Electrocardiographic abnormalities and cardiovascular physiology in athletic overtraining

A King Jr, YSA Lo, MK Chin, JX Li, RCH So

The term overtraining syndrome describes the condition in which an athlete suffers from various complications resulting from overtraining. These typically include overuse injuries, mood disturbances, and blood chemistry changes. Although there is no gold-standard criterion for diagnosing this syndrome, the best current indicators include prolonged fatigue, impaired athletic performance, and psychological changes. This article aims to provide some insights into the physiology of overtraining, with emphasis on its cardiovascular consequences. Impaired cardiac function may occur after prolonged exercise, and is documented by depressed left ventricular function and decreased maximum oxygen consumption. Electrocardiographic abnormalities which may be seen in overtraining are T wave inversions, ST segment depressions, and various arrhythmias. Well-conditioned athletes may also manifest the same abnormalities as a result of physiological cardiac hypertrophy. Electrocardiography and two-dimensional echocardiography should form an integral part of the screening performed by any sports medicine department overseeing the training of athletes.

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Introduction

The quest for gold medals and record-breaking feats in sport is the objective of most, if not all, of today's athletes, coaches, and sports institutions. This drive to further improve performance has made training much more vigorous and taxing. A relatively new, challenging, and bewildering condition has emerged—the problem of overtraining. The term overtraining syndrome describes the situation when an athlete suffers from various training complications, such as overuse injuries, mood disturbances, and blood chemistry changes.[1] This syndrome is not only responsible for athletes' failures and poor performances, but also represents a real health problem for the athlete. As competitive sport now forms an integral part of Hong Kong life, this article aims to provide some insights into the cardiovascular physiology which results from overtraining.

Incidence of overtraining

As there is no accepted gold standard for the diagnosis of this syndrome, the true incidence of overtraining is difficult to ascertain.[2] One study reported incidences of 10% in college swimmers training for up to 14 km daily and 60% in elite distance runners.[3] Some aspects of the cardiovascular system are commonly used to diagnose and monitor overtraining. The monitoring of morning heart rate, recovery heart rate, and resting blood pressure has been advocated.[3-5] Currently, the best indicators of overtraining appear to be prolonged fatigue of at least one week's duration, decrements in performance, and changes in psychological factors.[5,6]

Philippine Center for Sports Medicine, Rizal Memorial Sports Complex, Vito Cruz Street, Malate, Metro Manila, Philippines
A King Jr, MD, FPCP
Hong Kong Adventist Hospital and Heart Centre, Stubbs Road, Hong Kong
YSA Lo, MD, FRCPC
Sports Science Department, Hong Kong Sports Institute, Shatin, Hong Kong
MK Chin, PhD
RCH So, M Phil
Department of Sports Physiology and Biochemistry, Tianjin Institute of Physical Education, Ligizhuang, Hexi District, Tianjin, China
JX Li, MSc, MD
Correspondence to: Dr YSA Lo

Various symptoms and laboratory tests have been used to document or monitor overtraining. However, it has been suggested that close and continuous observation of athletes by coaches is sufficient, rather than any specific scientific tests.[5]

Cardiovascular alterations due to overtraining

As early as 1959, electrocardiographic (ECG) changes had been noted in swimmers after strenuous exercise.[7] Other authors have documented ECG abnormalities due to overtraining.[8] However, some have argued against the use of ECG changes as markers for overtraining, as vagal tone and cardiac hypertrophy in athletes may produce similar ECG findings.[9]

Documented ECG changes in overtrained athletes include T wave changes, ST segment changes, P-R and Q-T time interval changes, and arrhythmias. Various authors have reported enlarged T waves in overtrained athletes.[8,10,11] A low T wave amplitude has been observed in track athletes and was associated with overtraining or overtiredness.[12] Depressed or inverted T waves have been documented in the West and also in Chinese athletes suffering from frequent upper respiratory tract infections and injuries.[7,13,14] At the Mexico Olympics, T wave changes with ST segment depression—similar to that seen in coronary heart disease—were reported in athletes training at high altitude who complained of angina.[15] The same changes have also been reported to be associated with overtraining by other authors.[13]

