Therapeutic education of patients with coronary heart disease

Training guide for general practitioners

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ABSTRACT

The guide is one of outcomes of a pilot project on the therapeutic education of patients with coronary heart disease, carried out between 1998 and 2003, in the Udine and Volterra areas, Italy. The aim of the project was to train general practitioners as trainers of groups of patients or patients and family with established coronary heart disease. The goal was to improve the outcomes of secondary prevention measures. The work was coordinated by the Cardiac Rehabilitation Centre, Udine, Italy, WHO Collaborating Centre for Research and Training in Rehabilitation and Secondary Prevention in Cardiovascular Diseases. The training was based on the principles of therapeutic patient education. The training sessions focused on basic knowledge of the structure and functioning of the cardiovascular system, guide symptoms and signs of emerging acute cardiovascular conditions, preventing and managing cardiovascular risk factors and principles of treatment. The ways of communicating health in the therapeutic education of patient were briefly presented. This publication summarizes the content of these sessions. It is aimed at general practitioners and other specialists interested in therapeutic education of patients with coronary heart disease. The guide is reference material for training patients or patients and family to develop better skills in the daily management of their health condition to improve the quality of life and to reduce the clinical manifestations of the disease.

Keywords

CORONARY DISEASE – prevention and control
CHRONIC DISEASE – prevention and control
TRAINING MATERIALS
MANUALS
PATIENT EDUCATION
PHYSICIANS, FAMILY

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Printed by Tipografia Doretti, Udine, Italy
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Acknowledgements

The editorial and writing groups greatly appreciate the efforts and comprehensive support of Filippo Marelli, Roberto Ferri, Alberto Piotrowski and Alessandro Fanzutto, Agency for Health Service N.4 “Medio Friuli”, Udine, Italy in implementing the pilot project on the therapeutic education of patients with coronary heart disease in 1998–2003.

The editorial and writing groups wish to extend sincere appreciation to Julija Brazdzionyte, Kaunas University of Medicine, Lithuania; Janos Feher, National Institute of Internal Medicine, Hungary and International Society of Prevention in Clinical Medicine; Brian Gaffney, Health Promotion Agency for Northern Ireland, United Kingdom; Ursula Härtel, Germany; and Daniele Tonelli, Freelance Translator from Italian to English, Udine, Italy, for perusing the manuscript and providing very valuable technical advice.

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Preface

Coronary heart disease remains a major health problem in terms of premature mortality, morbidity and disability. Available rehabilitative and preventive measures include lifestyle modification, treatment with drugs and intervention procedures. Their appropriate application should result in a better quality of life for people with coronary heart disease and in improved cost–effectiveness of health care. However, the success of these measures depends largely on the skills of patients in the daily management of their condition.

Patients with coronary heart disease are often not adequately supported to develop skills in the day-to-day management of their health condition. Moreover, they are not always adequately informed about the need for adhering to therapy, opportunities for self-management, as well as therapeutic choices and limitations of treatment.

Patient education is a strategy to improve patients’ active participation in the process of disease management and to enhance the application of measures of rehabilitation and secondary prevention.

To improve the quality of patient education the concept of therapeutic patient education has been developed (1). The aim of therapeutic patient education is the prevention of complications and the improvement of quality of life. Patients are trained in skills useful in self-management of their health condition and in adapting treatment to personal situations. Therapeutic patient education is provided by health professionals trained in educating patients. Therefore training of health professionals plays a central role in developing effective patient education.

The Cardiac Rehabilitation Centre in Udine, Italy has special expertise in patient education and in training health personnel to provide adequate health education to patients with coronary heart disease. A WHO Collaborating Centre for Research and Training in Rehabilitation and Secondary Prevention in Cardiovascular Diseases was established in 1994 at the Cardiac Rehabilitation Centre to assist WHO in promoting the quality of cardiovascular disease prevention and management and to disseminate accumulated experience to other countries.

In 1998, the Centre initiated a dialogue among specialists involved in managing coronary heart disease about organizing a pilot project on therapeutic patient education. The goal was to contribute to reducing coronary heart disease morbidity and mortality and to improve the quality of life by enhancing the education of patients. The project followed up the recommendations of the WHO Working Group on Therapeutic Patient Education published in 1998 (1).

The aims of the project were: a) to improve the quality of life and to increase satisfaction with the health care received by promoting awareness and understanding of the lifestyle modifications required while improving the skills to implement these changes and adhere to a disease management plan; b) to raise the profile of cardiac rehabilitation and secondary prevention by disseminating good practice through effective partnership with other specialists and by initiating, stimulating and supporting cardiac rehabilitation and secondary prevention of coronary heart disease at all levels of care.

The project was based on the principles of the training-the-trainers approach. Training of general practitioners (GPs) as trainers of groups of patients with coronary heart disease and/or their families was the core of the project. The training course lasted 20 hours, divided into two weekly
sessions of about two hours each. After the training was completed, GPs held therapeutic education courses in outpatient clinics.

The project team consisted of cardiologists, diabetologists, psychologists, experts in medical communication and GPs. The Cardiac Rehabilitation Centre in Udine coordinated the teamwork. The project was implemented in two demonstration areas: Udine and Volterra, Italy. The project had the following partners: Diabetes Unit, Udine General Hospital; Cardiac Rehabilitation Centre and Neurological Rehabilitation Centre, Volterra (Pisa) General Hospital; Associazione Amici del Centro per la Riabilitazione del Cardiopatico (Cardioclub), Udine; and Lauro Papi Foundation for Cardiology, Volterra.

This publication stems out of this project. It is hoped that this work will encourage physicians to practise therapeutic education as a strategy for secondary prevention of coronary heart disease. Secondary prevention can afford significant benefits to patients’ health.

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Reference
In memory of Professor Giorgio Antonio Feruglio

This publication is dedicated to Professor Giorgio Antonio Feruglio on the tenth anniversary of his death on 2 January 1996. His approach to cardiology was innovative and visionary. He believed passionately in preventing heart disease and pioneered a comprehensive approach to coronary heart disease emphasizing the importance of primary health care, of empowering patients and the community as well as of interdisciplinary integration and collaboration. Professor Giorgio Antonio Feruglio was an excellent communication expert and educator who inspired several generations of health professionals to take up the practice of preventive cardiology. He was a very talented organizer. Under his leadership, a great number of research projects and programmes were designed and implemented.

Professor Giorgio Antonio Feruglio was born on 13 December 1927. He graduated (summa cum laude) from the Medical School of Padua and specialized in cardiology. He worked as a research fellow in cardiology at the Graduate Hospital of the University of Pennsylvania under the direction of Professor S. Bellet and at the Cardiovascular Unit of the University of Toronto (1956–1959).

In 1959, at the General Hospital of Udine, Italy, he was charged with organizing the Centre for Prevention of Cardiovascular Diseases (later designated as a WHO Collaborating Centre for Research and Training in Monitoring and Prevention of Cardiovascular Diseases) and the Department of Cardiology that have always been aligned with international progress.

He taught at the University of Trieste and the University of Udine. He founded and directed the first school in Italy for cardiovascular technicians. He was a co-founder and editor in chief of the Giornale Italiano di Cardiologia, the Rivista di Cardiologia Sociale and the Giornale di Cardiostimolazione. A Fellow of the American College of Cardiology (1965) and of the European Society of Cardiology (1988), he was President of the European Group of Cardiac Pacing, Vice-President of the International Society of Cardiac Pacing and Electrophysiology and a member of the Council of Clinical Cardiology of the International Society and Federation of Cardiology. He organized and chaired the second European Congress of Cardiac Pacing and Electrophysiology in Florence, Italy in 1981.

After founding (1963) the Associazione Nazionale Medici Cardiologi Ospedalieri and (1975) the Associazione Italiana di Cardiologia Preventiva e Sociale, he was president of these professional organizations several times. He was also a member of the Ministerial Committee for Cardiology and Cardiac Surgery, and of Consiglio Superiore di Sanita. The Departments of Cardiology, Cardiothoracic Surgery and Preventive and Rehabilitative Cardiology were first united under his leadership in 1992.

In 1977, he started the Martignacco project, which became the basis for the Project for the Prevention of Cardiovascular Diseases in the Region of Friuli-Venezia Giulia, Italy. He actively participated in the WHO MONICA (monitoring trends and determinants in cardiovascular disease) project, in the WHO comprehensive cardiovascular community control programmes and numerous other international projects including the Anturan Reinfarction Trial, the Trasilol Post-Acute Myocardial Infarction Study, the ISIS-2 (International Study of Infarction Survival) and the GISSI (Gruppo Italiano per lo Studio della Sopravvivenza nell’Infarto Miocardico) and GISSI-2 studies.
**Introduction**

This guide has been written for the therapeutic education of patients with coronary heart disease (clinically manifested myocardial ischaemia, a history of angina pectoris, after acute myocardial infarction, coronary artery bypass graft surgery or percutaneous interventions).

The treatment targets of a patient with coronary heart disease are: better quality of life, improved well-being and the prevention of future coronary events. To achieve them, a patient needs a combination of therapies including lifestyle changes, pharmaceutical treatment, revascularization interventions as well as long-term follow-up and continuity between preventive and curative care.

Implementing these approaches requires active and knowledgeable participation of patients and their families. Patients’ understanding of their health condition is a major factor of the extent to which they agree to and follow a prescribed treatment: thereby patients need to learn about the nature of the disease. It is easier for a patient to follow a prescribed treatment correctly if adequate health counselling and information on action mechanism of intervention measures are provided. Patient education and counselling are therefore an essential part of disease management.

Therapeutic education provides information to help patients participate in the choice of treatment, advises on how to adopt a healthy lifestyle and to adhere to follow up of a disease treatment regimen, and recommends personal strategies for coping with their illness.

This guide is aimed at general practitioners (GPs) as trainers. Given their role as primary health care providers, GPs are the key for effective patient education. They should be proactive, offering their patients health counselling and improving their knowledge for managing current health problems and reducing the risk of future adverse events. The publication was designed as a reference material for preparing training seminars for groups of patients or their individual therapeutic education and health counselling. It was written following the concept and principles of therapeutic patient education outlined in the 1998 report of a WHO Working Group on Therapeutic Patient Education.
General introduction for the trainer

The guide for the therapeutic education of patients and their families aims to supply the content of most important aspects of the daily management of coronary heart disease. The subjects covered in the guide are based on patients’ needs and were chosen based on three decades of experience in patient education at the Cardiac Rehabilitation Centre in Udine, enriched by the advice of GPs involved in the project. The publication aims to provide health information that is evidence based, relevant and acceptable to the patients.

The publication consists of five chapters, reflecting the five training sessions of the project:

The first session provides basic knowledge about the structure and function of the cardiovascular system, the pathophysiology of atherosclerosis and the principles for managing coronary heart disease. An attempt was made to write the text in a simplified manner to allow GPs to communicate information so that patients can understand the information easily despite its complexity.

The second session advises on how to train patients on guide symptoms and signs indicating emerging acute cardiovascular problems.

The third session provides information on major risk factors of coronary heart disease and advice on modifying lifestyle for managing blood lipids, reducing elevated blood pressure and elevated blood glucose, stopping smoking, increasing physical activity and coping with stressful situations.

The fourth session covers the principles of pharmaceutical treatment of coronary heart disease and provides more detailed information on main classes of drugs. This session is written more for GPs than for patients to assist them in discussing with patients their individual disease management plan.

The fifth session addresses health psychology and the communication process in therapeutic patient education.

This guide does not address cardiopulmonary resuscitation. Boxed text provides additional in-depth information for GPs that might be useful in preparing their own training material.

The chapters of this publication are written based on the structure of training sessions used in the project. To ensure the consistency and homogeneity of the sessions and to teach in an interactive manner, trainers are recommended to use a standardized structure for each session.

Each session begins with an introduction, which includes the subject to be presented (“This session aims to give information about: “subject title”), description of the knowledge required in order to continue training (“Previous knowledge required”), the objectives of the session (“At the end of the session, the participant should be able to understand.”) and a very short summary of the information to be presented during the session (“Content: information and advice on …”). There are no special recommendations on audiovisual material. This aspect depends on the local situation, the relevance and availability of material and the preferences of the trainer.

In order to get patients and their families actively involved in the training process, the trainer should introduce himself or herself at the beginning of the session, explain his or her role as a
trainer and invite the participants to introduce themselves. Then the aim of the session is introduced, including information about the subject to be addressed, and the content of the session is presented. It is advisable to use simple words and explanations and to provide examples to ensure that the patient understands the subject. It is very important at the end of each session to evaluate whether the audience understood the information provided. For good feedback, the trainer is recommended to encourage the audience to ask questions and ask the audience questions.
1. First session. The cardiovascular system and atherosclerosis of the coronary arteries

Introduction

Subject
This session explains the structure and functioning of the heart, the blood vessels including the coronary arteries and how atherosclerosis of the coronary arteries develops. The principles for managing coronary heart disease are presented.

Previous knowledge required
None

Objectives
At the end of the session, the participant (patient or patient and family) should be able to understand:

- what the heart and the blood vessels are and how they function
- what the coronary arteries are
- what atherosclerosis is
- what happens when the coronary arteries are affected by atherosclerosis
- the basic principles for managing coronary heart disease in the long term.

Content summary
Information on the structure and functioning of the heart and blood vessels, how atherosclerosis of the coronary arteries develops and the basic principles for managing coronary heart disease.
Content

The human body is made up of billions of cells that join together to form tissues, organs and systems (such as the respiratory and digestive systems). These are nourished by oxygen and nutritive substances (such as fat, carbohydrate and protein) delivered by the blood. This essential supply must never be interrupted. Blood travels through the body by way of the circulatory system (also called the cardiovascular system), which consists of the heart and blood vessels. The heart sends blood throughout the body. From the moment it starts until the moment it stops beating, the human heart never stops working. In an average lifetime it beats billions of times without the slightest pause for rest. This capacity to maintain life has given rise over time to an air of mystery surrounding the heart. Modern technology has unravelled this mystery to some extent, but the heart retains its aura of fascination.

1.1 The structure of the heart

Position. The heart is situated in the centre of the chest, in the space between the two lungs, behind the sternum, in front of the spine and above the diaphragm (a large, flat muscle used for breathing).

Shape and size. The heart is shaped like a cone or a large upside-down pear. A normal adult heart is about the size of a clenched fist and weighs just over 300 grams.

Construction. The heart has to hold a liquid (blood). Thus, like any container, it is a hollow organ surrounded by a wall. The wall of the heart consists of muscle called myocardium. The heart is enclosed in a sac called pericardium.

The heart is divided into four chambers: two atria (right and left) and two ventricles (right and left) lined with a thin layer of cells called endocardium (similar to a wall lined with tiles). Going back to the image of a cone, the two atria are located at the top, which corresponds to the base of the cone. The atria are separated by a wall (the interatrial septum) to prevent blood from flowing between them. They serve as a storehouse for blood returning to the heart and act as additional pumps that assist in filling the ventricles.

The ventricles form the body and the apex of the cone and are also separated by a wall (the interventricular septum) to prevent blood from flowing between them. The function of the ventricles is to pump blood.

Blood flows between the atria and ventricles via circular openings with valves. The valve between the atrium and ventricle on the right side is called tricuspid because it comprises three cusps or flaps of endocardium. The valve between the atrium and ventricle on the left side is called bicuspid (it is formed of two cusps of endocardium) or mitral because, when open, it resembles a bishop’s hat or mitre. Both valves can open only towards the ventricles, ensuring that blood only flows one way.

1.2 The circulatory system

The circulatory system (the cardiovascular system) – the heart and blood vessels – is similar to a closed system of a pump and pipes such as a system of central heating with outflow and inflow pipes to the boiler. The circulatory system branches out all over the body, enabling blood to be
carried from the heart to all parts of the body and then returned to the heart. If all the blood vessels of an adult were placed end to end, they would be almost 100 000 km long.

The function of the circulatory system is to transport nutrients and oxygen to the tissues and to take away the waste products of metabolism (metabolism is a process of breaking down nutrients to produce energy and using energy to carry out the necessary functions in the body) including carbon dioxide.

1.2.1 Blood vessels

The main vessels of the circulatory system start from and return to the heart. The vessels carrying blood from the heart are called arteries. Those that bring the blood back to the heart are called veins; and those linking the arteries to the veins are called capillaries.

**Arteries.** The outflow vessels from the heart are the pulmonary artery (from the right ventricle) and the aorta (from the left ventricle). In the opening between the ventricles and the outflow vessels there are semilunar (“half-moon”) valves, called pulmonary and aortic valve respectively, that open only towards the blood vessels. The valves are three-cusped: they have three flap-like folds of endocardium. They prevent blood from flowing back from arteries into the ventricles when they relax.

The artery wall is composed of three layers of tissue: an external support layer of connective tissue, a middle strong elastic layer of muscle and an internal layer (lining) of cells forming a kind of wall tiling. This layer, called endothelium, is very smooth, allowing the blood to flow easily with minimal resistance.

The function of larger arteries is to “distribute” the blood to smaller arteries. The small arteries (arterioles) distribute the blood to the capillaries. The walls of arterioles, by constricting or relaxing, narrow or dilate their lumen and in this way regulate blood flow.

**Veins.** The inflow vessels to the heart are the two cava veins (inferior vena cava and superior vena cava) joining the right atrium and the four pulmonary veins joining the left atrium. The cava veins drain blood from the lower and upper parts of the body. The pulmonary veins return blood from the lungs to the heart.

The vein wall is also made up of three layers of tissue. They are thinner than those of the arteries because the pressure of the blood inside is lower. The blood must flow in the correct direction and not backwards. This is guaranteed by the presence of semilunar valves inside the veins, which act like bulkheads, ensuring that the blood flows in one direction only. The valves are also necessary to ensure that the blood flows against the force of gravity. Since the walls of the veins are relatively thin, the blood in them can be seen as a blue colour through the skin.

The veins collect the blood and also act as a reservoir that can contain different volumes of blood without changing pressure.

**Capillaries.** Capillaries are tiny blood vessels connecting the arterioles to the veins. In the capillaries, gases, nutrients and waste products of metabolism are exchanged between the blood and the pulmonary alveoli (small air sacs in the lungs) and between the blood and tissue cells.
Unlike the arteries and the veins, the capillaries are very thin and the red blood cells have to line up single file to pass through. The capillary walls are made up of only one layer of cells to permit the exchange of gases. This process takes place in two modalities:

- **blood-tissue cells**: the blood releases oxygen into the cells and takes out carbon dioxide; and
- **blood-pulmonary alveoli**: in the lungs, carbon dioxide is released from the blood into the alveoli and oxygen is transferred from the alveoli to the blood; the carbon dioxide-rich air is forced out from the alveoli during exhalation; the air rich in oxygen enters the alveoli during inhalation.

### Composition and function of blood

Blood is liquid tissue composed of cells and plasma. The cells include:

- red blood cells (erythrocytes) for the transport of gases (oxygen and carbon dioxide);
- white blood cells (leukocytes) that defend the body against external agents or antigens such as bacteria, viruses or fungi; and
- platelets (thrombocytes), which play a fundamental role in forming blood clots to stop bleeding.

Plasma is 90% water, and the rest is organic and inorganic substances:

- organic substances, including proteins (albumin, globulins, fibrinogen and others) produced by the liver; glucose, fat (lipoproteins containing triglycerides and cholesterol); hormones from the pituitary, thyroid, adrenal glands, pancreas and gonads; amino-acids and catabolites (waste products of cellular metabolism such as urea, uric acid, ammonia and others); and
- inorganic substances, including ions (sodium, calcium, magnesium, potassium, chloride, iodide, bicarbonate and others).

The blood, like a chemical motorway, connects all the organs in the body and has two major transport functions:

- it transports to every cell the necessary substances for the metabolic process, coming from the intestine (nutrients), liver (stored nutrients), the endocrine system (hormones secreted within the tissue) and lungs (oxygen); and
- transports the waste products of metabolism, which are broken down by and/or expelled from the liver, kidneys and lungs (carbon dioxide).

The other important functions of blood are to defend the body against infection or other external agents and to maintain a relatively stable body temperature as well as acid-base balance.

The chemical transporter of the two gases in the blood is a protein called haemoglobin, found in the red blood cells. When the blood passes through the lungs, oxygen attaches to the haemoglobin. When the blood passes through the tissues, oxygen is released from the haemoglobin to the cells. Then the haemoglobin bonds with carbon dioxide and takes it away from cells.
As a result of this mechanism, blood in the pulmonary veins and the aorta and its branches is rich in oxygen, because these vessels are downstream from the blood-pulmonary alveolus interface and upstream from the blood-tissue cell interface. Blood in the venae cavae and their affluents and the pulmonary artery is rich in carbon dioxide because these vessels are downstream from the blood-tissue cell interface and upstream from the blood-pulmonary alveolus interface.

1.2.2 Pulmonary and systemic circulation

The circulatory system of the body is organized as two connected systems: the pulmonary (lungs) and the systemic circulation. The two systems of circulation are not separate but, being part of a connected system, form a single loop.

The pulmonary circulation runs blood through the lungs. It includes the right atrium, the right ventricle, the pulmonary artery, the pulmonary capillaries and the pulmonary veins. The pulmonary circulation starts in the right ventricle of the heart, which pumps the blood into the pulmonary artery. The artery divides into two branches, right and left, leading to the right and left lung respectively, where they subdivide into vessels getting progressively thinner and thinner (like a tree structure spreading out from trunk to branches), finishing up as arterioles terminating in capillaries, which form a net around the alveoli of the lung, where they meet the air breathed in. With an identical process in reverse, the capillaries come together forming venules (small veins), which in turn come together to form veins that become larger and larger, finishing up as four pulmonary veins (two for each lung) that join the left atrium of the heart.

The systemic circulation supplies blood to all tissues of the body except the lungs. It includes the left atrium, the left ventricle, the artery called the aorta and its branches, the capillaries and the systemic venous system with the venae cavae. The systemic circulation starts in the left ventricle, which pumps blood into the aorta. The aorta climbs up and then curves in an arch, issuing arteries to the upper limbs and the brain, then continues through the chest and abdomen. Before dividing into the two branches for the lower limbs, it issues branches to other organs. Inside each organ, the vessels divide up into finer and finer vessels, ending in a network of capillaries that surround each cell. As in the lungs, the capillaries then join to form venules, and these join to make larger and larger veins, leading to the venae cavae joining the right atrium of the heart.

To understand how the heart functions as an oxygen pump, the blood circulation in the heart can be divided into the circulation in the right heart (the right atrium and the right ventricle) and the left heart (the left atrium and the left ventricle). Carbon dioxide-rich and oxygen-poor blood flows from the body into the right atrium. Through the right atrium it flows into the right ventricle. The right ventricle pumps the blood to the lungs, where the blood releases carbon dioxide and picks up oxygen. The oxygenated blood returns to the heart and enters the left atrium. Through the left atrium it flows into the left ventricle. The left ventricle pumps the oxygen-rich blood to all parts of the body.

1.3 The functioning of the cardiovascular system

The heart is the motor of the circulatory system. Its muscle works like a pump, pushing the blood to flow through the arteries and veins. This function is closely linked to the electrical properties of the cells of the myocardium and depends on the conduction of electrical signals within the myocardium.
The blood flows through the heart in two alternate phases known as:

- **diastole**: relaxation of the myocardium of the ventricles, allowing blood to flow into the cavity of the ventricles filling them; and
- **systole**: contraction of the myocardium, ejecting the blood from the cavity of the ventricles.

The sequence of the pumping function of the heart is as follows:

- Blood enters the atria from the venae cavae and pulmonary veins.
- The tricuspid and mitral valves open, allowing the blood from the atria to flow into the ventricles (diastole); at the same time the pulmonary and aortic valves are closed to prevent back flow from the pulmonary and aortic arteries into the ventricles.
- The myocardium of the ventricles contracts (systole), inducing in sequence: closure of the tricuspid and mitral valves (to prevent blood from flowing back into the atria), opening of the pulmonary and aortic valves and ejection of the blood into the pulmonary artery and the aorta.
- The myocardium of the ventricles relaxes (diastole), and the sequence is repeated.

The filling and contraction of atria and ventricles are timed to pump blood most efficiently and to respond to the changing demand of blood supply to various organs. The steady pumping action is ensured by the following four characteristics of the heart: **contractility** (inotropism), **excitability** (batmotropism), **conductibility** (dromotropism) and **rhythmicity** (chronotropism).

**Contractility**: Contractility is the strength of the heartbeat. Each contraction ejects about 60 ml of blood; if there are 80 contractions per minute, the left ventricle ejects 4800 ml (60 ml times 80 contractions) in one minute, approximately the whole amount of blood in the human body.

The initial intermittent blood flow, caused by the alternate systolic-diastolic movement of the heart, is made continuous by the main arteries, which fully dilate when the heart contracts and recoil when the heart relaxes, thanks to their elastic walls, giving the blood a further thrust towards the peripheral vessels. We can feel the arteries expanding and recoiling. As this is synchronized with the beating of the heart, the heart rate can be measured by counting the alternate expansion and recoil of the arteries. This is what we do when we measure the pulse.

**Stroke volume and cardiac output**

The amount of blood pumped in one heartbeat is called stroke volume. The amount of blood pumped in one minute is called cardiac output. Thus, cardiac output is determined by the stroke volume and the number of heartbeats per minute (the heart rate). The stroke volume and the heart rate are regulated by mechanical (such as the length of fibres of the heart muscle), neural and chemical factors.

As described earlier, the arterioles are small peripheral vessels that determine the flow of blood in the capillaries. They regulate the blood flow, dilating and constricting because of the action of the muscular layer of their walls, which is subject to the effect of the sympathetic autonomic nervous system and stimulating substances in the blood (such as the hormone angiotensin II, the most powerful constrictor of vessels). Resistance of peripheral arterioles to the blood flow is important in maintaining blood pressure.
Blood pressure

Arterial blood pressure is the force of blood against artery walls as the heart pumps the blood through the arteries. The blood pressure can be expressed as a numerical value in millimetres of mercury (the height of a column of mercury). The cardiac output and the resistance that the blood encounters flowing through peripheral arterioles are two factors influencing blood pressure.

Systolic (or maximum) pressure is the maximum pressure in the arteries during systole, when the blood is ejected from the heart. Diastolic (or minimum) pressure is the lowest pressure in the arteries during diastole, when the heart is relaxing.

The systolic pressure results from the systolic expansion of the aorta wall, spreading out like a wave along its branches and is mainly determined by the stroke volume and heart rate. The resistance exerted by the arterioles on the blood as it flows through them mainly determines the diastolic pressure.

Ensuring the normal functioning of the body requires responding to changes in the environment and at the same time maintaining equilibrium in the internal environment (ensuring stability of temperature, blood pressure, chemical composition and other characteristics). This capability of the body is called homeostasis and is dependent on various regulatory mechanisms.

Blood pressure should be maintained within certain relatively narrow levels to ensure adequate blood flow. When necessary, the regulatory mechanisms of homeostasis can modify blood pressure by adjusting the output of the heart through changes in heart rate and stroke volume and through changes in the volume of blood distributed to various parts of the body. Blood pressure can also be modified by varying arteriolar resistance and thereby increasing or decreasing the diameter of vessels. Blood pressure changes continually during the day.

- It increases during physical stress, including effort and activity, and during mental stress, both voluntary and involuntary.
- It decreases during physical rest and mental relaxation.
- It varies according to the circadian rhythm (day versus night and asleep versus awake), temperature, body posture, environmental noise, drugs taken, diet and other factors.

Excitability and conductibility. The muscles of the body, including the muscle of the heart, contract only when stimulated by a nerve impulse (an electrical signal). The process of transmitting a stimulus from the nerve cell to the muscle cell and induction of an impulse in the muscle cell is called excitation. The capacity of the heart muscle cells to respond to electric stimuli is called myocardial excitability.

The muscles of the body receive impulses to contract from the brain (the central nervous system). The heart has its own “nervous” system to stimulate the cells of the heart muscle to contract. The heart has two important distinguishing features regarding electrical activity. One feature that distinguishes the heart muscle from other muscles is that an electrical excitation occurring in one heart muscle cell can spread to other cells. Another distinctive feature is the presence in the heart muscle of specialized muscle cells that generate periodically an action potential (a nerve impulse). They are located in the right atrium and are called the sinoatrial node. The cells are self-excitatory and autonomic: that is, they initiate a rhythmic nerve impulse.
independently of the central nervous system. However, the self-induced rhythm can be modified by neural and hormonal factors. This regulation of the heart rate adjusts cardiac output to the changing needs of the body. The sinoatrial node is the governing part of the heart’s “nervous” system that conducts electrical impulses throughout the heart. The other parts of the system are: the atrioventricular node (located at the boundary between the atria and the ventricles); the bundle of His; the bundle branches and the Purkinje fibres (fibres spread out over the ventricles, connecting to the endocardium).

