30. Heart rhythm disturbances

Authors

Agneta Ståhle, PT, PhD, Associate Professor, Department of Neurobiology, Care Sciences and Society, Division of Physiotherapy, Karolinska Institutet, Stockholm, Sweden

Lennart Bergfeldt, MD, PhD, Professor, Sahlgrenska Academy, Gothenburg University and Sahlgrenska University Hospital, Gothenburg, Sweden

Anders Englund, MD, PhD, Associate Professor, Arrhythmia Center and Karolinska Institutet, South Hospital, Stockholm, Sweden

Knut Gjesdal, MD, PhD, Professor, Department of Cardiology, Ullevål University Hospital, Oslo, Norway

Summary

Heart rhythm disturbances is a collective description of deviations in the heart's electrical impulse generation and/or conduction. Included in this concept at one end of the spectrum are extra beats, which do not usually produce symptoms and are therefore perceived as a normal variant in the absence of other heart disease, and at the other end directly life-threatening ventricular fibrillation or interruptions in the impulse generation. Many patients with arrhythmia problems have an underlying cardiovascular disease such as hypertension, coronary heart disease and/or heart failure, which is why care must be taken when prescribing physical activity. The principles for exercise training that apply to other heart patients are also applicable to patients with arrhythmias. The prescription must always include definition of the frequency, duration and intensity.

Training method	Intensity	Frequency (times/week)	Duration (min./session)
Aerobic training	50–80% of VO_2 max* RPE** 9–15/20	2–3	45–60
Strength training	40–60% of 1 RM*** 1–3 sets of 8–10 exercises with 12–15 reps/set RPE 11–13/20	2–3	30–40
Muscle endurance training	40–80% of 1 RM*** > 15 reps/set RPE 9–15/20	2–3	45–60

* VO_2 max = Maximal Oxygen Uptake.

** Rating of Perceived Exertion, according to the Borg scale.

*** RM = Repetition Maximum, which corresponds to the maximum weight that can be lifted throughout the entire exercise movement one time.

Suitable activities include brisk walking, jogging, cycling, swimming, exercise or aquafit classes, skiing, skating, dance and ball sports, depending on the individual's interests. For patients with an implantable cardioverter defribrillator (ICD), activities like jogging in proximity to heavily trafficked roadways, swimming and cycling involve a certain risk of injury to themselves or others as there is a 10 to 20 second delay between detection of the arrhythmia and generation of the ICD shock.

Definition

The heart is a muscular pump through which blood flow is controlled by valves (one-way valves). In order for the pumping motion to occur, an electrical activation (electromechanical coupling) is required. This originates in the heart's own electrical system which is made up of a generator (the sinoatrial node), a reserve generator and filter between the atrium and ventricle (the AV node), and a main cable (the His bundle), which branches into three main divisions – one to the right– and two to the left ventricle. The heart is also a secretory organ with hormones that affect the heart itself and other organs such as the kidneys.

Heart rhythm disturbances is a collective description of deviations in the heart's electrical impulse generation and/or conduction. At one end of the spectrum are extra beats, which do not usually produce symptoms and are therefore perceived as a normal variant in the absence of other heart disease, and at the other end are directly life-threatening ventricular fibrillation or interruptions in the impulse generation. Deviations in the heart's electrical activity can give rise to both too slow a rhythm (bradycardia) and a racing heart (tachycardia). An estimated 1–1.5 per cent of the population has some form of heart rhythm disorder that will at some point become the object of assessment and/or treatment. Commonly occurring disorders are atrial fibrillation (approx. 1 per cent of the entire population, but approx. 10 per cent of the population over 80 years), bradycardia that requires artificial pacing (0.3 per cent) and attacks of regular atrial tachycardia

(0.5–1.0 per cent of the adult population). The extent of life-threatening ventricular tachycardia is more difficult to estimate. In Sweden, about 30,000 people per year are diagnosed with acute myocardial infarction. Against this background, it is important to know that approximately 30 per cent of those who die a sudden death in acute myocardial infarction had no earlier symptoms of coronary heart disease.

