Heart of the athlete

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When sports medicine was in its infancy in the early 1900s, and before anyone had heard of an exercise physiologist, some physicians had been concerned about the effects of vigorous exercise on the heart. Among them were Schott¹, Henschen², Kienbock³, Barach⁴, and Lee⁵ who had made and recorded observations on swimmers, cross-country skiers and wrestlers. With the general increase in sports activity that followed World War I, many physicians expressed concerns about the long-term effects of sports participation on the adolescent heart. Their apprehension was embodied in a concept of 'athlete's heart' that stemmed back to an erroneous calculation of the German cardiologist Beneke⁶, published in 1879, that resulted in his statement that the growth of the left ventricle was disproportionate to the increase in diameter of the ascending aorta during adolescence. This error was pointed out by Karpovich⁷, but the concept and the term lingered on.

Studies of the trainability of athletes by cardiologists and physiologists following World War II demonstrated the relationships between the heart rate attained during exercise and oxygen consumption, giving rise to the concept of maximal oxygen consumption $\dot{V}O_{2\max}$ ⁸. Aerobic and anaerobic performance were defined and differentiated. Maximum attainable heart rates were related to chronological age. Training schedules to help individuals reach and sustain rates close to the maximum were described. Retrospective and prospective studies of different populations indicated that regular vigorous physical exercise that resulted in training the cardiovascular system not only improved its ability to perform more efficiently but enabled individuals to continue to be physically active longer in their lives, and perhaps to be able to extend their lives.

This, in turn, led to the development of physical training for persons who had suffered myocardial damage from arteriosclerotic cardiovascular disease, the so-called cardiovascular rehabilitation programmes⁹. Improvement in myocardial function, reduction in second myocardial infarctions and increased longevity were reported widely. One voice of caution came from Hellerstein¹⁰ who showed that some victims of this disease who survived the initial attack had sustained a level of muscle damage that

prevented any increase of maximum $\dot{V}O_2$ by training. Attempts to train these persons beyond levels of basic activity might shorten their lives.

The generally optimistic feeling generated by all this activity suggested that anyone who had an apparently normally functioning heart, as demonstrated, for example, by electrocardiography and a cycle ergometer and treadmill test, could train his/her heart to perform feats of endurance formerly not believed to be possible for the average individual by prolonged and intensive training. The concept that you could not damage but only strengthen such a heart by that type of training was encouraged by exercise physiologists and others in the coaching and training professions. What was lost from view were the well documented observations on differences between individuals in their capacities for increasing strength in skeletal muscle, which was apparently based chiefly on their genetic potential. Would it not seem likely that similar differences in the potential of heart muscle between individuals might also be present? Strong patterns of achievement in different sports among certain families from generation to generation appear to bear this out.

Early studies of the effects of vigorous exercise on the heart reached a peak with the publication in 1924 of *Heart and Athletics* by Deutsch and Kauf¹¹. The Heart Station in Vienna had been for many years the central place for the examination of the hearts of Austrian athletes, and these two physicians based their observations on the examinations of thousands of athletes of both sexes and a range of ages. In their introduction they stated:

Although a number of viewpoints concerning the influence of athletic training upon the heart are found in the medical literature, there is no uniform conception of this question, with the result that judgement of athletic qualification is handled dissimilarly by different physicians. . . . The research considered here was therefore planned with the idea of laying down uniform, generally valid directions for those formulating an opinion concerning the hearts of those who engage in athletics.

The result was the presentation of several thousand measurements of heart sizes using orthodiagraphy of male and female athletes who were competitive in 16 different sports. They measured only the transverse diameter of the heart and used for comparison the figures for a normal population published by Haudek¹² and their own studies on normal heart sizes. They noted gross body weight,

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age and breadth of lung shadows in all subjects. They found that among those who exercised only for pleasure heart sizes were in the same range as other normals, but 16% of the men and 12% of the women had greater than average heart diameters. The average heart size for male competitors and champion swimmers exceeded normal by 30–40% and for female champions and competitors there were 4–12% increases. Older athletes and those who had trained longer had the largest transverse diameters. The greatest average increases were in the Nordic skiers followed by oarsmen, cyclists, wrestlers, mountain climbers, runners, weight-lifters and throwers, soccer players, boxers and fencers. In spite of the enlargements noted, however, Deutsch and Kauf concluded that the enlargements were not clinically significant and not dangerous to the athletes. They felt that the enlargements were due to dilatation rather than hypertrophy, but also noted that even after cessation of training 'the original size is never completely reached.'

Although Deutsch and Kauf believed that the enlargements they noted were not pathological, they believed in remeasurement of heart size every 6 months or even more often. They described 14 athletes whose heart sizes showed progressive increase and in these cases advised cessation of training until the heart returned to the initially measured size. They made the interesting statement, 'The normal man bears a marked loading of his heart without any change being produced in it; on the contrary the inferior value heart suffers an often increasing enlargement early, even with slight claims made upon it.' This is the first, and only, statement that I have found suggesting that some hearts although not diseased are not as strong as others. Wondering about constitutional predisposition to large hearts they examined 100 pairs of brothers and sisters and 12 sets of three members of the same family. They found 61 people with significantly enlarged hearts and in 91.5% of these cases both family members had enlarged hearts. None of the three swimming champions in one family, on the other hand, showed enlarged hearts in spite of years of strenuous training.