**Action potential**

Action potential refers to a short reversal of electric polarization of the membrane of a nerve or a muscle cell. It starts at one spot of the cell membrane and then spreads throughout the cell. In the nerve cell an action potential results in the production of the nerve impulse; in the muscle cell it results in the contraction of the cell.

To maintain the functions of the cell, molecules and ions (electrically charged chemical elements) move across the cell membrane (the outermost layer of the cell). When the cell is resting, the concentrations of positively and negatively charged ions are unequal on either side of the cell membrane. This causes a transmembrane voltage difference (potential). The potential is called the resting membrane potential. The inside of the cell is electrically negative (there are more negatively charged ions inside the cell) relative to the outside, and therefore the potential is said to be negative. In tissues, capable of being stimulated (nerve or muscle cells), a chemical or electrical stimulus can activate the ion channels in the membrane and change its polarity: positively charged sodium ions start moving through the membrane channels into the cell and the potential becomes positive. This reversal of membrane polarity is called depolarization. It refers to the generation of electric current, called action potential. Then the cell membrane starts recovering its original polarity. This process is called repolarisation, during which the membrane resting potential is re-established. The electric activation of a muscle cell is followed by its mechanical contraction.

The underlying biomolecular mechanism of this process represents the movement through the cell membrane channels of three ions: sodium, calcium and potassium. Depolarization takes place due to the inflow of sodium across the cell membrane; the outflow of potassium ions ensures membrane repolarization so that the process can be repeated; and calcium, interacting with the contracting proteins, leads to the contraction of a muscle cell.

The resting membrane potential corresponds to the diastole of the myocardium, and the action potential corresponds to the systole.

The conduction system of the heart synchronizes the cardiac cycle. Excitation starts as repetitive spontaneous self-excitation of cells in the sinoatrial node and spreads to the myocardium of the atria (causing the atria to contract and push the blood into the ventricles); then, it is channelled into the atrioventricular node and spreads out through the conduction system to the myocardium of the ventricles (causing a coordinated ventricular contraction or systole). Then ventricles relax (diastole) and blood starts flowing again in the atria. This process of systole and diastole is called cardiac cycle. Such a cycle ensures productive pumping of blood.

**Rhythmicity.** The sinoatrial node is the pacemaker of the heart. It determines the heart rate: the number of beats in one minute. Although the sinoatrial node generates spontaneously an action
potential at the rate of about 60 per minute, the heart rate is not constant but varies considerably below and above this level. This variability in the heart rate is influenced by the tone of the nervous system called the autonomic nervous system.

**Autonomic nervous system.** The autonomic nervous system helps the body adjust to changes in the environment: that is, it supports homeostasis. The autonomic nervous system transmits nerve impulses from the central nervous system regulating the function of the smooth muscles in the organs (including the heart muscle) and vessels walls. The chemical substances transmitting nerve impulses are called neurotransmitters. They are of two types: catecholamines and acetylcholines.

The autonomic nervous system regulates a great variety of vital body functions: the beating of the heart, breathing, blood pressure, the work of the digestive system and the secretion of glands (such as the production of insulin – a hormone produced by the pancreas and involved in metabolizing sugar) and others. Evidence shows that the conscious mind can influence the autonomic nervous system. However, it was earlier believed that these functions could not be influenced by the will of an individual and thus believed to be basically involuntary (“I can’t stop my heart from beating or stop it from beating rapidly if I am agitated”), governing themselves without our conscious mind. The term autonomic has therefore been used historically.

The autonomic nervous system is divided into the sympathetic and the parasympathetic systems.

- The sympathetic nervous system increases heart rate, heart function and the consumption of oxygen by the heart as well as myocardial excitability. It increases blood pressure, dilates bronchi and stimulates the function of the digestive system and the urinary bladder. It acts during waking hours and in times of physical and/or mental stress. The sympathetic nervous system prepares the body for emergency situations (when fight-or-flight response is needed) such as physical danger, shortage of water, extreme weather conditions or other stressful situations.

- The parasympathetic nervous system reduces heart rate, function and the consumption of oxygen by the heart. It reduces blood pressure and relaxes the digestive system and the urinary bladder. The parasympathetic nervous system acts during sleep and during physical rest and mental relaxation. It keeps the body running smoothly with minimum expenditure of energy (rest-and-repair reaction).

The nerve fibres of the autonomic nervous system innervate the sinoatrial node and release chemical substances that influence the heart rate and its contractility. The heart receives nerve fibres from both the sympathetic and the parasympathetic systems. The walls of most blood vessels are innervated only by sympathetic nerves.

### 1.4 Coronary arteries and coronary circulation

The arteries supplying blood to the walls of the heart are called coronary because they form a kind of crown (in Latin: corona) around the heart. The coronary arteries branch off the aorta just above the aortic valve and encircle the heart externally. There are two: right and left.

The left artery supplies blood to the left ventricle. The artery, after the common trunk, divides into a circumflex branch and an anterior descending branch, which runs through the furrow
between the two ventricles. The right artery supplies the right side of the heart and part of the left ventricle. Usually, it has only one main branch.

These vessels are situated on the epicardium, the external surface of the heart. They are relatively large: the diameter varies from 1.5 mm to 5 mm in the main branches. Intramural (inside the myocardium) twigs branch off, forming the capillary network where gases, nutrients and the waste products of metabolism are exchanged with the cells of the muscle. Following the exchange, the blood flows into larger and larger veins, which become more superficial and finally flow into the coronary sinus (a large vein), which opens into the right atrium.

The coronary arteries transport the blood which gives the myocardium, as all the other organs, oxygen, nutrients and takes away the waste products of metabolism to ensure that the heart maintains its pumping function. Blood flows through the coronary arteries mostly during diastole. The heart has the highest demand for oxygen compared to any other part of the body. The oxygen consumed by the heart depends on its muscle mass, contractility, systolic blood pressure and the heart rate. The heart extracts about 75% of the oxygen from the blood flowing through the coronary arteries. The ability of the myocardium to extract more oxygen is very limited. Therefore when the oxygen demand of the myocardium rises, increased oxygen supply to the myocardium depends on increased blood supply by coronary arteries. The supply of blood by coronary arteries increases through an autoregulatory mechanism that is influenced by neural, metabolic and hormonal factors. Coronary arteries are innervated by both the sympathetic and the parasympathetic nervous systems. Stimulation of the sympathetic system results in dilatation of coronary arteries.

### 1.5 Atherosclerosis of the coronary arteries

#### 1.5.1 The process of developing atherosclerosis

Coronary artery disease is a process affecting the walls of the arteries of the heart resulting in an obstruction to the flow of blood through these vessels. The normal state of the vessel wall creates no obstacle to the flow of blood. The lumen of a coronary artery can be narrowed (luminal narrowing is called stenosis) when a segment of the wall thickens. This process of arteries thickening and narrowing is called coronary atherosclerosis and is brought about by two linked processes called atherogenesis and thrombogenesis. Knowing the concepts of atherogenesis and thrombogenesis is useful in understanding the intervention measures for preventing and treating atherosclerosis.

Atherogenesis is the formation of atherosclerotic plaque (atheroma) on the internal layer of the wall of the artery. Thrombogenesis is the formation of a blood clot (thrombus) in a vessel by the thrombocytes circulating in the blood.

Atherogenesis starts with fatty deposits. Fibrous tissue then grows around a fatty deposit creating an atherosclerotic plaque, which protrudes into the lumen of the artery narrowing it. Plaques vary in shape, size and composition, even in the same person. An atherosclerotic plaque can contain little fat (cholesterol) and have a thick fibrous shell or can be swollen with fat and surrounded by a thin fibrous shell. The former type of a plaque is unlikely to ulcerate and/or desquamate and therefore is termed stable. The latter type can ulcerate and/or desquamate (which means that it loses its lining of cells or “tiles”) and is therefore considered unstable.
When a plaque full of cholesterol ulcerates or desquamates, it releases the cholesterol content and various fragments of the plaque to the circulation. This triggers the formation of a blood clot by thrombocytes circulating in the blood. The atherosclerotic plaque exposes itself to the aggregating thrombocytes, allowing them to form a blood clot, which leads to varying degrees of obstruction of the lumen. The thrombus can obstruct the vessel like a cork, jeopardizing blood flow in that vessel.

Atherosclerosis can affect one or all coronary arteries and their branches. This process can be diffused or localized to a varying degree. Atherogenesis is slow (it may take up to 20–30 years from the first fatty streaks before the coronary arteries are critically narrowed), whereas thrombogenesis may be quite rapid.

Clinical manifestations of coronary atherosclerosis usually develop when the coronary arteries are narrowed at least by 75%. Atherosclerotic plaques compromise blood flow in the coronary arteries, especially when the demand for oxygen is increased. The disease can manifest in the following forms: chest pain (angina pectoris), heart attack (myocardial infarction), heart rhythm disturbances (arrhythmia), sudden cardiac death or congestive heart failure.

The severity of coronary heart disease varies widely. Some people have no symptoms, some have occasional mild chest pain, some have more pronounced chest pain, and some have such severe symptoms that carrying out daily activities is difficult. The symptoms are caused by myocardial ischaemia.

1.5.2 Myocardial ischaemia

Ischaemia means a reduction of blood flow. Myocardial ischaemia is a deficiency of blood flow to the heart muscle when blood flow in the coronary arteries does not provide enough oxygen to meet the needs of the myocardium. The most common cause of myocardial ischaemia is coronary atherosclerosis. Insufficient blood flow most often results from the chronic or acute (for example, thrombus formation) narrowing or occlusion of the lumen of a segment of a coronary artery due to advanced atherosclerotic process. A reduction or loss of blood flow to the heart muscle makes the heart muscle below the narrowing or occlusion temporarily or permanently ischaemic. The myocardium cells starved for oxygen start suffering within minutes, and irreversible damage to the cells may occur in about 40 minutes. The longer re-establishing blood supply takes, the more significant the damage. The dead heart muscle cells cannot be replaced by new muscle cells. They are replaced by scar tissue, which is less elastic and does not contract as the muscle. As a consequence, the pumping function is less effective. In some cases, the scarred area becomes thin and dilated as a sack (it is termed an aneurysm), which further weakens the pumping function of the heart.

Blood flow can resume in the affected region either through collateral arteries from the distal vessels beds or by reopening the vessel segment that was occluded. If blood flow is re-established within one hour, loss of cell viability does not occur or is minimal.

1.6 Risk factors

The cause of atherosclerosis is not known. However, a wealth of data from various studies exists on risk factors or conditions that are associated with an increased risk of atherogenesis. Risk factors can be divided into those that cannot be changed and those that can be modified or managed.
Atherogenic risk

Coronary risk factors are features that have been associated with an increased risk of coronary artery disease. Their presence identifies persons who are more likely to develop the disease in the future. The overall risk of having a heart attack is much higher if a person has several risk factors. Risk factors multiply each other’s effect of increasing the risk of coronary heart disease. The negative impact of more than one risk factor is not arithmetic but exponential. It can be quantified as follows:

- The presence of one risk factor implies that the atherogenic risk is 1 on a relative scale.
- The presence of three risk factors implies that the relative atherogenic risk is 9 and not 3, because the risk does not grow arithmetically ($1 + 1 + 1 = 3$) but exponentially ($3 \times 3 = 9$).

Conversely, eliminating two of three risk factors would reduce the relative atherogenic risk from 9 to 1.

Risk factors that cannot be changed include age, gender and a family history of early coronary heart disease.

- **Age.** The risk of atherosclerotic cardiovascular disease increases sharply with age (45 years or older for men and 55 years or older for women).
- **Gender.** At all ages men have a higher risk of developing atherosclerosis than women.
- **Family history of early coronary heart disease.** This includes having a brother or father who had coronary heart disease before the age of 55 or a sister or mother before the age of 65.

Modifiable risk factors include disorders of blood lipids (dyslipidaemia), smoking, elevated blood pressure, obesity, diabetes, sedentary lifestyle and stress.

- **Disorders of blood lipids (dyslipidaemia).** There is a causal link between an elevated level of blood cholesterol and coronary heart disease. Cholesterol is the main ingredient of atherosclerotic plaque. The higher the level of blood cholesterol, the higher the risk of developing a heart attack. If other risk factors (especially high blood pressure and smoking) are also present, the risk of coronary heart disease is even higher.
- **Smoking.** The health hazards of tobacco are very well established. Tobacco and tobacco smoke contain substances that increase the risk of cardiovascular diseases and other diseases. The risk of coronary heart disease is 2–4 times higher among smokers than among nonsmokers. Smoking significantly increases the risk if other risk factors are present. Passive smoking also increases the risk of coronary heart disease.
- **High blood pressure.** Elevated values of both systolic and diastolic blood pressure are an important and well-established determinant of the risk of a heart attack. Lowering blood pressure decreases the risk of heart attack and stroke.
- **Obesity.** Obesity, particularly abdominal, is linked with various risk factors, especially disorders of glucose metabolism, high blood pressure and dyslipidaemia. Even a relatively small decrease in body weight may reduce blood pressure and improve the blood lipid profile and glucose regulation.
Diabetes. Diabetes increases significantly the risk of coronary heart disease. People with diabetes are considered to have the same risk of a coronary event as people with established coronary disease since diabetes accelerates atherogenesis.

Sedentary lifestyle. Sedentary lifestyle is a risk factor for coronary heart disease. Physical activity provides many health benefits. Physical exercise ameliorates the blood lipid profile and lowers blood pressure. It helps control body weight and blood glucose and in coping better with stress.

Stress. Various psychosocial factors such as stress, hostility, anger and others have been associated with the risk of coronary heart disease. There is also some evidence that there are links between stress, health-related behaviour, and the development of coronary heart disease. For example, some people in stressful situations eat more, start smoking or reduce their physical activity.

A person with coronary risk factors can be compared to a poorly maintained car with ineffective brakes, which is statistically more likely to be involved in an accident in the future compared with a well-maintained car with good brakes. People with coronary heart disease (a history of heart attack, clinical evidence of myocardial ischaemia or a history of percutaneous transluminal coronary angioplasty or coronary artery bypass graft surgery) are at very high risk of new coronary events. Special attention should be paid to managing these people’s risk factors.

Evidence shows that, among people with established coronary heart disease, changes in lifestyle – especially stopping smoking, eating a healthy diet and increasing physical activity – are likely to reduce the risk of both recurrent clinical manifestations of coronary heart disease and the occurrence of other vascular events, especially stroke. These changes may also reduce the need for medication and the risk of other diseases (such as lung cancer). Deriving maximum benefit from reducing risk factors requires tackling them all at the same time.

1.7 Diagnosing atherosclerosis of the coronary arteries

Coronary heart disease is frequently a silent disorder since symptoms do not occur until advanced stages of the atherosclerotic process. Various diagnostic tests aim to determine whether the coronary arteries are narrowed from atherosclerosis, to assess the extent of the narrowing and evaluate the clinical manifestations of the disease. New tests are constantly being developed to recognize the disease as early and accurately as possible.

The diagnostic tests may be indirect or direct. They may be nonprovocative or provocative, inducing symptoms and signs, and noninvasive or invasive (for example, using a catheter).

The basic indirect, nonprovocative and noninvasive tests include electrocardiogram, dynamic electrocardiogram and resting echocardiogram.

Electrocardiogram. The electrical activity of the heart is evaluated by an instrument called electrocardiograph which makes graphic records. Such a record is called an electrocardiogram (ECG). It shows the heart's rate and rhythm. An ECG also can give information about current or past heart attacks, enlargement of the heart and whether the heart muscle is getting enough oxygen-rich blood.

Dynamic electrocardiogram (Holter monitoring). This test is the ambulatory recording of an ECG, for a longer period of time (24–48 hours), to determine whether there are transient
problems (problems that come and go) such as heart rhythm disturbances or a shortage of oxygen to the heart muscle. The test is also useful for correlating the symptoms and signs of heart disease.

**Resting echocardiogram.** This is a test to provide information about heart functioning (for example, determining whether there is persistent alteration in the movement of a segment of the myocardium; evaluating the ejection fraction – the fraction of the diastolic blood volume that is ejected from the heart during systole) and the structure of the heart (for example, assessing its dimensions and the status of the heart valves). An echocardiogram is produced by moving a device that sends sound waves across the chest wall. The sound waves bounce off (“echo”) the heart. Then they are displayed on a computer screen as a two-dimensional picture.

The basic indirect, noninvasive and provocative tests include an exercise stress test, myocardial scintigraphy and a stress echocardiography test.

**Exercise stress test.** This test is used to record and analyse the ECG during exercise, since certain heart problems show up only when the heart works hard. After blood pressure and heart rate are measured and a resting ECG is taken, blood pressure, heart rate and an ECG are recorded at the end of each stage of exercise as well as at the onset of the ischaemic response of the myocardium (angina and/or ECG changes). The exercise stress test is performed with a cycle ergometer (constant pedalling rate with gradually increasing load) or with a treadmill (walking while gradually increasing the speed and angle of incline of the ramp).

**Myocardial scintigraphy.** This is a test to detect the extent of the myocardium not receiving an adequate supply of oxygen. It is performed with exercise or with a drug that produces the same increase of blood flow as exercise. A small amount of a radioactive substance (tracer) is injected into the blood. The substance mixes with the blood and enters the cells of the myocardium. The heart is imaged using a special camera. If part of the myocardium does have a reduced blood supply, this part will accumulate less tracer and this can be seen in the images.

**Stress echocardiography test.** This is a test to detect an abnormal movement of a segment of the heart muscle. The test is done by comparing an echocardiogram obtained at rest with an echocardiogram obtained after stress induced either by exercise or by drug administration.

The basic diagnostic direct and invasive tests include coronary angiography and an electrophysiology study.

**Coronary angiography.** Coronary angiography visualizes the coronary arteries. This enables to study the structure of the coronary arteries, including the location, size and type of atherosclerotic plaques. The test assesses the extent of arteries occlusion and helps in determining whether percutaneous transluminal coronary angioplasty or coronary artery bypass graft surgery is indicated. The test is performed by injecting radiopaque contrast dye through a fine tube (catheter) put into an artery of the leg or the arm and then producing X-ray images (angiograms) of the lumen of the coronary arteries while the heart is working. This test remains the most certain way to diagnose and assess the extent of coronary lesions.

**An electrophysiology study.** This test studies abnormalities of the conduction system of the heart that are responsible for most disturbances of heart rhythm. Special electrode catheters are inserted into the heart through peripheral blood vessels and positioned in specific areas. Then the electric signals of the heart are recorded and analysed.
1.8 Managing atherosclerosis of the coronary arteries

Coronary heart disease is a chronic disease and requires lifelong management. The aims of treatment are to:

- reduce the clinical manifestations: relieve symptoms and reduce coronary events
- slow, stop or even reverse the progression of atherosclerosis.

The disease is treated in several ways, depending on the seriousness of the disease and clinical manifestations. Management strategies include:

- changing health-related lifestyle to manage the risk factors of atherosclerosis (discussed in the chapter on risk factors);
- administering drugs to:
  - reduce the workload on the heart, thereby reducing the heart’s demand for oxygen;
  - increase or re-establish coronary blood flow, thereby improving oxygen supply to the myocardium;
  - reduce the risk of thrombogenesis;
  - manage risk factors (such as elevated blood cholesterol) when lifestyle measures are insufficient;
- applying intervention procedures to relieve coronary artery obstruction caused by atherosclerotic plaque (with or without thrombosis) to ensure an adequate blood supply to the myocardium and prevent myocardial infarction. There are three main procedures.
  - Treatment with drugs: thrombolysis – a drug (enzyme) is injected (intravenously or through intracoronary route) to dissolve the blood clot in the affected coronary artery. This treatment acts on the thrombus, “dissolving” it. This treatment re-establishes blood flow but has no effect on the atherosclerotic plaque.
  - Invasive treatment: percutaneous transluminal coronary angioplasty – this is a procedure during which a thin balloon dilatation catheter is inserted into a femoral, radial or brachial artery and advanced to the narrowed segment of the affected coronary artery. Then the device is inflated to stretch the artery to reopen it for blood flow (the plaque is pushed outward). A stent (a small metal coil) might be put into the artery where the blockage was to hold the artery open. This treatment acts on the plaque (reduces the thickness of the atherosclerotic plaque compressing it), thereby increasing the space for blood flow. Other forms of angioplasty have also been developed such as lasers or drills to mechanically clear the narrowed segment of the artery. If angioplasty does not widen the artery or if complications occur, coronary artery bypass graft surgery may be needed.
  - Surgical treatment: coronary artery bypass graft does not act on the atherosclerotic plaque but bypasses the occluded or narrowed coronary artery segment like a bridge, thus creating an alternate route for blood to flow. One or more grafts (vessels – veins from the legs or the internal mammary artery) are implanted between the aorta and the affected coronary artery. Bypass surgery relieves the symptoms of heart disease but does not cure it.
Intervention to relieve arterial occlusion in an urgent situation (impending infarction) should be rapid. It is most effective if given within an hour after the onset of symptoms. The sooner treatment starts, the greater the chance of full recovery. In such a situation there are three options:

- percutaneous transluminal coronary angioplasty – the best option but not always available at a nearby health care institution;
- thrombolysis – generally more available than percutaneous transluminal coronary angioplasty;
- coronary artery bypass graft surgery if percutaneous transluminal coronary angioplasty fails.

When the intervention procedure to ensure long-term patency of the affected coronary arteries is elective, the choice of procedure (percutaneous transluminal coronary angioplasty or coronary artery bypass graft surgery) to apply depends on several criteria. Coronary anatomy and clinical assessment will be the most important decision criteria.
2. Second session. Guide symptoms and signs in heart disease

Introduction

Subject
The aim of this session is to train the patient and his or her family to recognize and evaluate the guide symptoms and signs, which may enable the patient with coronary heart disease to assess the seriousness of an acute health condition, recognize a life-threatening situation early and take the best possible action to prevent avoidable complications.

Previous knowledge required
Knowledge of the structure and the functioning of the heart and blood vessels and how coronary atherosclerosis develops.

Objectives
At the end of the session, the participant (patient or patient and family) should be able:

• to recognize the guide symptoms and signs of heart disease and describe them correctly
• to evaluate the seriousness of symptoms and signs
• to determine the most suitable action to be taken regarding his or her health situation.

Content summary
Information on guide symptoms (dyspnoea, chest pain, palpitation, vertigo, lipothymia and syncope) and signs (pulse and blood pressure) and advice on the action to be taken, including the kind of decisions, options and instructions in relation to the symptoms experienced and signs observed.
Content

As described earlier, atherosclerotic plaques can be classified as stable and unstable. Stable atherosclerotic plaques manifest as predictable chest pain and are managed by administering appropriate medication, relevant intervention procedures (such as percutaneous transluminal coronary angioplasty or coronary artery bypass graft surgery) and by changes in health-related lifestyle. Unstable plaques can cause a sudden severe health problem (for example, myocardial infarction) that might be life-threatening. Such a situation requires rapid and aggressive management. Early hospitalization in case of acute myocardial infarction can save lives and increase the probability of successful treatment. In terms of emergencies and the need to take decisions, such an acute manifestation of coronary heart disease may be compared, for example, with a situation of a trauma, fracture or haemorrhage.

Symptoms are alterations in the normal perception of one’s body in case of illness. The patient may refer to them as health problems or troubles. Symptoms are subjective. Signs are indicators of health status. They can be assessed and measured by an observer (in this case, by a patient or his or her family): that is, they are objective.

Symptoms and signs indicate manifestation of a disease. Table 1 shows the guide cardiovascular symptoms and signs.

### Table 1. Guide symptoms and signs

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Signs</th>
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<tbody>
<tr>
<td>Dyspnoea (a feeling of shortness of breath or uncomfortable feeling of</td>
<td>Pulse</td>
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<tr>
<td>breathing)</td>
<td>Blood pressure</td>
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<td>Chest pain</td>
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<td>Palpitation (an uncomfortable awareness of the heart beating)</td>
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<td>Vertigo (dizziness), lipothymia (the beginning of fainting) and syncope</td>
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<tr>
<td>(a transitory loss of consciousness)</td>
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2.1 Symptoms

The patient or the patient and family must be taught to: 1) recognize, 2) evaluate, 3) emphasize a symptom, 4) decide whether or not it constitutes an acute health problem and 5) take action.

1. **Recognition: what is it?**

The aim is to provide a comprehensive description of a symptom so that the patient can recognize it.

2. **Evaluation: how is it?**

The aim is to teach the patient to contextualize and to measure a symptom. The basic measurement parameters need to be given: for example, distance, number of steps, how many pillows to sleep on, body weight, volume of urine. The following symptom characteristics should
be evaluated: intensity, duration, frequency, relation to treatment, when and how a symptom appears and disappears and, if possible, pulse and blood pressure measurements during the symptom.

3. Emphasis: how serious is it?

The aim is to help the patient assess the seriousness of a symptom in relation to its history, characteristics and association with other guide symptoms. This is probably the most difficult stage in training the patient. However, the patient should be made aware that a symptom is relevant if it never occurred before or if the symptom worsens. Generally, a symptom is more serious if it worsens rapidly than if it worsens slowly. Similarly, a more intense and prolonged symptom is more serious than a less intense and shorter one. Finally, an isolated symptom is less serious than one associated with other symptoms.

Ideally, the patient should be able to recognize a symptom (for example, dyspnoea is recognized as difficult or laboured breathing) and be able to describe it in such terms as exercise tolerance (stairs or running), duration and intensity. The patient should be able to emphasize the symptom. For example, assessing the seriousness of dyspnoea, the patient should recognize whether it had not occurred before and then appeared or it had occurred and then got worse. The patient also should assess the symptom in relation to variation in duration, intensity and evolution. For example, a threshold of dyspnoea that declines from tolerating climbing 20 steps to tolerating 5 steps is more serious than a reduction in tolerance from 20 to 15 steps; a reduction in tolerance is more serious if it takes place within one day rather than over a month; dyspnoea at rest is more serious than dyspnoea as a result of effort. Dyspnoea in association with chest pain is more serious than if it occurs alone. This is what is meant by assessing the relevance (emphasis) of each guide symptom in relation to variation that can basically be described as rapid versus slow, large versus small and long versus short, and in relation to the association of two or more guide symptoms. Further, the patient should understand that a symptom accompanied by abnormal signs (for example, dyspnoea accompanied by slow or rapid and irregular pulse and/or abnormal blood pressure) is more relevant than a symptom with normal signs (for example, dyspnoea with normal pulse and normal blood pressure).

4. Decision: is it a problem? What should I do about it?

The patient has recognized and evaluated the symptom and assessed its seriousness and now must decide whether there is an acute problem and, if so, what to do. The patient may decide as follows.

• There is no acute serious health problem and there is no need to take immediate action.
• There is a serious acute problem requiring calling the medical emergency service number.
• There is a grey situation: the patient does not know how serious the problem is and does not know what to do. In such a situation, the patient should make an appointment with his or her GP or consult the physician on call.

5. Action: how should I react?

The patient should be taught how to ask for help. The following advice is useful.

• His or her GP has a telephone number and the hours during which the GP can be contacted directly or indirectly (such as a phone answering service). The GP has office
hours and makes house calls; details about office hours and arrangements for house calls must be requested. Outside these hours, an on-call physician should be contacted.

- The on-call physician service, if available, can be contacted.
- The medical emergency service has a phone number active 24 hours a day, 7 days a week. There is a central operating service and an operator will request information about the service required (such as an ambulance or other).
- The GP or on-call physician may decide to refer the patient to a specialist. The specialist will also have certain times and arrangements for visits.
- Finally, the patient should keep a file with selected records of his or her health condition (hospital discharge letters, reports of health care visits, data from clinical examinations and details of treatment). The GP can help the patient gather this documentation.

Training patients on how to recognize, evaluate, emphasize symptoms and take relevant action requires remembering the following points.

- Break up symptom assessment into five phases: recognition, evaluation, giving emphasis, decision and action.
- Illustrating all possible situations and appropriate decisions is very difficult.
- Providing examples and discussing them with patients can be very helpful.
- Teach the patient or the patient and family that prolonged chest pain or brief but rapidly worsening chest pain, acute and worsening dyspnoea or marked and/or poorly tolerated palpitation and syncope require the medical emergency service.
- Emphasize the need to seek medical attention if the above symptoms are present, since early diagnosis and treatment may be life saving.
- Slight dyspnoea and brief chest pain that appear or worsen slowly, slight vertigo with low pulse and low blood pressure indicate that the GP or on-call physician should be contacted.
- In contrast, “sighing” dyspnoea, clearly atypical chest pain, well-tolerated brief or momentary palpitation or occasional slight postural vertigo without accompanying variation in pulse and blood pressure can be considered as a minor health problem or no problem at all.
- Dubious symptoms will likely be the most frequent. The role of the GP here is fundamental: over time, a patient well trained by a GP will learn not to worry without cause.

