# NURSING ASSISTANCE TO THE PATIENT WITH ACUTE MYOCARDIAL INFARCTION; NURSING IMPLICATIONS

Cimmino Olimpia<sup>1</sup>, Falconio Lucio Marcello<sup>2</sup>, Ruocco Vincenzo<sup>3</sup>, Sanselmo Salvatore<sup>3</sup>, Cacace Simona<sup>3</sup>, Sanselmo Mirko<sup>3</sup>, Pontillo Ermelinda<sup>3</sup>, Addeo Domenico<sup>4</sup>

<sup>1</sup>Degree in nursing sciences, coordination master's degree Integrated assistance master in hospital territory, regional contact person for SDO flow, Head of HUB VACCIBALI ASL NAPOLI 1 CENTRO, Monitoring NSG.

<sup>2</sup>Degree in Pharmacy ASL NAPOLI 2 NORD

<sup>3</sup> Degree in nursing Sciences HEAD OF ASL NAPOLI 1 CENTRO

<sup>4</sup> Degree in Pharmacy ASL NAPOLI 1 CENTRO

**KEYWORDS:** ECG (ElectroCardiogram) - AMI (Acute Myocardial Infarction) - WHO (World Health Organization) - ACS (Acute Coronary Syndrome) - WHO (World Health Organization)

# ABSTRACT

In this treatise we intend to deepen the theme of Acute Myocardial Infarction (AMI), one of its main risk factors at the cardiovascular level, namely smoking, and in particular, deepen the role of the nurse in patient education. after the ischemic event and implement strategies aimed at smoking cessation.

If we look at the data from the World Health Organization (WHO), it is striking that the main cause of mortality in the modern world is heart ischemia which alone causes 7 million and 400 thousand deaths; while in second place we find stroke and cerebral vasculopathies with 6 million and 700 thousand deaths (Cesta 2014).

Having established that tobacco plays a primary role in our society and that its active or passive use has a negative impact on the health of the individual (WHO 2014), we will first briefly mention the anatomy and physiology of the cardiovascular system, then we will discuss the implications between heart and smoking and, in particular, between heart attack and smoking, trying to understand - through authoritative sources such as the WHO and databases - where the roots of such a widespread habit lie and its impact on the system cardiovascular.

Later we will address the educational issue of secondary prevention, we will examine the strategies implemented for smoking cessation. By dealing directly with patients, acquaintances and friends we realized how, despite the widespread information on the dangers of smoking, several people, even knowing the possible consequences, not only do not try to quit smoking, but almost underestimate the damage that can be caused by this "dangerous" habit of theirs is paradoxical. We believe that the role of the nurse in the post-heart attack moment is of extreme importance not only to provide specific direct assistance, also because that relationship of trust is created and to make him understand the risks to the patient in case he decides to continue smoking, but also because a correct education and the use of some strategies, personalized to each patient, to quit smoking, are essential in reducing the risk of a relapse. The choice of the topic dealt with in this work was dictated by a strong SOCIAL motivation.

Although I am a smoker, noting the importance and the close relationship between health and a habit such as smoking, I have always been interested in being able to deepen my knowledge on the effects of the cardiovascular system and the problems that orbit around it. We still believe that an in-depth study on an issue so debated today and which will still be discussed for a long time can give the opportunity to approach smoking patients in a more conscious, personalized and adequate way and thus making treatment a better means. comprehensive within the health sector.

We believe that this work can enhance our personal and professional background, allowing us in the near future to prevent, identify and deal more effectively with clinical problems involving both doctors and nurses. Finally, the hope is to acquire greater critical capacity in problematic cardiological situations and to have a greater capacity for acceptance and respect for the patient's will.

This work aims to highlight and deepen the cardiovascular problems secondary to the phenomenon of smoking, implementing a therapy for smoking cessation.

The methodology is based on articles researched from databases, on journals specialized in cardiology and on textbooks.

From the observed articles it emerged that the nurse must use a multisystemic, multifaceted and multidisciplinary approach that includes different roles, namely: member of a working group, role of health promoter, role of teacher and communicator, of educator and expert in nursing care, and taking a look at post-ischemic nursing care.

In this treatise we intend to deepen the issue of Acute Myocardial Infarction (AMI), one of its main cardiovascular risk factors, namely smoking, and in particular, deepen the role of the nurse in patient education after the ischemic event and in implementing strategies aimed at smoking cessation. If we look at the data from the World Health Organization (WHO), it is striking that the main cause of mortality in the modern world is heart ischemia which alone causes 7 million and 400 thousand deaths; while in second place we find stroke and cerebral vasculopathies with 6 million and 700 thousand deaths (Cesta 2014).

Having established that tobacco plays a primary role in our society and that its active or passive use has a negative impact on the health of the individual (WHO 2014), we will first briefly mention the anatomy and physiology of the cardiovascular system, then we will discuss the implications between heart and smoking and, in particular, between heart attack and smoking, trying to understand - through authoritative sources such as the WHO and databases

- where the roots of a widespread habit and its impact on the cardiovascular system lie. Later I will address the educational issue of secondary prevention, I will examine the strategies implemented for smoking cessation.

By dealing directly with patients, acquaintances and friends we realized how, despite the information seems to be more than abundant on the dangers of smoking, several people, even knowing the possible consequences, not only do not try to quit smoking, but underestimate in the damage that can be caused by this "dangerous" habit of theirs is almost paradoxical.

We believe that the role of the nurse in the post-heart attack moment is of extreme importance not only to make the patient aware of the risks in case he decides to continue smoking, but also because proper education and the use of some strategies, customized to each patient, to quit smoking, are essential in reducing the risk of a relapse.

# CARDIOCIRCULATORY SYSTEM

#### Anatomy of the cardiovascular system

It consists of a series of tubular-shaped hollow structures (the vessels), within which a defined quantity of fluid flows which is pushed by the heart.

There is a blood circulatory system and a lymphatic circulatory system:

- 1) the first is characterized by the heart and the presence of a series of vessels within which blood flows, that is, arteries, capillaries and veins;
- the second is characterized by a series of lymphatic vessels and lymphoid organs that transport the lymph, ie a certain quantity of liquid that cannot be recovered once the exchange of substances from the bloodstream has taken place;
- 3) through the lymphatic vessels this fluid enters the lymphoid organs (spleen, thymus and lymph nodes) and then returns to the bloodstream.
- 4) Vessels originate from the heart, which lead towards the periphery, which take the name of arteries, while the vessels that return from the periphery to the heart are called veins. Arteries peripherally divide into multiple vessels and reduce in size to facilitate blood diffusion.

The branched veins become fewer and larger until they reach the heart to carry all the waste substances. The heart is contained within the thoracic cavity, which starts from the spinous process of the seventh cervical vertebra and reaches the lower edge of the costal arch.

What divides the thoracic cavity from the abdominal area is the diaphragm; the heart occupies the central region of the thoracic cavity and is located in the lower and anterior part.

Specifically within the thoracic cavity there are three spaces: two very large spaces that are occupied by the lungs and pleurae (pleuropulmonary spaces), and a central median space called the mediastinum, where there are a series of splanchnic organs including the heart, which occupies the front and bottom.

The heart is in direct contact with the posterior wall of the rib cage, rests at the level of the diaphragm muscle and is surrounded by a white structure.

The heart has a frontal and two-dimensional triangular shape, with a pointed part that looks down towards the left, a flatter part facing upwards and to the right. The whitish structure surrounding the heart is the pericardium, a kind of sac that has an externally very



strong fibrous wall (fibrous pericardium) and an internal serous (serous pericardium). The outermost part through ligament structures is connected to muscular (inferiorly) or bony (anteriorly) components anchoring the heart in one position and preventing it from moving during the movements of the body.

The serous component consists of a visceral sheet, which adheres intimately to the heart, called the epicardium, and a parietal sheet, which lines the inner wall of the fibrous pericardium. The serous pericardium is constantly lubricated allowing the sliding of the heart inside the pericardium and therefore an adequate contractile activity.

#### Anatomy of the heart

Two faces can be recognized in the heart: an anterior face, called sternocostal, due to the close relationships it contracts with the sternum and the median part of the ribs, and a postero-inferior or diaphragmatic face, which rests on the diaphragm muscle.

The three-dimensional heart has a shape similar to a slightly flattened cone at the front; we recognize two margins, a left margin of obtuse shape and a right margin of acute shape, a base (above) and an apex (below), between which an axis is drawn, called the anatomical axis of the heart, to study it.

Both the anterior and posterior faces have sulci: there is a transversal groove, called the coronary sulcus, since it surrounds the heart as a sort of crown, both on the anterior and posterior faces;

two other sulci that run perpendicular to the coronary sulcus and that lead towards the coronary sulcus both on the anterior and posterior faces towards the apex, i.e. the interventricular sulci (because they are near the ventricles).