Cause

Cardiac arrhythmias, another general description, can be primary or secondary. Primary electrical problems can be congenital, caused by extra pathways outside the regular conduction system (WPW syndrome), or acquired, extra impulse pathway within the heart's own electrical system (AV nodal reentry tachycardia). Functional and structural changes in the pores (ionic channels) that control the flow of electrically charged particles (above all sodium, potassium and calcium ions) across the cell membrane are also among the primary arrhythmias. An example of such a disease condition is congenital long QT syndrome, which involves an increased risk for serious arrhythmias and fainting. By secondary problems are meant disorders in the heart muscle and/or valve function that have consequences for the heart's electrical function, as well as other diseases, such as toxic goitre, which can lead to arrhythmia.

Risk factors

There is a genetic origin to certain types of arrhythmia, for example, long QT syndrome, certain types of atrial fibrillation, and some types of ventricular arrhythmias. In most patients with atrial fibrillation and ventricular arrhythmias, however, there is an underlying cardiovascular disease, such as hypertension and coronary heart disease. Because there are a number of predisposing factors, at least with respect to the last disease condition noted, such as diabetes, smoking, overweight, blood lipid disorders, etc., these must also be seen as risk factors for arrhythmias. It is suspected that strenuous physical exercise can predispose to atrial fibrillation.

Pathophysiological mechanisms

The cause of too slow a heart rhythm (bradycardia) requiring pacemaker treatment is just as often disorders in sinoatrial node function (sinus bradycardia, sinus pause), blocking conduction of the impulse between the atrium and ventricle (AV block). When it comes to a rapid heart rhythm (tachycardia), the predominant mechanism is an electrical circuit (a reconnection or reentry mechanism) This electrical circuit can be relatively stable, such as in AV nodal reentry tachycardia and WPW syndrome, as well as some ventricular tachycardias associated with scarring after myocardial infarction, but can also vary as with atrial fibrillation and ventricular arrhythmias in relation to congenital or acquired long QT syndrome. The latter includes effects of drugs and thickening of the heart muscle as a result of hypertension, heart failure and cardiomyopathy (disease of the heart muscle).

Abnormal impulse generation is less common as a cause of atrial arrhythmias, but is relevant in the initiation of ventricular arrhythmia with respect to acquired long QT syndrome.

Common symptoms

Heart palpitations involve the patient's perception of his or her heart rhythm, but are not necessarily a symptom of arrhythmia, as palpitations also occur in sinus tachycardia of a purely physiological nature. Sudden onset palpitations are the most predominant symptom in patients with tachycardias but who are otherwise in good cardiac health. Shortness of breath, pressure across the chest or chest pain and disturbances in consciousness (dizziness and/or fainting) are more common during rapid heart rhythms in individuals with other concurrent heart disease. In the case of atrial fibrillation, impaired performance capacity is a common symptom. When it comes to bradycardia-related symptoms, sudden onset dizziness or fainting is most common, but shortness of breath, fatigue and impaired performance capacity upon exertion are also common symptoms that lead the patient to seek medical attention.

The heart's pumping capacity, that is its ability to meet the varying demands of the body is seen in the cardiac output (heart minute volume), which at rest is 4–5 litres per minute depending on body size, and can increase to 25–30 litres per minute during maximal exertion. This ability to adjust is primarily dependent on variations in heart rate, which at rest is most often between 50 and 70 beats per minute and up to 170–200 per minute at maximal exertion. The heart's stroke volume (volume per beat) can increase by approximately 50 per cent. Another important factor to consider in this context is that 85 per cent of the blood supply to the heart itself takes place in the heart's electrical resting phase (diastole), and when heart rate increases, regardless of the cause, this mainly reduces the heart's resting phase. In concrete terms, this means that while a heart rate that is too slow yields good filling of the heart's chambers, it yields poor adjustment to increased demands, whereas a high heart rate reduces the filling and places high demands on the heart muscle's energy supply since every heart beat expends energy, while the time for supplying this energy and oxygen is reduced, relatively speaking. Both ends of this heart rate spectrum can be life-threatening.

In summary, however, the patient's symptoms are due firstly to the heart rate, secondly to cardiac function in general, and thirdly to the patient's general fitness, which can vary from one instance to another.