One study found a slightly higher incidence of premature atrial contractions (PACs) in 60 endurance-trained athletes, compared with non-athletes.[16] Twenty-four-hour ECG monitoring found 100% of a group of runners to have PACs, compared with only 51% in the control group.[17] In addition, 25% of runners were found to have atrial couplets (two successive PACs) compared with none in the control group. A Thai study noted changes such as prolonged Q-T interval, first degree AV block, and inverted T waves in 133 of 451 Thai half-marathon runners after a 21.1 km race.[18]

Biochemical alterations

The exact mechanism of ECG changes in overtraining is probably multifactorial. It is important to understand that an ECG is a recording of electrical activity, which is mainly affected by ions, both positively and negatively charged [e.g. potassium (K^+), hydrogen (H^+), magnesium (Mg^{++})]. A number of factors in the overtrained athlete may alter ionic activity and give rise to ECG changes. These include nutritional status and hydration, electrolyte balance [e.g. sodium (Na^+), K^+, Mg^{++}, calcium (Ca^{++})], neurogenic or autonomic stimulation (sympathetic system), myocardial blood flow (ischaemia), hypoxia (decreased O_2 delivery to the myocardium despite adequate blood flow), pH, and structural changes in the heart, such as hypertrophy of the left ventricle.

A number of overtraining studies have documented weight loss.[9,19] Some authors have suggested that the loss is caused by chronic dehydration rather than true body composition changes.[20] This would explain the increase in morning heart rates and decreased fluid volume seen.[21] With decreased intravascular volume, there is a concomitant reduction in cardiac filling and output. This may explain the reduction in peak oxygen consumption (VO_2) seen in overtraining.[1] Dehydration may cause orthostatic hypotension—whether relative coronary hypoperfusion occurs as a result remains to be elucidated. In addition, dehydration is usually accompanied by electrolyte disturbances. Subnormal levels of K^+ and Mg^{++} have been reported.[22,23] Hypokalaemia is known to flatten T waves.

Heightened sympathetic tone

Most overtrained athletes present with signs and symptoms of increased sympathetic activity. Overtrained athletes have been shown to be anaemic and iron deficient, and various authors have documented low serum ferritin levels in association with overtraining.[24,25] One study found 22.4% and 39.5% incidences of iron deficiency anaemia in adult and young Chinese athletes, respectively.[23] Both dehydration and anaemia may trigger increased sympathetic stimulation as a compensatory mechanism to improve myocardial performance. There are other causes of increased sympathetic tone in overtraining, such as trauma and injuries. A major sign and symptom of overtraining is the development of depressive moods. These are associated with increased basal concentrations of plasma norepinephrine and epinephrine in relation to the degree of anxiety.[26] Other studies have shown that epinephrine may cause a decreased T wave amplitude and that larger doses may cause ST depression.[27] People with increased sympathetic drive often have T wave inversions and ST depression (Reynold's syndrome).[28] The endogenous release of epinephrine has also been shown to cause premature ventricular contractions, ventricular tachycardia, and fibrillation.[29] This is particularly relevant for individuals with susceptible hearts. An increase in blood pressure as a result of sym-

pathetic stimulation has also been shown to be a sensitising factor for epinephrine-induced arrhythmias.[30] It has been suggested that increased training loads may lead to increased resting and exercise blood pressure levels, sensitising the myocardium to epinephrine, and may be responsible for the arrhythmias seen in overtraining.

Muscular acidosis

The effects of exhaustive exercise include acidosis in sketetal muscle and blood, and are well documented.[31] It is also known that coronary artery occlusion and other causes of ischaemia may produce myocardial acidosis.[32] However, there is scant information on the effects of exhaustive exercise on myocardial intracellular pH in normal subjects. Nevertheless, the potential for exercise-induced acidosis exists and may be compounded by any physiological or pathological factors capable of compromising blood flow.[33] Perfusion experiments on animals have shown that acidosis increases T wave amplitude, whereas alkalosis causes a reduction or inversion of the T wave.[34] It is well established that chronic hyperventilation with resultant hypokalaemic alkalosis is characterised by total body potassium depletion, and is a very potent cause of ST-T wave changes and arrhythmias.[35]