2.1.1 Dyspnoea

1. Recognition

Dyspnoea is awareness of the effort of breathing or shortness of breath; breathing is rapid but not deep. It is a common feature of heart disease (especially heart failure) and/or lung disease. It can be acute or chronic. Acute and worsening dyspnoea is a typical symptom of acute pulmonary oedema: the accumulation of liquid in the lungs that prevents exchange of gases. The result is a painful sensation of air hunger accompanied by cough with foamy expectoration, sometimes streaked with blood.
The grading of dyspnoea due to heart failure

Dyspnoea can be classified according to seriousness (New York Heart Association classification) as follows:

**Class I.** No limitation of physical activity: ordinary activity does not cause undue fatigue, dyspnoea, palpitation or anginal pain.

**Class II.** Slight limitation of physical activity: comfortable at rest, but ordinary physical activity causes fatigue, dyspnoea, palpitation or anginal pain.

**Class III.** Marked limitation of physical activity: comfortable at rest, but less than ordinary activity causes fatigue, palpitation, dyspnoea or anginal pain.

**Class IV.** Inability to carry on any physical activity without discomfort: symptoms of heart failure or the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.


2. Evaluation: questions to be asked about dyspnoea

2.1. During or after exercise? After how many steps? After walking what distance?

2.2. At rest? On how many pillows do I sleep? Is night-time dyspnoea reduced if I sit up?

2.3. Is it slight or intense?

2.4. How long does it last?

2.5. Have I taken my medicine?

2.6. Do I have a cough, catarrh or fever?

2.7. How are my pulse and blood pressure during dyspnoea?

3. Emphasis

*By variation*

3.1. I did not have it before but now I do.

- Is it acute and worsening?
- Is it slight, brief or occasional?
- Does it follow slight or intense effort?

3.2. I had it before but it is getting worse.

- Worse in terms of intensity? Worse in terms of duration?
- Has my level of exercise tolerance dropped? How much? Over what period of time?

*By association*

3.3. Do I have other symptoms (chest pain, palpitation or vertigo) together with dyspnoea?
3.4. Have I noticed without changing eating habits an increase in body weight and a reduction in urine produced during one day? Are my legs swollen?

3.5. Are my pulse and blood pressure abnormal during dyspnoea?

4 and 5. Examples of decision and action options

- No cause for concern: “sighing” dyspnoea. In this case I can talk to my GP.
- Clear cause for concern: acute and worsening dyspnoea. I need immediate help. I must call the medical emergency service number.
- A grey situation: other forms of dyspnoea. According to the situation, I will call my GP or the on-call physician in the nearest future or I will need to talk to my GP at some point.

2.1.2 Chest pain

1. Recognition

Chest pain can result from myocardial ischaemia, cardiac problems not related to ischaemia (such as inflammation of the pericardium) or other causes (disorders of the digestive system, of the respiratory system or neuromusculoskeletal or mental causes).

Brief pain (less than 15 minutes) resulting from myocardial ischaemia is called angina pectoris. Angina generally disappears within minutes after the cause is removed (such as physical effort) and/or taking nitroglycerine.

This recurring chest pain or discomfort is not a heart attack but a sign of an increased risk of heart attack. Angina pectoris indicates that some part of the heart muscle is not receiving an adequate blood supply.

The term “pain” is used in a wide sense (including feelings of oppression, heaviness and similar expressions the patient does not always identify as pain). Angina is chest discomfort that could be constricting, heavy, oppressive or burning and is felt deep in the chest, generally behind the sternum. It may spread out across the chest towards the arms (left rather than right) and towards the neck and lower jaw; pain spreading to the shoulder and to the area between the shoulder blades has been described. In the arms, the pain spreads across the forearms to the wrist and then the little finger. Occasionally the pain can only be felt in the arm or can start in the arm and spread to the chest. At times it can be felt in the epigastrium (the upper middle part of the abdomen). It is felt rather often only in the lower jaw or teeth.

Angina is caused by transient myocardial ischaemia, which is the result of a discrepancy between the demand for and supply of oxygen on the part of the myocardium and the coronary blood flow, respectively. In other words, angina might occur when the heart muscle needs more oxygen than the coronary arteries can supply.

There are several types of angina, caused by different mechanisms:

- The conditions (such as effort) that bring about an increase in heart rate and blood pressure increase the myocardial demand for oxygen, which is not met by a corresponding increase in coronary blood flow. Blood flow in such cases is limited by the atherosclerotic narrowing of the coronary lumen. This is called fixed coronary stenosis (luminal narrowing) and secondary angina.
• When the discrepancy between the oxygen demand and supply does not result from a rise in heart rate and blood pressure but arises from a primary transient narrowing of the lumen caused by constriction of the coronary arteries, this is called dynamic coronary stenosis and primary angina (for example, spontaneous angina).

• When both mechanisms are present, it is called mixed angina (for example, caused by effort in the cold).

Angina can arise during effort or at rest. It can appear upon lying down or getting out of bed in the morning. It can be triggered by emotions, especially anger, and by exposure to cold. It can arise after meals or result from a combination of factors: for example, if the patient attempts to climb stairs after a meal.

Angina is defined as stable when it has been present for more than one month with steady features. Angina is defined as unstable when it appears for the first time (or for less than one month) or when pre-existing angina is worsening.

Prolonged pain (more than 15–20 minutes) indicates persistent myocardial ischaemia and may indicate the onset of myocardial infarction.

The pain of myocardial infarction usually lasts more than 15 minutes, is often stronger and can be accompanied by nausea, vomiting, sweating and dyspnoea. Duration of pain is not an absolute value and may vary substantially.

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**Functional classification of angina pectoris**

The functional classification of angina pectoris by the Canadian Cardiovascular Society includes four classes of angina:

**Class I.** Ordinary physical activity (such as walking and climbing stairs) does not cause angina. Angina occurs with strenuous, rapid or prolonged effort at work or recreation.

**Class II.** Slight limitation of ordinary activity. Angina occurs on walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, or in cold, in wind, or under emotional stress; or only during the few hours after awakening. Walking more than two blocks on the level or when climbing more than one flight of ordinary stairs at a normal pace and in normal conditions can elicit angina.

**Class III.** Marked limitation of ordinary physical activity. Angina occurs on walking one to two blocks on the level or climbing one flight of stairs at a normal pace and in normal conditions.

**Class IV.** Inability to perform any physical activity without discomfort. Angina symptoms may be present at rest.

2. Evaluation: questions to be asked about chest pain

2.1. How long does it last? More or less than 15 minutes? Does it disappear rapidly when nitroglycerine is taken correctly?

2.2. Does it follow effort (effort equivalents include emotions such as anger)? After how many steps? After walking what distance?

2.3. Does it occur during activity or at rest?

2.4. Is it like other episodes already noted as being of cardiac or non-cardiac origin?

2.5. Does it change with breathing, changes in posture, digital pressure or belching?

2.6. Have I taken my medication?

2.7. How are my pulse and blood pressure during the symptom?

3. Emphasis

By variation

3.1. I did not have it before but now I do.
   - How long does it last? The duration of more than or less than 15 minutes (infarction versus angina) is merely a guideline: dyspnoea, sweating, nausea or vomiting should be evaluated.
   - When did it first appear? More or less than one month (stable versus unstable angina)?
   - Do I get it when resting or after effort?
   - Do I get it following slight or intense effort?
   - Is it brief but frequent and worsening or brief and infrequent?

3.2. I had it before and it is getting worse.
   - Worse in terms of frequency (number of episodes)? Of intensity? Of duration?
   - Has my level of exercise tolerance dropped? By how much? Since when (degree of instability of angina)?

By association

3.3. Do I have other symptoms (dyspnoea, palpitation or vertigo) together with chest pain?

3.4. Are my pulse and blood pressure abnormal during the symptom?

4 and 5. Examples of decision and action options

- No cause for concern: a momentary twinge; chest pain definitely of chest wall origin (such as when I cough or apply pressure or pain is related to posture). In this case I can talk to my GP.
- Clear cause for concern: prolonged or brief but rapidly worsening chest pain (of suspected cardiac origin). I need immediate hospitalization. I call the medical emergency service number.
- A grey situation: other forms of chest pain. According to the situation, I will call my GP or the on-call physician in the nearest future or I will need to talk to my GP at some point.
2.1.3 Palpitation

1. Recognition

Palpitation of the heart is defined as an uncomfortable awareness of the heartbeat. The patient may note the sensation of missing a beat or more intense beating, rapid or slow beating, in regular or irregular sequence, beginning and ending suddenly or gradually. The heartbeat can be felt by palpation of the radial pulse. Direct auscultation of the heart by the physician and an ECG carried out during occurrence of the symptom can lead to a precise diagnosis.

2. Evaluation: questions to be asked about palpitation

2.1. How is my pulse during the symptom? Slow (bradycardia): less than 60 beats per minute, “normal” (60–100 beats per minute) or rapid (tachycardia): exceeding 100 beats per minute? Is it regular (rhythmic) or irregular (arrhythmic): does it have a stronger beat (does it jump); does it miss a beat or is it completely irregular?

2.2. How long do episodes of palpitation last? How do these episodes appear and disappear (gradually or suddenly)?

2.3. What is my blood pressure during the symptom? There are many possible causes of bradycardia, such as abnormalities of the conduction system of the heart, heart attack or the use of some medications. On the other hand, physically well-trained people might have a low heart rate and experience no health problems. In certain cases, when the heart rate is very slow, the body might get insufficient blood flow.

Tachycardia can result from heart disease or from a number of situations other than heart disease: fever, anaemia or excessive functional activity of the thyroid gland (hyperthyroidism). The heart rate can increase normally above 100 beats per minute during and after physical effort or during stressful situations. Such tachycardia does not represent any danger to healthy people.

3. Emphasis

By variation

3.1. I did not have it before but now I do.
   • If the pulse is irregular, are the beats missed frequently or seldom?
   • Are these episodes of palpitation characterized by slow beating (bradycardia), slow and arrhythmic beating (bradyarrhythmia), rapid beating (tachycardia) or rapid and arrhythmic beating (tachyarrhythmia)? Are they prolonged or brief? Are they frequent or infrequent?
   • Does palpitation follow effort?

3.2. I had such episodes before but they are getting worse.
   • Worse in terms of frequency? Worse in terms of duration?

By association

3.3. Do I have other symptoms (dyspnoea, chest pain, vertigo, lipothymia or syncope) together with palpitation?

3.4. Is my blood pressure abnormal during the symptom?
4 and 5. Examples of decision and action options

- No cause for concern: occasional palpitation of extrasystolic (premature contraction of the heart) origin; transitory and well-tolerated increase of heart rate beginning and ending gradually and caused by effort or fever or by emotion including anger or anxiety. In this case I can talk to my GP.

- Clear cause for concern: slow or rapid palpitation or palpitation associated with other guide symptoms. I need immediate hospitalization. I call the medical emergency service number.

- A grey situation: other forms of palpitation. According to the situation, I will call my GP or the on-call physician in the nearest future or I will talk to my GP at some point.

2.1.4 Vertigo, lipothymia and syncope

1. Recognition

Vertigo is an objective or subjective disorientation in space; lipothymia is the beginning of fainting without leading to complete loss of consciousness; syncope is the sudden brief loss of consciousness, usually caused by a temporary reduction or interruption of blood flow to the brain.

Syncope can be caused by a number of factors. It may be the result of a critical reduction in blood flow from cardiac causes (cardiac syncope), a local or general vascular change (non-cardiac or vascular syncope) or both (mixed syncope). Non-cardiac or non-vascular syncope also may result from primary changes in cerebral function, changes in brain metabolism or mentally disordered behaviour.

**Syncope**

In general, vasodepressive syncope (fainting) is the most common (30–55% of cases) form. Cardiac disorders are the cause of syncope in 20% of cases. The other frequent causes are postural hypotension (caused by low blood pressure upon standing up) and syncope caused by disorders of blood circulation in the brain.

Cardiac syncope may result from a mechanical obstacle to the emptying and/or filling of the ventricle (mechanical cardiac syncope) or from arrhythmia (arrhythmic cardiac syncope) – the most common cause of cardiac syncope – or both.

Mechanical cardiac syncope may be caused by various heart diseases such as aortic valve stenosis and prosthetic valve dysfunction. Arrhythmic cardiac syncope may be caused by marked bradycardia or bradyarrhythmia, tachycardia or tachyarrhythmia or the malfunctioning of a pacemaker.

2. Evaluation: questions to be asked about the symptom

2.1. What do (or did) I have? Vertigo, lipothymia or syncope?

2.2. How long did it last?

2.3. How did it appear or disappear? Suddenly or gradually? Was it linked to changes in position, surroundings, emotion, pain, digestion, urination or defecation? What did people in attendance notice?
2.4. Did I fall and hurt myself?
2.5. How were my pulse and blood pressure during the symptom?

3. Emphasis

By variation
3.1. Is the symptom intense (do I feel really ill)? Is the symptom long-lasting or brief or momentary? Do I have this symptom frequently or rarely?

By association
3.2. Do I have other symptoms (palpitation, chest pain or dyspnoea) together with vertigo or lipothymia?
3.3. Is the pulse slow or rapid during the symptom? Is blood pressure low during the symptom?

4 and 5. Examples of decision and action options

- No cause for concern: occasional, postural vertigo with normal pulse and blood pressure. In this case I can talk to my GP.
- Clear cause for concern: syncope. I need immediate help. I (they) call the medical emergency service number.
- A grey situation: other forms. According to the situation, I will call my GP or the on-call physician in the nearest future or I will need to talk to my GP at some point.

2.1.5 Summary of symptom assessment

What are the guide symptoms?
Dyspnoea, chest pain, palpitation, vertigo, lipothymia and syncope.

How should I deal with the symptoms?
Recognize, evaluate, give emphasis, decide and take action.

What should I evaluate?
The circumstances (effort, rest or other), intensity, duration and frequency of the symptom and, if possible, signs (pulse and blood pressure).

What should I emphasize?
The type and extent of symptom variation, association with other cardiac symptoms and abnormal pulse and blood pressure.

What action should I take?
Depending on the situation, my choices are to:
- stay calm because there is no cause for concern
- call the medical emergency service
- call my GP
- call the on-call physician.

I have the documentation regarding my case. I have the contact telephone numbers. I know how to contact the medical emergency service and which hospital to go to. I realize that prolonged or
brief but rapidly worsening chest pain (of suspected cardiac origin), acute and worsening
dyspnoea, marked and/or poorly tolerated palpitation and syncope require calling the medical
emergency service.

2.2 Signs

The patient or the patient and family must be taught, in theory and practice, how to take and
evaluate the pulse and to measure and evaluate blood pressure. Here are the steps.
1. What does the sign mean?
2. How and what do I measure?
3. What action I should take?

2.2.1 The pulse

1. What does it mean?

The pulse is the expansion and recoil of the artery wall caused by the pressure wave from heart
contraction. It indicates the frequency with which blood is ejected from the heart and can be felt
by palpation of large or medium-size peripheral arteries.

2. How is it measured?

The pulse can be measured by palpation of a superficial artery. The easiest place to take the
pulse is on the radial artery on the thumb side of the wrist, where the pulsation can be felt
clearly. The first, second and third fingers should be placed over the left radial artery with slight
pressure.

3. What is measured?

Pulse rate: the number of beats in one minute. The number of beats in 15 seconds can be
counted and multiplied by four. “Normal” values ranging between 60 and 100 beats in one
minute depend on the person’s age and physical fitness. A heart rate of less than 60 beats per
minute is called bradycardia (slow heart beating). A heart rate exceeding 100 beats per minute is
called tachycardia (rapid heartbeating).

Pulse rhythm: the pulse may be regular (rhythmic) or irregular (arrhythmic). Rhythmic pulse is
when heartbeat is felt at regular intervals. Arrhythmic pulse is when varying intervals separate
heartbeats.

4. What should I do?

- If the pulse is abnormal in terms of rate and/or rhythm, a physician should be consulted.
- If the pulse rate is below 60 beats per minute or above 100 beats per minute, and if this is
  associated with symptoms such as dyspnoea, chest pain, vertigo, lipothymia or syncope,
  calling the medical emergency service number may be necessary.
2.2.2 Blood pressure

1. What does it mean?

There are two numbers in a blood pressure reading: systolic and diastolic. Systolic (or maximum) pressure is the maximum pressure in the arteries during systole (contraction of the heart). Diastolic (or minimum) pressure is the minimum pressure in the arteries during diastole (relaxation of the heart). A typical reading might be, for example, 120 mmHg (systolic) and 80 mmHg (diastolic).

2. How is it measured?

Blood pressure is measured with a sphygmomanometer. Mercury or anaeroid (not containing liquid) devices are used as well as automated devices. The latter type is easier for domestic use. Anaeroid devices could become inaccurate without this being noticed.

The following are guiding steps in blood pressure measurement.

- Blood pressure is measured on the brachial artery – the large artery of the arm.
- Blood pressure should be measured after at least five minutes of rest.
- The cuff of the sphygmomanometer is wrapped firmly around the arm, with the lower edge about 2 cm above the fold of the elbow.
- If a stethoscope is used, the membrane must be placed over the brachial artery (inside of the elbow, to be located by palpation).
- If an automated device is used, the sensor (usually near the rubber tube of the cuff) must be placed over the brachial artery.
- The cuff is inflated to a pressure that blocks the flow of blood in the artery.
- As the pressure in the cuff is slowly released, blood starts flowing again and the flow can be heard with the stethoscope. The number at which blood starts flowing indicates maximum (systolic) pressure (for example, 120 mmHg).
- While the cuff is slowly deflated, a series of strong sounds will be heard, followed by a sudden passage from strong sounds to weaker sounds: the number at which the last strong sound disappears indicates minimum (diastolic) pressure (for example, 80 mmHg).

3. What is measured?

Systolic and diastolic blood pressure readings should be measured and recorded. Blood pressure levels can vary according to body mass index (BMI), daily activities and various other factors.

If systolic blood pressure is 140 mmHg or higher and/or diastolic blood pressure is 90 mmHg or higher in a series of measurements, then such a blood pressure level is defined as high blood pressure.

4. What action should I take?

- If high blood pressure is an occasional finding, it should be measured again at rest.
- If blood pressure is consistently elevated, a GP should be consulted.
3. Third session. Risk factors for coronary heart disease

Introduction

Subject

This session provides information on major modifiable risk factors. As described earlier, the main modifiable risk factors are: disorders of blood lipid levels (dyslipidaemia), smoking, high blood pressure (hypertension), diabetes, sedentary lifestyle, obesity and psychosocial risk factors. The strongest evidence of association between risk factors and coronary heart disease is for high cholesterol, high blood pressure and smoking.

This session is divided into six sections to cover the following risk factors: dyslipidaemia, smoking, hypertension, diabetes, sedentary lifestyle and stress.

Previous knowledge required

Basic understanding of the structure and the functioning of the heart and blood vessels and how atherosclerosis of the coronary arteries develops.

Objectives

At the end of the session, the participant (patient or patient and family) should be able to understand:

- the major risk factors
- how they contribute to the development of coronary heart disease
- how they are measured
- how they can be managed
- modification in health-related lifestyle.

Content summary

Definition of risk factors, influence of risk factors on the cardiovascular system, their assessment and advice on their management with focus on health-related lifestyle modification.
3.1 Dyslipidaemia (disorders of blood lipid levels)

Introduction

Subject

The purpose of this section of the session is to give information on blood lipids, on their role in the development of atherosclerotic coronary heart disease and on the importance of reducing them if elevated; and to advise on strategies for doing this.

Previous knowledge required

Basic understanding of the structure and the functioning of the heart and blood vessels and how atherosclerosis of the coronary arteries develops.

Objectives

At the end of the session, the participant (patient or patient and family) should be able to understand:

- what blood lipids are;
- how their levels are measured;
- familial types of dyslipidaemia;
- secondary causes of dyslipidaemia;
- desirable blood lipid levels and recommended levels in relation to clinical situations;
- how to improve blood lipid levels by modifying health-related lifestyle and treating with drugs.

Content summary

Information on blood lipids and advice on modifying health-related lifestyle to prevent and manage blood lipids disorders.
3.1.1 Definition of blood lipids

In the body, the major classes of lipids are triglycerides, phospholipids and steroids. The most prevalent steroid is cholesterol. Lipids are fatty substances and therefore cannot be dissolved in blood. They are transported in the blood being encapsulated in particles containing protein, which are water-soluble. These lipid-protein complexes are called lipoproteins. Lipoproteins are classified according to the density in ultracentrifugation and to their lipid content. There are the following classes of lipoproteins: chylomicrons, very-low-density lipoproteins (VLDL), low-density lipoproteins (LDL), intermediate-density lipoproteins (IDL) and high-density lipoproteins (HDL). Each lipoprotein contains different amount of cholesterol. Chylomicrons and VLDL carry mainly triglycerides; LDL and HDL carry mainly cholesterol.

Digestion of lipids

When dietary lipids (fat) enter the digestive tract, they are emulsified by bile salts, hydrolysed by pancreatic enzymes to release fatty acids and then absorbed by the lining cells of the small intestine. There the fatty acids are recombined into triglycerides, which are incorporated into chylomicrons. A chylomicron is composed of cholesterol and triglyceride that is surrounded by a lipoprotein. Chylomicrons are secreted into the intestinal lymph. The lymph passes chylomicrons through the thoracic duct into the systemic circulation. In the blood, chylomicrons come into contact with an enzyme located on the internal surface of capillaries. The enzyme breaks up the triglycerides contained in chylomicrons, releasing fatty acids. The fatty acids are either used by skeletal muscle as energy or they are taken up by fat cells (adipocytes). There the fatty acids are resynthesized into triglycerides and stored for future energy needs. The chylomicron remnants, rich in cholesterol, re-enter the systemic circulation and are taken up by the liver. The liver uses cholesterol for the synthesis of bile acids and VLDL. The synthesized VLDL carry triglycerides to fat and muscle cells. The remaining VLDL fragments become IDL enriched with cholesterol. The liver takes IDL, where they are converted into VLDL or LDL.

3.1.2 Relationship between lipids and coronary heart disease

Cholesterol. Cholesterol is a naturally occurring substance found in the blood and tissues. Cholesterol is necessary for the following four functions: it is a component of the cellular membrane (cholesterol joining “soft” phospholipids adds cohesive strength, similar to the metal net that strengthens a wall), it is the basis of the molecular structure of some hormones, it is used in producing the bile salts and it is used in producing vitamin D. Most cholesterol is in the cells, and only about 7% of it circulates in the blood (plasma cholesterol). It is the plasma cholesterol that is involved in the formation of atherosclerotic plaques.

The importance of cholesterol in atherogenesis is shown by its presence in the atherosclerotic plaque. To understand the atheroma-causing effect of cholesterol, the medical terms and the types of plasma cholesterol distinguished as being “good” and “bad”, the following cholesterol “loop” may be useful.
There are two sources of blood cholesterol: endogenous sources (cholesterol made in the body) and exogenous sources (cholesterol coming from food).

The body can produce all the cholesterol needed. Cholesterol is synthesized primarily by the liver and passed from the liver into the blood to be used in the normal functioning of the organism. Cholesterol can be also produced by the lining cells of the small intestine and by individual cells. Being a fatty substance, cholesterol is not water-soluble and therefore cannot be transported alone by the blood; the liver produces special proteins that can transport cholesterol. These lipid-protein particles contain mainly fat and are called LDL. They move in the blood, carrying about 70% of the total plasma cholesterol; when they reach the capillaries, they deposit the cholesterol on the cell surface receptors to be used by the cells.

Any cholesterol excess (that is, cholesterol that is not used by cells) is loaded onto lipid-protein particles that have more protein (these proteins are also produced by the liver). These particles are called HDL. The excess cholesterol is carried back to the liver, where it is stored or used in the synthesis of bile salts. HDL carry only about 20% of the total plasma cholesterol.

Thus, the LDL are cholesterol carriers and the HDL are cholesterol cleaners. For this reason, the HDL cholesterol is called “good” cholesterol and the LDL cholesterol is called “bad” cholesterol since, under certain conditions, LDL cholesterol can be deposited on the artery wall, contributing to the formation of atherosclerotic plaques.

- When the mechanism of cholesterol synthesis and transport is balanced, no excess cholesterol is left circulating in the blood.
- The mechanism may become imbalanced from increasing LDL (caused by such factors as a high intake of saturated fatty acids) or decreasing HDL (caused by such factors as sedentary lifestyle). Instead of being carried away, the excess cholesterol remains stuck to the artery wall, causing the onset of atherosclerosis. Circulating LDL cholesterol filters through the internal layer of cells of the arterial wall into the space under the layer where the atherosclerotic process starts. There is a causal relationship between total cholesterol, LDL cholesterol and coronary heart disease. Severe atherosclerosis can develop if LDL cholesterol is significantly elevated, even in the absence of other risk factors. In contrast, other risk factors seemingly produce little atherosclerosis in the absence of a certain elevation of the LDL cholesterol level.
The third component of the total plasma cholesterol (approximately 10%) is the VLDL, which are also produced by the liver. VLDL contain mostly triglycerides: they, like cholesterol, cannot circulate in the blood without being encapsulated in a lipid-protein particle. VLDL deliver triglycerides to muscle and fat cells. The remaining fragments become IDL.

**Triglycerides.** Triglycerides are a fatty substance naturally present in blood and tissues. Their essential function is to be a reserve of energy. Calories that are absorbed but not consumed are stored as triglycerides, the principal constituents of fat cells. They are used when energy is needed. Triglycerides in the blood come from the liver and food.

A high level of triglycerides in the blood is not necessarily a risk factor for atherosclerosis. Excess triglycerides can damage the arteries, although the correlation between elevated triglycerides and an increased risk of coronary heart disease is not as strong as that for cholesterol. However, in some people triglycerides are part of lipoproteins with a high level of cholesterol. Such lipoproteins are a risk factor for atherosclerosis. Further, it is increasingly recognized that high triglycerides are often associated with other conditions that increase the risk of atherosclerosis, including obesity, low HDL cholesterol, insulin resistance and some other.

### 3.1.3 Measuring lipid levels

All people with a history of coronary heart disease or of other types of atherosclerotic disease (such as stroke or peripheral vascular disease) are at very high risk of developing new clinical manifestations. On the other hand, studies have shown that rational and continuous management of dyslipidaemia among people with coronary heart disease can lead to an increase in survival rates and a reduction in the frequency of new expressions of the disease. Effective treatment of elevated cholesterol has been shown to reduce progression and probably even lead to regression of coronary atherosclerotic plaques. Even people who have had a heart attack may decrease the risk of future attacks, since reducing blood cholesterol can reduce the cholesterol content in plaques, making them more stable, thus, decreasing the possibility of their ulceration or desquamation.

All patients must therefore have their lipid profile assessed to select if needed, the most appropriate treatment (with lifestyle modification and/or drugs). Assessing the lipid profile includes measuring total plasma cholesterol and its fractions: HDL cholesterol, triglycerides and LDL cholesterol. Lipid levels are determined through chemical analysis of a blood sample taken from a finger prick or from a vein after fasting for 9–12 hours and abstaining from alcohol consumption for 72 hours (the triglyceride level varies notably in individuals in relation to diet and alcohol consumption).

**Total blood cholesterol.** The total cholesterol level is assessed with reference not to a “normal” level but rather to a “desirable” or “optimal” level. Several factors influence the cholesterol level, including age, sex, family history, diseases and lifestyle. The level of cholesterol increases with age, reaching a plateau in men at about 60 years, whereas in women it continues to rise with age. Men tend to have higher LDL and lower HDL cholesterol levels than women (estrogen increases the HDL cholesterol level), especially before the age of 50. People who are overweight or obese are more likely to have a higher total cholesterol and lower HDL cholesterol level. The location of the excess weight also seems to play a role. People in whom the extra weight is in the abdominal area tend to have a higher level of cholesterol as compared with people whose extra weight is on the legs and buttocks.
According to the European guidelines on cardiovascular prevention prepared in 2003 by the European Society of Cardiology Committee for Practice Guidelines to improve the quality of clinical practice and patient care in Europe (1), the optimal or desirable level of total blood cholesterol is below 190 mg/dl (<5.0 mmol/l). For people with clinically manifested cardiovascular disease and for people with diabetes, total cholesterol should be below 175 mg/dl (<4.5 mmol/l).

Abnormal blood cholesterol levels may develop under certain circumstances in pregnant women and in people who have recently experienced trauma, heart surgery or other major surgery, acute serious illness or heart attack; in these cases, the lipid profile is altered, making the results unreliable, and baseline levels do not return for about six weeks.

**HDL cholesterol.** No specific optimal or desirable levels are established for HDL cholesterol. The level is used as a marker of increased risk. The HDL cholesterol level lower than 46 mg/dl (<1.2 mmol/l) in women and lower than 40 mg/dl (<1.0 mmol/l) in men indicates an increased cardiovascular risk.