Diagnosis

Manual pulse-taking, electrocardiography (ECG) conducted as resting ECG or long-term ECG monitoring and electrophysiological recording and stimulation methods, in part from outside the heart via the oesophagus and in part from inside the heart via blood vessels, for example, in the groin, are important diagnostic tools. Stress tests have a poor capacity (sensitivity) to provoke (diagnose) both tachycardia and bradycardia, but are a valuable method for assessing the patient's overall aerobic fitness and possible presence of coronary heart disease.

Treatment

A correct diagnosis is decisive in the care of these patients. Once a link between a rhythm disturbance and symptoms has been established and an assessment of the level of danger and prognosis has been made, it is often possible to refrain from other treatment. When it comes to bradycardias, the decision is usually a pacemaker in a secondary prophylactic aim (when arrhythmia is already producing symptoms) and less often in a primary prophylactic aim (before symptoms appear). For tachycardias, it is usually a question of no treatment at all, drug therapy, ablation therapy (an invasive catheter technique in which the electrical disorder or pathway is treated with heat), surgery (e.g. maze surgery for atrial fibrillation), or combinations of treatments such as a pacemaker plus drug therapy, drug therapy plus ablation, etc. Implantable cardiac defribillators (ICDs) are used both to treat ventricular arrhythmia relapses and to prevent sudden arrhythmia death in high-risk patients.

Effects of physical activity

Long-term effects

Exercise training has effects on the autonomic nervous system's parasympathetic system, which can affect heart rhythm disturbances in different directions. An increase in vagal activity, particularly at night, can give rise to both sinus pauses and AV blocks, mainly in the form of AV block II (Wenckebach type). In a physically fit person, this rhythm disturbance is of little prognostic importance as long as conditions during physical exertion are completely normal (1).

A less common subgroup of atrial fibrillation presents primarily at night and is considered to be linked to vagal dominance. In the case of this condition, exercise training can possibly predispose, even if scientific evidence here is lacking. In animal trials, vagal activity during concurrent sympathetic stimulation and arrhythmia provocation has been shown to have a beneficial effect in that the fibrillation threshold in the ventricles is raised and it becomes harder to induce fibrillation. This has nevertheless not been convincingly documented in humans. Because good aerobic fitness can improve a person's tolerance to disturbances in cardiac function, good overall fitness is worth striving for. After many years of training at the competitive level, arrhythmias are relatively common (2), but the mechanism behind this has not been established. This applies in particular to atrial fibrillation (3, 4).

Effects of arrhythmia

A tendency to bradycardia is often linked to an inability to increase the heart rate sufficiently in connection with exertion (chronotropic incompetence). This results in reduced maximal performance capacity. Certain tachycardias, and tachycardias in certain people, present especially in connection with physical and/or mental exertion, which the patient has usually observed. As a rule, one's performance capacity decreases in connection with the tachycardia.

Exercise training for different arrhythmia

Pronounced sinus arrhythmia

Young, physically fit persons often have a slow and uneven resting heart rate. This is due mainly to breathing-related vagal reflexes and disappears when the heart rate increases during activity.

Permanent atrial fibrillation

In a randomised clinical study of 30 patients with chronic atrial fibrillation, Hegbom and colleagues showed that 2 months of aerobic fitness and strength training yielded an increase in performance capacity on the exercise bike (41% at 17/20 on the Borg scale) and better heart rate control (5). Heart symptoms as well as quality of life were also significantly improved (6).

Atrial flutter

In the case of atrial flutter, the atrium beats regularly at approximately 250 beats per minute. The AV node normally blocks the impulses so that every second, third or fourth impulse is conducted. In physical exertion, there is always a risk that conduction of the impulses will increase to 1:1 (7). This gives worse circulation and many people experience a drop in blood pressure and shortness of breath. These patients may therefore require drugs to slow the AV conduction while they are exercising (beta blockers, calcium antagonists). These patients can be cured with ablation treatment and possible drugs against the flutter can be withdrawn.