Myocardial dysfunction

An important question is whether the ECG changes seen in overtraining are markers of organic and structural changes brought about by overworking the heart. Are these changes warnings of actual myocardial damage? An instance of heart attack has been reported during altitude training in an athlete with a previously normal coronary angiogram.[36] A similar instance in a 20-year-old national class swimmer occurred during a period of overtraining.[37] Others have noted that creatine phosphokinase-MB subunits may be elevated in asymptomatic long distance runners.[4] Myocardial damage has been detected by echocardiography and myocardial scintigram in athletes with ECG repolarisation changes.[38] These findings raise the question as to whether exhaustive exercise and overtraining can produce transient myocardial ischaemia, causing alterations in cardiac structure and function.[39] Studies in race horses suggest that myocardial function may be limited by maximal blood flow during periods of extreme exercise.[40] During such exercise, coronary flow may also be limited by α-adrenergic–mediated coronary vasoconstriction. Two studies conducted in dogs have shown that α-adrenergic constriction might limit maximum oxygen delivery to the myocardium during extreme exercise.[41,42] A similar response has been found in patients with coronary disease, although not in normal subjects.[43] Currently, there is no evidence that athletic performance in normal humans is limited by maximal blood flow in the coronary circulation.

Many authors have documented impaired cardiac function following prolonged exhaustive exercise.[44-46] The subjects were all marathon runners tested after an exhaustive run. Two runners presented with signs and symptoms of congestive heart failure. One study found decreased cardiac stroke volume in a 24-year-old man who had run across the United States.[44] Significantly reduced stroke volumes and ejection phase indices of left ventricular global function were found in 13 experienced marathon runners following a competitive 24-hour run.[45] It was concluded that strenuous physical exertion can actually impair myocardial performance. Likewise, a transient (24- to 48-hour) mild depression of left ventricular function in ultra-marathon runners has been observed.[46] Others have observed an increase in end-diastolic volume and increased duration of mechanical systole at exhaustion, produced by prolonged exercise in young men, indicative of possible transient left ventricular dysfunction.[47] It has been suggested that the increase in morning heart rates observed in long distance runners on consecutive days of a 500 km run was secondary to cardiac fatigue.[4] It is also possible that decreased cardiac output secondary to lowered myocardial performance may explain the findings of decreased peak VO_2 reported in overtrained athletes.[48] It is possible that these findings were simply a result of decreased cardiac filling due to dehydration and not a decrease in myocardial contractile performance.[49]

Animal studies which document reduction of myocardial performance after being overworked have been conducted. Depressed intrinsic contractile functioning has been demonstrated in isolated trabecular muscles taken from the left ventricle of rats which had been run to exhaustion on a treadmill.[50] Depressed length-tension and force-velocity functions were observed. This study also documented that the inotropic response to exogenous norepinephrine was lower in the hearts of exhausted rats. Exhaustive swimming exercises in rats produce a significant reduction in the Ca^{++} transport capacity of the sarcoplasmic reticulum and mitochondrial membrane system, providing a possible explanation for the contractile dysfunction seen after exhaustive exercise.[51]

Acidosis has long been known to decrease myocardial performance. Myocardium, in contrast to skel-

etal muscle, has a much greater sensitivity to agents which reduce sarcolemmal Ca^{++} influx, including lactate formed in acidosis.[52] Although lactate studies in overtrained athletes show diminishing values (probably as a result of glycogen depletion), no definitive pH studies have been done on the human heart under conditions of overtraining or following exhaustive exercise. Regardless, the acidosis present in coronary artery occlusion cannot be explained by any increase in tissue lactate concentration. Over 40 organic acids are readily detectable in rat myocardium.[53] Virtually every one of these acids increases in concentration within four minutes of an ischaemic state. The simplistic view that acidosis is due solely to lactate production in the myocardium (or other tissues) should be discarded.[33]