The septum that internally divides the two ventricles is instead called anterior interventricular sulcus and posterior interventricular sulcus (based on the face where it is located).

At the level of the base of the heart, on the anterior face there is the presence of large vessels which correspond, from right to left, to the superior vena cava, to the origin of the aorta artery and to the origin of the pulmonary artery.

On the posterior side we recognize the same structures plus others, from the right: the outlet of the superior vena cava, the aorta, the pulmonary artery, which divides into two branches to reach the lungs, then the outlet of the inferior vena cava and the presence of pulmonary veins that carry blood from the lung to the left atrium.

As regards the anterior and posterior aspect of the ventricular region, there is the presence of vascular structures that run inside the adipose tissue along the interventricular sulcus and along the coronary sulcus. These structures are the arterial branches and the venous branches that lead to supply the heart; then there are two coronary arteries, right coronary artery and left coronary artery, they supply the heart muscle and originate from the aorta artery: the right coronary artery enters the coronary sulcus, moves to the right, reaches the right (acute) margin, ad reaches the meeting point of the posterior interventricular sulcus, where it bends into a U and forms the posterior interventricular branch; during this path the right coronary artery gives branches for the right atrial region and for the right ventricular region, from an important marginal branch on the acute margin and continues to give branches for the right latero-posterior part.

The left coronary artery also originates from the aorta, and runs behind the aortic trunk; it then exits laterally to the pulmonary arterial trunk, where it divides into two branches: one that runs along the anterior interventricular sulcus, and a circumflex branch that arises inside the coronary sulcus, reaching the point where the coronary sulcus meets the interventricular sulcus rear.

These arteries are terminal arteries: they do not exchange blood between them, this means that the collaterals that will supply the individual portions of the heart muscle will be the only ones to bring nourishment to that area of the heart muscle.

Internally the heart has atrial and ventricular cavities; they are divided right atrium, left atrium, right ventricle, left ventricle, and 8 have a particular relationship between them:

the right atrium communicates with the right ventricle through the right atrio-ventricular ostium, the left atrium communicates with the left ventricle with the left atrio-ventricular ostium;

the two ventricles are separated by septa, which prevent the passage of blood from the right half to the left half.

The heart has muscular walls of different diameters: in the right ventricle the muscular wall is thin, in the left ventricle it is thicker, as is the septum that divides the ventricles; the reason for the difference in muscle diameter is due to the two different circulations (large and small), i.e. from the left half (the thicker one) the large circulation (or systemic circulation) will start, so the blood will be pushed away from the heart and from the right half (thin) will start the small circulation (or pulmonary circulation), in which the blood is pushed to the area near the lungs. The atria have a smooth wall, with the exception of the two offshoots leading towards the front which are called auricles; they have small muscle regions that tend to propagate inwards, forming the combed muscles.

The musculature of the ventricles gives rise to reliefs with a very different morphology from that of the atria; the reliefs give life to the flesh trabeculae which can be classified into three categories: type I flesh trabeculae, or papillary trabeculae, which are represented by the introflexions inside the ventricular cavity of the musculature, which has a base adhering to the wall and an apex that leads inside the ventricular cavity;

type II carneal trabecula, which forms a sort of bridge, with two points anchored to the wall itself; flesh trabeculae of type III, some simple reliefs.

The papillary muscles are the most interesting, since from the apex of these muscles some tendon-like structures in the shape of a cord, called tendon cords, come off and reach the two atrioventricular valves.

The atrioventricular valves are the valve systems that modulate the passage from the atrial cavity to the ventricular cavity.

These have a different characteristic between the right and left halves: in the right half there is the presence of three triangular cusps (therefore it is called tricuspid), in the left half there is the presence of two cusps (called mitral or bicuspid).

These regulate the passage of blood and open when

blood is pushed from the atrial to the ventricular cavity; once the blood has arrived in the ventricle, the contraction of the ventricle begins and these valves close without being able to tip over so that the blood does not come back.

57

This is due to the presence of tendon cords that pull the cusps downwards. Blood from the ventricular cavity is then pushed into the large arteries originating from the right and left ventricles.

There are other valves also between the ventricles and the large arteries: these valves, called semilunar, when the blood is pushed inside the vessel, squeeze and allow the outflow of blood inside the vessel.

Once the ventricular contraction is over, the blood would tend to go back, but the blood meets the lower part of the semilunar valves which is pocket-shaped, making the cusps of the semilunar valves come into contact with each other, blocking the reflux of blood to the interior of the ventricular cavity.

The valve-type structures are anchored to an intermediate zone between the atrium and the ventricle, where the atrioventricular ostia and the arterial-type ostia are anchored; this structure, of a connective type, also constitutes a sort of central skeleton to which the heart muscles can adhere and therefore allow a correct distribution of the fibers which, thanks to their arrangement, will ensure adequate contraction for the function that the heart performs. blood vessels are divided into arteries (blood to the periphery), veins (blood to the heart) and capillaries (nutritional exchanges).

The vessels are structured in internal or intimate cassock, medium cassock, and external or adventitious cassock.

Thanks to the diameter, the arteries are divided into: large caliber arteries, with a diameter that reaches 6 mm, medium caliber, when the diameter reaches 0.1 mm, and small caliber, when the diameter reaches about 20 microns.

Large caliber arteries are elastic arteries, because they contain a considerable amount of elastic tissue, while medium and small caliber arteries have non-elastic tissue; this means that the great artery is capable of, through an expansion generated by the strong pressure it undergoes, to receive the blood which is pushed into the first part; this elasticity of the great arteries is also important because when the ventricular thrust ends, thanks to the closure of the valves, it allows the blood to be pushed.

In the arteries of medium and small caliber it is instead necessary to control through the smooth muscle, located at the level of the middle layer of these arteries according to the needs of the organism. Veins can also be classified into small, medium and large caliber veins. The only structural difference that is found with respect to the arteries is that the veins of the lower part of the body have semilunar shaped valve systems similar to those located at the origins of the large arteries. The main arteries are the pulmonary artery and the aorta.

The aorta is the most important artery and can be divided into several portions: the first originates from the left ventricle and moves upwards and for this direction it is called the ascending artery, after which it undergoes a posterior flexion going to form an arch with a lower cavity, which runs above the left bronchus, and moves downwards, and this portion is called the thoracic descending artery, because contained within the thoracic cavity, it will pass the diaphragm muscle descending into the abdominal cavity, going to constitute the abdominal descending tract.

From the ascending aorta the originating vessels are the coronaries; in the curved section there are a series of collaterals that go to supply the head, neck and upper limbs; other collaterals depart from the descending thoracic aorta divided into parietal collaterals, which supply the chest wall, and visceral vessels, which supply the organs inside the thoracic cavity; also from the descending abdominal tract there are parietal branches and visceral branches, the latter divided into: uneven visceral branches that originate from the anterior region of the abdominal aorta, i.e. celiac tripod, immediately below the diaphragm, superior mesenteric artery, inferior mesenteric artery, from the celiac tripod there is blood supply to the liver, stomach and spleen, from the mesenteric the small intestine and colon; even visceral branches, i.e. renal arteries and genital arteries;

the abdominal aorta ends in a bifurcation with the two iliac arteries. The main arterial branches that supply the head originate from the arch of the aorta, which are three, are represented by a single trunk called the brachiocephalic trunk, which divides into two, i.e. on one side into a right subclavian artery (which leads to the right) and a right common carotid artery, followed by the left common carotid artery and the left subclavian artery.

The subclavian moves towards the upper limb, the common carotid towards the head; the latter divides into two large branches, an external carotid which vascularises the external surface of the skull and an internal carotid which vascularises the interior of the skull.

There is another artery that rises towards the skull, namely the vertebral artery, which detaches from the subclavian artery, running between the transverse processes of the vertebrae and moving inside the skull, in order to vascularize the nervous structures which are found within the neurocranium. The upper limb receives blood from the subclavian artery;

in the arm there are: the axillary artery which is positioned under the armpit, the brachial artery, which is in contact with the humerus, which divides into two collateral branches that lead towards the forearm following the radius and ulna , i.e. radial artery and ulnar artery, and then branch out to form a network inside the hand.

The lower limb receives blood from the abdominal aorta, which becomes the iliac artery, which moves towards the thigh first and then towards the leg, giving life to the femoral artery anterior to the knee and popliteal artery posterior to the knee, which is divides into the terminal branches which are anterior tibial artery and posterior tibial artery.