Patients with pacemaker-ICD

An artificial pacemaker sends impulses to the heart at the rate set at the hospital and most have an activity sensor that increases the stimulation during physical exertion. The most common sensors respond to vibrations or movement and resting heart rate, maximal heart rate, and how quickly the heart rate should increase and decrease can be programmed. Different types of exercise training produce different responses in heart rate: running increases the heart rate a lot, cycling a bit less, and swimming yields a weaker stimulus and can in fact lead to a drop in blood pressure during exertion. An active person with a pacemaker should therefore adjust the programming to his or her activities. Occasionally other sensor systems are needed, for example, respiratory-controlled or impedance-controlled systems (the resistance in the system decreases when sympathetic tone increases).

Problems can also arise with exertion in ICD patients. The system can sometimes have trouble distinguishing between when the heart is beating rapidly due to exertion and serious arrhythmias that must be treated with pacing or shock. This can lead to the patient receiving shock unnecessarily. With proper programming, however, it generally works well, and according to a large controlled study ICD patients have good results from exercising (8).

Patients with tetraplegia

Spinal injuries in vertebrae Th 1–4 have an impact on the sympathetic innervation of the heart. This results in a poorer pulse increase and sometimes a drop in blood pressure during exercise. Arm training can improve the metabolism (9), however, and training with electrical stimulation of paralysed muscles yields better oxygen uptake, increased muscle and less fatty tissue (10). In most of these patients, however, an increase in heart rate cannot be used as a measure of exercise intensity (11).

Arrhythmias that occur in connection with exercise

Arrhythmias sometimes present during exercise load, and a rapid start and high load is sometimes used as a provocation test. Supraventricular arrhythmias induced by intensive exercise do not affect future risk of cardiac death (12). When a patient experiences arrhythmia during training or competition, the current activity should be stopped so that the stimulation from the sympathetic nervous system decreases, which reduces and normalises the heart rate. In atrial fibrillation and flutter in particular, continued activity can lead to a very strong and potentially dangerous heart rhythm.

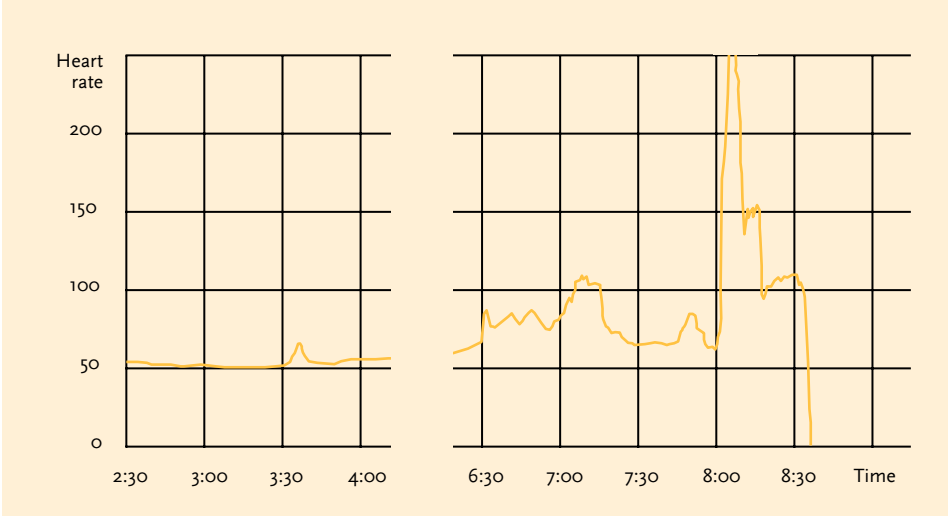

Figure 1. Trend curve from 24-hour ECG in a 50-year-old man with atrial fibrillation. At 8:10 he has an attack, and at 8:20 he attempts to "run it off". Maximal heart rate is 275 beats per minute.

Indications

Because good aerobic fitness can improve one's tolerance for disturbances in cardiac function, regular physical activity, preferably with the goal of endurance training, is of particular importance for this group of patients. If it has been present for a long time, a tendency to arrhythmia often has a negative impact on the level of physical activity, that is, the patient is afraid to exert him/herself for fear of provoking an arrhythmia or other symptoms during exertion, such as dizziness or fainting, resulting in a deterioration in aerobic fitness. In such cases, supervised training is especially important in the beginning and preferably with a physiotherapist specialised in heart diseases.