Competition occurs between H^+ ions and Ca^{++} for troponin binding sites, and an increase in H^+ within a physiological range serves to reduce Ca^{++} binding to calmodulin.[54,55] Conversely, alkalosis is known to increase muscle contractility. A reduction in the rate of sarcoplasmic reticulum Ca^{++} uptake and impairment of calcium-induced cardiac relaxation by acidosis may be the mechanism by which acidosis has a negative effect on muscle contractility.[33] It has been shown that increasing H^+ ion concentrations serve to substantially increase the Ca^{++} requirement for myofilament activation.[54,56] The non-invasive measurement of intracellular pH is a critical area of research and perhaps through the use of newer technology, a clearer understanding of possible exercise-induced reduction in pH in cardiac muscle in humans will emerge.[33]

Discussion

The data presented show that there are deleterious effects of exhaustive exercise and overtraining on the human heart. Although most of the data presented are based on effects following exhaustive exercise and not necessarily those of overtraining, there is a strong likelihood that the same situation occurs with both conditions.

There remain many uncertainties and unanswered questions on the effects of overtraining on the heart. A compounding problem is the fact that well-conditioned athletes may also display the same ECG abnormalities because of physiological cardiac hypertrophy. Furthermore, an ECG may be normal in an overtrained athlete.

Notwithstanding the uncertainties associated with ECG technology, all athletes should probably have baseline ECGs performed before commencing intensive training, as well as during peak training periods. Whenever an abnormal ECG is obtained in potentially overtrained athletes, clinical correlation should be obtained. The exercise physiologist can present other laboratory data or markers of overtraining, while the coach can provide actual observations of an athlete's performance, training intensity, and behaviour to give a more accurate diagnosis.

A two-dimensional (2D) echocardiogram would also be helpful to document physiological hypertrophy and/or reduced cardiac contractile performance, a possible sign of overtraining. A simple 2D echocardiograph is relatively inexpensive and should form an integral part of any sports medicine department overseeing the training of athletes. This diagnostic tool may also help screen athletes for hypertrophic and dilated cardiomyopathy, both being conditions which predispose to sudden death. Electrocardiographic exercise stress-testing may be performed in selected individuals, especially in older overtrained athletes who have other risk factors for coronary heart disease. It is known that the two cardiomyopathy conditions may be missed completely, despite a good physical examination and 12-lead ECG. In this way, we may make better judgements and decisions concerning the safety and welfare of athletes and also establish a better database for use in future research projects.

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References

1. Stone MH, Keith RE, Kearney JT, Heck SJ, Wilson GD, Triplett NT. Overtraining: a review of signs, symptoms and possible causes. J Appl Sport Sci Res 1991;5:35-50.
2. Budgett RS. Overtraining syndrome. Br J Sports Med 1990;24:231-6.
3. Morgan WP, O'Connor PJ, Sparling PB, Pate RR. Psychological characterization of the elite female distance runners. Int J Sports Med 1987;8(2 Suppl):124S-131S.
4. Dressendorfer RH, Timmis GC, Gordon ST. Creatine kinase MB isoenzyme responses to repeated long-distance running [abstract]. Circulation 1985;66(2 Suppl):185S.
5. Brown RL. Overtraining of athletes: a round table. Phys Sportsmed 1983;11:92-110.
6. Costill DL. Inside running: basics of sports physiology. Indianapolis: Benchmark Press Inc., 1986.