There are two venous systems: a superior venous system (or suprradiaphragmatic), which is received in a large venous vascular structure, that is the superior vena cava, which collects blood from the head, upper limbs and organs of the thoracic cavity; a lower venous system, which collects blood in the lower limbs, from the organs of the abdominal wall; the inferior venous system is received by the inferior vena cava. Unlike arteries, which run deeply, veins can be found both deep and on the surface below the connective tissue. The superior vena cava is a trunk that originates from the confluence of two large veins: the left brachiocephalic vein and the right brachiocephalic vein, which in turn originate from the jugular and subclavian veins (on all sides). Connected to the head is the internal jugular vein, which is very large, which collects the blood that reaches the brain and the external jugular vein, which is smaller, which collects venous blood from the external part of the head; the latter flows directly into the subclavian vein, before it joins the internal jugular vein.

In the upper limb there are several superficial veins, which are useful for drawing blood;

there are several superficial veins that start from the hand up to the root of the arm, and the two main veins of the upper limb are: the basilic vein, which reaches the elbow region and then goes deep, and the cephalic vein, which is bring externally over the bicep and then go deep to the level of the shoulder.

The inferior vena cava collects blood from the entire subdiaphragmatic region; it is formed at the confluence of the two iliac veins, which collect blood from the lower limb, then there is the internal iliac vein which collects blood from the lower part of the abdominal cavity, the renal veins and the genital veins.

The drainage of most of the splanchnic organs present inside the abdominal cavity occurs through another system, called the portal vein system: it passes through the liver, which acts as a filter, and then re-enters the superior vena cava, completing the drainage of the subdiaphragmatic area.

In the lower limb there is a system called the saphenous veins: there is a small saphenous vein that leads posteriorly to the leg to merge into the popliteal vein, which is located in the fold of the knee, and the great saphenous vein which runs medially to become deep in the inguinal region and then reaches the level of the femoral vein.

Saphenous veins are known for certain pathologies, given their morphology:

presenting valvular structures that help the blood to reach the heart by muscle contraction; the increase in volume of muscle tissue inside the vein causes the internal volume of the veins to decrease, the pressure rises, the semilunar valves close, the upper ones open and the blood passes;

the pathology intervenes when the veins lose the functionality of the semilunar valves, so the blood remains at the bottom causing a swelling evident superficially.

#### Physiology of the cardiovascular system

The heart is composed of a right and a left portion giving life to the systemic circulation (large circle) and to the pulmonary circulation (small circle); The functions of the two circles are complementary and they come together (being placed one after the other) at the level of the heart. The pulmonary circulation starts from the right ventricle through an arterial trunk called the common trunk of the pulmonary artery which is short and then divides into the two right and left pulmonary arteries that supply the lung.

The blood circulating in the pulmonary artery is poor in oxygen and goes to the lung to oxygenate itself: there is a network of arteries that branch out repeatedly and eventually there is a network of capillaries that envelop the pulmonary alveoli.





The wall of the alveoli of the capillaries is thin and oxygen can pass from inside the alveolar air, through the barriers, to the blood, where it is dissolved and reaches the red blood cell. The red blood cell is thus enriched with oxygen and releases carbon dioxide.

This network of capillaries pours into a network of veins, which then return to the heart. The oxygenated blood through these veins returns to the left atrium of the heart, passes into the left ventricle and here begins the great circulation. From the left ventricle the aorta artery arises, the major artery of the human body which distributes, through numerous branches, the oxygen-rich blood to all organs, including the lung (bronchial arteries).

A network of capillaries is then created at the level of the organs, which is drained (the blood is recaptured) by the veins: a subdiaphragmatic one, the inferior vena cava and a suprradiaphragmatic one, the superior vena cava. You then return to the right atrium again.

The pulmonary circulation then originates from the right ventricle. In both circulations the arteries have some characteristics.

In both circulations the arteries increase in number as they move away from the heart (through the emission of collaterals) while their caliber progressively decreases. When an artery terminates, instead of emitting collaterals, it bifurcates at an angle of about 60 ° (e.g. the iliac arteries for the aorta) into two terminal daughter arteries of equal caliber to each other (about 76% of the caliber of origin). While the collateral arteries are arteries emitted roughly at right angles (with caliber less than 50% of the origin).

During its path and the subdivision into the various arteries, the diameter of the daughter arteries, collateral emitted, is progressively reduced.

This means that the blood is distributed to all tissues. There is a decrease in the flow velocity from the aorta to its collaterals and terminals. The transverse section of the aorta has a given area (considering for example that the diameter of a 70 kg person is about 30-32 mm, it can be calculated about 7 cm2). Contractions of the heart muscle are generated by signals that originate within the muscle itself. myogenetic contractile activity.

The ability of the heart to generate signals that activate its contraction in a cyclic way is defined AU-THORITHMICITY, and is due to the action of small modified muscle cells, called autorhythmic cells, essential for the action, that is:

1. Pacemaker cells: trigger the action potential and establish the heart rhythm. They are concentrated in two specific areas of the myocardium: the sinoatrial node (located in the upper wall of the right atrium, near the outlet of the superior vena cava) and the atrioventricular node, located in the interatrial septum near the tricuspid valve. AS cells have a higher spontaneous depolarization. Action potentials of the SA node initiate depolarization of the AV node.

59

 Conduction fibers: conduct action potentials and propagate them in the heart with a highly coordinated sequence of conduction system cells.

Then the contraction of the heart will begin in the sinoatrial node (or peacemaker), and gives rise to the depolarization which will then be transported throughout the heart, through bundles of specific conducting muscle fibers called internodal; they, usually three in number, run along the wall of the atrium, and reach another agglomeration of cells, called nodal structure or atrioventricular node (or bundle of His) due to the position in which it is located, that is, at the base of the atrium right at the limit with the atrial septum;

from here the atrioventricular node allows the origin of another bundle of fibers, which first enters the fibrous structure of the interventricular septum, that is the bundle of His, when it meets the myocardial muscle mass that constitutes the septum interventricular divides into two branches, which run one on the right wall and one on the left wall of the septum, until reaching the tip of the heart; from here these bundles of fibers divide into fine structures that reach all the single cells of the myocardium giving life to the subendothelial network of the Purkinje fibers ;

depolarization follows the path just explained: it originates from the sinoatrial node, transfers the depolarization of cells to the atrial cavities allowing them to contract, reaches the atrioventricular node where it undergoes a short slowdown so that the fibers that make up the atrial wall have already contracted and from here the depolarization quickly reaches the tip of the heart, from where the myocardial cells are stimulated.

The pacemaker cells of the SA node receive afferents from neurons of the autonomic nervous system, both orthosympathetic and parasympathetic. With the influence of the garden, the heart accelerates its beats and the action potential reaches the diastolic depolarization threshold earlier.

Conversely, with the influence of para , the heart slows down and the potential is activated more slowly with a consequent decrease in heart rate.

#### **Orthosympathic Control Mechanism**

Noradrenaline binds to SA node 1 receptors and activates the AMP system as a second messenger. The intracellular increase of AMPc stimulates the opening of funny channels and T -type Ca channels, increase in spontaneous depolarization rate and decrease in repolarization.

The onset threshold for PA is reached faster by increasing the PA rate, heart rate, and cardiac output.

# **Control Mechanism By The Parasympathic**

The Ach binds to the muscarinic receptors placed on the cells of the SA node, causes the opening for the potassium channels and at the same time the closure of the T-type Ca channels and the funny channels . decrease of the spontaneous depolarization rate and a hyperpolarization of the membrane. Heart rate slows and cardiac output tends to decrease.

# THEORETICAL FRAMEWORK

#### Infarction Of The Acute Myocardium

The blood Acute myocardial infarction (AMI) is a condition in which necrosis of a portion of myocardial tissue occurs, following an ischemia, or the lack of oxygen supply (hypoxia and anoxia) to the district, caused by an occlusion arterial. The evolution takes place in a few hours and is divided into: occlusion of the vessel, ischemia, edema, haemorrhage, necrosis and cicatricial evolution.

The obstruction may be due to the formation of a thrombus or plaque within a coronary artery (ather-osclerosis).

It is a multifactorial disease that affects the arteries of medium and large caliber and leads to a gradual accumulation in the intestine of macrophages, smooth muscle cells, lipids and collagen; it is identified as chronic inflammation localized in the intimate vascular tunic and triggered by prolonged endothelial damage.

The classic atherosclerotic lesion is plaque or atheroma;

On a macroscopic level, atherosclerosis shows itself with 3 lesions: lipid stria, fibrous plaque and complicated plaque.

LIPID STRIEs are 1-2 mm elongated lesions, yellowish in color and sharp edges, which stand out against the white color of the intima; only flat and have a smooth and continuous surface.

Histologically, the lipid striae contain lipids and macrophages. They do not reduce the vessel lumen and do not compromise its structural integrity. In the presence of cardiovascular risk factors they can progress into more advanced lesions.