Prescription

The principles for exercise training applicable to other heart patients are also applicable for patients with arrhythmias. The prescription must always include definition of the frequency, duration and intensity. Many patients with arrhythmia problems consequently have other underlying heart disease, such as heart failure and/or coronary heart disease, which is why consideration must be taken to this when prescribing physical activity and training (13). For more on this, see the chapters on Heart Failure and Coronary Heart Disease.

The general goal of exercise training in heart disease is to improve aerobic fitness through loading the central circulatory system. When it comes to the central circulatory system, training is effective and less strenuous if as large muscle groups as possible are engaged in the training. One effective and non-injurious way to conduct this training is in intervals, alternating between harder and easier intervals of 3–5 minutes (14). In order to improve aerobic fitness in healthy, previously physically inactive individuals, a training intensity of about 50 per cent of the individual's maximal oxygen uptake (corresponding to light to moderate breathlessness) for 30 minutes, 3 times per week, appears to be completely sufficient to achieve an improvement of between 5 and 10 per cent (15). Each training session should begin with a warm-up phase and end with a relatively long cool-down phase, regardless of the activity being done. The cool-down phase is of particular importance for patients with arrhythmia problems as arrhythmias generally appear in this phase of training (13, 16). The principle of interval training should be applied both in group exercise classes and cycling, aquafit and other forms of training.

All training should begin with a successive warm-up of 6–10 minutes at an intensity of up to 50 per cent of maximal capacity and an exertion level of "very light to light", a perceived level of 9–11 on the Borg RPE scale (17). After warm-up, come 3 loading exercise sessions of 4–5 minutes each at an intensity of up to 50–80 per cent of maximal capacity and with an exertion level of "somewhat hard to hard (heavy)", 13–15 on the RPE scale. Between the loading intervals, follow lighter 4–5-minute intervals at an intensity of up to 50 per cent of maximal capacity and an exertion level of 9–11 on the RPE scale. All training should finish off with a successive cool-down and stretching of at least 6, and optimally 10, minutes.

Supervised training includes individual adaptation of both the load and the time for this during the training period itself. For patients with arrhythmia problems, it can be beneficial to begin by lengthening the loading interval by 2–3 minutes before the load level is increased, that is, a somewhat longer training session than those noted above.

Strength training, which used to be considered contraindicated for patients with coronary heart disease and heart failure, has been shown in recent studies to be a safe and effective way to train (18, 19). The requirement is that the load not exceed 60 per cent of 1 RM (Repetition Maximum = the weight that can be lifted through the entire exercise movement once) and that the number of repetitions per set be somewhat higher (12–15) than in traditional strength training. Sometimes strength training must be preceded by other training in order to enable fitness training, such as a brisk walk. One example of this is exercise training for patients with heart failure, whose muscles may be so weakened that lighter strength training, or peripheral muscle training (with a focus on endurance training), is the only type of exercise initially tolerated by the patient. The load level in muscular endurance training can be determined with the help of Borg's RPE scale or by establishing an RM. Here, the number of repetitions should be higher than 15 in each set, see also the chapter on Heart Failure.

Using a relatively high load enables one to achieve the beneficial effects of physical training more rapidly, but not all elderly persons or patients with concurrent heart failure can handle the heavier load. For these people, a parallel assessment of the central- and peripheral exertion levels should be made. Here, one chooses a lower central load (intensity up to 50–60 per cent of maximal capacity, exertion level 10–11 on the RPE scale), but can have a higher intensity in the peripheral exercise (exertion level 13–15 on the RPE scale).

Table 1. Description of training methods for patients with heart rhythm disturbances.

Training method	Intensity	Frequency (times/week)	Duration (min./session)
Aerobic fitness training	50–80% of VO_2 max* RPE** 9–15/20	2–3	45–60
Strength training	40–60% of 1 RM*** 1–3 sets of 8–10 exercises with 12–15 reps/set RPE 11–13/20	2–3	30–40
Muscle endurance training	40–80% of 1 RM*** > 15 reps/set RPE 9–15/20	2–3	45–60

* VO_2 max = Maximal Oxygen Uptake.