**LDL cholesterol.** If the triglyceride level is below 400 mg/dl, the LDL cholesterol level can be calculated (in mg/dl) using the following formula:

\[
\text{LDL cholesterol} = \text{total cholesterol} - (\text{HDL cholesterol} + (\text{triglycerides}/5))
\]

According to the European guidelines on cardiovascular prevention prepared in 2003 by the European Society of Cardiology Committee for Practice Guidelines (1), the optimal level for a healthy adult is less than 115 mg/dl (<3.0 mmol/l); for people with cardiovascular disease and for people with diabetes, the level should be less than 100 mg/l (<2.5 mmol/l).

A high level of total cholesterol is typically due to a high level of LDL cholesterol. The combination of high levels of total and LDL cholesterol with a low level of HDL cholesterol further increases the risk of atherosclerosis and heart attack.

**Triglycerides.** A level higher than 150 mg/dl (>1.7 mmol/l) indicates increased cardiovascular risk.

### 3.1.4 Familial types of dyslipidaemia

High lipid levels can run in families. Inherited disorders of lipid metabolism are present from birth. In familial hyperlipidaemia (high levels of blood lipids), the total cholesterol level usually exceeds 290 mg/dl and LDL cholesterol exceeds 190 mg/dl. People with this relatively common disorder (1 in 500 people) have a high risk of atherosclerosis with early-onset coronary heart disease.

In these cases, the cholesterol level is high not because of poor diet but rather because of a genetic defect. Such a defect can lead to excessive production or decreased removal of LDL cholesterol. If there is a family history of coronary heart disease manifesting below the age of 50 and/or the presence of fat deposits at the extensor tendons of the fingers and the Achilles’ tendons, the diagnosis is almost certain. In such cases, a healthy diet alone is often not sufficient to reduce the cholesterol level and lipid-lowering drugs must be administered.

A high triglyceride level (hypertriglyceridaemia) also can be inherited. Cholesterol and triglyceride levels both being high is known as familial combined hyperlipidaemia. Similarly, a
very low or a very high HDL cholesterol level can be inherited. People from families with a low HDL cholesterol level have a higher incidence of coronary heart disease, whereas people from families with a high HDL cholesterol level have a lower probability of a heart attack compared with the general population.

### 3.1.5 Secondary causes of dyslipidaemia

High cholesterol levels can be also seen in connection with some causes. The causes can be hormonal diseases such as diabetes or low functional activity of the thyroid gland (hypothyroidism), liver or kidney diseases, alcohol abuse or chronic medication (such as cortisone) use. A high triglyceride level can often be caused by obesity, excessive alcohol intake, diabetes mellitus, kidney disease, intake of estrogen-containing medications such as contraceptives or medication used for hormone replacement therapy. People with diabetes often have a lower level of HDL cholesterol.

Recognizing the secondary origin of high lipid levels is important, since treating the underlying cause significantly improves the lipid profile.

### 3.1.6 Ways of improving the lipid profile

As described above, improving the lipid profile can slow or even reverse the development of atherosclerotic plaque. The main goal of treatment is to lower LDL cholesterol.

Nutrition, familial predisposition and metabolic disorders are the factors influencing blood lipids. In most cases their levels are defined primarily by a combination of all three factors.

The following strategies are to be used in improving the lipid profile:

- diet;
- regular physical activity;
- weight loss in overweight or obese people;
- treatment with drugs.

Dietary therapy is the cornerstone of the treatment of lipid disorders. Losing excess weight and being engaged in regular physical activity ameliorate the blood lipid profile. Sometimes diet and exercise alone are not enough to manage lipid levels. In such cases drugs should be used. If lipid-lowering drugs are needed, they should be supplementary to a healthy diet and adequate physical activity. The need for drug therapy is determined by assessing the probability of coronary heart disease based on the individual’s risk factors.

**Diet.** The levels of total cholesterol and its fractions are affected by intake of calories and the types of fat eaten.

- Consuming large amounts of any fat increases body weight if energy intake exceeds energy expenditure. Excess body weight lowers HDL cholesterol, increases LDL cholesterol and triglycerides.
- Consuming large amounts of certain fat is linked to the risk of atherosclerosis: dietary saturated fat and trans-fatty acids can significantly increase total and LDL cholesterol, whereas diets high in unsaturated fat can lower LDL cholesterol.
- Dietary cholesterol may increase total plasma cholesterol and LDL cholesterol, but the increase induced is far less than that induced by dietary saturated fat and trans-
fatty acids. There are wide variations in individual responses to dietary cholesterol with regard to blood cholesterol. For some people, reduced cholesterol consumption results in a decrease in blood cholesterol level, although limited; for others, dietary cholesterol has little impact on it.

### Dietary fatty acids

Dietary fat is a mixture of triglycerides (90%), cholesterol and phospholipids. A triglyceride has three fatty-acid molecules. These molecules are composed mostly of carbon and hydrogen atoms. Fatty acids differ in the length of the carbon chain (the number of carbon atoms) and the number of double bonds between carbon atoms. Each carbon atom has four valences for bonding: two for carbon atoms and two for hydrogen. Depending on the number of double bonds in the molecule, fatty acids are classified as saturated, monounsaturated and polyunsaturated.

- **Saturated fatty acids**: when a fatty acid molecule has two hydrogen atoms attached to every carbon atom, there are no double bonds between carbon atoms. It is said that the fatty acid molecule is saturated with hydrogen atoms.

- **Monounsaturated fatty acids**: some fatty acids are missing one pair of hydrogen atoms in the carbon chain. This is called an “unsaturation”. Such fatty acids have one double bond between carbon atoms and are termed monounsaturated fatty acids.

- **Polyunsaturated fatty acids**: fatty acids with two or more “unsaturations” and, accordingly, two or more double bonds between carbon atoms. Depending on where in the carbon chain the double bonds are located, polyunsaturated fatty acids are classified as n-3 (also called omega-3) and n-6 (also called omega-6) polyunsaturated fatty acids. One refers to a polyunsaturated fatty acid as n-3 polyunsaturated fatty acid when the first double link is located at the third carbon atom in the carbon chain and as n-6 when the first double link is located at the sixth carbon atom.

- A double bond of an unsaturated fatty acid molecule can have a cis- or trans-geometrical configuration. Although trans-fatty acids occur in small amounts naturally in dairy products as a result of partial hydrogenation of the unsaturated fatty acids of grass by bacterial fermentation in the rumen, they are mainly produced by industry. As vegetable oils are liquid at room temperature and can become rancid easily, manufacturers produce more solid oils to prolong the shelf-life of products. Unsaturated oils are “hardened” partially hydrogenating them. In this process, hydrogen atoms are put in the carbon chain and unsaturated cis-fatty acids adapt a trans-geometrical configuration like a saturated fatty acid that has a higher melting temperature.

In human nutrition the following fatty acids are quantitatively important:

- saturated fatty acids: stearic acid, palmitic acid, myristic acid and lauric acid;
- monounsaturated fatty acids: oleic acid and elaidic acid;
- polyunsaturated omega-3 fatty acids: alpha-linolenic acid and two prolonged carbon chain acids: eicosapentaenoic acid and docosahexaenoic acid;
- polyunsaturated omega-6 fatty acids: linoleic acid.

Dietary triglycerides – the main component of dietary fat – contain a mixture of all three classes of fatty acids but in varying proportions: for example, dietary saturated fat such as fat of animal origin comprises a mixture of various fatty acids, saturated fatty acids being dominant.
A healthy diet should be based on eating appropriate portions of healthy food. It should focus on a variety of foods (to provide a wide range of nutrients in sufficient amounts) originating mainly from plants. At least 400 g (five or six portions every day; one portion is equivalent to one apple or other fruit) of a variety of vegetables and fruit, preferably fresh and local, should be eaten every day. The consumption of whole grain breads and cereals should be encouraged. Fish consumption once or twice a week is recommended.

It is recommended to eat poultry or lean meat, legumes such as beans or lentils, fish, and to consume dairy products that are low in fat or fat-free. Total salt intake should not be more than one teaspoon (5 grams) per day, including the salt in bread and processed, cured and preserved foods. The intake of saturated fat and trans-fatty acids should be limited. It is recommended to select foods that are low in sugar and to eat refined sugar sparingly, limiting the frequency of sugary drinks and sweets.

A healthy diet does not impose the use of certain foods or the total avoidance of others. Nevertheless, attention to what one eats is essential to avoid excessive calorie intake resulting from consumption of energy-dense foods.

**Atherogenic effect of fatty acids**

Fatty acids can be classified according to their atherogenic effect. To this end, how the blood cholesterol responds to the intake of a fatty acid is compared with how the blood cholesterol responds to the intake of oleic fatty acid (the level of blood cholesterol response to the intake of oleic acid or to the intake of carbohydrates is the same; therefore oleic acid is classified as neutral versus the blood cholesterol).

Saturated fatty acids are atherogenic. They raise the “bad” LDL cholesterol. Several mechanisms have been proposed on how these acids raise LDL cholesterol. Saturated fatty acids probably suppress the activity of LDL receptors located on the surface of liver cells. Primarily via these receptors, the liver intakes most circulating LDL particles.

Trans-fatty acids are more atherogenic than saturated fatty acids. They increase LDL cholesterol and lower HDL cholesterol.

Unsaturated fatty acids (both monounsaturated and polyunsaturated) do not raise blood cholesterol. They can lower LDL cholesterol. Polyunsaturated omega-6 fatty acids lower LDL somewhat more effectively than monounsaturated fatty acids. Polyunsaturated omega-3 fatty acids can reduce the triglyceride level. Eicosapentaenoic acid and docosahexaenoic acid significantly lower triglyceride level and can increase HDL cholesterol. On the other hand, they can also increase LDL cholesterol. There is limited evidence that eicosapentaenoic acid and docosahexaenoic acid prevent coronary heart disease through other mechanisms than affecting lipids.

Dietary fat supplies a substantial proportion of energy. It adds flavour to food and provides essential fatty acids, some of which promote absorption of the fat-soluble vitamins.

The amount of dietary fat required depends on people’s energy needs. The needs include energy for processing food and for basal metabolism as well as for physical activity. The intake of calories should be balanced with energy expenditure to keep body weight within optimal range.
A medium-sized adult needs a daily intake of energy ranging from 1800 kcal for those with a sedentary lifestyle up to 3000 kcal for those with an active lifestyle. A healthy diet should be based on the following balanced percentages of energy resources: it should derive 15–30% of total calories from fat, 10–15% from protein and the remaining proportion from carbohydrate. A high intake of fat (greater than 35 percent of calories) makes it more difficult to avoid consuming excess calories and generally increases saturated fat intake. A low consumption of fat may provide an inadequate supply of essential fatty acids and vitamin E and may unfavourably influence the levels of HDL cholesterol and triglycerides.

Around half of total calories from fat should come from monounsaturated fat. The remainder should come from a mix of saturated fat and polyunsaturated fat: that is, saturated fat should supply less than 10% of total daily energy. Daily intake of cholesterol should not exceed 300 mg. The intake of trans-fatty acids should be limited to less than 1% of daily energy intake. For some patients with an elevated level of LDL cholesterol a more stringent lipid-lowering diet might be needed. In such cases, the intake of saturated fat should not exceed 7% of the total calories. Daily intake of cholesterol should not exceed 200 mg.

The most effective natural way of lowering blood cholesterol is to reduce the amount of saturated fat consumed. Major dietary sources of saturated fat are foods mostly of animal origin such as dairy and meat products. Foods high in saturated fat generally also contain substantial amounts of cholesterol. Consumption of meat such as beef or pork should be limited. It is useful to eat meatless meals several times a week. Fried foods, full-fat cheese, milk and cream should be avoided. It is recommended to replace these foods with low-fat or fat-free products and to choose in food preparation steaming, grilling, boiling, microwave cooking.

The first step in reducing the level of triglycerides is a diet low in saturated fat and cholesterol with a limited amount of sweets. Alcohol consumption should be limited. Other lifestyle modifications include taking regular physical exercise, losing excess weight (since triglycerides are energy reserve and the main component of adipose cells, reducing these cells often immediately reduces the triglyceride level) and avoiding smoking. In people with diabetes mellitus, good control of blood glucose is important.

For reducing coronary heart disease risk, polyunsaturated fatty acids, especially linoleic acid (polyunsaturated omega-6 fatty acid) are the most effective replacement for saturated fatty acids. Linoleic acid lowers circulating LDL increasing the clearance of LDL cholesterol from the blood through enhanced LDL receptor activity.

There is evidence of the cardiovascular benefits of eicosapentaenoic and docosahexaenoic acids (polyunsaturated omega-3 fatty acids with prolonged carbon chain) as well as alpha-linolenic acid (polyunsaturated omega-3 fatty acid).

Monounsaturated fatty acids reduce LDL cholesterol and help maintain the level of the protective HDL cholesterol.

Polyunsaturated fatty acids come from two main sources: plants (however, palm, palm kernel and coconut oils are rich in saturated fatty acids) and oily fish. Sunflower seed oil is very rich in linoleic acid. Flaxseed, canola (low-erucic-acid rapeseed) and soybean oils are rich in alpha-linolenic acid. Fish and fish oil, especially oil from deep-sea ocean fish are rich in eicosapentaenoic acid and docosahexaenoic acid. Monounsaturated fatty acids are found mostly in olive, canola and peanut oil and avocado.
Dietary fat may be “visible” or “invisible”. Visible fat is clearly apparent: cooking oils, spreads, or marbling or fat on meats and its products. “Invisible” fat is found in the cells of animal tissue (meat, meat products, dairy products and fish). Fat can be also hidden, having been incorporated into the food during preparation. Many bakery products, such as cakes, biscuits and some breads are sources of saturated fat. Shortening, dressing and products such as cookies, bread and fried and snack foods may contain trans-fatty acids.

Many margarine manufacturers have reduced the level of trans-fatty acids in their products. However, it is useful to seek information (for example checking the labels of margarine) to ensure that processed foods do not contain trans-fatty acids. A food product containing trans-fatty acids has ingredients termed hydrogenated or partially hydrogenated.

A diet rich in fibre (such as from unrefined plant products) reduces the risk of coronary heart disease. There is evidence that water-soluble fibre (oat or corn bran, beans and other legumes, plums, citrus fruits, apples and others) can lower cholesterol. There is also a potential link between a fibre-rich diet and reduced risk of type 2 diabetes.

The calories derived from saturated fat can be partly replaced by calories derived from complex carbohydrate. Carbohydrate is found in fruit, vegetables, milk and grains. It is recommended to eat a wide variety of whole-grain products, vegetables and fruit. Vegetables and fruit provide not only sugar and starch as energy source for the body but also fibre, micronutrients (especially potassium) and various phytonutrients. Whole-grain products are rich in dietary fibre, minerals, vitamins and other important micronutrients. In the grain refining process many of these ingredients are lost.

However, a very high intake of carbohydrate might unfavourably change the level of HDL and induce hypertriglyceridaemia, mainly by enhancing hepatic synthesis of VLDL.

It is recommended to limit intake of highly refined carbohydrate (for example, simple sugars added to manufactured foods and sugars naturally present in juices, honey and syrups) to prevent unnecessary increase in calories consumed that might lead to weight gain.

Although consuming limited quantities of alcohol has some protective effect against coronary heart disease, other health risks such as a possible substantial increase in the triglyceride level and weight gain associated with alcohol do not support a recommendation on its use. If alcohol is consumed, intake should be limited to no more than two standard drinks (each containing 10 g of alcohol) per day (3).

The input of a dietician can be extremely important both in drawing up an individual diet plan and in helping the patient stick to the diet over the long term. After a diet is initiated, compliance and response should be monitored for 1–6 months.

**Physical activity.** A healthy diet should be complemented by 30–60 minutes of moderate physical activity on most days of the week.

Regular moderate intensity physical activity such as walking, gardening or a structured physical exercise programme has been shown to have positive effects on risk factors by improving physical fitness, blood lipid profile, insulin sensitivity and by reducing body weight and blood pressure. Exercise reduces resistance of peripheral arterioles and improves blood flow in coronary arteries.
**Weight loss in overweight.** A stable weight is linked to the balance between energy intake (food) and energy expenditure (physical activity): people should eat as many calories as they burn in daily activities to maintain their weight stable. Healthy eating habits, combined with regular physical activity, are the most effective way to control body weight.

BMI is an index to define the approximate total body fat. This index is calculated using the following formula: weight in kg divided by height in meters, squared. For example, a person weighing 80 kg who is 160 cm tall has a BMI of 31.25 ($80/(1.60 \times 1.60) = 31.25$). The normal weight range is from 20 to 25. A BMI between 25 and 30 indicates overweight and BMI over 30 indicates obesity (3).

Those who need to reduce body weight should aim at a slow weight loss decreasing the intake of calories and engaging in aerobic moderate-intensity physical activity for 60–90 minutes daily. These changes in lifestyle can also help raise the HDL level, reduce triglycerides and LDL. People with coronary heart disease should consult their GP before starting a vigorous physical activity programme.

**Calorie needs.** To understand how the calorie requirement of an individual is determined, we can compare the body to the internal combustion engine. In the same way that the engine uses petrol to produce energy to drive the car along the road, our body uses food as fuel to function both when at rest (we need energy to breathe and energy for the heart to work) and above all when moving and working (we need much more energy when large muscles are working). If the amount of energy received from the food consumed exceeds the energy needs, only part of the energy will be used. The excess calories will be stored in the form of fat, leading to weight gain.

**Energy measurement**

The energy is measured in units called calories. For example, heat produced by a piece of burning coal is measured in calories. In the same way, the energy produced by our body “burning” food is measured in calories.

One kcal (1000 cal) is the energy needed to raise the temperature of one kg of water by one degree Celsius.

Energy can also be measured in joules. One kcal is equal to 4.2 kJ.

The food substances from which the body derives energy are called macronutrients. They are: protein, carbohydrate and fat. They provide different quantities of calories. One gram of protein or carbohydrate provides 4 calories. One gram of fat provides 9 calories. Sometimes diets include alcohol, which is an energy-dense substance: one gram provides 7 calories, almost as much as one gram of fat.

For weight control it is important to consider not the proportion of macronutrients (fat, protein, carbohydrate) in the diet but the total calories consumed. It is advisable to select foods that are rich in vitamins, minerals but are lower in calories (such as many kinds of vegetables or fruit). It is important to pay attention to portion sizes.
Drug therapy. The selection and dosage of drugs to treat an elevated level of cholesterol depends mainly on the baseline LDL cholesterol level. In most cases, the decision to use drugs will be taken after a thorough attempt to correct the level through diet and physical activity.

When the cholesterol level has been reduced to within the individual optimal limits by means of diet, physical activity and/or drug treatment, these measures must be continued. Indeed, if treatment is suspended, the cholesterol level will return to baseline within a few weeks, losing all the benefits obtained.
3.2 Smoking

Introduction

Subject
The purpose of this section of the session is to provide information on the risks associated with smoking, to support giving up the habit and to encourage former smokers not to take it up again.

Previous knowledge required
Basic understanding of the structure and the functioning of the heart and blood vessels and how atherosclerosis of the coronary arteries develops.

Objectives
At the end of the session, the participant (patient or patient and family) should be able to understand:

- the association between smoking and disease;
- the mechanisms of the acute and chronic damage to the cardiovascular system caused by smoking;
- the effects of nicotine;
- the damage to health caused by low-tar cigarettes, pipe and cigars;
- the damage to health caused by passive smoking;
- the benefits of quitting;
- how to give up smoking;
- how to avoid taking smoking up again.

Content summary
Information on the harmful effects of smoking on health and advice on how to quit smoking.
3.2.1 Smoking-related diseases

Numerous studies have consistently demonstrated the harmful effect of smoking on health. Tobacco smoking adversely affects tissues and organs. Smoking has been identified as the single most important preventable cause of premature death, due in particular to cancer (especially of the respiratory tract: throat and bronchi) and coronary heart disease. In general, smokers have twice the risk of cardiovascular disease of nonsmokers. Among women the risk is even higher, about four times that of nonsmokers.

Tobacco smoke has many substances that harm health: tar, carbon monoxide, nicotine, ammonia, benzene and others. Tar and carbon monoxide cause most of the damage to health. Tar is responsible for cancer of the respiratory system and chronic respiratory disease. Carbon monoxide reduces the oxygen-carrying capacity of the blood red cells and impairs the supply of oxygen to tissues. The heart, brain, muscles and other organs receive less oxygen. Nicotine is harmful for the cardiovascular and nervous systems. It plays a central role in tobacco dependence.

The relationship between smoking and the damage to health is of the dose-response type, related both to the intensity of the exposure (the degree of smoke inhalation and number of cigarettes smoked in a day) and its duration (number of years of smoking).

3.2.2 How smoking causes cardiovascular damage

Smoking raises the risk of coronary heart disease substantially. Smoking multiplies that risk if other risk factors are present. In addition, smoking has direct harmful effects on the heart and blood vessels.

The damage to the cardiovascular system caused by smoking can be divided into acute and long-term.

Acute damage. Smoking facilitates myocardial ischemia and increases susceptibility to arrhythmia after smoking even a single cigarette.

The acute ischaemic effect is produced by nicotine. Nicotine stimulates the sympathetic nervous system: the higher levels of noradrenalin and adrenalin increase heart rate, raise blood pressure and cause arterioles to constrict. That is, when someone smokes, the heart beats more quickly and arterial pressure increases. This increases the demand for oxygen by the myocardium. At the same time, the lumen of the arteries is reduced and so they allow less blood through. The effects of carbon monoxide, as described earlier, further reduce the supply of oxygen to the myocardium.

Nicotine also damages blood vessels walls and increases the likelihood of a blood clot and of cardiac arrhythmia.

Long-term damage. Smoking stimulates atherogenesis and thrombogenesis encouraging the formation of atherosclerotic plaques and increasing the risk of blood clots developing on these plaques;

The atherogenic and thrombogenic effects are caused chiefly by carbon monoxide and are mediated by direct damage to the lining of the arteries walls, a decrease in HDL cholesterol and
an increase in the ability of the blood to coagulate. Smoking in all likelihood changes LDL cholesterol to a form that promotes the build-up of deposits in the walls of the coronary arteries.

### 3.2.3 The effects of nicotine on the nervous system

Nicotine has a powerful stimulating effect on the central nervous system. It is a very addictive substance.

**Dopamine**

In the brain, nicotine acts on acetylcholine receptors, increasing the release of the neurotransmitter dopamine. This substance is involved in the production by the brain of feelings of pleasure. This body reaction is similar to the reaction a person experiences when using drugs such as opiates. The increased release of dopamine plays a key role in the desire to continue smoking. When tobacco smoke is inhaled, nicotine reaches the brain tissue very quickly. The level of nicotine in the brain remains high for about two hours.

Smokers develop tolerance to nicotine. When a person becomes tolerant to a drug, larger doses are needed to achieve the same effect. Smokers therefore tend to increase the dose of nicotine: they smoke more frequently or inhale more smoke.

Nicotine is capable of creating dependence. It causes all the well-known symptoms of withdrawal (“craving for cigarettes”) when a person stops smoking (the central nervous system needs to readapt without nicotine). The symptoms of withdrawal are headaches, dizziness, irritability, disturbed sleep, tiredness, upset stomach, low ability to concentrate, depression and others. They usually decline within a month after quitting smoking.

Smokers’ craving for nicotine is strongest when the level of nicotine is low. A reliable indicator of the degree of dependence on nicotine is the time that elapses between waking up and smoking the first cigarette. If this is less than half an hour, the degree of dependence is high.

### 3.2.4 Damage to health caused by smoking low-tar cigarettes, pipe or cigars

Some people switch to low-tar (“light”) cigarettes. The advantage of switching is minimal, especially compared with that of quitting. They also often end up smoking more or inhaling more frequently and more deeply to obtain the same level of nicotine in the blood. There is no way of smoking without damaging health.

Many people consider pipes and cigars to be a safe substitute for cigarettes. This is absolutely false. They would generally be less harmful if those who smoke them did not inhale and, thereby limiting the damage to their lungs. Further, toxic compounds present in smoke still cause their characteristic harmful effects on health. For example, nicotine is absorbed to the bloodstream through the mucus lining of the mouth and the upper respiratory tract; carcinogenic substances in smoke cause mouth cancer.

Occasional smokers are at lower health risk compared with heavy smokers. However, occasional smokers tend to inhale more and take more puffs. Since nicotine is very addictive, they face a high risk of increasing the frequency of smoking.
3.2.5 Damage to health caused by passive smoking

Sidestream smoke emitted from burning tobacco is a major component of environmental tobacco smoke. The way passive smoking increases the risk of coronary heart disease has been well documented.

The mechanisms of how smoke damages to health are the same as those for active smoking; indeed, a passive smoker inhales smoke that is heavily laden with carbon monoxide and the products of nicotine degradation. Since passive smoking seems to stimulate an increase in the volume of atherosclerotic plaques, those suffering from coronary heart disease are recommended to stay away from smoky places and avoid tobacco smoke at home, at work and in transportation settings.

3.2.6 The benefits of stopping tobacco use

Smoking cessation is an important component of coronary heart disease prevention and management. Studies of primary prevention have shown that the risk of coronary heart disease falls to the same level as that of nonsmokers several years after giving up completely. The risk of lung cancer and other types of cancer also gradually declines. However, the reduction of cancer risk to the same level as nonsmokers takes longer – more than 10 years.

The mortality of people who stop smoking after an acute coronary event is about 45% lower than among those who continue to smoke. The benefit of quitting is even greater among people who have undergone coronary surgery.

There are two types of decline in the risk of coronary heart disease after quitting smoking: rapid and gradual. The risk of new coronary events among smokers declines rapidly after quitting because a number of effects on cardiovascular health are reversible within days or weeks. However, changes such as smooth cell proliferation or fatty deposits in the internal layer of the arterial wall reverse gradually over a longer period of time.

After people stop smoking, blood pressure and heart rate decline. The bronchi start relaxing and coughing improves in several months. Stopping smoking also improves sleep.

3.2.7 How to give up smoking

Health personnel should advise all smokers to quit. It is important to understand the importance of smoking cessation and to know how difficult quitting could be (4). Patients may find it useful to remember and practise the five D’s (5) when confronted with the urge to smoke:

- **Delay**, even for a short while.
- **Drink** water.
- **Deep** breathing.
- **Do** something different.
- **Discuss** the craving with another person.
The following behavioural tips (5) for quitting smoking tailored to each individual might be helpful:

- Write out a list of reasons to quit and display it prominently, such as on the refrigerator;
- Get rid of all tobacco products, ashtrays, lighters, matches, etc. from all areas you inhabit;
- Clean all clothes to remove tobacco smell;
- Enlist the support of nonsmoking friends, relatives and workmates;
- Change habits associated with smoking: for example, instead of smoking after meals, chew on a toothpick or change rooms;
- Change the environmental cues: for example, the telephone often causes a reflex action to smoke, so move the telephone to another place to change the cue;
- Keep hands busy, such as knitting, gardening, drawing or making origami;
- Change the daily routine to minimize the association of tobacco with certain activities or times of the day;
- Sit in nonsmoking areas;
- Escape situations if a potential relapse cannot be avoided: for example, go to the bathroom;
- Breathe deeply;
- Engage in positive self-talk;
- Try to avoid stressful situations in the immediate period after stopping;
- Replace the urge to smoke with another activity, such as going for a walk;
- Try daily exercise, such as walking, to keep yourself occupied, to relieve stress, to help maintain a positive frame of mind and to become fit;
- Set aside the money normally spent on cigarettes to buy something as a reward;
- Do not drink alcoholic beverages, because these are associated with relapse;
- Avoid, even temporarily, social situations normally associated with smoking. Practise saying “No thank you, I don’t smoke”;
- Ask other smokers not to give cigarettes, offer to buy cigarettes or smoke in your presence;
- Think positively and remember your reasons for quitting in the first place;
- View quitting as a day-at-a-time process rather than an immediate lifelong commitment.

Since the nervous system of a smoker adjusts to nicotine and smoking is also linked to social habits, smoking cessation often requires repeated interventions. Many people who achieved stopping smoking for the long term have previously experienced failure in quitting smoking several times.
Most people who give up smoking do so without outside help. Most people find stopping all at once easy, whereas others find stopping gradually easier. The fact of being hospitalized due a heart attack or a cardiovascular procedure (percutaneous transluminal coronary angioplasty or coronary artery surgery) and the fear of new episodes constitute good deterrents to continuing to smoke.

People find stopping smoking a challenge, mainly because of withdrawal symptoms. For those who cannot give up on their own, help is available, either pharmaceutical therapy (such as nicotine replacement therapy or buproprion) or behavioural psychotherapy (such as practical counselling and social support).

**Pharmaceutical therapy to support quitting smoking**

**Nicotine replacement therapy**
Nicotine replacement therapy does not contain harmful tobacco substances other than nicotine. It is less addictive than tobacco. Very few people become addicted to nicotine replacement therapy. Nicotine replacement therapy is available as chewing gum, skin patches, sublingual (for release under the tongue) tablets, nasal spray and inhalators. Skin patches provide a gradual intake of nicotine, whereas gum, inhalator or spray give a higher nicotine level. The choice of the form of nicotine replacement therapy depends on personal preference and the accessibility of nicotine replacement therapy products.