The FIBROUS PLATE (atheroma) is a circumscribed thickening, which protrudes into the lumevascular, up to 1.5 cm long; the fibrous capsule of the lining is made up of smooth muscle cells and dense connective tissue; underneath are macrophages, smooth muscle cells migrated from the media and a few T lymphocytes.

These muscle cells become capable of producing cell matrix proteins, including collagen. Deeper, a necrotic nucleus is observed containing lipids, cellular debris and cell -foam.

The latter, originating from macrophages, are filled with lipids. In the periphery of the plates there are small newly formed vessels. The uncomplicated plaque is lined on the luminary side with endothelial cells. Atheromatous plaques have constant distribution.

Plaque ulcerates when macrophages in the lesion release metalloproteases that weaken the fibrous capsule.

Intra -plaque hemorrhage, the result of the rupture of newly formed vessels, also causes plaque ulceration because the accumulation of blood causes an increase in volume. The rupture of the capsule causes the release into the circulation of solid fragments (emboli) which can stop in the smallest vessels and cause is-

#### chemia.

The contact between blood and the contents of the plaque evokes the haemostatic response with the formation of a thrombus which can rapidly occlude the vessel causing necrosis of the downstream tissue.

Deposition of calcium salts in plaques is often observed in a process similar to ossification. Plaque can weaken the arterial wall which, under blood pressure, dilates to form an aneurysm.

The occlusion can be complete or incomplete, of intermittent or persistent duration. When the thrombus completely occludes the lumen of the coronary vessel for a prolonged period of time, transmural cardiac ischemia occurs which corresponds to the clinical picture of acute myocardial infarction with elevated STEMI.

intracoronary thrombus does not

determines a complete and persistent occlusion, the clinical picture of unstable angina or myocardial infarction without elevated ST (NSTEMI) is realized ( Miceli 2005).

However, it takes a certain period of time for an area of the myocardium to experience a heart attack. Initially, ischemia develops; over time, the lack of oxygen causes a heart attack, ie cellular necrosis (Smeltzer 2010, p. 871).

Although biochemical and functional changes occur immediately at the onset of ischemia, severe loss of myocardial contractility occurs within 60 seconds, while other changes take longer; for example, the irreversible damage occurs after at least 20-40 minutes from the complete stop of the blood flow. (Fuster, Alexander and O'Rourke 2006, p. 1468).

Heart attack is part of that syndrome called "Acute Coronary Syndrome", SCA, which also includes unstable angina as both represent continuum of the same process. (Porter 2014). Anamnesis, laboratory diagnosis, signs and symptoms In this chapter I intend to deepen the anamnestic research to be carried out, which tests to look for, but not only, I will highlight the main signs and symptoms of a heart attack related to the smoker patient.

A typical acute myocardial infarction is diagnosed primarily by history, as the most important factor is whether you currently smoke (if you are an active smoker, former smoker or non-smoker), how much you smoke (when you started, if you stopped smoking) later, when you have resumed), how many cigarettes or packs you smoke daily, to know if you have previously had heart or respiratory problems and if you have recurrent cough, phlegm or bronchitis.

It is necessary to measure vital parameters (in particular blood pressure), check the level of LDL and HDL for the possible development of atherosclerotic processes caused by tobacco, carry out blood clotting tests - through platelet counts - and tests on coagulation factors (PTT and INR).

The diagnosis of AMI should be considered in men over thirty-five and in women over fifty who complain mainly of chest pain that must be differentiated from pain due to pneumonia, pulmonary embolism, pericarditis, rib fracture, pain in the chest muscles after trauma or after physical activity, acute aortic dissection, renal colic and various gastrointestinal pathologies.

Once the medical history has been taken, the most important diagnostic investigation in the patient with suspected AMI will be carried out, namely the ECG , which should be performed within 10 minutes of when the patient reports the painful symptom or when he arrives in the emergency room. The ECG can be used to diagnose myocardial ischaemia and heart attack, conduction rhythm disturbances, heart chamber enlargement, electrolyte imbalances, and drug toxicity. The standard 12-lead ECG uses electrodes placed on the patient's extremities and chest to evaluate the heart from 12 different points of view. The standard 12-lead ECG consists of three standard bipolar leads (called I, II, III), three unipolar leads (to VR, to VL, to VF) and six unipolar precordial leads (C1 to C6). The exact point of contact with the skin of the leads is not really that important; it is important that the electrodes adhere well to the skin. The chest leads are positioned at specific points to ensure a faithful registration. All leads, except the precordial ones, show the heart from the frontal plane. The precordial leads show the heart from the horizontal plane. Each lead covers a specific area of the myocardium and provides an electrocardiographic photograph of the electrochemical activity of the cell membranes. The ECG measures the differences between the electrical potential of the electrode for each lead and reports it in graphical form, creating the standard ECG complex, called PQRST. At a later stage, the value of CK-MB, myoglobin and troponin will be evaluated (Smeltzer 2010). The key symptom of an AMI is typically deep, retrosternal visceral pain, described as constricting or oppressive, often radiating to the back, jaw, or left arm. Pain can also be very mild and about 20% of acute heart attacks are silent or not recognized by the patient as a pathological event (Porter 2014). Other signs and symptoms that characterize a heart attack are chest problems and heart palpitations; tachypnea, wheezing and shallow breathing; pale, cold, sweaty and sticky skin, as well as anxiety, restlessness, and dizziness may indicate increased sympathetic stimulation or decreased cardiac contractility (Smeltzer 2010, p. 871). In severe episodes, the patient appears distressed and may feel a sense of imminent death; nausea and vomiting may occur. On clinical examination, the patient is usually restless and anxious, with pale, cold and sweaty skin; peripheral or central cyanosis may occur while the pulse may be filiform and the BP variable (Porter 2014). Generally in a smoker there is an increase in vascular resistance and arterial pressure, heart rate and, consequently, also in output, increasing cardiac output. This causes higher cardiac work and higher myocardial oxygen consumption. Since carbon monoxide is in circulation, it binds more easily to hemoglobin and this results in a lower supply of oxygen to the myocardium. Smoking can also induce coronary vasoconstriction and helps to further decrease the oxygen supply and this sets the stage for a possible heart attack. In the medium and long term, smoking also leads to the formation of atheromatous plaques through alterations of the endothelium and coagulation, causing narrowing of the lumen of blood vessels which, once obstructed, lead to necrosis of the area where no oxygen reaches and, subsequently, heart attack of the same. This picture inevitably leads to a series of "warning" symptoms that should be known in advance by the patient (Ibidem). Infarct epidemiology Within this chapter, the theme of the epidemiology of infarction will be analyzed in depth. According to

the latest Eurostat data, just over 1.9 million people died from diseases of the circulatory system (mainly heart attacks and strokes), while 1.3 million died from cancer. These were the two leading causes of death in the EU, 28 responsible for 37% and 26% of all deaths respectively. Diseases of the circulatory system were the leading cause of death in all EU Member States, with the exception of Denmark, France, the Netherlands and the UK, where cancer was the leading killer. Cardiovascular disease is a class of conditions that include heart and blood vessel disorders or disorders and that include: coronary heart disease - in turn including unstable angina, variable angina, Syndrome X, silent ischemia and acute coronary syndromes including heart attack - cerebrovascular disease, peripheral arterial disease, rheumatic heart disease, congenital heart disease, deep vein thrombosis and pulmonary embolism (WHO 2015). Worldwide 32.4 million people are affected by acute myocardial infarction every year, of which 6.7 million die, according to data updated to 2012 (WHO 2014). As far as Europe is concerned, the situation regarding the health of citizens could improve, especially if certain eating and other habits are changed. The "black list" of wrong behaviors includes the excess of fatty foods, the little sport practiced, the little fruit and vegetables, the few fibers, but above all too much stress: pointed out as one of the main causes of the new millennium of various diseases. This is what numerous experts said during the "Winning Hearth ", organized on February 14, 2014 in Brussels by the European Heart Network in association with the European Society of Cardiology (ESC) and the European Community. In particular, according to recently published data by British Heart Foundation, around 4 million people die each year from cardiovascular disease, which is at the origin of about half of all deaths in Europe. Of these 4 million, 1.9 million die from heart attacks. In Germany, for example, every year 1% of citizens go to their doctor accusing attacks of angina pectoris; what is most frightening, however, is that after one year 10% of these people die or have a non-fatal heart attack (Medina 2014). Another argument should be made for patients who previously suffered from a heart attack: they are the group at the highest risk for coronary occlusions and brain events. Heart attack "survivors" have a calculated risk of death that is around 5% per year; six times higher risk of people of the same age who have not had cardiovascular disease. Unfortunately cardiac infarcts, heart failure and cerebral strokes continue to occupy the first place among the causes of death, so much so that annually about 30,000 people undergo an MI (Ibidem). According to the "Health 2020" report, the decline in deaths is increasingly offset by an increase in chronic diseases caused by changing living and working conditions, demographic changes, medical and technical progress and a way of unhealthy living - too little exercise, too much food, alcohol, TOBACCO - which were once fatal. This aspect, in addition to suffering and the often limited quality of life, also weighs on the health system and the economy (FSC 2013). Despite these pitiless statistics, there is considerable scientific evidence showing how specific interventions can reduce the risk of future ischemic events in patients who have already suffered from cardiovascular problems. If these interventions were promptly implemented,

JOURNAL OF ADVANCED HEALTH CARE (ISSN 2704-7970) - 2022 - VOLUME 4 - ISSUE III

about a third of fatal or non-fatal heart attacks could be prevented (WHO 2014). Furthermore, conditions such as AMI and stroke that are associated with often recurring and cost-related morbid events, if prevented in time, can reduce future costs. The results of the cost-effectiveness analyzes of secondary prevention indicate that secondary prevention measures are very cost-effective compared to many other routine medical interventions.

