. The recommendations of the 26th Bethesda Conference may be useful in this regard (52).

Conclusions

Although ventricular hypertrophy is sometimes suggested as the commonest cause of sudden, exercise-related death in the young adult, the total number of exercise-induced deaths is very small, and their relationship to either ventricular hypertrophy in general or cardiomyopathy in particular is far from established. There is a rare inherited form of myocardial dystrophy that predisposes to sudden death, but this is not peculiar to athletes. Indeed, because those affected have a low physical work capacity, the affected individuals are unlikely to have become involved in endurance sport. Mass pre-participatory laboratory screening of endurance athletes leads to an unacceptable toll of false positive diagnoses, and detailed examination of an athlete's heart is warranted only if there is a strong family history of early cardiac death or other significant findings in the history and clinical examination. In general, the hypertrophied heart of the athlete is a beneficial adaptation to endurance training; it increases both overall survival and quality-adjusted life expectancy, enhancing physical work capacity and reducing the likelihood of dependency in old age.

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