Nicotine replacement therapy can produce various side-effects. Irritation in the mouth, coughing and running nose are most frequent side-effects in those using a nicotine inhaler. Nasal spray can cause nasal irritation, congestion and temporary changes in smell and taste. Skin patches can cause skin irritation and sometimes disturb sleep. Chewing gum can cause sore mouth and dyspepsia.

Nicotine replacement therapy should be used with caution in patients within the first two weeks of acute myocardial infarction, with serious angina and with serious arrhythmia.

**Buproprion**
Buproprion, a drug used to treat depression is also used to support quitting smoking. It is as effective as nicotine replacement therapy in reducing withdrawal symptoms. The mechanism of action is not clear, but it is not connected to its antidepressant effect. Buproprion is available as sustained-release capsules. Treatment with this drug should start two weeks before quitting smoking. The most common side-effects are insomnia and dry mouth. It is contraindicated in people with a history of seizures as well as in pregnancy. It can be used for up to six months.


Nicotine replacement therapy reduces withdrawal symptoms. It helps stopping smoking, breaking the process in the following two phases: stopping smoking and then stopping using nicotine. Therapy is continued for 2–6 months. Nicotine replacement therapy can be discontinued gradually.
3.2.8 Preventing starting again after quitting

Smoking dependence is a chronic condition and relapses may occur after quitting. Various factors might trigger a relapse. Most common ones are low mood or depression, withdrawal symptoms, weight gain and feeling deprived.

Most relapses occur within the first months after quitting. The risk of starting again may increase in the long term (after months or even years), especially in specific stressful situations (such as returning to work after a coronary event).

Various interventions can prevent relapse, such as support and encouragement by health personnel, family members and friends. For those who feel deprived, it is useful to explain that such feelings are common and transient. People with depression can receive counselling and appropriate medication. In severe cases a visit to a specialist might be helpful. It is very important that health personnel encourage maintaining abstinence among ex-smokers who report gaining weight; advice on a healthy diet and physical activity should be provided.

Those who quit smoking should be informed that a cigarette offered innocently by a friend or colleague carries the risk of starting again. Even a puff increases the urge to start smoking again. A person quitting smoking must therefore know how to resist social pressure to take a cigarette. The ex-smoker must be able to recognize situations that carry a high risk of starting again and learn how to deal with them. For example, if the desire to smoke is high after meals, such behavioural habits as leaving the table quickly after the meal, going for a walk or cleaning teeth might be very helpful.
3.3 Hypertension

Introduction

Subject
The aim of this section of the session is to provide information on hypertension and its effects on the cardiovascular system and to support changes in health-related lifestyle to reduce blood pressure.

Previous knowledge required
Basic understanding of the structure and the functioning of the heart and blood vessels and how atherosclerosis of the coronary arteries develops.

Objectives
At the end of the session, the participant (patient or patient and family) should be able to understand:

- normal readings of blood pressure;
- blood pressure variability and the frequency, causes, symptoms and potential complications of hypertension;
- how to reduce high blood pressure by modifying health-related lifestyle and using drug therapy;
- the long-term benefits of blood pressure lowering.

Content summary
Information about levels of blood pressure, their variability, causes, symptoms and potential complications of hypertension, advice on modifying health-related lifestyle to lower high blood pressure.
3.3.1 Blood pressure levels and variability

As described earlier, the heart has to pump blood continuously to ensure circulation and thereby to distribute oxygen and nutrients to all tissues of the body. As the heart contracts and relaxes (or beats), it imparts a force to the blood that is expressed as a pressure against the walls of the blood vessels through which it flows. The maximum pressure coincides with the contraction of the heart (systole) and is called systolic pressure; the minimum pressure coincides with its relaxation (diastole) and is called diastolic pressure.

Blood pressure fluctuates considerably during the day. It is lowest during sleep and rises when a person gets up. Blood pressure varies during the day in relation to various factors: the position of the body (standing, seated or lying), physical activity, emotional status, and other factors. The blood pressure often differs 10–20 mmHg when it is measured at different times, even minutes apart. Because of this normal variation, doctors do not diagnose a patient as having high blood pressure unless repeated measurements show it to be consistently elevated.

Under normal conditions, blood pressure stays around the same level when a person is sitting or standing still. The optimal blood pressure level should not exceed 120/80 mmHg (6). Exercise or emotional response can increase the blood pressure to allow more blood to be pumped to help the body cope with increased activity or stress. The body can tolerate such a temporary rise in blood pressure. One elevated blood pressure reading should not cause panic. However, this necessitates repeated measurements over several days and weeks followed by appropriate health intervention measures if necessary. If the systolic blood pressure level is persistently equal to or higher than 140 mmHg and/or if the diastolic blood pressure level is persistently equal to or higher than 90 mmHg, the doctor will diagnose a patient as having high blood pressure. The medical term for high blood pressure is hypertension. If a person is being treated for high blood pressure, such a person is still considered as having hypertension even if blood pressure levels are in the normal range.

3.3.2 Hypertension: frequency, causes, symptoms and effects

Between 15–30% of the adult population are hypertensive. Unfortunately, only half of them are aware of having high blood pressure and, of these, only half take steps to lower it.

In about 90% of cases the cause of hypertension is unknown. This type of hypertension is called essential or primary hypertension. When the cause is known, it is called secondary hypertension. High blood pressure might be secondary to a disorder of the endocrine system, kidney disease, congenital or acquired heart disease.

Certain factors are associated with high blood pressure. People who smoke, consume a high quantity of salt, misuse alcohol, experience chronic psychosocial stress or are not physically active are at a higher risk of developing high blood pressure. Also, people who have a history of hypertension in the family have a higher risk of developing high blood pressure. Though some people are at a higher risk of developing hypertension, anyone of any age and background can develop high blood pressure.

Obesity is common among people with hypertension. Obesity can contribute to hypertension in several possible ways. Obese persons with elevated blood pressure have greater resistance in their peripheral arterioles. Obesity leads to a greater cardiac output because the heart has to
pump more blood to supply the excess tissue. The increased cardiac output then can raise the blood pressure. Finally, obesity may be associated with a tendency for the kidneys to retain salt.

Hypertension is usually asymptomatic. In other words, most people with high blood pressure do not experience any symptoms. Such people would not know that their blood pressure is high unless blood pressure is checked. The disease progresses silently and may remain unnoticed for many years. If high blood pressure is not controlled adequately and continuously, it can cause serious health problems in the long term. Unfortunately, rather often a person’s blood pressure is measured for the first time when he or she receives health care because of a complication with high blood pressure.

Sometimes high blood pressure may cause headache, dizziness, shortness of breath, blurred vision or feeling tired or generally not feeling well. The symptoms can prompt people to consult a doctor and improve their compliance with treatment.

People with high blood pressure have an increased resistance of the peripheral arterioles. This increased resistance causes the heart muscle to work harder to pump the blood through blood vessels. Blood pressure tends to increase with age. The blood vessels lose elasticity as they are affected by atherosclerosis. High blood pressure increases the heart’s workload, oxygen demand and can strain the heart. The heart muscle can thicken and become stiffer and the dimensions of the heart can increase. Heart enlargement may be a forerunner of congestive heart failure, abnormal heart rate or irregular beating (arrhythmia). Untreated high blood pressure increases the risk of heart attack, stroke, kidney failure and retina damage. It also promotes the development of atherosclerosis, including the coronary arteries, especially in people with high levels of blood lipids. The higher the pressure and the longer it is not treated, the more frequent these complications are. The risk associated with high blood pressure is higher among people who have other risk factors such as smoking, obesity, high blood cholesterol or conditions such as diabetes, previous strokes or heart attacks.

3.3.3 Long-term benefit of reducing high blood pressure

Properly managing high blood pressure can reduce significantly the incidence and seriousness of the potential health complications. The treatment of hypertension has been associated with an approximate 40% reduction in the risk of stroke and 15% reduction in the risk of myocardial infarction. Lowering blood pressure has been shown to be beneficial even among people who already have heart disease. Controlling blood pressure also limits the development of atherosclerosis.

3.3.4 How to lower blood pressure

After diagnosing high blood pressure, the doctor examines the patient and orders tests to exclude secondary hypertension and to evaluate to which extent the high blood pressure has affected the heart and blood vessels. The results of this assessment, the level of blood pressure and the presence of other risk factors and conditions will determine individual hypertension management choices.

According to the European guidelines on cardiovascular prevention prepared in 2003 by the European Society of Cardiology Committee for Practice Guidelines (1), the goal of high blood pressure management is a blood pressure of less than 140/90 mmHg, but for people with diabetes and individuals at high total risk of cardiovascular disease, the blood pressure target should be lower.
Primary hypertension has no cure. However, effective measures can bring blood pressure to normal range. Controlling blood pressure requires a lifelong commitment to a healthy lifestyle and treatment with drugs, if necessary.

A healthy lifestyle includes weight control, limiting alcohol intake, dietary changes (decreasing sodium and increasing potassium intake), nonsmoking, increasing physical activity, better coping with stress and checking blood pressure regularly. It is also important to control the levels of blood lipids and blood glucose, if necessary (7).

**Diet.** A healthy diet should take into account the following.

- Energy intake should be adequate to energy needs (to maintain the normal weight within the limits of BMI 20–25 kg/m²).
- Salt intake should be limited to less than 5 grams (or 2 grams (85 mmol) of sodium) per day.

**Salt intake**
The average daily salt consumption in industrialized countries is about 10–12 grams per day. In total, about 75% of an average person’s daily salt intake comes from preserved and convenience food.

Both direct (adding salt to food or cooking) and indirect (salty processed foods such as sausages) consumption should be limited. Processed food products containing baking soda, baking powder or soy sauce usually have high levels of sodium. Canned food also might have a high amount of salt. Reading food labels is useful in selecting food products with less salt. The amount of salt can be estimated from the information provided on the labels of most purchased processed foods. The words salt (chemically termed NaCl or sodium chloride), sodium (chemically termed Na or natrium) on food labels are used to inform about the salt content. One gram of salt corresponds to 0.4 grams of sodium.

It is recommended to choose foods that are unsalted or have little salt such as fresh or frozen fruits, vegetables, cereals and skim milk. Instead of salt, various herbs, spices, vinegar or juices could be used.

- A healthy diet should be rich in potassium. Most vegetables and fruits are a good source of potassium, especially oranges, bananas, tomatoes and nuts.
- The consumption of alcohol should be limited.

**Physical activity.** Regular physical activity or a regular exercise programme of moderate intensity may help lower blood pressure over the long term. It also helps reduce weight and manage stress. Physical activity should take at least 30 minutes on most days of the week. Examples include walking, cycling, jogging, swimming, gardening or exercise classes.

**Nonsmoking.** Since smoking among people with hypertension substantially increases the risk of a stroke or a heart attack, smoking cessation is extremely important. If a person finds stopping smoking difficult, special smoking cessation programmes and/or a specific medication might be helpful.
**Reducing stress.** Managing stress may be helpful in lowering blood pressure. Avoiding or reducing the situations that are particularly demanding mentally and emotionally may accomplish this. Regular physical activity, adequate rest and sufficient sleep are important measures in managing stress. In some cases, the use of various relaxation techniques might be useful.

**Checking blood pressure.** Regular appointments with the GP should be made to check blood pressure and assess treatment efficacy. Regular blood pressure checks with the GP are necessary to ensure that blood pressure is properly controlled.

Some people benefit from self-measurement of blood pressure at home. Semiautomated or automated electronic devices that are relatively simple to use allow people to measure their own blood pressure (2). They appear to be affordable and the preferred option. Aneroid devices should be considered only if calibrated at regular intervals (such as every six months).

**Blood pressure-lowering medication.** If lifestyle measures do not control blood pressure adequately, treatment with drugs may be necessary. The most suitable drug or drugs will be prescribed. Some of the drugs may bring about adverse effects. The patient should consult with his or her physician rather than getting discouraged and stopping taking prescribed drugs. The patient should be advised not to stop taking the prescribed drug if blood pressure is brought back to normal, explaining that this means that blood pressure is effectively controlled and treatment should be continued. While the patient is taking drugs, lifestyle measures should be continued.
3.4 Diabetes

Introduction

Subject
The aim of this section of the session is to explain about diabetes mellitus as a major risk factor for coronary heart disease and provide information on how to control blood glucose and reduce the risk of coronary heart disease.

Previous knowledge required
Basic understanding of the structure and the functioning of the heart and blood vessels and how atherosclerosis of the coronary arteries develops.

Objectives
At the end of the session, the participant (patient or patient and family) should be able to understand:

- what diabetes is;
- what the metabolic syndrome is;
- the relationship between diabetes, the metabolic syndrome and coronary heart disease;
- how to manage the blood glucose level;
- how to reduce the risk of coronary heart disease in people with the metabolic syndrome or diabetes;
- the importance of a healthy diet.

Content summary
Information and practical advice on secondary prevention of coronary heart disease for people with diabetes.
Content

Diabetes is called mellitus (honey in Latin) because the urine of the affected people is sweet like honey. It is a disease linked to the utilization of sugar, the simplest energy source of the body. People with diabetes have a disorder of the regulation of the blood glucose level.

Carbohydrate eaten is broken down in the digestive system into glucose and other simple sugars and then absorbed by the intestine. From here the sugar is then dispersed via the bloodstream to all the organs and tissues including muscle, brain and fatty (adipose) tissue. It is used immediately by muscle or stored as energy reserve in the form of fatty acids (in adipose tissue) and of glycogen (in the liver and muscle).

Energy is needed for cells to survive and function. Glucose is the primary source of energy. To use or store glucose, the body needs insulin, a hormone produced by the pancreas when the level of glucose in blood rises after a meal. Insulin is the key that opens the door to cells for the blood glucose, permitting it to enter the cell and be used to produce energy or stored as energy reserve. Most tissues (except a few such as the brain and nervous system tissues) rely completely on insulin interaction for glucose to enter cells.

3.4.1 Types of diabetes

There are two major types of diabetes mellitus: type 1 (previously called insulin-dependent because it requires insulin for therapy) and type 2 (previously called non-insulin-dependent because it often does not require insulin for therapy, at least at the early stages).

Type 1 diabetes typically emerges in childhood and adolescence. This is a relatively infrequent disease, affecting one to two people per thousand. It results from the destruction of the pancreas cells that produce insulin. Insulin is lacking in such people. In this case, the person can only survive by receiving injections of insulin. Being a protein, insulin cannot be taken orally since, like all proteins, it would be digested by the stomach and lose its properties.

Type 2 diabetes is the most common type of diabetes and typically arises in adulthood. It usually appears after the age of 35–40 and tends to run in families affecting several members of the same family. The incidence of type 2 diabetes is 3–4%, higher after the age of 60. In type 2 diabetes, the body does not respond properly to insulin and insulin secretion is impaired.

Type 2 diabetes is insidious since initially there are no symptoms. The only way to recognize the disease in time, before symptoms or complications appear, is through a blood test for the level of glucose. Overweight (especially abdominal adiposity), physically inactive people as well as smokers and people with hypertension have an increased risk of developing this type of diabetes. A high saturated fat intake has been associated with an increased risk of abnormalities of glucose regulation as well.

3.4.2 Diagnostic tests for the status of glucose regulation

Disorders of glucose regulation (Table 2) can be divided into two categories: diabetes mellitus and impaired glucose regulation. The latter category can be divided into: impaired fasting glucose (IFG) and impaired glucose tolerance (IGT) (8). Two tests are used to define these categories.
• A fasting plasma glucose test measures the venous plasma glucose level after eight hours of fasting;
• In the oral glucose tolerance test, venous plasma glucose is measured after fasting at least eight hours and then two hours after drinking 75 grams of glucose.

### Table 2. Diagnostic categories of glucose regulation disorders

<table>
<thead>
<tr>
<th>Diagnostic category</th>
<th>Glucose measurement</th>
<th>Blood glucose level (venous plasma, mg/dl (mmol/l))</th>
</tr>
</thead>
</table>
| Diabetes mellitus                    | Fasting and/or Two hours after glucose load | ≥126 (≥7.0)  
|                                      |                                      | ≥200 (≥11.1)  |
| Impaired glucose regulation          | **Impaired glucose tolerance** (IGT) | Fasting (if measured) | <126 (<7.0)  
|                                      | Two hours after glucose load         | 140–200 (7.8–11.1) |
|                                      | **Impaired fasting glycaemia or impaired fasting glucose** (IFG) | Fasting | 110–126 (6.1–7.0)  
|                                      | Two hours after glucose load (if measured) | <140 (<7.8)  |


A fasting blood glucose level of less than 110 mg/dl (or 6.1 mmol/l) is categorized as “normal” (normoglycaemia).

IGT is defined as a condition in which the fasting blood glucose level is within normal range, but the blood glucose level is high two hours after taking an oral glucose load; IFG is defined as a condition in which fasting glucose levels are between 110 mg/dl (6.1 mmol/l) and 126 mg/dl (7 mmol/l).

Glucose regulation disorders are active many years before glucose builds up in the blood reaching the levels considered diagnostic for glucose intolerance and diabetes. Persons with IGT or IFG are at increased risk of developing type 2 diabetes. Impaired glucose regulation increases significantly the risk for cardiovascular disease.

Glucose levels equal or higher than 126 mg/dl (or 7 mmol/l) are called hyperglycaemia and are considered to diagnose diabetes, if confirmed with a similar result on a subsequent day. People with diabetes, especially those with onset of diabetes in adult life, have a higher risk of coronary heart disease. Strictly controlling blood glucose levels reduces the risk of coronary heart disease.

### 3.4.3 Insulin resistance

In type 2 diabetes, insulin is produced but the blood glucose level remains high. At first the body reacts, producing more insulin and creating an elevated level of insulin (this condition is called
hyperinsulinism). This insulin, however, is unable to perform its function. Why does this happen? Because the body has become resistant to the effect of insulin, requiring more insulin to move glucose from into cells.

More simply, if two people (one without and one with insulin resistance) are given 10 units of insulin, the blood glucose concentration of the person without insulin resistance might typically decline from 150 mg/dl (8.3 mmol/l) to 70 mg/dl (3.9 mmol/l), whereas the blood glucose concentration of the person with insulin resistance would decline from 150 mg/dl (8.3 mmol/l) to only 130 mg/dl (7.2 mmol/l).

How does insulin resistance arise? While it is often associated with familial, likely genetic predisposition, insulin resistance is worsened by overweight or obesity (or simply weight gain) and lack of physical activity. Insulin resistance by itself is an important cardiovascular risk factor. This condition is associated with atherosclerosis even before diabetes is diagnosed. As many as 50% of persons with newly diagnosed diabetes could already have experienced a cardiovascular complication.

For this reason, to reduce the risk of cardiovascular disease it is not sufficient to control blood sugar when it is elevated, but it is necessary to prevent the progression of insulin resistance by all means, particularly by adhering to an appropriate lifestyle.

Insulin resistance and hyperinsulinism can be reduced through an appropriate diet and increased physical activity. These are the first measures to be undertaken. A modest weight loss, about 5% (for instance from 80 to 75 kg) and moderate-intensity, regular physical activity (such as walking 3 km in 30 minutes on most days of the week or every day) can be very effective in reducing insulin resistance and thus the risk of cardiovascular disease. Only when these lifestyle measures are insufficient oral medications or insulin should be resorted to.

If insulin resistance is not corrected and is allowed to go on for years, the pancreas finally “gives up” and starts producing less insulin. Glucose then begins to build up in the blood, at first after meals, then also while fasting and classic symptoms of diabetes begin to appear.

The body tries to dilute the high concentration of glucose by drawing water from cells. The body will also try to eliminate excess blood glucose with urine. This causes frequent urination and polyuria (an abnormally high quantity of urine produced in one day). Excessive thirst (polydipsia) will develop. Irritation or inflammation of the external genitals, the urinary tract and the skin is frequent. People with diabetes have too much sugar in the blood but the body does not have sufficient energy to function. Since the cells will be starved, they will send signals to eat more, making patients very hungry. Tiredness, rapid, unexplainable weight loss and blurred vision will appear.

At this point, oral medications stimulating the pancreas to produce more insulin or aiming to reduce insulin resistance may still be effective in reducing blood glucose. However, diet and physical activity can reduce insulin resistance better than drugs.

In the end, drugs lose their effectiveness because the pancreas, even if stimulated, can no longer produce enough insulin to maintain normal levels of blood glucose. At this point, insulin injections become necessary and this type of diabetes behaves more like type 1.
3.4.4 Metabolic syndrome

Insulin resistance and hyperinsulinaemia are often associated with other metabolic changes: impaired glucose regulation or diabetes, hypertension, obesity (especially abdominal obesity: when the waist circumference is larger than 102 cm among men or 88 cm among women), dyslipidaemia (in particular increase in triglycerides, reduction of HDL cholesterol and sometimes increase in LDL cholesterol) and a tendency for blood clotting. This is the metabolic syndrome (also called plurimetabolic syndrome). Each feature of the syndrome represents a single cardiovascular risk factor, whose aggregation confers additional overall risk. The factor common to all these metabolic changes is probably insulin resistance and the subsequent compensatory hyperinsulinism, since the risk of cardiovascular disease increases well before the blood glucose level starts to rise.

The syndrome increases significantly the risk of coronary heart disease. Changes caused by the metabolic syndrome lead to atherosclerotic narrowing of the arteries and thus coronary heart disease. This explains why the risk of myocardial infarction or stroke is significantly greater among persons with diabetes or impaired glucose regulation.

3.4.5 Hyperglycaemia and hypoglycaemia

What happens if insulin resistance and high blood glucose (a high level of blood glucose is called hyperglycaemia) are not reduced? Prolonged hyperglycaemia can damage the circulatory system (large and small arteries) and nerves. Hyperglycaemia is a key reason of diabetes complications, which affect various organs: kidneys, eyes and nerves, in particular of the legs, causing foot problems. It can lead to kidney failure, blindness and amputation of limbs. Damage caused by hyperglycaemia is cumulative. It may not be noticed before complications manifest clinically.

Hyperglycaemia can turn into an acute complex medical condition. As described earlier, although the level of glucose in blood is high, the cells cannot take it and are starved of energy. To provide energy to cells the body starts metabolizing fatty acids. This process leads to accumulation of by-products called ketones that interfere with the acid-base balance of the body. This can lead to renal failure, loss of consciousness and even death.

Hypoglycaemia is a sudden drop of blood glucose to an unacceptably low level, typically causing unpleasant symptoms. This is not necessarily a serious problem, although it can be frightening when it happens for the first time. People with diabetes are advised to keep a supply of sugar (such as candy or orange juice) at hand to consume at once when symptoms occur. Hypoglycaemia is far more frequent among people with diabetes who are treated with insulin but can also occur among people with diabetes taking oral glucose-lowering drugs. It does not occur among people treated with diet and exercise only.

3.4.6 Diabetes management

The goal of treatment of people with diabetes is achieving a blood glucose level as near to normal level as possible. Strong clinical evidence indicates that the risk of cardiovascular disease among people with diabetes can be further reduced if, together with control of blood glucose, efforts are made to lower blood pressure, to manage dyslipidaemia and to reduce the risk of the blood to clot.

In diabetes management, diet and physical activity are extremely important. People with diabetes need to be on a healthy diet, have a healthy weight and be physically active every day. Those
with diabetes who smoke are often very dependent on tobacco. Tobacco smoke impairs glucose tolerance. Tobacco is very harmful for people with diabetes. All diabetes complications, including cardiovascular disease, eyes, kidney damage and foot problems, are worse in people who smoke.

Effective management of patients with diabetes or metabolic syndrome includes the following purposes:

- reducing overweight and increasing physical activity to reduce insulin resistance;
- quitting smoking;
- managing blood pressure, ideally keeping the level below 130/80 mmHg;
- managing dyslipidaemia, bringing LDL cholesterol below 100 mg/dl and lowering triglycerides below 150 mg/dl;
- reducing the risk of thrombosis: low-dose aspirin is recommended to lower the risk of blood clotting; and
- monitoring the blood glucose level is essential; if a healthy diet and physical activity are not sufficient in achieving satisfactory blood glucose levels, glucose-lowering drugs will be prescribed, but these are effective only when combined with a healthy diet and adequate continual physical activity.

These goals are within reach when people with diabetes are aware of the risks inherent in the disease and are prepared to collaborate with physicians and other health care personnel to modify their lifestyle, especially smoking, nutrition and physical activity habits.

### 3.4.7 Blood glucose control

As described above, it is very important to monitor whether treatment measures ensure that the blood glucose level is kept as close to normal as possible. The two most commonly used blood tests to assess blood glucose control are: the haemoglobin A1c (glycosylated haemoglobin A) test and the test of blood glucose concentration.

The haemoglobin A1c test is a metabolic control index used to assess diabetes management over time. It is a simple blood test that indicates the average level of blood sugar over the past three months. The normal value of the test varies from one laboratory to another but is about 6%. It corresponds to an average glucose level of 135 mg/dl (7.5 mmol/l). A 1% change in the haemoglobin A1c level corresponds to about a 30 mg/dl change in the blood glucose level.

Values of less than 7% can reduce cardiovascular risk among people with diabetes by about 50%. The closer the results of the haemoglobin A1c test to 6%, the better the diabetes control.

<table>
<thead>
<tr>
<th><strong>Haemoglobin A1c</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>There are several variants of haemoglobin, 90% of which is haemoglobin A. Haemoglobin A1c is a subtype of haemoglobin A that has glucose attached. The haemoglobin A1c test measures the percentage of haemoglobin A molecules in the red blood cells that have glucose attached. Once attached, glucose remains in these cells throughout their life cycle, which is 3–4 months. Checking this haemoglobin therefore assesses the blood glucose level over the past several months.</td>
</tr>
</tbody>
</table>
Blood glucose concentration for day-by-day control of diabetes management is self-tested. The test is usually done before meals and/or at bedtime. The concentration of blood glucose before meals should be 80–120 mg/dl and 100–140 mg/dl at bedtime.

3.4.8 Dietary therapy

Diet represents the mainstay of therapy of diabetes or the metabolic syndrome. Diet should be balanced with physical activity and, if necessary, glucose-lowering drug therapy. In the early stages of diabetes diet, combined with adequate physical activity is the sole therapy. The objectives of dietary therapy are:

- to maintain levels of blood glucose as near as possible to normal, avoiding situations of hypoglycaemia or hyperglycaemia;
- to maintain normal levels of blood cholesterol and triglycerides;
- to limit salt intake to the recommended levels; and
- to attain optimal body weight (the weight that the patient and physician recognize as being achievable and sustainable).

Optimal body weight may not necessarily correspond to the ideal weight calculated using the BMI (a BMI between 20 and 25). Even a small weight loss (5–10%) among people with diabetes is enough to improve not only control of blood sugar but also other cardiovascular risk factors such as high blood pressure.

The word diet brings to mind the idea of restrictions. There are many types of diets, such as low-calorie, low-fat, high-fat, low-carbohydrate, low-salt and others. The real meaning of diet, however, is to provide nutrients in sufficient quantity and quality within calorie needs to reach and maintain a good state of health. A healthy diet means developing good eating habits to be maintained over time. With the exception of sucrose, dietary recommendations follow the same line as dietary recommendations for coronary disease prevention.

It is important to limit the intake of saturated fat (the intake should not exceed 10% of total energy). The percentage of calories from carbohydrate in the diet of persons with diabetes should be about 55–60% of total daily calorie. Carbohydrates from whole grains, fruit, vegetables (to ensure adequate intake of non-starch polysaccharides) and low-fat milk is recommended.

Up to date there is no agreement on the use of sugar and sugar containing foods in the meal planning of people with diabetes or the metabolic syndrome. The consumption of sucrose cannot be unrestricted: the patient should discuss with his/her doctor or dietician the use of sugar in the diet. It is recommended that every individual should test his/her “tolerance” to such a food, monitoring its effects upon their blood glucose level.
3.5 Sedentary lifestyle

Introduction

Subject
The aim of this session is to provide information on the role of physical activity in the secondary prevention of coronary heart disease and to advise on how to achieve adequate physical activity.

Previous knowledge required
Basic understanding of the structure and the functioning of the heart and blood vessels and how atherosclerosis of the coronary arteries develops.

Objectives
At the end of the session, the participant (patient or patient and family) should be able to understand:

- the health enhancing effects of physical activity
- how much physical activity is needed
- the importance of physical activity for secondary prevention of coronary heart disease
- how to exercise
- the effect of drugs on the heart rate during exercise.

Content summary
Information on the negative effects of sedentary lifestyle on health, benefits of physical activity, ideas and advice on how to increase physical activity, recommendations on how to engage in physical activity for people with coronary heart disease.
**Content**

Sedentary lifestyle is low levels of physical activity at work, home and during leisure time. Such lifestyle includes only light physical activity related to typical day-to-day tasks. Lack of physical activity is associated with an increased risk of cardiovascular disease, overweight and obesity, diabetes and cancer.