## **Risk factors**

It is not possible to draw a picture of cardiovascular disease without taking into account the distribution of risk factors and the prevalence of conditions at risk. There are several known factors that increase the person's risk of developing a heart attack condition and that predispose the body to get sick. The most important are:

- Age: the risk of heart attack, as with almost all cardiovascular diseases, increases with age.
- Gender: Atherosclerosis and heart attack are more common in men than women for the decades of youth and maturity. After female menopause, the risk of atherosclerosis and heart attack is similar in men and women.
- Familiarity: those who have cases of acute cardiovascular disease in their family history are at greater risk of heart attack, especially if the cardiovascular pathology of the joint has manifested itself at a young age by modifiable factors.
- Nutrition: A diet that is too high in calories and fat contributes to raising the level of cholesterol and other fats (lipids) in the blood, making atherosclerosis and heart attack much more likely. A healthy and balanced diet has a great value in terms of prevention of cardiovascular diseases.
- Arterial hypertension: "high blood pressure" or arterial hypertension can have various causes and affects a large portion of the population over the age of 50. It is associated with an increased likelihood of developing atherosclerosis and its complications, such as heart or brain infarction. It creates an increase in cardiac work which over time translates into a progressive malfunction of the heart and leads to the appearance of cardiocirculatory decompensation.
- Diabetes: the excess of glucose in the blood damages the arteries and promotes atherosclerosis, myocardial and cerebral infarction and the damage of important organs such as the kidneys, with the appearance of renal failure, in turn associated with increased cardiovascular risk.
- Tobacco addiction: According to the World Health Organization, tobacco smoke represents the single main avoidable risk factor for early death, illness and disability. The overall health consequences of smoking are very serious, as they lead to an overall reduction in estimated life expectancy of 10 years compared to non-smokers. Every moment is good to quit smoking: scientific evidence shows that cessation of exposure to smoke halves the risk of myocardial infarction after one year of abstention; after 15 years the risk becomes equal to that of a non-smoker. Smokers who quit before the age of 50 cut their risk of dying in half in the next 15 years compared to those who continue to smoke.

#### Smoking

This chapter will discuss the epidemiology of tobacco smoking, the phenomenon of addiction and the effects of smoking on the cardiovascular system. Addiction to smoking, or smoking, represents one of the greatest public health problems worldwide and is one of the major risk factors in the development of neoplastic, cardiovascular and respiratory diseases. Cigarette smoking continues to be a serious health hazard and contributes significantly to cardiovascular morbidity and mortality. Smoking affects all stages of atherosclerosis from endothelial dysfunction with acute clinical events, the latter largely thrombotic. Both active and passive (environmental) exposure to cigarette smoke predispose to cardiovascular events. Whether there is a distinct dose-dependent direct correlation between cigarette smoke exposure and risk is questionable, as some recent experimental clinical studies have demonstrated a non-linear relationship with cigarette smoke exposure. Recent experimental and clinical data support the hypothesis that exposure to cigarette smoke increases oxidative stress as a potential mechanism for initiating cardiovascular dysfunction.

#### **Addiction to tobacco**

Smoking has a high potential for addiction: the first symptoms can appear already after consuming a few cigarettes. In the majority of young people, occasional consumption evolves to become regular. Matteo Pacini, Stefania Pasquariello and Domenico Enea affirm that "10% of those who approach tobacco smoke develop a desire to smoke already two days after the first cigarette, to become 25% after two months" (quoted in Amato and 2013 Plan, page 167). Many believe they can control their consumption, but underestimate the addictive potential of smoking; all 8 smokers started smoking occasionally, but very few people manage to keep their consumption low for life (Amato and Piano 2013). Smoking also reduces the performance of young and fit people: those who smoke, in fact, have a shorter breath; sportsmen who smoke give less oxygen to their organs, get tired earlier and have a higher heart rate at rest. It takes a short time for nicotine to yellow your teeth and cause bad breath, as well as invade clothes and hair with a pregnant smell. Girls who use the birth control pill should not smoke, because the combination of the pill and tobacco drastically increases the risk of dangerous heart disease and thrombosis. The quantitative boundary within which these elements cease to indicate a pleasant habit and denote loss of control has not yet been defined, so that some low levels of consumption may correspond to an addiction (Amato and Piano 2013, p. 167). In the case of tobacco smoke, physical dependence is mainly caused by a neurotoxic: nicotine, which in combination with a psycho -behavioral complex corresponds to the physiopathological core of addiction. As one of the most addictive chemicals, it reaches the brain in seconds, affecting perceptions and mood for a limited period of time. Symptoms of addiction can also occur long before one switches to daily cigarette consumption; in fact, after just a few cigarettes, one tends to connect smoking to an expectation, so one smokes to relax, reward oneself, energize oneself, overcome moments of stress, etc. However, the feeling of relaxation that often comes from smoking does not amplify pleasant

withdrawal by taking nicotine. The relationship that binds the smoker to smoking is complex and articulated on several factors: from the gratifying effects of nicotine, to gestures, to the pharyngeal stimulation, to the role of the cigarette in managing moments of stress up to the emotional memory that can link it to object. In the beginning, smoking is the result of a substantially free and voluntary choice and causes a hedonic effect on those who use it: Later it tends instead to turn into a compulsive need, in order not so much to reproduce the initial effects, but rather to avoid the disturbances caused by its lack, transforming from hedonia to an additive effect. This phenomenon, called drug addiction or addiction, is common to most drugs and is one of their most fearful and, at the same time, most complex aspects. A group of experts from the World Health Organization defined drug addiction as «a psychic and sometimes even physical state, resulting from the interaction between a living organism and a drug, characterized by changes in behavior and other reactions, which include the drive to to take the drug in a continuous and operative way, in order to recover its psychic effects and to avoid the disturbances caused by its deprivation "(WHO1973). In addition to appearing among drugs in the Diagnostic and Statistical Manual of Mental Disorders IV Edition (DMS - IV) and in the Pocket Guide to the ICD - 10 Classification of Mental and Behavioral Disorders (1994), nicotine has also been classified as such by the World Health Organization. Implicit in the above definition is the concept that drug addiction involves the inability to maintain a state of physical and mental well-being without taking a drug. It follows that drug addiction is a disease which, paradoxically, is relieved by the same agent that is the cause. The unifying feature of drugs is the ability to free the mind from the constraints that keep it on the perhaps narrow but safe terrain of normal behavior; as a result of addiction to the drug, sensations of pleasure, of liberation from physical and mental suffering, of strength and escape from reality. Tobacco determines, through nicotine, very complex mental effects, attributable to its coupling point, represented by neuronal ganglia that modulate various central and peripheral nervous functions. These effects, which make smoking "pleasant, desirable and sometimes even useful", are contained in a framework which, depending on the circumstances, can respond to the need for reassurance or, conversely, for stimulation. At the same time, there is an improvement in concentration and learning ability. Probably the spread of smoking is explained by the fact that it does not satisfy only limited needs, as happens with other drugs, but to bring pleasure to larger areas. With tobacco you go from a few puffs of the first cigarette, which give a sense of daze and fun, up to 20/40 and, in extreme cases, even 80 cigarettes a day, which no longer cause any discomfort. Drug addiction leads back to a homeostatic reaction, which despite being a natural defensive process, must not lead to underestimating its extent. Reacting to the drug, the organism recovers its own functional state, but recovers it through adjustments that must be counterbalanced by a force of the opposite sign; it is an unstable equilibrium, different from the physiological one, because it requires the presence of drugs to be maintained. If the latter fails,

sensations, but rather eases the unpleasant effects of

difficulty in concentration, bradycardia, even marked, and so on (Silvestrini et al. 2003). Other effects induced by nicotine are for example malaise, sweating, vomiting which disappear in a relatively short period thanks to the addiction of the body, acceleration of the heartbeat, decrease in skin temperature caused by vasoconstriction and risk of thrombosis through hormonal mechanisms (League Against Cancer 2011). In the long run, these effects can damage the cardiovascular system. The intensity of drug addiction also varies from person to person, as well as from drug to drug; some become addicted after a few exposures to a drug, others only after prolonged exposure. Others get rid of drug addiction easily, others fail even when they suffer the devastating effects. Susceptibility to drug addiction is likely to be linked to hereditary factors, although this is difficult to ascertain with certainty. Generally speaking, there are two extreme types, among which all the others are placed: the one with a strong constitutional predisposition and a relatively modest influence of external factors: that, on the contrary, in which the latter play the fundamental role (Silvestrini et al. 2003).