In the above studies Deutsch and Kauf did not discuss the total concept of overtraining, which was not well understood then any more than it is now. The principal physical finding in the overtrained athlete is unusual fatigue in accustomed exercise with an increase in the resting heart rate to abnormal levels and failure of the rate attained during exercise to return even to the high resting level for many hours. This is often accompanied by slight ankle oedema. Arrhythmias sometimes occur. I am not aware of any published reports of measurements of heart sizes in overtrained athletes with comparison to their pre-trained or normally trained heart sizes. I suspect that the 14 athletes with progressive enlargements who were rested and recovered were overtrained even though there was no mention of their resting pulse rates.

Early signs of heart failure from any pathology include some dilatation of the heart, rapid pulse rate,

failure of pulse rate to return to resting level after exercise and slight ankle oedema. It is only recently that we have had a marker in the peripheral blood for heart failure – the atrial natriuretic peptide (ANP). The response of this factor to graded exercise is rapid and very sensitive. Since subjects with true physical overtraining are not seen commonly there is little chance that any have been evaluated by checking their ANP level before and after exercise.

Case reports

Reports of two cases at the 30th Annual Meeting of the American College of Sports Medicine in 1989 suggest the two patients concerned possessed hearts that Deutsch and Kauf might have described as being of 'inferior value.' The histories and associated findings indicate that they were overtrained with loads that many other athletes find acceptable.

*The first case*¹³ is that of a 25-year-old female marathon runner seen in the emergency room of the Lenox Hill Hospital in New York City with 30 min of dyspnoea, palpitations and light-headedness after running 3 miles. She reported that she ran 60 miles per week and that 2 years earlier had suffered a similar attack after running which was not treated medically and resolved itself after rest. She also said that her father has suffered ventricular tachycardia in the apparent absence of coronary artery disease. On admission her heart rate was 230 beats/min with a blood pressure of 100/70 mmHg. There was no heart murmur. Her electrocardiogram showed a wide-complex tachycardia with left bundle branch morphology. Because the tachycardia was not controlled by the administration of a β -blocker a cardiac catheter was inserted to look for evidence of cardiac pathology. A suspicious-looking area of ventricular myocardium was biopsied and showed circumscribed myocardial necrosis that appeared to be of recent origin. She responded gradually to conservative management and was able to leave the hospital under the care of her physician the following week.

*The second case*¹⁴ is that of a 28-year-old female runner who presented with fatigue and loss of endurance over a period of 2 months and bilateral leg swelling which she had had for 2 weeks. She had been amenorrhoeic for 2 years. She had competed frequently in 10K runs and had a personal best marathon time of 3h 17min. She was working as an instructor at a physical fitness centre and exercising about 3h daily, including swimming, biking and running a minimum of 1h per day. This is a typical triathlon training schedule. She thought her usual weight was about 48kg but admitted food avoidance and fears of being too fat. She denied having bulimia. She was seen for a hamstring strain following a marathon 9 months previously, and 2 weeks after that had suffered two episodes of haematuria following 9 mile runs. At the time of presentation her haemoglobin was 12g/dl.

On examination she had a preadolescent body type at a height of 162cm and weight of 41.4kg. She appeared much older than her stated age with wrinkled dry skin and thinning coarse scalp hair. Her resting heart rate was 100 beats/min, blood pressure 90/60 mmHg and her legs showed pitting oedema to her knees. Haemoglobin was 8.2g/dl, serum albumin 3.8g/dl, protein 5.2g/dl, and blood urea nitrogen 12mg/dl. Serum ferritin was less than 5g/dl. No electrocardiogram was recorded.

The two physicians agreed that this woman manifested several of the criteria for a diagnosis of anorexia nervosa. One suggested that 'cardiopulmonary limitations' should

be considered in the differential diagnosis because of fatigue and decreased exercise tolerance but there was no other evidence to substantiate this, although her tachycardia was not explained. Peripheral oedema occurs in about one-third of anorexics. They recommended an increase in her caloric intake, a decrease in exercise to walking 20 min three times weekly, iron supplementation and evaluation regarding her amenorrhoea, osteoporosis, anorectic behaviour and psychiatric status. She continued to run for 1 h per day, however, but stopped biking and swimming. She took an iron supplement but continued to lose weight and did not follow the other recommendations.

Discussion

The first patient was apparently clearly suffering from heart failure with documented evidence of myocardial damage. Her previous similar but less severe episode may have left some myocardial damage which was not discovered in the later episode because of other over-riding factors. Unless she has now modified her exercise substantially by stopping marathon training it may well happen again, and perhaps with a more serious consequence.

The second patient clearly manifested anorectic behaviour, but this did not appear sufficient by itself to account for all her complaints and findings. Specific evidence of her functional cardiac status is lacking because no studies for such an evaluation were performed. The decreased heart size and reduced function observed as a result of starvation may not return to normal with refeeding; the mortality rate of anorexia is about 2% in our population. This patient may have been suffering from first-degree heart failure in addition to her other problems.

Psychological factors are involved in states of overtraining, but physicians dealing with this problem might consider whether they are secondary responses to the physical overtraining, just as they are in patients suffering heart failure due to other causes.

The study of overtraining is particularly difficult because of the scarcity of clearly defined case material in any clinic or training setting. Certainly treatment

has been handicapped by lack of any consensus on causative factors other than sheer overwork. One approach to the study of this condition as a cardiovascular problem is practical with the technology available today and might prove valuable.

In my view the majority of top-class athletes today are overtrained and overworked the year around. The frequency of disability observed and the cyclic nature of their performances provide practical evidence of this. Perhaps a practical system of physical monitoring would be more successful in reducing this toll than the current methods of control which appear to be driven more by prestige and money.

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