Physical activity is any body movement caused by large skeletal muscles that results in an expenditure of energy (burning calories). Physical activity covers walking, cycling, occupational physical activity, leisure activities including sports and recreational activities, household work and commuting activities. Although a large percentage of people’s total energy is used for processing food and for basal metabolism, physical activity is an important determinant of total energy expenditure: it might account for 15–40% of energy expenditure. Any physical activity burns calories, but vigorous physical activity burns calories at a higher rate.

Physical fitness refers to the ability to meet the physical demands of work and leisure, that is, it reflects the physical working capacity. Exercise (exercise training or physical training) is a category of physical activity. Physical training is structured and planned body movements aimed to improve physical fitness.

### 3.5.1 The health-enhancing effects of physical activity

The physical activity required at work and home has been declining. In countries around the world, between 60% and 85% of adults are simply not active enough to benefit their health.

Men and women of all ages benefit from physical activity. Evidence shows that regular physical activity protects and promotes health as well as improves psychological wellbeing. When a person is engaged in regular physical activity, the musculoskeletal, cardiovascular and respiratory systems adapt to increase the physical capacity of the body. Physical activity requires support of the cardiovascular and respiratory systems to sustain the activity for longer periods. Regular physical activity of moderate or vigorous intensity influences significantly the amount of fat, muscle and bone tissue in the body. It increases the fitness of the cardiovascular and respiratory systems.

A programme of regular physical activity at moderate to vigorous intensity lasting at least 2–3 months can reduce:

- blood pressure
- resting heart rate
- LDL cholesterol and triglyceride level
- blood glucose level
- body fat and body weight if appropriate diet is followed
- the effects of stress on the body
- blood tendency to coagulate
- anxiety and depression.
Such a programme can improve:

- the flow of blood to the muscles, including the heart muscle
- oxygen use by the skeletal muscles
- the efficiency of the heart muscle
- HDL cholesterol
- insulin resistance
- psychological wellbeing.

There is evidence that regular moderate physical activity reduces the risk of coronary heart disease. Physical activity not only facilitates several biological mechanisms related to prevention of atherogenesis and thrombogenesis (reducing triglycerides and LDL cholesterol, raising HDL cholesterol, lowering blood pressure, improving insulin sensitivity and preventing or reducing overweight) but also improves the function of the endothelium of vessels walls. Regular physical activity leads to an increase in activity of the parasympathetic nervous system.

Long-term regular physical activity of moderate to vigorous intensity increases the mass and contractility of the heart (makes the heart muscle stronger) and decreases heart rate. This process is called heart conditioning. Physical activity reduces resistance of peripheral arterioles and enhances dilation of vessels. The maximum cardiac output and the amount of oxygen extracted from blood increase. Physical training also improves physical capacity and increases maximum oxygen uptake in persons with coronary heart disease complicated by heart failure. This improvement is related to higher oxygen uptake by exercising skeletal muscle and lower resistance of peripheral arterioles.

**Physical activity and oxygen consumption**

Physical activity is a major determinant of maximum oxygen consumption. Moderate to vigorous activity of skeletal muscle greatly increases the overall metabolism. Body tissues use oxygen and glucose at higher rates. During physical activity (exercise), oxygen consumption increases linearly with the intensity of the activity. The uptake of oxygen from the blood increases to the point at which the tissues are not able to extract more oxygen. This is called maximum oxygen uptake. It partially depends on physical training. The maximum oxygen intake is used as an estimate of fitness of the cardiovascular and respiratory systems.

The current recommendation of at least 30 minutes of at least moderate-intensity physical activity on most days of the week is considered sufficient to reduce the risk of cardiovascular disease. Among patients who have experienced myocardial infarction, this goal will reduce cardiac mortality by about 20–30%.

The benefits of physical activity can be attained whether a person is engaged in sport, planned exercise, household chores, work-related physical tasks, gardening or other leisure time physical activity. What matters is the duration rather than the type of physical activity. The ideal seems to be the equivalent of walking 25 km per week, which corresponds to brisk walking for 5 km five days per week.
Studies show that even the most inactive people can gain significant health benefits if they are engaged in regular physical activity. Physical activity does not need to be strenuous or prolonged. In sedentary people, even a modest increase in their physical activity produces health benefits.

### 3.5.2 Optimal frequency and intensity of physical activity

How much physical activity is required to obtain health benefit? Almost all research so far has shown that physical activity of moderate intensity provides health benefit. Greater benefits are achieved by engaging in continued physical activity of more vigorous intensity or longer duration. Vigorous physical activity raises the fitness of the cardiovascular and respiratory systems.

The intensity and duration of physical activity to produce a health benefit are related to the level of physical activity at baseline. For sedentary people, the exercise will be intense and the heart rate will be high, while the same exercise physically trained people will perform level easily and with a lower heart rate.

The minimum aim should be to carry out moderate intensity physical activity for 30–60 minutes per day on most days of the week (optimally daily): walking, jogging, cycling or other aerobic activities that involve the large muscle groups (muscle building is not an aerobic exercise and may increase blood pressure). The 30 minutes of physical activity of moderate intensity may be done in a single session or accumulated in bouts of 8–10 minutes during the course of the day. For example, short walks of about 10 minutes can be taken, such as:

- parking the car about 1.5 km from the destination and walking the rest of the way;
- getting off the bus two stops early on the way to work for 20 minutes of walking and then one stop early on the way home for another 10 minutes of walking;
- alternatively, other types of activity include gardening, cycling or swimming might be taken.

Physical activity of moderate intensity, such as brisk walking, causes a person to breathe faster than normal but still be able to talk easily. However, people who are unaccustomed to regular exercise or have a high cardiovascular risk profile should avoid sudden and high-intensity physical activity.

### 3.5.3 Physical activity for secondary prevention of coronary heart disease

Physical activity is important for people with established coronary heart disease. Regular physical activity can improve the functioning of the heart and reduce the clinical manifestations of atherosclerotic coronary heart disease (9).

Patients with coronary heart disease tend to limit their physical activity being afraid of increasing the risk of inducing new events due to physical effort or being afraid of worsening their heart condition.

Physical activity must be started gradually and continued regularly. Occasional heroic physical effort does not bring any benefit and can be extremely dangerous to health! It might take several weeks to reach the goal of 30–60 minutes.
The person with coronary heart disease should be advised to start the programme by being physically active for a few minutes with the aim of carrying out moderate-intensity physical activity for at least 30 minutes, in addition to usual activity, per day on most days of the week. Moderate physical activity means a lifestyle that includes physical activity equivalent to brisk walking (5–6 km per hour).

Activities at low-to-moderate intensity include:
- walking
- taking the stairs instead of the elevator
- gardening
- household work
- exercising at home and/or in a gym
- dancing
- swimming
- bicycling.

If a person starts having chest pain or discomfort, feels faint or light-headed or becomes extremely out of breath while exercising, the person should stop exercising at once and talk to the physician as soon as possible.

Patients should be advised to consult a physician before starting (or greatly increasing) physical activity if they have high blood pressure, experience pain or pressure in the chest and/or shoulder, feel dizzy or have a history of fainting, get breathless after mild physical effort, are middle-aged or older and have not been physically active or plan a vigorous physical activity programme. In such people, sudden vigorous physical activity can provoke an acute clinical manifestation of coronary heart disease.

Patients will benefit from a supervised programme of cardiac rehabilitation that adapts the type and amount of exercise prescribed to the individual’s capacity for physical effort. A supervised programme considers the safe conduct of physical training to prevent injuries and to recognize symptoms that might indicate unsuitability of the programme and early warning signs of an acute cardiac condition. Such a programme is available in cardiac rehabilitation centres. If the patient prefers physical training at home, clear and detailed guidance and regular check-ups by a GP should be ensured.

For those who have already experienced clinical manifestations of coronary heart disease, the intensity of the exercise to be taken should be decided individually, in strict accordance with a few simple rules.

- Physical activity must not cause symptoms such as chest pain or shortness of breath; if a person has the sensation of shortness of breath during exercise to the point of not being able to speak, he or she should stop immediately. The person should never try to determine whether the symptoms are of cardiac origin by continuing to exercise.
- The maximum recommended intensity of physical activity should be based on individual tolerance; in this respect, reference to the results of an exercise stress test or of a 24-hour electrocardiographic monitoring (Holter monitoring) of the heart work can
be useful. These tests will give a heart rate that should not be exceeded during exercise. For health safety reasons (avoiding provoking an acute cardiovascular event) it is advisable not to exceed 85% of the maximum heart rate recorded during these tests. For example if the exercise stress test gives a result of 130 heart beats per minute, it is best not to exceed a pulse rate of 110 beats per minute during physical activity or exercise. Patients should therefore learn to take their pulse.

- Patients with cardiovascular disease should avoid physical activity that requires substantial muscular tension, such as weightlifting or pushing a car. These people should also avoid competitive sport.

### 3.5.4 Procedures when exercising

When people start physical exercise, it is important to warm up the muscles first. This normally leads to slight sweating. Exercise should be done correctly, especially regarding the intensity and pace, to avoid muscular pain or joint problems. Strenuous physical activity should be avoided for at least two hours after a meal.

Physical exercise should never be stopped suddenly; the intensity and pace should be reduced gradually and followed by 5–10 minutes of cooling down.

Physical exercise should be adapted to the weather avoiding excessive heat or cold. The person should dress appropriately. To prevent dehydration adequate amount of fluid should be consumed during and after the physical activity.

### 3.5.5 The effect of drugs on the heart rate during physical activity

Treatment with some drugs such as beta-blockers reduces heart rate at rest and limits the increase in heart rate during exercise.

In patients who receive this treatment, the recommendations about the maximal heart rate during effort have to derive from exercise stress testing or Holter monitoring carried out during therapy. It is better to exercise while the drug is having its full effect.

### 3.5.6 Importance of motivation

One reason frequently cited for failing to exercise regularly is “lack of time”; very often time that could be spent exercising is spent on activities considered more important. In fact, in view of the long-term benefits, time spent exercising should be regarded as an investment that can be withdrawn in the future as better health. Further, all age groups can benefit, including elderly people. In any case, to obtain maximum advantage, physical activity should be treated as a pleasure and done without exhaustion. Choosing an enjoyable activity, being engaged in physical activity with friends or family members are useful in increasing motivation. A way to start increasing physical activity could be introducing into everyday routine activities, which require more energy.
3.6 Stress

Introduction

Subject

The aim of this section of the session is to provide information on the role of stress in cardiovascular disease and possibilities of stress management.

Previous knowledge required

Basic understanding of the structure and the function the heart and blood vessels and how atherosclerosis of coronary vessels develops.

Objectives

At the end of the session, the participant (patient or patient and family) should be able to understand:

- what stress is
- what causes stress
- the reactions to stress
- the consequences of long-term stress
- the consequences of stress for the cardiovascular system
- what personality type is most at risk
- how to reduce the consequences of stress.

Content summary

Information on stress, ideas and advice on coping with stress.
Various psychosocial factors may contribute to the development of atherosclerotic coronary heart disease and the onset of clinical events. They include stress, lack of social support, certain personality types and emotional patterns, low socioeconomic status and others. Evidence also shows that these factors tend to cluster in the same individual.

3.6.1 Definition of stress

Certain environmental physical or psychological stimuli are perceived by the body as potential dangers. The brain evaluates them as an alarm signal and triggers series of biological responses. The original purpose of this reaction was to give the necessary support to behavioural reactions (fight-or-flight response) to stimuli that might cause direct damage.

The stimuli capable of triggering stress are linked to the individual’s manner to cope with stress. Stress can be classified as chronic (such as stress at work, family conflicts) and acute (such as acute anxiety).

Given the complexity of the structures under consideration, the number of stressful stimuli is large and response to these stimuli varies markedly from person to person. Further, the same person may consider a particular incident as stressful one day and not the next. Events such as illness or the death of a family member are generally very stressful for anyone; stressful events that are less dramatic but occur on a daily or almost daily basis might include arguments at work or at home, driving in heavy traffic, continuous phone calls or going to the physician.

The following list of the most stressful events (stressors) was drawn up based on the subjective ratings of a population sample. The scale is relative, with 100 being the death of one’s spouse.

<table>
<thead>
<tr>
<th>Social readjustment rating scales</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Death of spouse</td>
<td>100</td>
</tr>
<tr>
<td>Divorce</td>
<td>73</td>
</tr>
<tr>
<td>Marital separation</td>
<td>65</td>
</tr>
<tr>
<td>Jail sentence</td>
<td>63</td>
</tr>
<tr>
<td>Death of a close relative</td>
<td>63</td>
</tr>
<tr>
<td>Illness or personal injury</td>
<td>53</td>
</tr>
<tr>
<td>Marriage</td>
<td>50</td>
</tr>
<tr>
<td>Redundancy</td>
<td>47</td>
</tr>
<tr>
<td>Reconciliation with spouse</td>
<td>45</td>
</tr>
<tr>
<td>Retirement</td>
<td>45</td>
</tr>
</tbody>
</table>


These are the most stressful life events, meaning a sudden change in life defined at a specific point in time. Life patterns also can cause chronic stress, such as excessive workload, shortage of time causing insufficient sleep or chronic problems in personal relationships.
3.6.2 Reactions to stress

Reactions to stress may be either biological or behavioural, and these may often be linked. For example, an external threat is a stressful stimulus that requires the fight-or-flight response (behavioural reaction). The body preparing for this reaction increases the production of certain hormones and activates the sympathetic autonomic nervous system (biological reaction). The release of hormones such as adrenalin generates immediate biological changes such as increasing heart rate, blood pressure, respiration and sweating and enhancing muscle contraction.

The biological reaction involves not only the endocrine system and the autonomic nervous system but also the immune system. The effects of stressful stimuli on the immune system occur over a longer term.

3.6.3 Consequences of chronic stress

In daily life, biological and behavioural reactions to stress occur repeatedly. In a stressful situation, the body needs to adapt rapidly to deal with the stressful stimulus modifying various parameters of homeostasis and mobilizing energy to respond to the perceived danger. These reactions may negatively affect the physical and mental health status of the individual especially if the body cannot restore its energy reserves. In particular, the continuing stressful stimuli without periods of recuperation may contribute to full exhaustion of energy to cope with the strain, to disturbances in the regulation of metabolism and to development of ill health.

Long-term stress has been shown to be linked to elevated blood cholesterol levels, possibly by affecting eating habits. For example, people in stressful situations might console themselves by eating tasty foods that often have high levels of fat. This also might lead to excessive body weight. Numerous studies indicate that long-term stress might be association with the risk of coronary heart disease. However there is no clear consensus of the relationship between stress and the development of coronary heart disease.

3.6.4 Consequences of stress for the cardiovascular system

The mechanisms of how stress can harm the cardiovascular system are not entirely understood.

Stress can contribute to coronary heart disease in two ways. The first is an unhealthy lifestyle – reduced physical activity, smoking, alcohol abuse, unhealthy eating habits and low compliance with preventive health interventions such as cardiac medicine – which is more prevalent in people with chronic stress.

The second is the link between stressful stimuli and corresponding physiological reactions, such as high heart rate and elevated blood pressure during an acute stress episode. Increased production of certain hormones involved in the stress response (such as adrenalin, noradrenalin and cortisone) can increase heart rate and blood pressure or the level of lipids and glucose in the blood and therefore indirectly cause the development of atherosclerosis. The activation of certain mechanisms also can promote the development of blood clots in vessels.

Social isolation and depression are apparently independent predictors of poor outcome in survivors of a myocardial infarction.
3.6.5 Which personality type is most at risk?

One problem still unresolved in assessing how stress influences health is how to measure stress levels. In fact, no test is capable of quantifying stress as a risk for cardiovascular disease.

Some features of the character of a person most at cardiovascular risk have been identified: hostility, anger, aggressiveness and others.

3.6.6 What are the most common causes of stress?

Stress is most often caused by the feeling of being unable to deal with problems that are perceived as being:

- too numerous, such as problems in personal relationships occurring at the same time as an unusually high number of work tasks;
- too prolonged, such as ongoing conflict with colleagues or family members;
- too difficult: such as taking on tasks required to be completed under time pressure.

3.6.7 Early warning signs of stress

The typical signs of stress are:

- tiredness: one of the earliest signs is the feeling upon waking that one has not rested well;
- problems in relationships with others, such as not enjoying being with other people, refusing to meet new people and deterioration of existing relationships with family members and colleagues;
- feeling disheartened: inability to solve even minor problems, lack of initiative and turning in on oneself;
- difficulty in concentrating;
- self-harming behaviour (such as drinking and smoking);
- headache, upset stomach and other frequent illness.

3.6.8 Stress management

Stress is unavoidable. Since no therapy is capable of eliminating the negative effects of a reaction to stress that is meant to be physiological, it is advisable to reduce stress by avoiding, whenever possible, the situations that are most demanding emotionally or by learning to deal with them in a less harmful way.

Interventions recommended to reduce stress include health behaviour modification, learning how to cope with stress, relaxation training, physical exercise programmes, psychosocial support and others.

The following simple advice may be useful to minimize the harmful consequences of stress.

- Change the individual’s reactions to stress. This requires the individual evaluating his or her behaviour in stressful situations. Keeping a diary for a few days may be useful; the person is advised to note how he or she reacts and feels during and after a stressful situation.
• Do less but do it better! People very often identify shortage of time as a source of stress. Therefore assessing daily and weekly commitments is useful. It is advisable to set out priorities and to keep some time every day for relaxation.

• Sometimes avoiding stressful situations is best, at least until the person can handle them comfortably.

Muscle relaxation can be very helpful in reducing reactions to stressful stimuli. The best known is autogenic training. Some muscle relaxation techniques are complicated and require the help of a psychologist. However, some simple exercises can be useful. For example, taking 15–20 minutes of rest (once or twice a day) in relaxing surroundings, without distractions, closing the eyes and breathing in and out slowly several times. The muscles should be relaxed gradually, starting with the feet and working up to the head. Then the deep breathing exercises are repeated.

Regular physical exercise has been also shown to be an effective means of managing stress.

Meeting people with similar problems can reduce the feeling of being stressed; talking about stressful situations or problems can help to solve them.

In more complex cases, it is advisable to consult a GP to try drug therapy or to consult a psychologist for advice on modifying lifestyle, stress management techniques and social counselling.
References


Introduction

Subject
The purpose of this session is to provide information on pharmaceutical therapy: what the drugs are, how they work, the desired effects, the unwanted (adverse) effects and their significance.

Previous knowledge required
Knowledge of the structure and the functioning of the heart and blood vessels and how atherosclerosis of coronary vessels develops. Knowledge of the risk factors for cardiovascular disease.

Objectives
At the end of the session, the participant (patient or patient and family) should be able to understand:

- the general guidelines and principles of pharmaceutical therapy;
- the importance of complying with treatment guidelines;
- the definition of the active ingredients of drug preparations;
- how drugs work (pharmacodynamics) and how they move through the body (pharmokinetics);
- the desired effects (indications) and the unwanted effects (side-effects) of drugs;
- the main drugs used for coronary heart disease.

Content summary
Information, knowledge and advice on drug therapy.
Content

Pharmaceutical therapy means treatment with drugs. Understanding drugs requires knowing what they are and how they work.

Drugs are chemicals used in preventing, diagnosing and treating disease. They are substances derived from high-level molecular research to identify the chemical structure and in-depth studies to evaluate the desired effects on the organism, the optimal dosage as well as the toxicity and adverse effects.

Pharmacodynamics (the study of the basis of action of drugs and their effects) and pharmacokinetics (the study of the movement of drugs through the body) provide understanding on how drugs work and act on organisms.

4.1 Pharmacodynamics

Pharmacodynamics study the biological effects produced by drugs: how drugs act to bring about therapeutic effects. This topic requires some understanding of molecular biology, a science that studies the functioning of cells at the molecular level, an invisible microcosm. Comparing a cell with a room may be helpful in understanding the mechanisms by which drugs work at the cellular level.

Room. The walls of a room delimit the interior, separating it clearly from the outside. The walls cannot be crossed or breached. However, the doors allow us to go into and out of the room and the windows allow light and air to penetrate. Without these means of access, we would be imprisoned in a dark, enclosed space. Further, the absence of electricity and telephones would prevent us from lighting the room artificially and communicating with the outside.

Cell. In a cell, the membrane corresponds to the room’s walls. Membranes could be said to act as a selective barrier to the passage of molecules. The room’s doors and windows are the equivalent of membrane channels that allow substances needed by the cell to cross the membrane. The electrical switches and the telephone are equivalent to the receptors contained on the membrane, which allow signals (information) sent by the autonomic nervous system and/or substances (messengers) circulating in the blood to reach the inside of the cell. These substances arrive at the outside of the cell by means of the capillaries, which can be compared with a corridor leading to a room.

Drugs bring about various specific therapeutic effects changing the way in which a cell normally functions. They stimulate, inhibit and/or modulate the receptors (a receptor is a structure that specifically recognizes and binds a substance acting as a messenger), the membrane channels and messengers (such as a neurotransmitter, drug, hormone and others). Drugs can cause unwanted effects (side-effects), which can be more or less unpleasant and dangerous. The adverse drug effects are generally known. However, it is not always possible to anticipate them. The prescribing physician should always assess the benefits and risks (therapeutic effects versus adverse effects) of a prescribed drug. Adverse effects should be reported to the physician immediately. The physician must always be consulted before any decision to stop taking the prescribed drug is made.
4.2 Pharmacokinetics

Pharmacokinetics is the study of how drugs move through the body. It covers absorption, distribution, metabolism (biotransformation) and excretion of drugs.

**Pathways of entry.** Drugs can be administered in various ways. The way a drug is administered constitutes the pathway of entry. The most widely used pathways of entry are the interfaces between: the intestine and capillaries (when drug tablets, drops or capsules are given by mouth), mucous membranes and capillaries (for example, sublingual application of nitroglycerine), the skin and capillaries (when drugs – patches, ointments – are given through the skin).

The pathway of entry affects the onset and duration of the therapeutic effect. For example, when rapid absorption is needed, drugs are administered sublingually or by inhalation. Each pathway of entry has advantages and disadvantages. The oral route is usually safer. Some drugs have delayed or reduced absorption when taken on a full stomach. Such drugs therefore need to be taken on an empty stomach. In contrast, many drugs have fewer side-effects on the digestive system when taken with food. Patients should be encouraged to talk to their doctor about the best time and way to take a prescription drug, especially if more than one drug is taken.

**Pathway of transport.** The pathway of transport is the blood. A drug enters the bloodstream through the capillaries of the entry interfaces and reaches the tissues (the interface between capillaries and cells). The dose (quantity) of the active substance contained in the drug preparation is calibrated so that, within a certain period of time, the substance will enter the bloodstream and reach and maintain the therapeutic range: an optimal drug concentration in the blood to achieve the desired therapeutic effect. An excessive dose of the drug will exceed the upper limit of the therapeutic range and adverse effects may appear together with the desired effects. Conversely, an insufficient dose will fail to reach the lower limit of the therapeutic range. This means that the desired therapeutic effect is not achieved but unwanted effects may still appear. Thus, an inadequate dose provides no benefit and is potentially harmful.

The systemic absorption of a drug depends on its physical and chemical properties, the nature of the drug product and the anatomy and physiological functions at the site of drug absorption. For systemic absorption, a drug must pass from the absorption site through or around one or more layers of cells to access the systemic circulation. To be absorbed into the cell, a drug must cross the cell membrane.

**Pathways of elimination.** The pathways of elimination are the liver and kidneys. Most drugs are either eliminated through the kidneys or metabolized in the liver and excreted through the kidneys. While circulating, the drug repeatedly crosses the liver, which is the body’s chemical laboratory. There the drug is broken down into inactive substances devoid of therapeutic effects, suitable for being expelled from the organism with the urine.

4.3 Drug products

Drugs are not generally given as a pure chemical substance but rather mixed into a compound that might have various forms such as a tablet, spray or capsule. A finished drug preparation includes the active substance and certain ingredients that constitute the dosage form. The finished drug preparation should meet the treatment objective by delivering the drug with maximum bioavailability (the measurement of the rate and extent of active drug that reaches the systemic circulation) and minimum adverse effects.
Several factors are taken into consideration, to determine the appropriate preparation of a drug: pharmacokinetics, bioavailability, route of administration, desired dose and drug dosage form. When a drug preparation is prescribed, it is very important to evaluate compliance and acceptability by the patient as well as cost. Knowledge of the pharmacokinetic profile of a drug is necessary to estimate the appropriate quantity of the active substance in the product and the release rate that will maintain a desired drug level in the blood. The goal is to attain a drug concentration in the blood sufficient to achieve the desired therapeutic effects with minimal adverse reactions (the therapeutic range).

A drug may have several names. Each drug has the chemical name of the molecule of the chemical substance. Since the chemical name is usually very complex, a drug can get a short defined and approved name. Such a name is known as the generic name. Manufacturers give a proprietary name to a finished drug preparation. Each preparation of a given drug may carry a different proprietary name.

Drugs are prescribed according to the nature of the patient’s disease and related health problems. Patients should use drugs only as advised by the doctor and should not take more or less drug than prescribed, in order to obtain the desired effects and to minimize the chances of unwanted reactions. Since drugs may interfere with other drugs, the physician should always be aware of any drugs taken in combination. Side-effects should be reported immediately.

If a special diet is prescribed, it should be followed carefully. The dosage of a drug might be reduced if lifestyle changes are effective.

When a disease does not cause symptoms, as often happens, some patients tend to forget about their medication. Patients also tend to fail to take their medication if side-effects appear (for example, some blood pressure-lowering drugs may cause impotence or fatigue). Understandably, these side-effects may profoundly affect the patient’s quality of life and his or her compliance with treatment.

When a dosing regimen requires a frequency of 2-4 times a day, patients often forget to take one or more doses. One daily dose tends to improve compliance. With this in mind, pharmaceutical companies have enabled rapidly eliminated drugs to be taken in a single daily dose by putting them into special containers that release the active ingredient gradually as they pass through the intestine. These products bear the label retard (delayed) or chrono (timed) alongside their commercial names. Rapidly eliminated drugs can be produced as patches for the skin-capillary interface, thus ensuring their constant absorption throughout 24 hours.

### 4.4 Drugs

The following classes of drugs used for preventing and managing coronary heart disease are covered: beta-adrenergic receptor blockers (beta-blockers), calcium channel blockers (calcium antagonists), angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor antagonists, antiplatelet agents and lipid-lowering agents.

#### 4.4.1 Beta-blockers (beta-adrenergic receptor-blocking agents)

Beta-blockers are used to reduce blood pressure, prevent angina attack, reduce the risk of a subsequent heart attack, manage irregular heartbeat and treat heart failure. Some beta-blockers
are also used to treat migraine, reduce symptoms linked to anxiety and manage some other conditions.

Names

<table>
<thead>
<tr>
<th>Generic names</th>
<th>Examples of proprietary names</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atenolol</td>
<td>Atenol, Seles-beta, Tenormin</td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>Concor, Monocor, Zebeta</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>Carvipress, Dilatrend, Eucardic</td>
</tr>
<tr>
<td>Labetolol</td>
<td>Normodyne, Trandate</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>Lopresor, Lopresor Retard, Seloken, Seloken Retard, Toprol-XL</td>
</tr>
<tr>
<td>Nadolol</td>
<td>Corgard</td>
</tr>
<tr>
<td>Propanolol</td>
<td>Inderal</td>
</tr>
<tr>
<td>Sotalol</td>
<td>Rytmo-Beta, Sotacor, Sotalex</td>
</tr>
</tbody>
</table>

Pharmacodynamics

Beta-blockers reduce myocardial oxygen demand by decreasing blood pressure, heart rate and making the heart muscle contract with less force. They widen arterioles increasing their diameter and thus allowing blood to flow under less pressure. This results in lower blood pressure.

Biomolecular mechanism

Beta-blockers block the transmission of nerve impulses of the sympathetic nervous system that influences (see session 1) the constriction and dilation of most blood vessels. Beta-blockers blocking the sympathetic nervous system prevent its excitant effects and allow the calming effects of the parasympathetic system to prevail in the heart and the peripheral arterioles. Thus, beta-blockers create a protective “shell” around the heart, which can then carry out its normal work calmly and undisturbed because it is protected from stressful stimuli.

Properly known as beta-adrenergic receptor-blocking agents, these drugs block the action of noradrenalin and similar substances. Noradrenalin is a chemical released by a nerve when it is stimulated. Noradrenalin, stimulating beta-adrenergic receptors (also called sympathetic receptors), transmits messages between nerves and muscles or between one set of nerves and another in the sympathetic nervous system. Beta-adrenergic receptors are found on various cells of the body. In the heart, when activated, these receptors transmit messages to the heart to speed up and increase the force of the myocardium contraction.