#### Nursing role

In this chapter, the intent is to deepen, among all the various competences of the nurse, the role of health promoter, since, thanks to this specific role, the nurse is allowed to help the subject to fulfill the care of himself for a satisfying quality of life. Health reflects the object to which the promotion is aimed. WHO describes health as "a state of complete physical, social and mental well-being, and not simply the absence of disease or infirmity" (Kickbusch & Nutbeam, 1998). The meaning of health takes on a perspective that is traced back to multiple dimensions of the human being and, more precisely to a biological, psychological, social and spiritual pattern in which human beings themselves are inserted in a network of relationships and influence the health both through specific and individual interactions (Simonelli & Simonelli, 2010). Health is considered a means aimed at an objective which, can be traced back and can be considered, a resource that allows people to lead a productive life on an individual, economic and social level (Simonelli & Simonelli, 2010). The state of health, as mentioned at the beginning of this chapter, reflects a state of complete well-being and, precisely for this reason, well-being has been defined as the equivalent of health. Some authors argue that well-being has four components, which can be traced back to the ability to adapt and adapt to changing situations, the ability to exercise one's skills in the best possible way, the explicit declaration of feeling good and the feeling that everything is part of a whole that is in harmony with the others (Simonelli & Simonelli, 2010). With this reference, the accent is placed on the role of care providers and, more specifically, on nurses, who possess the skills to promote and enable positive changes oriented towards wellbeing and health (Smeltzer et al., 2010). If guaranteed and promoted, health therefore becomes a resource for daily life, a positive concept that enhances the individual and social resources of the individual aimed at reaching their human potential (Kickbusch & Nutbeam, 1998). According to the Ottawa Charter, within the concept of health promotion,

health is seen as a resource of daily life rather than the goal of living. The state of health is achieved thanks to the ability of individuals to develop and mobilize their resources in the best possible way, so that they can satisfy both personal (mental and physical) and external (material and social) qualities (Kickbusch & Nutbeam , 1998). Nurses possess and develop specific professional skills. These competences are briefly described below:

- "Role of expert in nursing care: as experts in nursing care, nurses are responsible, within the health system, for their professional actions and related decisions and evaluations.
- Role of communicator: as communicators, nurses allow the development of relationships of trust in their context and transmit information in a targeted manner.
- Role of team member: as team members, nurses participate effectively and efficiently in interdisciplinary and interprofessional groups Role of managers: as managers, nurses take on specialist management, contribute to the effectiveness of the organization and develop their professional career.
- Role of health promoter (Health Advocate): As health promoters, nurses rely responsibly on their expert knowledge and leverage their influence in the interests of the health and quality of life of patients / clients and society as a whole.
- Role of learner and teacher: As apprentices and teachers, nurses are committed to lifelong learning based on reflective practice and to the development, transmission and application of evidence-based knowledge.
- Role linked to professional membership: as belonging to their professional category, nurses are committed to the health and quality of life of individuals and society.

They are bound by professional ethics and the care of their health. " ("SUPSI - Department of Business Economics, Health and Social Affairs - Skills of the SUP nurse", sd) As you can see, the nurse at the end of the "Bachelor in Nursing Care" study cycle must master multiple professional skills, including whose role as health promoter; a role that has a greater value in this chapter. Nurses, designed to promote the health of the individual user, undertake to act with respect to problems related to health and quality of life, the interests of users and those of the people most dear to them. Within nursing care programs, nurses integrate health promotion and disease prevention practices. They also try to allow the patient and their relatives to use an individual and targeted approach in order to take advantage of means to prevent and deal with the disease and to maintain the highest possible level of quality of life. Finally, they participate in the development of concepts concerning health promotion and disease prevention ("SUPSI - Department of Business Economics, Health and Social - Skills of the SUP nurse", sd) Reporting these skills, allows us to understand how much the role of the nurse, is fundamental in maintaining a state of well-being, health and a satisfactory quality of life in the person in need of help. People nowadays seem to know more and more about their health and are showing more and more interest in promoting it. Healthcare professionals have made a considerable effort over the years to reach out and motivate members of various groups to promote their health, prevent disease and practice self-care ( Smeltzer et al., 2010). However, sometimes health is not fully perceived in all subjects. Often, we are confronted with the adoption of inappropriate behaviors that negatively affect the health of individual subjects. Stress, anxiety, depressive symptoms, inappropriate diet, lack of exercise, smoking and therefore, highrisk behaviors, are all lifestyle aspects that have a negative effect on health (Karmali et al ..., 2014). The task of the nurses therefore becomes that of involving each individual user, stimulating him to adopt behaviors and habits that promote his own state of health. The objective is therefore to motivate people to improve the way they live, modifying, if necessary, risk behaviors, adopting the healthier ones instead (De Lorenzi et al., 2010). Due to the importance that society assigns to health and the responsibility that each person has in maintaining and promoting it, the members of the health team and, in particular, nurses, are obliged to constantly make available educational initiatives aimed at maintaining a self-care and to promote health since, without adequate knowledge, people find it difficult to make decisions about their own health (Molinari et al., 2006). More attention will then be paid to the role of therapeutic education in promoting the health of the individual user who, at times, does not adhere to the therapeutic regimen offered to cope with their illness and, in this case, to the IMA.

Nursing management of the person with AMI Chest pain is a symptom that frequently requires the use of all the emergency structures operating, both in the territory and in the hospital. Chest pain management according to ACLS guidelines of American Cardiac Life Support, provides that chest pain is managed following schemes that take into account temporal and sequential intervention logics.

The timeliness of the therapeutic intervention aimed at re-establishing coronary recanalization is an important element for the prognosis of the subject affected by AMI.

The patient affected by AMI can arrive at the hospital in different ways and using extremely different times The most common are: - arrival following a call and subsequent prescription from the family doctor 68 direct arrival of the patient to the emergency room by own means or transported from family members - arrival after calling the emergency health service (118). Recourse to the 118 territorial emergency service is always recommended in the case of a typical onset symptomatology / or of high clinical severity (pain, loss of consciousness, severe dyspnea, imperceptible arterial pulses).

In relation to the concentration of sudden cardiac arrest deaths in the first and second hour after the first event, different knowledge is of fundamental importance, namely; - to know the risk factors for ischemic heart disease (smoking, arterial hypertension, diabetes mellitus, dyslipdemia, family history) that make the cardiac etiology of chest pain more likely - to know the characteristic symptoms of a heart attack to alert the emergency system territorial 118 - knowledge of cardiopulmonary resuscitation (BLS)

#### The nurse rescuer

The nurse, during the territorial rescue phase, is responsible for the correct functioning of the medical

equipment of the rescue vehicle. He must check the quantity and quality of medical-health devices, monitors, defibrillators, respirators, drugs. In addition, it must provide both basic life support (blsd) and advanced cardiac life support (als - acls). Once the nurse arrives in the field, he must carry out an early diagnosis with subsequent therapy, therefore he will carry out: 1) Control of vital parameters = through the evaluation of: - state of consciousness (using the glasgow scale ) - Respiratory frequency and type of respiration = provides essential data to ascertain the effectiveness of breathing and to detect adventitious or abnormal breath sounds that may be indicative of acute pulmonary edema or heart failure secondary to ischemic disease - Blood pressure - Radial pulse = (rate and rhythm providing a range of data for detect the presence of cardiac arrhythmias, changes in blood volume, and compromise of the cardiovascular system - Apical pulse = (frequency and rhythm) when the perideric pulse is irregular, weak or extremely rapid. (pallor, redness, diaphoresis, peripheral cyanosis and marbling) Venous access: positioning of a needle cannula of adequate caliber for the vein - use of a defibrillator monitor; considering the high incidence of VF in the first hour of the ischemic event) 12-lead ECG performed in the field with computerized interpretations by the electrocardiograph or transmission to medical personnel in order to stratify patients with chest pain 3) Administration of drugs = above all in the presence of a subject with persistent agor without hypotension or signs of collapse, it is always advisable to administer a nitrate by the sl route (trinitrine or carvasin) which allows a reduction in pain especially if there is hypertension.