7. Carlile FF, Carlile UB. T wave changes in ECG associated with prolonged periods of strenuous exercise in sportsmen with special reference to training swimmers. Phys Educ J 1959;17:10-20.
8. Kuipers HS, Keizer HA. Overtraining in elite athletes: review and directions for the future. Sports Med 1988;6:79-92.
9. Hollmann WH, Hettinger TH. Muskulare ermudung. Sportsmedizin: arbeits und trainings-grundlagen. Stuttgart: Schattauer, 1990.
10. Michael ED. Overtraining in athletes. J Sports Med Phys Fitness 1961;1:97-8.
11. Foster C, Pollock M, Farrel P, Maksud M, Anholm J. Training responses of speed skaters during a competitive season. Res Quart Exerc Sports 1982;53:243-6.
12. Stegeman JR. Exercise physiology: physiologic basis of work and sports. Chicago: Yearbook Medical Publishers Inc., 1981.
13. Counsilman JE. All that yardage. Swim Tech 1988;24:19-24.
14. Fu YY, Liang KT. T wave changes in 7 middle long distance runners. Chin Med J 1980;60:543-5.
15. Kuramoto K, Ikai M, Asahina K, Kuroda Y, Ogawa S. Strenuous exercise ECG of top class swimmers in Mexico City. Jpn Heart J 1967;8:291-300.
16. Beswick FW, Jordan RC. Cardiological observations at the 6th British Empire and Commonwealth Games. Br Heart J 1961;23:113-29.
17. Talan DA, Bauernfeind RA, Ashley WW, Kanakis C, Rosen KM. Twenty-four continuous ECG recordings in long distance runners. Chest 1982;82:19-24.
18. Thongyai KT, Tiyapan AE, Banchmen KK. ECG finding in Thai half marathon runners. J Sports Med Assoc Thailand 1988;1:3-6.
19. Stamford BA. Avoiding and recovering from overtraining. Phys Sportsmed 1983;11:180.
20. Dressendorfer RH, Wade CE, Scaff JH. Increased morning heart rate in runners: a valid sign of overtraining? Phys Sportsmed 1985;13:77-86.
21. Lamb DR. Physiology of exercise: responses and adaptation. New York: McMillan Publishing Co., 1984.
22. Strauzenberg SE, Schneider F, Donath R, Zerbes H, Kohler E. The problem of dieting in training and athletic performance. Bibl Nutr Dieta 1979;27:133-42.
23. Chen, JD. Sports nutrition: present status and future prospect in China. In: Chong YH, Ng KW, Tee ES, Mohd IN, editors. Proceedings of the Sixth Asian Congress of Nutrition: Nutritional Challenges and Frontiers Towards the Year 2000; 1991 Sept 16-19; Malaysia. Kuala Lumpur: National Society of Malaysia Press, 1991:432-5.
24. Belcastro AN, Dallaire J, McKenzie DC, Schutz R, Smith M, Wenger HA. Canadian Association of Sports Sciences (CASS): overstress study. Ontario: Ontario-Gloucester Publishing Co., 1991.
25. Flynn MG, Pizza FX, Boone JB. Indices of overtraining syndrome during a running season. Med Sci Sports Exerc 1990;22(2 Suppl):131S.
26. Axelrod JS, Reisine TD. Stress hormones: their interaction and regulation. Science 1984;224:452-9.
27. Weiner NS. Norepinephrine, epinephrine and the sympathomimetic amines. In: Gilman AE, Goodman LA, editors. The pharmacological basis of therapeutics. New York: McMillan Publishing Co., 1980:160-5.
28. Ellestad MH. Stress testing: principles and practice. Philadelphia: FA Davis Publishing Co., 1986.
29. Dresel PE, MacCannell KI, Nickerson M. Cardiac arrhythmias induced by minimal doses of epinephrine in cyclopropane-anaesthetized dogs. Circ Res 1960;8:948-55.
30. Benfey BG, Varma DR. Interactions of sympathomimetic drugs, propranolol and phentolamine, on a trial refractory period and contractility. Br J Pharmacol 1967;30:603-11.
31. Osnes JB, Hermansen LA. Acid-base balance after maximal exercise of short duration. J Appl Physiol 1972;32:59-63.
32. Ichihara K, Haga N, Abiko Y. Is ischemia-induced pH decrease of dog myocardium respiratory or metabolic acidosis? Am J Physiol Heart Circ Physiol 1984;246:652-7.