The sympathetic receptors stick out of the external surface of the cell membrane like “buttons”. They are called adrenergic because they are inclined to link up with noradrenalin, a hormone released during stress. A beta-adrenergic receptor behaves like an electric switch, in that it can be turned “on or off”. The receptor is “on” and “excites” the cell when linked to the noradrenalin (receptor-noradrenalin complex); it is “off” and “relaxing” for the cell when it is detached from the noradrenalin (a free receptor).

The biomolecular mechanism is based on competitive antagonism between two substances and takes place as follows. When beta-blocker molecules reach the external surface of the cell membrane, they link up with the sympathetic receptors, keeping them switched off. Noradrenalin, finding its place occupied by the beta-blocker, cannot link up (through competitive antagonism) and activate the receptor.
**Desired effects**
The desired effect is protecting the heart from overwork by cancelling the excitant effects of the sympathetic system, thereby achieving:

- reduced heart rate
- reduced work rate and oxygen consumption by the myocardium
- reduced excitability of the myocardium
- reduced blood pressure.

**Therapeutic uses**
Therapeutic uses include:

- preventing secondary angina caused by effort by reducing the heart rate and work rate, thereby reducing the demand for oxygen;
- preventing and treating certain types of arrhythmia (irregular heartbeat) caused by physical and/or mental stress by reducing the excitability of the myocardium;
- managing arterial hypertension;
- “protecting the heart”: the results of numerous clinical trials conducted with beta-blockers following heart attacks showed significant reduction in morbidity and mortality, including sudden death in patients treated with beta-blockers compared with untreated patients; and
- managing heart failure.

**Dosage regimen**
The pharmacokinetic features of beta-blockers vary from drug to drug. Most are eliminated slowly. Some are available in a programmed-release container to ensure a slow elimination. Depending on the speed of elimination, beta-blockers are taken once or twice daily. If taken as a single dose, they should be taken in the morning upon awakening. This affords the most benefits from the beta-blocker during waking hours, when the sympathetic nervous system is more active for two reasons:

- because of the circadian rhythm (day versus night and asleep versus awake) the sympathetic-parasympathetic balance (see session 1) is physiologically programmed so that the excitant effects of the former are dominant during the day, whereas the calming effects of the latter are stronger at night;
- because of daily activities involving physical and/or mental stress (voluntary and involuntary).

When beta-blockers are taken regularly, the body gets used to them. These drugs should be never stopped abruptly since sudden withdrawal could lead to recurrence of symptoms or even a heart attack. When treatment needs to be stopped, this should be done gradually under medical supervision.

**Side-effects**
Since various body tissues have beta-adrenergic receptors, the response to beta-blockers is therefore multiple. Beta-blockers block the beta-adrenerceptors not only in the vessels and the heart, but also in the bronchi, liver and other organs. A reduced sympathetic tone affects functions of all structures and organs.
The most frequent side-effects are: fatigue or tiredness; bronchial constriction; cold extremities caused by the constriction of the arterioles in the skin; minor depression; impotence (a urologist needs to be consulted to exclude other causes) or reduction in libido; skin rash and dry eyes; sleep disturbances, nightmares; digestive tract problems.

The most significant adverse effect of beta-blockers is the risk of provoking breathing difficulties as a result of blocking beta-receptors in the lungs, which might lead to bronchial constriction and narrowing the air passages in the lungs; beta-blockers may bring to the surface a predisposition to bronchial asthma that had gone unnoticed. All beta-blockers are therefore prescribed with caution to people suffering from asthma, bronchitis or other forms of respiratory disease. These side-effects are functional and disappear when treatment is suspended. Sometimes the heart rate can become too slow causing dizziness or fainting.

Contraindications
Contraindications include asthma, chronic bronchitis, emphysema or other conditions that affect breathing, acute heart failure, severe peripheral vascular disease, advanced heart block, low pulse rate (bradycardia), major depression.

Beta-blockers should be taken with caution in the presence of poor circulation to the hands and feet (Raynaud’s disease), diabetes (patient cannot detect when the blood sugar level becomes low), impaired kidney or liver function, stable heart failure.

4.4.2 Calcium antagonists (calcium channel-blocking drugs)

These drugs are used to treat primary and secondary angina, to lower high blood pressure and to treat certain types of heartbeat disturbances. There are two types of calcium channel blockers. One group is the dihydropyridines. Drugs of this group are termed vasoselective since they affect the cells of the muscle layer of the vessel wall more than the cells of the heart muscle. The other group is the non-dihydropyridines. They are termed cardioselective since they act almost equally on myocardial tissue and the cells of the muscle layer of the vessel wall.

Names

<table>
<thead>
<tr>
<th>Generic names</th>
<th>Examples of proprietary names</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dihydropyridines or vasoselectives</strong></td>
<td></td>
</tr>
<tr>
<td>Amlodipine, felodipine, isradipine, lacidipine, manidipine, nicardipine, nifedipine, nitrendipine and others</td>
<td>Adalat, Anifed, Antacal, Baypress, Feloday, Iperten, Lacipil, Lacirex, Lomir, Norvasc, Prevek, Vascoman, Vasodin and others</td>
</tr>
<tr>
<td><strong>Non-dihydropyridines or cardioselectives</strong></td>
<td></td>
</tr>
<tr>
<td>Diltiazem</td>
<td>Altiazem, Angizem, Cardizem, Dizene, Tildiem</td>
</tr>
<tr>
<td>Gallopamil</td>
<td>Procorum</td>
</tr>
<tr>
<td>Verapamil</td>
<td>Calan, Isoptin, Isoptin Press, Isoptin Retard, Quasar, Verelan</td>
</tr>
</tbody>
</table>

Pharmacodynamics

Vasoselective calcium channel-blocking drugs act by relaxing the smooth muscles of the walls of the arterioles, decrease resistance to blood flow. As the blood flows with less resistance the work of the heart and its consumption of oxygen are reduced. These drugs also increase coronary blood flow. They do not slow heart rate and do not have antiarrhythmic properties.
Cardioselective calcium channel-blocking drugs lower the myocardial oxygen demand by decreasing the force of the heart’s pumping action (contraction of the myocardium) and heart rate.

**Biomolecular mechanism**

Calcium channel-blocking drugs decrease smooth muscle contractility inhibiting calcium movement across the cell membrane through calcium channels. Calcium ions regulate the contraction of the muscle cell: they activate contractile proteins in the cell.

The calcium channels, which are like gates in the cell membrane, modulate the amount of calcium ions entering the muscle cell and its function as follows:

- the more calcium enters (if most of the channels are open), the greater the contractile force.
- the less calcium enters (if most of the channels are closed), the lesser the contractile force.

The number of open calcium channels depends on the autonomic nervous system. The sympathetic nervous system increases, the parasympathetic nervous system decreases the number of open calcium channels.

The walls of the arteries contain, as described earlier, an elastic layer of muscle that, by tightening and relaxing, narrows or dilates the vessel lumen, enhancing or reducing the flow of blood. When more calcium ions enter cells, the muscle layer of the peripheral arterioles, including coronaries over-contracts. This might cause a significant obstacle to the flow of blood and might facilitate respectively myocardial ischaemia and/or increase blood pressure.

In the heart, the quantity of calcium ions that enter the cells modulates heart rate and the work of the myocardium. The more calcium enters, the faster the heart rate and the greater the strength of the myocardium’s contraction (work) and its consumption of oxygen; the less calcium enters, the slower the heart rate and the lesser the contractile force of the myocardium and consumption of oxygen.

Vasoselective calcium antagonists plug the calcium ions channels of the cells of the muscle layer of peripheral arterioles and coronary arteries. Cardioselective calcium antagonists act on the calcium ions channels of the myocardium cells and the cells of the conduction system of the heart.

**Desired effects**

The desired effects of dihydropyridines (vasoselective calcium antagonists) are relieving coronary artery spasm by dilation of coronary arteries and lowering blood pressure by reducing peripheral resistance to the blood flow. The desired effects of non-dihydropyridines (cardioselective calcium antagonists) are lowering myocardial oxygen demand by reducing heart rate, myocardial contractility and lowering blood pressure.

**Therapeutic uses**

Therapeutic uses of vasoselective calcium antagonists include preventing primary angina and reducing blood pressure. This group of calcium channel blockers is also used in Raynaud’s syndrome, a condition of poor blood flow to the extremities.
Cardioselective calcium antagonists are used for prevention of secondary (effort) angina and to manage certain arrhythmias.

Unlike the beta-blockers, calcium channel blockers have not been shown to reduce the risk of a heart attack in people with unstable angina. For this reason, calcium channel blockers are usually prescribed for people with angina who do not respond to or cannot take beta-blockers.

**Dosage regimen**

Many calcium channel blockers come in a short-acting and a long-acting (sustained-release) form. The short-acting form may have adverse long-term consequences such as stroke or heart attack. These effects are presumably due to the wide fluctuations in the blood pressure and heart rate that occur during treatment. The fluctuations result from the rapid onset and short duration of drug action. When the calcium channel blockers are used in sustained-release preparations, however, less fluctuation in blood pressure and heart rate occurs. Accordingly, the sustained-release forms of calcium channel blockers are probably safer for long-term use.

Some evidence suggests that stopping calcium-channel blocking drugs suddenly may worsen angina.

**Side-effects**

The most frequent side-effects are: leg or ankle swelling; hot flushes, tenderness or bleeding of the gums; constipation; skin rush; hair loss; headache; excessive lowering of heart rate (non-dihydropyridines) and blood pressure; depressing the function of the myocardium.

**Contraindications**

Calcium channel blockers should be used with caution in the following situations: liver or kidney problems (only applies to some drugs); very low blood pressure; diabetes (applies to nifedipine); heart failure (applied to cardioselectives); breastfeeding. Contraindications include hypersensitivity, certain impairments of the conduction system of the heart (cardioselectives), recent myocardial infarction (vasoselectives), pregnancy.

**4.4.3 Angiotensin-converting enzyme inhibitors**

Angiotensin-converting enzyme (ACE) inhibitors are used to lower high blood pressure, to treat heart failure and acute myocardial infarction and in secondary prevention of coronary heart disease and other forms of atherosclerosis.

**Names**

<table>
<thead>
<tr>
<th>Classes</th>
<th>Generic names</th>
<th>Examples of proprietary names</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Class I: captopril</strong></td>
<td>Captopril</td>
<td>Capoten</td>
</tr>
<tr>
<td></td>
<td>Benazepril</td>
<td>Cibacen</td>
</tr>
<tr>
<td></td>
<td>Cilazapril</td>
<td>Inibace, Initiss</td>
</tr>
<tr>
<td></td>
<td>Delapril</td>
<td>Delaket</td>
</tr>
<tr>
<td></td>
<td>Enalapril</td>
<td>Converten, Enapren, Naprilene, Vasotec</td>
</tr>
<tr>
<td></td>
<td>Fosinopril</td>
<td>Eliten, Fosipres, Tensogard</td>
</tr>
<tr>
<td></td>
<td>Moexipril</td>
<td>Femipres, Univasc</td>
</tr>
<tr>
<td></td>
<td>Perindopril</td>
<td>Aceon, Conversyl, Pro captan</td>
</tr>
<tr>
<td></td>
<td>Quinapril</td>
<td>Accupril, Acequin, Quinazil</td>
</tr>
<tr>
<td><strong>Class II: prodrugs</strong> (a prodrug means that the active ingredient remains metabolically inactive until it is modified in the liver)</td>
<td>Ramipril</td>
<td>Altace, Quark, Triatec, Unipril</td>
</tr>
<tr>
<td><strong>Class III: water-soluble</strong></td>
<td>Lisinopril</td>
<td>Alapril, Prinivil, Zestril</td>
</tr>
</tbody>
</table>
They differ in chemical structure and pharmacokinetic properties. Based on chemical structure, there are three groups of drugs (sulfhydryl-containing, carboxyl-containing and phosphinyl-containing inhibitors). They also can be classified as drugs that are active in their own right or drugs that need biotransformation by the liver.

**Pharmacodynamics**

ACE inhibitors reduce peripheral arterial resistance and decrease blood volume. These drugs inhibit the production of ACE, an enzyme participating in the metabolism of angiotensin II, which acts as vasoconstrictor and of bradykinin, which counteracts the effects of angiotensin II.

Angiotensin II is a substance that makes vessels narrow and thus increases blood pressure. It also stimulates the release of the hormone aldosterone, which promotes the reabsorption of water and sodium by the kidneys. Moreover, angiotensin II plays a role in inflammation, neurohormonal activation and nitric oxide synthesis in endothelial cells of the vessel wall.

ACE inhibitors block the effect of angiotensin II, therefore vessels relax and widen leading to lower blood pressure. A lower blood pressure makes the pumping function of the heart easier.

**Biomolecular mechanism**

When the blood pressure drops, the kidneys produce an enzyme called renin. This enzyme converts the protein angiotensinogen (produce in the liver) into angiotensin I. This is then transformed into angiotensin II which causes vessel constriction interacting with angiotensin receptors of the smooth muscle of vessels walls.

ACE facilitates the conversion of angiotensin I into angiotensin II. ACE inhibitors block ACE production thereby reducing production of angiotensin II. The level of angiotensin II drops and its constricting effect on the blood vessels is prevented. As a consequence, the vessels (arteries and veins) dilate. A lower level of angiotensin II also interferes with aldosterone availability. Therefore more fluid is filtered from the vessels by the kidneys into the urine and blood volume decreases. Both mechanisms, dilation of vessels and decrease in blood volume, help to reduce blood pressure and to decrease the workload of the heart. ACE inhibitors are therefore very useful for reducing high blood pressure, enhancing blood flow to various organs and decreasing the amount of work the heart has to do.

**Figure 2. The renin-angiotensin system**

<table>
<thead>
<tr>
<th>THE RENIN – ANGIOTENSIN SYSTEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>LIVER</td>
</tr>
</tbody>
</table>

- Renin \downarrow\rightarrow Angiotensin Converting Enzyme (ACE) \downarrow\rightarrow ANGIOTENSINOGEN \Rightarrow ANGIOTENSIN I \Rightarrow ANGIOTENSIN II \downarrow

- • ARTERIOLAR CONSTRICITION
- • ↑ ALDOSTERONE
- ↑ sodium + ↑ water
ACE inhibitors also favour the activity of bradykinin that contributes to vessels dilation. ACE inhibitors inhibit kininase II which brakes down bradykinin and therefore increase the level of bradykinin. Bradykinin has a counteracting effect on angiotensin II and is involved in releasing nitric oxide.

Figure 3. Action of the angiotensin converting enzyme

\[
\begin{align*}
\text{active bradykinin} & \iff \text{ACE} \iff \text{Angiotensin I (inactive)} \\
\downarrow & \downarrow \\
\text{inactive bradykinin} & \iff \text{Angiotensin II (active)}
\end{align*}
\]

ACE inhibitors have also been shown to have an antiatherosclerotic effect by reducing the proliferation of smooth muscle cells in the walls of the vessels, improving vascular endothelial function, reducing inflammation and by other mechanisms. These drugs have some antithrombotic effects and decrease sympathetic tone. These multiple actions are very useful in the secondary prevention of coronary heart disease.

**Desired effects**
The desired effects are:

- Making the pumping function of the heart easier and reducing myocardial oxygen demand by decreased blood pressure and reduced volume of blood that the heart has to pump (reduced amount of venous blood returning to the heart);
- promoting vasodilation to improve coronary blood flow and vasodilator reserve;
- delaying the development of atherosclerosis.

**Therapeutic uses**
ACE inhibitors are used to treat high blood pressure and heart failure. ACE inhibitors may reverse heart muscle hypertrophy in patients with hypertension.

When given shortly after a heart attack, some ACE inhibitors can facilitate heart muscle recovery. Among people with diabetes, these drugs slow the process that leads to kidney damage. ACE inhibitors have been found to be beneficial in secondary prevention of coronary heart disease.

**Dosage regimen**
ACE inhibitors are eliminated slowly therefore it is sufficient to take them once or twice daily. Some people may feel dizzy or faint with the first dose due to a sudden drop in blood pressure. The first dose therefore should be taken at bedtime or when lying down. These drugs are usually started on a low dose, especially among patients with heart failure and normal or low blood pressure. ACE inhibitors should be taken on an empty stomach one hour before meals. The number of doses will depend on the type of ACE inhibitor and other considerations. Blood pressure, blood potassium concentration and kidney function should be checked regularly.

**Side-effects**
Annoying dry cough is a common side-effect. If it is misinterpreted, it might lead to unnecessary testing of the respiratory system. This effect is functional and disappears when the ACE
inhibitors are suspended. The most likely explanation is related to the inhibition of bradykinin degradation, which takes place simultaneously with the inhibition of angiotensin conversion.

For patients who develop chronic and disturbing cough on an ACE inhibitor, an angiotensin receptor antagonist drug is a good substitute. The angiotensin receptor antagonist drugs appear to have many of the advantages of ACE inhibitors without the associated cough.

There is a very rare life-threatening side-effect: swelling of the tongue and the lips (angioedema). Other side-effects include low blood pressure, acute kidney failure, skin rash, dizziness, a high potassium level, vomiting and diarrhoea, sore throat and fever.

These drugs should be used with care in patients with reduced kidney functioning, patients on a low-salt diet and with high potassium content, patients taking potassium-sparing diuretics and in breastfeeding women.

Contraindications
Contraindications include low blood pressure, hypersensitivity, history of angioedema, pregnancy, bilateral renal artery narrowing.

4.4.4 Angiotensin II receptor antagonists (sartans)
The angiotensin II receptor antagonists are a recently introduced subtype of ACE inhibitors.

Names

<table>
<thead>
<tr>
<th>Generic names</th>
<th>Examples of proprietary names</th>
</tr>
</thead>
<tbody>
<tr>
<td>Candesartan</td>
<td>Blopress, Ratacand</td>
</tr>
<tr>
<td>Irbesartan</td>
<td>Aprovel, Karvea</td>
</tr>
<tr>
<td>Losartan</td>
<td>Lortaan, Losaprex, Neolotan</td>
</tr>
<tr>
<td>Telmisartan</td>
<td>Pritor</td>
</tr>
<tr>
<td>Valsartan</td>
<td>Diovan, Tareg</td>
</tr>
</tbody>
</table>

Angiotensin II receptor antagonists produce effects similar to those of ACE inhibitors but do not increase the level of bradykinin. They therefore do not cause the cough or angioedema sometimes associated with conventional ACE inhibitors.

Pharmacodynamics
Angiotensin II receptor antagonists lower the blood pressure, preventing blood vessels from constriction through their interference with ACE action. Angiotensin II receptor antagonists do not interfere with the deactivation of bradykinin and therefore do not cause coughing.

Biomolecular mechanism
Angiotensin receptor antagonists do not interfere with ACE production. They work by blocking the angiotensin receptors. These drugs prevent ACE from binding to the angiotensin receptors located on the smooth muscles of blood vessels and the heart. When angiotensin II attaches to the angiotensin II receptors, it causes blood vessels to constrict. By blocking these receptors, angiotensin II receptor antagonists prevent the vasoconstrictive effect of angiotensin II and thus lower blood pressure.

Desired effects
The desired effect is reducing blood pressure and lowering myocardial oxygen demand.
Therapeutic uses
Angiotensin II receptor antagonists are used to treat hypertension and heart failure. They are particularly useful for patients who cannot tolerate ACE inhibitors due to cough.

Dosage regimen
Angiotensin II receptor antagonists are taken once or twice a day.

Side-effects
Headache, dizziness, fainting, sore throat, angioedema and diarrhoea.

Contraindications
Pregnancy and severe impairment of kidney or liver functioning.

4.4.5 Nitrates
Nitrates are used primarily to abort or prevent an angina attack. The main action is dilation of veins and arteries.

Names

<table>
<thead>
<tr>
<th>Generic names</th>
<th>Examples of proprietary names</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroglycerine: sublingual, oral spray or transdermal (patch or ointment)</td>
<td>Adesitrin, Dermatrans, Deponit, Martindale, Minitran, Nitraket, Nitroderm TTS, Nitroderm, Nitromin, Nitrosylon, Sustac, Transderm-Nitro, Triniplas, Trinitrina, Venitrin</td>
</tr>
<tr>
<td>Isosorbide dinitrate: sublingual, skin spray, tablets</td>
<td>Carvasin, Cedocard, Isordil, Isocard, Sorbitrate</td>
</tr>
<tr>
<td>Isosorbide mononitrate (active 12–24 hours) (biologically active metabolite of isosorbide dinitrate)</td>
<td>Imdur, Monoket, Ismo</td>
</tr>
</tbody>
</table>

Pharmacodynamics
Nitroglycerine dilates veins and arteries, particularly medium sized such as the coronary arteries. Widening the veins decreases the volume of blood returned to the heart, which makes the pumping function of the heart easier. Widening of peripheral arteries reduces blood pressure. As a result, the heart is working less and therefore the myocardium demands less oxygen. In addition, widening the coronary arteries increases the blood supply to the heart muscle and consequently the supply of oxygen.

Biomolecular mechanism
Nitrates replenish the body with nitric oxide. After entering the body through the skin or the lining of the mouth or of the intestine, the drug reacts chemically with the sulphydryl group of cysteine, an amino acid contained in the cell membrane and is transformed into nitric oxide (this substance is produced normally by the endothelial cells of the vessels). Nitric oxide released in the lining of veins and arteries walls causes the smooth muscles of the walls of the vessels to relax. This makes veins and arteries wider.

Desired effects
The desired effects include reducing the demand of oxygen for the myocardium and increasing the blood supply to the myocardium. The oxygen demand is reduced due to a decline in the volume of blood returning to the heart and a reduction in blood pressure due to widening of
peripheral arterioles. Thus, the workload of the left ventricle is reduced and less oxygen is needed for the myocardium. The blood supply to the myocardium is higher due to wider coronary arteries.

**Therapeutic uses**

Nitrates are used for the acute and preventive treatment of angina pectoris. They can be also used for treating hypertension.

Nitroglycerine sublingual preparation or oral spray is the drug of choice to treat an angina attack. It is also very effective for managing hypertensive crisis.

Angina is better prevented by long-acting drug preparations such as a transdermal format of nitroglycerine or isosorbide mono- or dinitrate preparations. However, sublingual nitroglycerine taken just before activities likely to cause angina (“predictable” angina) can effectively prevent it.

**Dosing regimen**

Nitroglycerine taken orally is absorbed and promptly metabolized by the liver. To achieve therapeutic effects, this drug is therefore administered as a sublingual tablet or spray, topical ointment or patch. For rapid action, nitroglycerine is available as a sublingual tablet or an oral spray. A sublingual tablet should be taken while seated or lying down to prevent the risk of fainting since the drug reduces blood pressure.

Sublingual absorption is rapid and angina pain starts disappearing in as little as 30 seconds. The attack is usually resolved in one to three minutes. The drug should be taken again after five minutes if the attack persists for a maximum of three doses. The effect of these preparations may last up to 30 minutes.

The spray form must be delivered under the tongue. Similarly, if the attack does not subside, it should be taken again in 5 and 10 minutes.

If angina is not relieved after three doses (that is, if the angina pain continues longer than 15 minutes), the patient should be taken to the hospital. Anyone with angina must have a rapidly acting nitroglycerine preparation with him or her at all times.

The sublingual tablets are very susceptible to moisture and therefore should not be kept in places where the level of moisture is high (such as in a bathroom). Nitroglycerine sublingual tablets cannot be stored for a long period of time, as they lose efficacy: the tablets should be replaced within eight weeks from opening the bottle.

Isosorbide mononitrate and isosorbide dinitrate are available as skin patches, paste or tablets. Sustained-release tablets of isosorbide mononitrate or dinitrate are taken one to four times a day (depending on the type of the preparation). They should be taken with water and not crushed.

Nitroglycerine ointments are active for 4–6 hours. When an ointment is applied, care should be taken not to put the ointment on the hands to prevent additional absorption.

The duration of action of the adhesive patches could be 24 hours. The patches are waterproof and therefore are not affected by taking a shower or bath.
Administration of long-acting nitrates leads to tolerance and the drug becomes ineffective. Nitrates are not transformed into active nitric oxide when the “reserves” of the sulfhydryl group of cysteine are exhausted. Daily administration should therefore be planned with a time gap (a period of time without taking the drug) of at least 4 hours every 24 hours. For example, nitroglycerine patches are applied for 12 hours and removed in the evening. The time gap allows the cell membrane to recharge itself with the sulfhydryl group and to reactivate the production of nitric oxide for the patch to be effective.

Use of this group of drugs should not be stopped suddenly after a long period of therapy in order to prevent a rebound of angina.

**Side-effects**

The side-effects include headache, flushing of the head and neck, palpitation, increased heart rate, nausea, vomiting, low blood pressure possibly leading to weakness, dizziness or syncope (fainting) with nitroglycerine sublingual tablet or oral spray. Headache, dizziness, flushing and fainting tend to decline with continued therapy.

**Contraindications**

Contraindications include significant aortic valve stenosis; idiopathic hypertrophic subaortic stenosis; use of sildenafil (such as Viagra) preparations (nitrates and sildenafil preparations cannot be taken together since this combination can significantly lower blood pressure); low blood pressure; allergy to nitrates; closed-angle glaucoma; malnutrition; reduced activity of the thyroid gland; severe kidney disease; severe liver disease.

### 4.4.6 Antiplatelet agents

Antiplatelet drugs impair blood clotting and therefore are used to reduce the risk of blood clotting in vessels.

**Names**

<table>
<thead>
<tr>
<th><strong>Generic names</strong></th>
<th><strong>Examples of proprietary names</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetylsalicylic acid (or aspirin)</td>
<td>Alka-Selzer, Ascriptin, Aspirin, Aspirinetta, Aspro, Bufferin, Cardioaspirin, Cardirene, Carin, Cemirit, Kilios, Vivin C</td>
</tr>
<tr>
<td>Dipyridamole</td>
<td>Persantin</td>
</tr>
<tr>
<td>Ticlopidine</td>
<td>Anagregal, Ticlopidina, Tiklid</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>Plavix</td>
</tr>
</tbody>
</table>

Various antiplatelet drugs are available. This section describes only acetylsalicylic acid (aspirin). Aspirin is the most widely used drug. Other agents (dipyridamole, ticlopidine and clopidogrel) might be prescribed alone or in combination. The use of aspirin with dipyridamole or ticlopidine or clopidogrel is limited to selected groups of patients and for limited periods of time.

**Pharmacodynamics**

Aspirin inhibits platelet activation and aggregation (joining together). The drug makes platelets less sticky and prevents platelets from aggregating at the site of a wound or injury.

**Biomolecular mechanism**

Aspirin prevents the formation of the thrombus by inhibiting the production of substances that activate the platelets to aggregate.
Along with coagulation, the aggregation of blood platelets is a defence mechanism in the case of rupture (such as a cut with a knife) of a vessel wall. It is aimed at blocking with a blood clot the blood’s way out.

Thrombosis is a pathological process in which a blood clot forms in the lumen of an intact blood vessel. Thrombus formation can be activated by a lesion of the endothelium; for example, desquamated or ulcered fibrous shell of unstable atheroma. The resulting blood clot produces an unwanted obstruction for blood flow in the affected vessel lumen.

Platelets form a thrombus by means of adhesion, activation and aggregation. These three successive steps can be simplified as follows: platelets circulate in the blood; when they come into contact with the endothelium, they behave according to its state. If the surface is smooth, the platelets slide and continue their journey. They stop on the areas where the “tiles” (cells of the endothelium) are absent. This breach of the endothelium exposes the “rough” underneath connective tissue that acts like glue. Platelets start sticking (adhesion) taking the form of a polyp (activation). Wrapping themselves tightly round each other (aggregation), they form the thin base of a thrombus. At the same time they “throw” substances (prostaglandins such as thromboxane) that “strike” and activate more platelets that continue to pass through in large numbers. These too clump, continuing to produce prostaglandins that activate even more platelets. The multiplication of the activation-aggregation process leads to the formation and growth of a blood clot that may obliterate the vessel completely.

**Prostaglandins**

Prostaglandins are a type of hormones found in most body tissues. Prostaglandins are membrane lipids. They are active at very low concentrations. Prostaglandins are involved in the process of pain, fever, blood coagulation and regulation of blood pressure. They act locally, that is, close to the cells that produce them, and are destroyed in a very short time. They also regulate the aggregation of platelets.

Aspirin blocks the synthesis of prostaglandins. The drug inactivates in platelets and endothelial cells cyclooxygenase, a key enzyme in prostaglandins synthesis. Platelets are not able to replenish cyclooxygenase and therefore the production of vasoconstricting and platelet activating and aggregating prostaglandins is blocked. This results in preventing the blood clot from growing.

Ticlopidine and clopidogrel inhibit the process of platelet activation and aggregation through a mechanism different from that of aspirin.

**Desired effect**

The desired effect is to prevent thrombogenesis in those at increased risk of thrombus formation such as in people with elevated risk of coronary heart disease or established coronary heart disease.