A generic sedation intervention with oral anxiolytics at a medium-low dose can be practiced right from the first approach with the person.

The administration of opioids (morphine), certainly more effective from an analgesic point of view, must be carried out in case of pain insensitive to nitroglycerin with attention due to the side effects ie depression of the breath, nausea vomiting, hypotension.

The administration of Aspirin at doses ranging from 165 to 325 mg, depending on the product available, is recommended, even in the absence of a perfectly defined diagnosis.

The systemic administration of antiarrhythmic drugs is not justified, because there is no evidence of a real preventive efficacy of malignant arrhythmias.

Finally, the administration of thrombolytic drugs is strongly recommended, thus increasing survival by as much as 17% 4) Administration of oxygen using a resovoir mask with high flow.

Oxygen therapy increases the surrounding oxygen and therefore also that available to the myocardial tissue. In uncomplicated IMA administer O2 4lt min. For the first 2 - 3 hours4 Operating instructions during transport.

The nurse team leader of an ambulance in addition to coordinating the rescue at the place of the event, carries out the triage by assigning a color code (green, yellow, red and in some situations black) and establishes the destination of the possible transport of the patient in the the most suitable first aid, which is not always the closest, but rather that of the structure which, according to the DEA level, is able to accommodate the particular type of patient. The nurse will also perform various tasks including:

- 1) Information to be transmitted to the operations center (severity code, arrival time, activation of the doctor and setting up of the resuscitation box if necessary)
- continuous monitoring and constant control of consciousness, breath, heart rate, blood pressure, peripheral O2 saturation
- 3) respect, comfort and safety of the user to be guaranteed with safe transport
- 4) maintain radio communication with the 118 operations center
- 5) in case of doubtful changes in the electrocardiographic trace, stop the vehicle to allow the monitor to correctly read the cardiac activity
- 6) Do not lose control of the person's evolution
- 7) Administration of drugs according to operational protocol ( asa, trinitina , oxygen therapy, morphine, diuretics)

#### The Tragedy Nurse

tragic nurse does not have to make a medical diagnosis, but rather must evaluate the user's condition and the elements that could produce a potential heart failure or the onset of complications within the short or medium term. Therefore, the timeliness of diagnosis and treatment must be a fundamentally important feature of the DEA to ensure the greatest chances of survival and a high quality of life for patients with this type of symptomatology. The nurse in this area will have to carry out various steps including:

1) Positioning of the person on the examination bed

- 2) Psychological counseling to the person and information to caregivers givers
- Detection of basic vital signs, including SATO2 and continuous monitoring, especially during the administration of prescribed drugs.
- Positioning of 1 or 2 venous accesses possibly on the same limb, in order to make the other limb accessible to the detection of PA (bloody or not).
- 5) Collection of the first blood samples with identification of the person by labeling, and subsequently collection as per established protocols.
- 6) 12-lead basic ECG and then as per established protocol or when pain recurs
- 7) Administration of drug therapy in collaboration with the doctor
- 8) Constant visual monitoring, in order to detect any hemodynamic or symptomatic changes in the person
- 9) Beginning of reperfusion treatment as early as possible ( Ptca / Thrombolysis ).

## CONCLUSIONS

The topic I dealt with is very topical not only in Italy but all over the world. This stimulated my curiosity in searching for articles not only on databases but also on books and specialized websites which are constantly updated. The scientific evidence of the role of smoking on the prognosis of the heart patient is indisputable and constitutes an assumption shared by all cardiologist specialists. The impact that smoking cessation has on reducing the risk of relapses is also well known and it is no coincidence that it appears in the

65

guidelines for secondary prevention of ischemic heart disease in the first place among the interventions to be implemented. Evidence shows that the role of nurses to address these issues and the doubts they bring with them constitute a pillar of the health system and communication is the most effective means available to them. Education in getting patients to accept compliance has a better effect if the work is done in collaboration with doctors; however occasional care, but carried out by experienced nurses, improves outcomes ; moreover, nurses contribute to raising the quality of patients in the management of the disease. A holistic patient-centered approach as well as being supported by evidence and literature is the key to effective and satisfactory care also with regard to the creation of a therapeutic alliance and priorities and in this way the preferences of patients emerge with most successful. Quitting smoking, avoiding relapses of a heart attack and setting up a lifestyle cannot be isolated events but constitute a process that, if set in the long term through continuity of care, improve the patient's quality of life and push him to a increasing autonomy.

# REFERENCES

- 1. ACS (American Cancer Society). 2014. Cigar Smoking. Atlanta: ACS.
- 2. Amato, Laura, and Pier Paolo Piano. 2013. Addiction. Rome: The Scientific Thought Publisher.
- Anne N. Thorndike, Nancy A. Rigotti et Al: Depressive Symptoms and Smoking Cessation After Hospitalization for Cardiovascular Disease. Arch Intern Med. 2008; 168 (2): 186-191
- 4. Arlette Closuit Jenzer Martignye , and Nicolas Broccard Büro . 2014. "Smoking and mental health". Swiss Association for Tobacco Prevention 135 (4): 1-4
- 5. Barnoya J, Glantz S. Tobacco addiction. In Topo
- 6. Barth J, Critchley J, Bengel J Psychosocial interventions for smoking cessation in patients with coronary heart disease . Cochrane Database Syst Rev. 2008 Jan 23; (1): CD006886.
- Barua RS, Ambrose JA, Eales -Reynolds LJ, DeVoe MC, Zervas JG, Saha DC Dysfunctional endothelial Nitric Oxide Biosynthesis in Healthy Smokers with impaired endotheliumdependent vasodilatation. Circul. 2001; 104: 1905-1910
- Benovitz NL Gourlay SG: Cardiovascular toxicity of nicotine: implications for nicotine replacement Therapy . J Am . Coll Cardiol 1997; 29: 1422-31
- Bolman C., De Vries H.: Psycho-social determinants and motivational phases in smoking behavior of cardiac inpatients . Prev Med . 1998 Sep - Oct; 27 (5 Pt 1): 738-47.
- 10. Branzi, Angelo, and Fernando Picchio. 2013. Core Curriculum Cardiology. Milan: McGraw Hill.
- Burstein AH, Clark DJ, O'Gorman M et Al: Lack of pharmacokinetic and pharmacodynamic interactions between a smoking cessation therapy, varenicline, and warfarin : an in vivo and in vitro study. J Clin Pharmacol. 2007 Nov; 47 (11): 1421-9 PMID : 17962429 [PubMed - indexed for MEDLINE
- 12. Calabrò, Raffaele, Andrea D'Andrea, and Berardo Sarubbi. 2006. Cardiology for exercise sciences and nursing sciences. Naples: Idelson -Gnocchi
- Campbell- Sherer D. Green Lee A. ACC / AHA guidelines update for the management of ST Segment elevation Myocardial Infarction . Am Fam Physician 2009 Jun 15; 79 (12): 1080-1086.
- Centers for Disease Control and Prevention . Annual Smoking- attributable mortality , years of potential life lost, and productivity losses - United States, 1997- 2001. MMVR 2005; 54: 625-628
- Cesaroni G.: Effect of the Italian smoking ban on population rates of acute coronary events. Circul.online Feb 11,2008; DOI: 10.1161 / CIRCULATIONAHA.107.729889
- 16. Citizens, Sergio and Elisabetta Sartarelli . 2011. A brief history of tobacco smoking. Rome: Somalia Salus.
- 17. Clavario P .: Tobacco smoking pharmacotherapy . A clinical cardiologist point of review . Monaldi Arch.Chest Dis . 2004; 62: 1, 22-28
- 18. Collaborative meta analysis of randomized trials of antiplatelet therapy for prevention of death , myocardial infarction , and stroke in high risk patients BMJ. 2002; 324: 71-86
- Critchley JA, Capewell S Mortality risk reduction associated with smoking cessation in patients with coronary heart disease . JAMA 2003; 290: 86-97
- De Rosa S, Pacileo M, Sasso L et Al: insights into pathophysiology of smoke related cardiovascular disease.Mon. Arch.Chest Dis 2008; 70: 59-67
- Department of Veterans affair . VA / DoD clinical practice guideline for the management of tobacco use 2004. www. guideline.gov/ (accessed 04.07.2008).
- 22. Doggrell SA .: Which is the best primary medication for long- term smoking cessation: nicotine replacement therapy, bupropion or varenicline ? Expert Opin Pharmacother 2007 Dec; 8 (17): 2903-15
- Doll R, Peto R, Boreham J, Sutherland I: Mortality in relation to smoking: 50 years ' observations on male British doctors.BMJ . 2004 Jun 26; 328 (7455): 1519. Epub 2004 Jun 22
- 24. Edwardson Sandra. 2007. "Patient education in heart failure ". Heart Length 3
- 25. Eisenberg Mark J Evaluation of Varenicline ( Champix ) in Smoking Cessation for Patients Post Acute Coronary Syndrome (EVITA) Trial ClinicalTrials.gov identifier : NCT00794573
- Eisenberg MJ, Filion KB, Yavin D et Al: Pharmacotherapies for smoking cessation : a meta- analysis of randomized controlled trials. CMAJ 2008; 179 (2): 135-44
- Freemantle N, Cleland J, Young P, Mason J, Harrison J. beta Blockade after myocardial infarction : systematic review and meta regression analysis . BMJ. 1999; 318: 1730-1737