33. Tibbits GF. Regulation of myocardial contractility in exhaustive exercises. Med Sci Sports Exerc 1985;17:529-37.
34. Barker PS, Shrader EL, Ronzonia ES. Effects of alkalosis and of acidosis upon human ECG. Am Heart J 1939;17:169-72.
35. Jacobs WF, Battle WE, Ronan JA. False positive ST-T wave changes secondary to hyperventilation and exercise: a cine-angiographic correlation. Ann Intern Med 1974;81:479-82.
36. Kindermann W, Keul J, Reindell H. Grundlagen Zur Bewertung leistungsphysiologyischer Anpassung suorgange. Dtsch Med Wochenschr 1974;99:1372-5.
37. Hanson PG, Vander Ark CR, Besozzi MC. Myocardial infarction in a national class swimmer. JAMA 1982;248:2313-4.
38. Frick MH, Korhola O, Nieminen M, Valle M. Ischemic electrocardiographic changes in an athlete with normal coronary arteries. Ann Clin Res 1975:7:264-8.
39. Thomas DP. Effects of acute and chronic exercise on myocardial ultrastructure. Med Sci Sports Exerc 1985;17:546-53.
40. Pascoe JR, Ferraro GL, Cannon JH, Arthur RM, Wheat JD. Exercise-induced pulmonary hemorrhage in racing thoroughbreds: a preliminary study. Am J Vet Res 1981;42:703-7.
41. Heyndricky GR, Muylaert P, Pannier JR. Alpha-adrenergic control of oxygen delivery to myocardium during exercise in conscious dogs. Am J Physiol Heart Circ Physiol 1982; 242:805-9.
42. Murray PA, Vatner SF. Alpha-2-adrenoreceptor attenuation of the coronary vascular response to severe exercise in the conscious dogs. Circ Res 1979;45:654-60.
43. Mudge GH, Goldberg S, Gunther S. Comparison of metabolic and vasoconstrictor stimuli on coronary vascular resistance in man. Circulation 1979;59:544-50.
44. Bruce RA, Kusumi F, Culver BH. Cardiac limitations to maximum O_2 transport and changes in components after jogging across the US. J Appl Physiol 1975;39:958-64.
45. Niemela KO, Palatsi IJ, Ikaheimo MJ. Evidence of impaired left ventricular performance after an uninterrupted competitive 24-hour run. Circulation 1984;70:350-6.
46. Douglas PS, O'Toole ML, Hiller WD, Reichek N. Left ventricular structure and function by echocardiography in ultraendurance athletes. Am J Cardiol 1986;58:805-9.
47. Ekelund LG, Holmgren AB, Ovanfors CO. Heart volume during prolonged exercise in the supine and sitting position. Acta Physiol Scand 1967;70:88-98.
48. Mikeselle KA, Dudley GA. Influence of intense endurance training on aerobic power of competitive distance runners. Med Sci Sports Exerc 1984;16:371-5.
49. Alpert JS, Pope LA, Ward A, Rippe JM. Athletic heart syndrome. Phys Sportsmed 1989;17:103-7.
50. Maher JT, Goodman AL, Francesconi RT, Bowero WD, Hartley LH, Angelakos ET. Responses of rat myocardium to exhaustive exercise. Am J Physiol Heart Circ Physiol 1972;222:207-12.
51. Pierce GN, Kutryk MJ, Dhalla KS, Beamish RE, Dhalla NS. Biochemical alterations in heart after exhaustive swimming in rats. J Appl Physiol 1984;57:326-31.
52. Rich TL, Langer GA. A comparison of excitation-contraction coupling in heart and skeletal muscle: an examination of "calcium-induced calcium release". J Mol Cell Cardiol 1975;7:747-65.

53. Haraguchi SI, Toshima HR, Matsumato I, Kuhara T, Shinka T. Changes of organic acids in rat heart muscle under ischemic-like condition. J Chromatogr 1982;227:1-9.
54. Blanchard EM, Pan BS, Solarzo RJ. The effect of acidotic pH on the ATPase activity and troponin Ca binding of rabbit skeletal myofilaments. J Biol Chem 1984;259:3181-6.
55. Haisch JR, Klee CB, Demaille JD. Effect of cations on affinity of calmodulin for Ca^{++} ions allow the specific activation of calmodulin-stimulated enzymes. Biochemistry 1981;20:3890-7.
56. Fabiato AD, Fabiato FF. Effects of pH on the myofilaments and the sarcoplasmic reticulum of skinned cells from cardiac and skeletal muscles. J Physiol 1978;276:233-55.