** Rating of Perceived Exertion, according to the Borg scale (17).

*** RM = Repetition Maximum. 1 RM corresponds to the maximum weight load that can be lifted through the entire exercise movement one time.

The choice of activity should always be preceded by a history of the patient's physical activity where consideration is given to fitness level, interests and requirements. Muscle training oriented to activities of daily life can be of particular benefit for the elderly, as declining muscle endurance and strength can prevent them from remaining socially self-reliant and living an independent life. Aerobic fitness training, which can be carried out in the form of brisk walks, jogging, cycling, swimming, exercise or aquafit classes, skiing, skating, dance or ball sports, depending on the individual's interests, should include 45–60 minutes of exercise, 2–3 times per week. For patients with implantable cardioverter defibrillators (ICDs), activities like jogging in proximity to heavily trafficked roadways, swimming and cycling can involve a certain risk of injury to themselves or others as there is a 10–20 second delay between detection of the arrhythmia and activation of the ICD shock (13). Other patients may also experience dizziness or disturbances to consciousness, and exercise training must be adapted with this in mind.

All training should be complemented with at least 30 minutes of physical activity daily, which need not be strenuous nor performed all at once and can include everything from regular moving about to walks and climbing stairs (20, 21). The goal is to achieve a daily energy expenditure of at least 660 kJ (approx. 150 kcal), a level for which there are documented health effects (10).

Functional mechanisms

Decreased heart rate variability is a risk factor for arrhythmia-related death in patients following a myocardial infarction (22). Aerobic fitness training in patients with coronary heart disease, or with heart failure, leads to increased heart rate variability as an expression of a relative increase in parasympathetic activity (23, 24). The latter study suggests a reduced arrhythmia risk, but larger studies than those currently available are needed to confirm this.

Functional tests

A patient history and physical examination complemented by an electrocardiogram (ECG) constitute minimum requirements before arrhythmia patients start training. If cardiovascular disturbance is suspected, or before training at a competitive level, echocardiography (ultrasound examination) of the heart is recommended to show possible structural heart disease and assess ventricular function. Echocardiography should be complemented with a stress ECG, which gives an overall assessment of function and can also give an idea of the tendency to arrhythmia in connection with maximal exertion.

All training at the physiotherapist should be preceded by some form of test in which aerobic and muscular fitness are evaluated before choosing the level of training. A stress ECG is recommended and should be conducted with current medication.

Interactions with drug therapy

Beta blockers and certain calcium antagonists (verapamil, diltiazem) are negative chronotropes, that is, they lead to a lowering of resting heart rate and reduced maximal heart rate. This generally limits maximal performance capacity, though the drugs themselves do not constitute a particular risk in connection with exertion. The underlying treatment indication (disease in question) is decisive for whether individual consultation is needed.

Contraindications

In general, the content and level/intensity of training should be adapted to the individual's requirements and conditions.

Absolute contraindications

Absolute contraindications are exercise-induced ventricular arrhythmias and atrial arrhythmias of high heart rate (>180–200 beats/minute), as well as recent onset and uninvestigated arrhythmia.

Relative contraindications

Tolerance for arrhythmias is normally reduced if the patient is hypoglycemic (low blood sugar level) and/or dehydrated. These factors are therefore important to consider in all types of exercise training, and especially in patients with heart disease, including those with isolated electrical disturbances.

Risks

Hypotension with impaired consciousness and in the worst case cardiac arrest.