**Therapeutic uses**

Aspirin is a fundamental element in managing coronary heart disease. A low dose of aspirin reduces the chance of the first heart attack as well as the chance of a new heart attack in patients who have already suffered one. Therapeutic uses also include preventing blood clotting after coronary artery bypass graft surgery or percutaneous transluminal coronary angioplasty.
Dosing regimen
Aspirin should be taken once a day, preferably after a meal to prevent heartburn. Aspirin comes in regular form, buffered to reduce acidity (such as Alka-Selzer) or enteric-coated (such as Ascriptin).

Side-effects
Although long-term use of a low dose of aspirin is safe and well tolerated, rare but potentially serious side-effects can occur, and aspirin should therefore only be used under a doctor’s supervision. Aspirin may cause gastric and duodenal problems with or without ulcer. These side-effects are less frequent with buffered or enteric-coated aspirin.

Contraindications
Active peptic ulcer disease and allergic reactions.

4.4.7 Lipid-lowering drugs
The main aim of treatment with lipid-lowering drugs is to reduce the LDL cholesterol level. Drugs available for lowering cholesterol include statins, bile acid sequestrants (resins), nicotinic acid, fibric acids (fibrates) and omega-3 fatty acids. Although they differ widely in their chemical structure and biomolecular mechanism of action, their way of action is based either on reducing cholesterol synthesis or interfering with cholesterol circulation.

The statins are very effective in lowering LDL cholesterol and, to a lesser extent, triglycerides. Bile acid sequestrants lower LDL cholesterol and can be used alone or in combination with statins. Fibric acid derivates are mainly used to treat high triglyceride and low HDL cholesterol. Nicotinic acid lowers LDL cholesterol and triglycerides and raises HDL cholesterol.

Even if these drugs are prescribed, treatment with lifestyle measures (a healthy diet, weight reduction and adequate physical activity) must continue. This will keep the drug dose as low as possible and lower the overall risk of coronary heart disease.

4.4.7.1 Statins

Names

<table>
<thead>
<tr>
<th>Generic names</th>
<th>Examples of proprietary names</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atorvastatin</td>
<td>Lipitor, Torvast, Totalip, Xarator</td>
</tr>
<tr>
<td>Fluvastatin</td>
<td>Lescol, Lipaxan, Primesin</td>
</tr>
<tr>
<td>Lovastatin</td>
<td>Altocor, Mevacor</td>
</tr>
<tr>
<td>Pravastatin</td>
<td>Aplactin, Prasterol, Pravaselect, Sanaprav, Selectin</td>
</tr>
<tr>
<td>Rosuvastatin</td>
<td>Cirantan, Crestor, Provisacor, Simestat</td>
</tr>
<tr>
<td>Simvastatin</td>
<td>Liponorm, Medipo, Sinvacor, Sivastin, Zocor</td>
</tr>
</tbody>
</table>

The major aim of statins is to lower the LDL cholesterol level. They reduce LDL cholesterol more than other drugs. Statins also reduce triglycerides and produce a modest increase in HDL cholesterol. Clinical trials with statins have shown good long-term results, with significant reductions in morbidity and mortality among treated patients.

Pharmacodynamics
Statins reduce the synthesis of cholesterol by the cells thus forcing them to use more circulating LDL cholesterol. This results in reducing the LDL cholesterol level in the blood.
**Biomolecular mechanism**
To satisfy their needs for cholesterol the cells either synthesize cholesterol or take it from LDL which transports cholesterol assembled by the liver. Statins inhibit the hydroxymethylglutaryl-coenzyme A (HMG-CoA) reductase, an enzyme that controls the rate of cholesterol synthesis. The production of cholesterol – primarily in the liver – slows down, resulting in the reduction of the release of LDL from the liver into the circulation and reduction of cholesterol in cells.

At the same time, as less cholesterol is synthesized, more LDL is cleared from blood to be used by cells and especially by the liver for cholesterol synthesis. This results in a lower LDL cholesterol concentration in the blood.

**Desired effects**
Statins reduce total cholesterol and LDL cholesterol by 20–40%. They also lower the triglyceride level (by 10–30%) and increase the level of HDL cholesterol (by 5–10%).

**Therapeutic uses**
The therapeutic use is to slow down the progression of atherosclerosis, attempting to prevent the formation of new plaques and to reduce the risk of new clinical events through the stabilization of existing plaques by reducing their lipid content and ensuring a thick fibrous covering of the plaque.

Statins are given to patients with high cholesterol levels if lifestyle changes do not adequately reduce blood cholesterol levels.

Statins can reduce the risk of dying from coronary heart disease by 25% if taken for at least five years. Many clinical studies have shown that the drugs of the most recent generation are safe and effective in the great majority of cases, with benefits extending to elderly patients.

The effectiveness of treatment can be evaluated two or three months after the beginning of treatment. Subsequently blood lipid levels should be monitored every 6–12 months. This will also help determine the lowest effective dose.

**Dosing regimen**
Statins are more effective if intake of saturated fatty acids and cholesterol is low. Hence, statins should be used in conjunction with a diet low in saturated fat and cholesterol. The drug should be taken in the evening, as the body synthesizes more cholesterol at night.

**Side-effects**
Statins are well tolerated and serious side-effects are infrequent. More common complications include: liver damage, disturbances of the digestive system and myopathy. Muscular damage might be aggravated if fibric acid derivatives or niacin are used as well. Muscular damage is measured by the rise of creatine kinase and liver damage is measured by the rise of transaminase. Creatine kinase and transaminase should be measured on a regular basis starting 4–6 weeks after the beginning of treatment.

Elevated creatine kinase indicates that such myopathy symptoms as muscle aches, weakness or nonspecific joint pains are induced by statins. In this case, treatment should be discontinued to prevent myocytolysis and kidney damage.

**Contraindications**
Patients with liver disease and pregnant or breastfeeding women should not use statins.
4.4.7.2 *Bile acid sequestrants (resins)*

**Names**

<table>
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<tr>
<th>Generic names</th>
<th>Examples of proprietary names</th>
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<tbody>
<tr>
<td>Cholestipol</td>
<td>Colestid</td>
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<tr>
<td>Cholestiramine</td>
<td>Questran</td>
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Bile acid sequestrants are used to lower the LDL cholesterol level.

**Pharmacodynamics**

Sequestrants prevent reabsorption of cholesterol containing bile acids from the intestine. The loss of bile acids stimulates the liver to produce more bile salts taking additional amount of circulating LDL cholesterol thus reducing its level in the blood.

**Biomolecular mechanism**

The liver synthesizes the bile salts using cholesterol. Bile salts enter the intestinal lumen together with the bile where they emulsify dietary fat transforming it from insoluble fat into water soluble. More than 90% of the bile salts are reabsorbed. They return to the liver, ready to re-enter the intestine along with the bile.

The intestinal lining cannot absorb the sequestrants. Therefore, linking up with the bile salts (through anion exchange) sequestrants prevent their reabsorption and cause bile acids to be expelled with the faeces. Along with the loss of the cholesterol contained in the bile salts, the liver takes an additional amount of LDL cholesterol out of the blood to compensate the loss from bile salts. This lowers the LDL cholesterol level in the blood.

**Desired effects**

Bile acid sequestrants decrease the level of LDL cholesterol by 15–30% and slightly increase HDL cholesterol.

**Therapeutic uses**

These drugs are used to lower LDL cholesterol. They are used as adjunctive therapy when treatment with statins is not sufficient. Statins and bile sequestrants combined can lower LDL cholesterol by more than 40%.

**Dosage regimen**

Sequestrants are inconvenient to take. Bile acid sequestrant powders must be mixed with water or other fluid such as juice and taken once or twice (rarely three times) daily with meals. Tablets must be taken with large amounts of fluids to avoid irritating the digestive system.

**Side-effects**

Since sequestrants are not absorbed, there is no risk of systemic toxicity. They reduce the reabsorption of fat-soluble vitamins: A, D, K and E and can cause various symptoms of the digestive system: constipation, abdominal pain, bloating, flatulence, nausea, and steatorrhoea (fatty faeces). They also interfere with the absorption of many other drugs. Other medications should therefore be taken at least one hour before or four to six hours after a sequestrant is taken.

**Contraindications**

Phenylketonuria, biliary obstruction and hypersensitivity to the drug.
4.4.7.3 Fibrates (fibric acid derivates)

Names

<table>
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<th>Generic names</th>
<th>Examples of proprietary names</th>
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<tbody>
<tr>
<td>Bezafibrate</td>
<td>Bezalip, Fulcro, Lipoclar, Lipsin</td>
</tr>
<tr>
<td>Ciprofibrate</td>
<td>Fibrocl, Gemlip, Lipozid, Modalim</td>
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<tr>
<td>Clofibrate</td>
<td>Atromid-S, Novofibrate</td>
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<tr>
<td>Fenofibrate</td>
<td>Nolipax, Scleril, Tilene, Tricor</td>
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<tr>
<td>Gemfibrozil</td>
<td>Lopid</td>
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Fibrates are primarily effective in decreasing the triglyceride level. This group of drugs also reduces LDL cholesterol but much less than statins. Fibrates raise HDL cholesterol.

Pharmacodynamics
The action mechanism of this group of drugs is very complex and might vary from drug to drug. Fibrates reduce the formation of triglycerides in the liver and also enhance triglycerides catabolism (the process of breaking them down to release energy) in the peripheral tissues.

Biomolecular mechanism
Fibrates work on a receptor related to the regulation of lipid metabolism. They increase the enzyme lipoproteinlipase, involved in the metabolism of triglycerides; they also influence the reduction of plasma fatty acids and the composition of lipoproteins. Fibrates improve coagulation and fibrinolysis.

Desired effects
The desired effect is reducing the level of triglycerides and LDL cholesterol and increasing HDL cholesterol.

Therapeutic uses
Fibrates are used mainly to lower the high level of triglycerides in patients with combined hyperlipidaemia. They also may be used to increase HDL cholesterol.

Dosing regimen
Fibrates are usually given twice a day 30 minutes before the morning and evening meals.

Side-effects
Most patients tolerate fibrates well. Side-effects include various symptoms of the digestive system, rashes and headache. Since fibrates increase cholesterol excretion in bile, use of fibrates increases the likelihood of developing cholesterol gallstones. These drugs can significantly intensify the effect of blood-thinning drugs (anticoagulants). If they are used in combination with statins, the risk of muscle damage might be increased. Since the kidneys eliminate fibrates, patients with renal impairment also risk muscle damage.

Contraindications
Reduced liver or kidney function and gallstones.
4.4.7.4 Nicotinic acid (niacin)

Names

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<th>Generic names</th>
<th>Examples of proprietary names</th>
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<tr>
<td>Nicotinic acid (niacin)</td>
<td>Niacor (immediate release form), Niaspan (sustained-release form)</td>
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Nicotinic acid lowers LDL cholesterol and triglycerides and significantly raises HDL cholesterol. It improves the ratio between LDL and HDL cholesterol.

Pharmacodynamics
Nicotinic acid (niacin) is vitamin B3. It acts as a coenzyme in many metabolic processes. An average diet contains 15–30 mg of niacin per day. High doses (1–3 grams a day) are needed to affect blood lipids.

Biomolecular mechanism
Nicotinic acid works by inhibiting the synthesis of lipoproteins and by decreasing the production of VLDL by the liver. It also decreases the release of free fatty acids from fat cells and thereby reduces the production of triglycerides by the liver.

Desired effects
High doses of nicotinic acid can significantly increase the HDL cholesterol level and reduce LDL cholesterol and triglyceride levels. However, long-term use is limited because of its adverse effects, especially vasodilation. Tolerating flushing and palpitation is difficult for patients.

Therapeutic uses
Nicotinic acid is most suited for patients who have only low HDL cholesterol in their lipid profile. Nicotinic acid is not as effective as statins in lowering the LDL cholesterol level. When a low HDL cholesterol level accompanies a high LDL cholesterol level, most doctors use a statin. If necessary, nicotinic acid can be added to a statin to further raise HDL cholesterol.

Dosage regimen
Oral nicotinic acid is rapidly absorbed (in 30–60 min) and rapidly eliminated. Nicotinic acid is available as regular tablets (immediate-release tablets) or slow and sustained-release capsules. The latter release nicotinic acid into the circulation gradually. The sustained-release capsules therefore cause a lower incidence of upset stomach and skin flushing than regular tablets.

It is recommended to start treatment with nicotinic acid with a low daily dose and gradually increase to an average daily dose of 1–3 grams. The immediate-release tablets are taken two or three times a day, the sustained-release tablets are taken at bedtime.

Side-effects
The most common side-effect of nicotinic acid is skin flushing and skin itching caused by dilation of vessels, likely to occur about half an hour after taking the drug. The extended-release drug formulations may cause less flushing. To decrease flushing, patients are advised to take aspirin or ibuprofen 30 minutes before taking nicotinic acid. Most patients develop tolerance after some time. Another side-effect is upset stomach, which can be partially alleviated by taking nicotinic acid with meals. Patients should avoid consuming alcohol or hot drinks around the time the tablet is taken.
Other side-effects include liver damage (caused more often by the sustained-release capsules), worsening glaucoma, increased blood sugar level in people with diabetes and increased level of uric acid, which may lead to gout.

**Contraindications**
Chronic liver disease, gout, diabetes (high doses of the drug) and peptic ulcer disease.

**4.4.7.5 Omega-3 polyunsaturated fatty acids**

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<tr>
<th>Names</th>
<th>Examples of proprietary names</th>
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<tr>
<td>Docosahexaenoic acid</td>
<td>Seacor</td>
</tr>
<tr>
<td>Eicosapentaenoic acid</td>
<td>Esapent, Eskim-3, MaxEPA</td>
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Eicosapentaenoic acid and docosahexaenoic acid in high doses reduce triglycerides and therefore are alternatives to fibrates or nicotinic acid. Recent clinical trials indicate that the intake of eicosapentaenoic acid docosahexaenoic acid reduces the risk of major clinical events among people with established coronary heart disease.

**Pharmacodynamics**
These fatty acids have a powerful effect on lowering the level of triglycerides; however, they might raise LDL cholesterol. Evidence shows that consumption of oil-rich fish that is rich in eicosapentaenoic and docosahexaenoic acids twice weekly reduces the risk of platelet aggregation and formation of blood clots and thus lowers the risk of thrombosis, stroke and myocardial infarction. These fatty acids influence endothelial function, the conduction system of the heart and have anti-inflammatory properties. Thus, regular intake of oily fish reduces the risk of cardiovascular disease in many different ways.

**Biomolecular mechanism**
High consumption of omega-3 polyunsaturated fatty acids reduces the production of VLDL by the liver and increases the amount of cholesterol carried by HDL.

**Desired effects**
The desired effects include:

- reducing the triglyceride level;
- very broad biological effects that cover various aspects of cardiovascular functioning (blood pressure, heart functioning and thrombogenesis);
- reducing the risk of sudden death by preventing cardiac arrhythmia and stabilizing atherosclerotic plaques.

**Therapeutic uses**
Omega-3 polyunsaturated fatty acids can be a useful adjunct in the treatment of hypertriglyceridaemia. Since they might raise LDL cholesterol both LDL cholesterol and triglyceride levels should therefore be monitored.
**Dosage regimen**
The dietary sources of eicosapentaenoic acid and docosahexaenoic acid include oily fish (salmon, herring, anchovies and others) walnuts and leafy green vegetables. It is recommended to eat fish at least twice a week. However, the level of mercury in fish might be a concern.

Another option is to take them encapsulated. Lowering the triglyceride level requires 3–12 grams a day of eicosapentaenoic acid and docosahexaenoic acid. This is a large number of capsules, which is inconvenient.

**Side-effects**
Breath and burps that smell of fish are a relatively common but harmless “adverse effect” of high intake of omega-3 fatty acid supplements (more than 3 grams a day). Sometimes high intake of these fatty acids can cause excessive bleeding. Caution should therefore be taken if a patient is taking blood-thinning drugs or has a blood coagulation disorder.
5. Fifth session. Communicating health in the therapeutic education of the chronic patient

Education provides an important opportunity for effective health initiatives. Faced with an ever-increasing demand for treatment, especially for chronic conditions, initiatives that can direct and educate chronic patients acquire significant therapeutic relevance. The World Health Organization defines therapeutic patient education as follows:

Therapeutic patient education should enable patients to acquire and maintain abilities that allow them to optimally manage their lives with their diseases. It is therefore a continuous process, integrated in health care. It is patient-centred; it includes organized awareness, information, self-care learning and psychosocial support regarding the disease, prescribed treatment, care, hospital and other health care settings, organizational information and behaviour related to health and illness. It is designed to help patients and their families understand the disease and treatment, cooperate with health care providers, live healthily and maintain or improve their quality of life.

Therapeutic patient education combines treatment with activities that were traditionally regarded as belonging to therapy. A central element in this definition is the complex and continuing process by which the patient’s assumption of joint responsibility for treatment, with the aim of improving his or her quality of life, becomes an indispensable aim. The contribution of psychology to therapeutic patient education, alluded to in the name itself, is relevant, both in the rehabilitative clinical sense and for all the aspects inherent to health education in general, as well as for the communicative strategies that can be used in this context.

Further, in recent years psychology has paid particular attention to questions of health, health care and prevention. This session examines the contribution of psychology to the health sciences, paying particular attention to the forms of communication that may be used in clinically directed educational strategies.

5.1 Social categories for health education

Psychologists have not limited their health activities simply to therapeutic intervention. Since the 1970s, the sector of health psychology has grown in importance; its practitioners promote the culture of health and the control of chronic diseases and facilitate access to health care. Health psychology may be described as:

the sum of specific contributions (scientific, professional and training) made by psychology to the promotion and maintenance of health, the prevention and treatment of disease and the identification of the etiological and diagnostic indicators of health, of disease and of the associated dysfunctions as well as the analysis and improvement of treatment systems.

Therapeutic education is an important part of health psychology, using concepts such as risk and danger, prevention and health promotion. The emergence of this area of study signals the decline of the traditional reductionist and dualist biomedical model and the move towards a biopsychosocial model with a systemic perspective. Attention is paid to the concept of health rather than that of illness, moving the axis around which clinical intervention revolves from illness to wellness. The term prevention, for example, emphasizes illness; literally prevention (from prae – venire meaning come first, anticipate) implies doing something so that an event regarded as negative or damaging will not take place. The recognizable meaning of the term promotion (from pro – movere, or move in favour) suggests the idea of a movement towards
self-realization, of completely new exploration that benefits the individual. The protagonist is the individual who acts to improve his or her own health.

5.2 The relationship between physician and patient

On a practical level, chronic illness raises some questions on what such a state can mean for patients and their families. In particular, all diseases with a nonfatal outcome but with a course accompanying the patient’s life from onset until death involve the establishment of conditions that will allow the patient to live with the illness and with treatments that last a long time. There may be important consequences in the way a person leads his or her daily life, such as the continual need to take medicine or periodic health checks. Such factors lead to cognitive restructuring of the patient’s perception of themselves, a self that may have undergone considerable physical changes and the organization of a new identity by recognizing the state of being ill.

**Education as part of treatment**

The education of the patient is an instrument of therapy that is equal to and ranks alongside other types of therapy and is an important tool in health care, especially in relation to the treatment of chronic diseases. This requires profound sensitivity towards aspects connected with interpersonal relationships, the needs of individuals, their expectations and the real possibilities of recovery and rehabilitation.

Looking at education as part of treatment permits a critical review of the usual physician-patient relationship, placing it in a psychological dimension that involves focusing on personal and emotional aspects and acknowledging the centrality of the person rather than the disease. It therefore presupposes an attitude in which, in an asymmetrical relationship between physician and patient, responsibility for treatment is transferred to the patient, who thus becomes an active participant in the therapeutic relationship.

Every conversation is characterized by asymmetrical relationships, not only because of the dynamics between the participants, role, status and the context but because each participant possesses different amounts of information on the subject being discussed. This asymmetry must therefore be exploited to create common spaces in which different cognitive systems (one technical and the other based on common sense) can meet and contribute to the joint construction of an understanding in which the aspects linked to the helping relationship carry the most weight.

It is also well known that communicative exchange is the element that contributes most to patient satisfaction, making the patient feel listened to and understood. Any interaction between patient and physician comes about as a result of a request for help. Whether these requests is made explicitly in relation to a particular illness or less explicitly, remember that the sick person is not simply a set of symptoms but rather expresses a lack of well-being that derives from “not being well”. The person who is listening and offering treatment should especially pay attention to this lack of well-being. Although symptoms can be very useful in making a medical diagnosis and offering treatment, the lack of well-being expresses a deeper “lack of ease” that affects the whole person and involves their feelings, sometimes in opposition to their rational understanding.

Appropriate use of language, both the choice of vocabulary and information content, becomes especially important. Using the language of the patient enables contact to be established between
the expert cognitive system of medical science and the representational systems of the patient to move towards the patient’s therapeutic education.

5.3 Preventing risk while promoting and communicating health

The central role communication plays in therapeutic patient education suggests some reflection on the significance of a specific area of communication: risk. The term risk is often used interchangeably with the term danger, but objectively they do not refer to the same thing. Danger can be defined as a threat to people and their possessions, while risk is a quantitative measure of harm, most frequently expressed as a probability of being harmed (3).

The communication of risk is described both as a simple exchange of scientific information between the interested parties and as a support for decisions and actions to be taken, including attempting to improve people’s ability to take appropriate decisions in the light of risks or opportunities.

Although the communication of risk covers various fields of application, in practical terms it is especially characterized by informative and educational aspects and by the dissemination of protective behaviour. In health care, health needs to be communicated actively as an element protecting against the risk of illness, and the communication itself should be regarded as an active process of jointly constructing knowledge and information and also attitudes, habits and behaviour that can contribute to the quest for and maintenance of health.

The communication of health requires a conscious, voluntary and deliberate effort aimed at promoting health. The communication of health may be thought of as a specific area of study, within which constructs can be identified, that may be useful in devising effective information strategies capable of achieving efficient educational activities. Every educational intervention regards communication as an essential element. Devising an adequate educational strategy requires adopting appropriate methods of communication capable of activating the cognitive resources of the people involved. Those who have studied the teaching and learning process describe this from a constructivist viewpoint, according to which reality is the result of subjective cognitive construction and in which cultural mediation between different systems of communication becomes crucial. It is therefore desirable, if not necessary, to examine how this mediation comes about.

5.4 When information is not enough: from persuasion to therapeutic education

Developing wide-reaching strategies capable of acting on large sections of the population and deeply rooted behaviour requires bringing together constructs from different sectors of application, such as those of information and education. It is well known that simple knowledge of risk factors is not always sufficient to encourage the adoption of protective behaviour. Most smokers, for example, know the risks to which they are exposing themselves; nevertheless, many continue to smoke. It is therefore useful to look at the informational acts aimed at educating that are capable of encouraging people, an active process that is education itself.

Information directed at educating requires methods, techniques and models that make the transmission of information effective and permit the communication to influence the people targeted. Although influence and persuasion are often seen as negative (4) or at least ambiguous
education is almost always influential and persuasive. Indeed education, especially when it is trying to influence deep-rooted behaviour such as habits, is education only if it is capable of influencing the cognitive system of the person at which it is aimed and therefore of convincing him or her. This does not imply that all means are acceptable provided that the goals are achieved, nor indeed that every initiative is valid provided that its aim is to protect health, but simply that all education must be influential and thus cannot be other than persuasive.

Bringing together elements that are useful in drawing up communicative and educational strategies capable of influencing people’s beliefs, behaviour and habits for the purposes of therapeutic education requires investigating the models that aim to describe the communicative processes themselves and how educational action is carried out. Communicating health and carrying out educational interventions requires referring to research into topics such as how attitudes are formed and changed, social influence and persuasion.

5.5 Persuading, influencing and changing attitudes

Tackling topics such as therapeutic education requires facing belief systems and attitudes that are often deeply rooted and difficult to change. To this end, it is useful to think about how such attitudes are formed and especially how they change or can be influenced. The concepts of persuasion, influence and change of attitude are often used interchangeably, in both everyday and scientific language, but they have very different meanings, even though they do have some points in common. Persuasion signifies an intentional act of communication, whereas the process of influence has a broader character, since it is not based solely on messages and arguments made up of words but also on behaviour and images perceived in non-intentional ways. Persuasion is only one of the many possible types of social influence, which constitutes a broader category of meaning that contains persuasion.

Further, a change in attitude is only one possible outcome of a process of social influence that may affect behaviour or feelings as well as ideas. In addition, attitudes change not only as a result of another person’s influence but also by reasoning, by behaviour and by the subject’s independent feelings. The study of attitudes focuses on the analysis of individual psychological processes, whereas the study of social influence pays particular attention to the social context and to the relations between the source and the target.

People are subject to persuasion both when they are paying attention and when they are not, but the way in which they are influenced in these two states is very different. Petty & Cacioppo (6) stated that persuasion can follow two different and parallel routes: central and peripheral.

The central route. This route is a process that requires considerable attention and involves careful reflection on the arguments and information relating to the message received. For this reason it uses considerable cognitive resources: paying attention, understanding, comparing, combining the new information with that already possessed and arriving at a new conclusion. The subject therefore participates consciously and knowingly and, as a result, forms opinions and attitudes. With the central route, the person receiving the message embarks on a careful and reasoned examination of the merits of the information that has been presented. With this route, the person can argue actively for and against the message, ask questions or seek new information.
The peripheral route. With the peripheral route, in contrast, the person receiving the message puts minimal attention and effort into processing the communication. On this route, people are persuaded through simple inductive elements, such as the pleasantness of the communicator or the opinions of others. Indeed, the presence of two routes (6) entails the idea borrowed from cognitive psychology according to which human beings are cognitive savers who, given their limited capacity to process information, try to save cognitive energy and frequently adopt strategies for simplifying complex problems.

The strong points of the model described above lie in the three ways in which the variables can act on the process of persuasion: persuasive arguments, peripheral elements and the factors that influence the degree or direction of the processing. The weaknesses lie in the parallel nature of the two routes, central and peripheral that, according to the model, are alternatives and cannot overlap.

A different model has been proposed (7) that recognizes the existence of two processes but largely reworks their content. There is the systematic processing of information, corresponding to the above described central route (6), which includes aspects such as understanding, reflection and the relationship with previous knowledge. The peripheral route is reformulated according to a process based on heuristics: the processes that can make information more easily available and are not related to the content of the message but rather are structured as simple rules for decision-making. That is, one relies on short cuts that, based on previous experience, allow one to take decisions quickly and without expending much cognitive energy. Many decisions are taken based on heuristics: for example, we often trust the authority of the source, accepting the truth of statements made by those we regard as experts and authorities; we accept as honest the opinions of people whom we trust; or we rely on the notion that the ideas and behaviour of the majority are more valid than those of a minority.

Heuristic rules can simplify decision-making, but using them requires the subject to have learned the rule in the course of past experience and observations (heuristics of availability), to recall the rule in the relevant situation (heuristics of accessibility) and to consider the rule reliable and functional (heuristics of reliability). The systematic and heuristic routes are not alternatives; on the contrary (7), they can interact and modify the outcome of the process of persuasion and information. Although therapeutic education should stress the need to pay attention to the systematic route, ignoring the heuristics that come into play in the processes of influence does not seem sensible.

Social psychologists have paid particular attention to the mechanisms that facilitate consent without pressure. In particular, some rules that have been identified for which there are corresponding heuristics are stated briefly here.

- The first principle is that of commitment (8), according to which people tend to maintain behaviour deriving from an earlier commitment. This probably results from the need to be consistent or perhaps from the desire to present a favourable image of oneself to others. According to this principle, a person who has taken up a particular position will thereafter tend to consent to requests for behaviour that are consistent with that position.

- The second principle is that of reciprocity, which relates to an implicit social rule: it is good to return favours, invitations and kindness. In this case, the heuristic principle might be expressed as follows: a person will tend to consent to a request to the degree to which their consent constitutes a return for someone else’s behaviour.
• The third principle is known as the social test, according to which a person will tend to consent to a request for behaviour to the degree to which other similar people have done it or are doing it, in which case we can speak of a heuristic of consent.

• The fourth principle is that of the authority from which the heuristic derives: people tend to follow the suggestions of an authority they regard as legitimate. People are often exposed to this kind of heuristic, especially when the benefits of a certain product, such as a toothbrush, are described by a man in a white coat or a professor of orthodontics.

• The fifth principle is that of scarcity, according to which the value of a product or service is determined by its availability. According to this principle, people try to take advantage of rare opportunities; this explains the way people rush to obtain drugs that are not yet available in their own country.

• The sixth principle is that of friendship or liking, according to which a person will tend to consent to requests made by friends or other people they like. One example in sales of household items is that of the Tupperware party or other personally sold product, but also the influence that a physician can have when he or she is also a friend.

Thus heuristics, techniques of influence and strategies of persuasion can become instruments of treatment in the hands of health care workers. The challenge is using these techniques consistently with therapeutic patient education, to move towards a situation in which the patient truly takes responsibility for his or her own health and well-being.
References


Bibliography