References

1. Bergfeldt L. Atrioventricular conduction disturbances. Cardiac Electrophysiology Review 1997;1:15-21.
2. Jensen-Urstad K, Bouvier F, Saltin B, Jensen-Urstad M. High prevalence of arrhythmias in elderly male athletes with a lifelong history of regular strenuous exercise. Heart 1998;79:161-4.
3. Mont L, Sambola A, Brugada J, Vacca M, Marrugat J, Elosua R, et al. Long-lasting sport practice and lone atrial fibrillation. Eur Heart J 2002;23:477-82.
4. Karjalainen J, Kujala UM, Kaprio J, Sarna S, Viitasalo M. Lone atrial fibrillation in vigorously exercising middle aged men. Case-control study. BMJ 1998;316:1784-5.
5. Hegbom F, Sire S, Heldal M, Orning OM, Stavem K, Gjesdal K. Short-term exercise training in patients with chronic atrial fibrillation. Effects on exercise capacity, AV conduction, and quality of life. J Cardiopulm Rehabil 2006;26:24-9.
6. Hegbom F, Stavem K, Sire S, Heldal M, Orning OM, Gjesdal K. Effects of short-term exercise training on symptoms and quality of life in patients with chronic atrial fibrillation. Int J Cardiol 2007;116:86-92.
7. van den Berg MP, Crijns HJ, Szabo BM, Brouwer J, Lie KI. Effect of exercise on cycle length in atrial flutter. Br Heart J 1995;73:263-4.
8. Vanhees L, Kornaat M, Defoor J, Aufdemkampe G, Schepers D, Stevens A, et al. Effect of exercise training in patients with an implantable cardioverter defibrillator. Eur Heart J 2004;25:1120-6.
9. de Groot PC, Hjeltnes N, Heijboer AC, Stal W, Birkeland K. Effect of training intensity on physical capacity, lipid profile and insulin sensitivity in early rehabilitation of spinal cord injured individuals. Spinal Cord 2003;41:673-9.
10. Hjeltnes N, Aksnes AK, Birkeland KI, Johansen J, Lannem A, Wallberg-Henriksson H. Improved body composition after 8 wk of electrically stimulated leg cycling in tetraplegic patients. Am J Physiol 1997;273:R1072-9.
11. Valent LJ, Dallmeijer AJ, Houdijk H, Slootman J, Janssen TW, Hollander AP, et al. The individual relationship between heart rate and oxygen uptake in people with a tetraplegia during exercise. Spinal Cord 2007;45:104-11.
12. Bunch TJ, Chandrasekaran K, Gersh BJ, Hammill SC, Hodge DO, Khan AH, et al. The prognostic significance of exercise-induced atrial arrhythmias. J Am Coll Cardiol 2004;43:1236-40.
13. Pashkow FJ, Schweikert RA, Wilkoff BL. Exercise testing and training in patients with malignant arrhythmias. Exerc Sport Sci Rev 1997;25:235-69.
14. Åstrand P-O, Rodahl K. Textbook of work physiology. 3. edn. Singapore: McGraw-Hill Co; 1986.
15. Pollock ML. The quantification of endurance training program. In: Wilmore JH, Ed. Exercise and sport sciences review. New York: Academic Press Inc; 1973. p. 155.
16. Dimsdale JE, Hartley LH, Guiney T, Ruskin JN, Greenblatt D. Postexercise peril. Plasma catecholamines and exercise. JAMA 1984;251:630-2.

17. Borg G. Perceived exertion as an indicator of somatic stress. Scand J Rehab Med 1970; 2:92-8.
18. McCartney N. Role of resistance training in heart disease. Med Sci Sports Exerc 1998;30:S396-402.
19. McCartney N. Acute responses to resistance training and safety. Med Sci Sports Exerc 1999;31:31-7.
20. US Department of Health and Human Services. Physical activity and health. A report of the Surgeon General. Atlanta: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 1996.
21. Lee IM, Sesso HD, Paffenbarger RSJ. Physical activity and coronary heart disease risk in men. Does the duration of exercise episodes predict risk? Circulation 2000;102:981-6.
22. Kleiger RE, Miller JP, Bigger JTJ, Moss AJ. The multicenter post-infarction research group. Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. Am J Cardiol 1987;59:256-62.
23. Ståhle A, Nordlander R, Bergfeldt L. Aerobic group training improves exercise capacity and heart rate variability in elderly patients with a recent coronary event. Eur Heart J 1999;20:1638-46.
24. Larsen AI, Gjesdal K, Hall C, Aukrust P, Aarsland T, Dickstein K. Effect of exercise training in patients with heart failure. A pilot study on autonomic balance assessed by heart rate variability. Eur J Cardiovasc Prev Rehabil 2004;11:162-7.