- 28. Frey P. et Al: impact of smoking on cardiovascular events in patients with coronary disease receiving contemporary medical therapy (from the Treating toNew Targets [TNT] and the Incremental decrease in End Points Through Aggressive Lipid lowering [IDEAL] Trials). Am J Cardio Dec 2010
- 29. Galanti LM: Tobacco smoking cessation management: integrating varenicline in current practice . Vascular Health and risk management 2008: 4 (4)
- 30. Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, Stein JH. Effects of smoking and smoking cessation on lipids and lipoproteins : outcomes from a randomized clinical trial. Am Heart J 2011 Jan ; 161 (1): 145-51
- 31. Greco G., Mocini D .: Treatment of smoking in secondary prevention. Ital Heart J 2001; 2 (1): 96-100
- Greco, Gabriella, and David Mocino . 2001. "Treatment of smoking in secondary prevention", Italian Heart Journal; 2 Suppl 1: 96-100.
- 33. Hays JT, Ebbert JO, Sood TO : Efficacy and safety of varenicline for smoking cessation . Am J Med 2008 Apr; 121 (4 suppl 1 ): s 32-42.
- 34. Hubbard R, Lewis S, Smith C et Al: Use of nicotine replacement therapy and the risk of acute myocardial infarction , stroke , and death . Tob . Control Dec 2005; 14 (6): 416-21
- 35. Humair, Jean Paul, and Jacques Cornuz. 2005. "Desaccoutumance au tabac". Vivre sans tabac. II Edition.
- 36. Smokers in Italy. ISTAT.www.istat.it/salastampa/comunicati/non\_calendario/20060110\_00/ (accessed 07.08.2008).
- Institute for Clinical Improvement . Tobacco use prevention and cessation for adults and mature adolescents 2004. www.icsi.org/ (accessed 04.07.2008)
- 38. Issa JS, Perez GH, Diament J, Zavattieri AG, de Oliveira KU Effectiveness of SustainedRelease Bupropion in the Treatment of Smoker Patients with Cardiovascular Disease . Arq Bras Cardiol 2007; 88 (4): 382-387
- 39. J. Taylor Hays, Jon O. Ebbert ,: Bupropion Sustained Release for Treatment of Tobacco Dependence . Mayo Clin Proc . 2003; 78: 1020-1024
- 40. Jaarsma T, R Halfens , H Huijer Abu Saad, K Dracup , T Gorgels , J van Ree, J Stappers . 1999. "Effects of education and support on self-care and resource utilization in patients with heart failure ". European Heart Journal 20 (9): 673-82
- JBI (The Joanna Briggs Institute ). 2001. "Smoking cessation interventions and strategies". Best Practice 5 (3): 1329-1874.
- 42. Karmali , Kunal , Philippa Davies, Fiona Taylor, Andrew Beswick , Nicole Martin and Shah Ebrahim. 2014. "Promoting patient uptake and adherence in cardiac rehabilitation". The Cochrane Collaboration, Issue 6: 1-63.
- 43. Kenfield SA, Stampfer MJ, Rosner BA, Colditz GA. Smoking and smoking cessation in relation to mortality in women . JAMA 2008; 299: 2037 -47
- 44. Kinjo K, Sato H, Sakata Y, Nakatani D, Mizuno H, Shimizu M et Al .: Impact of smoking status on long term mortality in patients with acute myocardial infarction .. Circ.J.69: 7-12
- Kristeller JL, Rossi JS, Ockene JK, Goldberg R, Prochaska JO: Processes of change in smoking cessation : a crossvalidation study in cardiac patients . J Subst Abuse . 1992; 4 (3): 263-76.
- 46. Lancaster T, Stead LF. Physician advice for smoking cessation . Cochrane Database of Systematic Reviews , 2004; Issue 2
- 47. Landau J., Ajani AE: Bupropion and bradycardia . Letters MJA 2008; 189 (3): 180
- 48. Lee AH Afessa B .: The association of nicotine replacement therapy with mortality in a medical intensive care unit . Crit . Care Med Jun 2007; 35 (6): 1517-21
- 49. Clinical guidelines to promote smoking cessation. ISS 2008. www.iss.it/ofad/fumo/cont.php?id=283 & lang = 1 & type = 3 (accessed 07.07.2008).
- 50. Clinical-organizational guidelines for the Piedmont Region. Cessation of tobacco smoking, 2007. www.snlg-iss.it (accessed on 04.07.2008)
- Mackay DF, Irfan MO, Haw S, Pell JP: Meta- analysis of the effect of comprehensive smoke -free legislation on acute coronary events . Heart online Aug 23 2010 doi : 10.1136 / hrt.2010.199026
- 52. McRobbie H., Hajek P.: Nicotine replacement therapy in patients with cardiovascular disease : guidelines for health professionals . Addiction 2001; 96: 1547-1551
- 53. Miceli, Domenico, Vincenzo Cirrincione, and Marino Scherillo. 2000. Management & Quality in Cardiology: the role of the nurse. Turin: Scientific Publisher Center.
- Moreno Ortigosa A, Ochoa Gómez FJ, Ramalle Gómara E et Al: Efficacy of an intervention in smoking cessation in patients with myocardial infarction. Med.Clin. 2000; 114 (6): 209-10
- 55. Mottillo S, Filion KB, Bélisle P et Al: Behavioral interventions for smoking cessation : a meta- analysis of randomized controlled trials. Eur . Heart J. 2009; 30: 718-730
- 56. New Zeland guidelines . Guidelines for smoking cessation 2002. www.nzgg.org.nz/ (accessed on 04.07.2008). Oberg EB, Fitzpatrick AL, Lafferty WE, LoGerfo Secondary prevention of myocardial infarction with nonpharmacologic strategies in a Medicaid cohort . JP.Prev Chronic Dis . 2009 Apr; 6 (2): A 52. Epub 2009 Mar 16.
- 57. Paciullo CA, Short MR, Steinke DT, Jennings HR: impact of nicotine replacement postoperative therapy on mortality following coronary artery bypass graft surgery . Ann . Pharmacother . Jul 2009; 43 (7): 1197-202
- Pinion M, Phillips C, Mulrow C. Use of lipid lowering drugs for primary prevention of coronary heart disease : meta analysis of randomized trials. BMJ. 2000; 321: 983 -986
- Polit Denise, and Cheryl Tatano Beck. 2008. Nursing research . Generating and Assessing Evidence for Nursing Practice . VIII Edition Philadelphia: Lippincott Williams & Wilkins
- 60. National Smoking Report. ISS 2006. www.iss.it/binary/ofad/cont/PACIFICI%2031%20maggio%202007.118094730 9.pdf (accessed 07.08.2008)...

67

- 61. Rea TD, Heckbert SR, Kaplan RC, Smith NL, Lemaitre RN, Psaty BM Smoking status and risk for recurrent coronary events after myocardial infarction. Ann Intern Med Sep 2002; 137 (6): 494-500
- 62. Reid RD, Pipe AL, Quinlan B: Promoting smoking cessation during hospitalization for coronary artery disease . Can J. Cardiol 2006 July ; 22 (9): 775-80 43.
- 63. Rice, Virginia Hill, Jamie Hartmann Boyce , and Lindsay Stead . 2013. "Nursing interventions for smoking cessation ". The Cochrane Collaboration, Issue 8: 1-8
- Ridker Paul M., Libby Peter Risk factors for atherothrombotic disease in Braunwald E. Heart Diseases. Seventh edition. Elsevier - Masson 2007
- 65. Rigotti NA, Munafo MR, Stead LF: Interventions for smoking cessation in hospitalized patients . Cochrane database Syst . Rev. 2007 Jul . 18; (3): CD001837
- 66. Rigotti NA, Pipe AL, Benowitz NL .et Al: Efficacy and safety of varenicline for smoking cessation in patients with cardiovascular disease : a randomized trial. Circul 2010; 121: 221-229
- 67. Smith PM, Burgess E. Smoking cessation initiated during hospital stay for patients with coronary artery disease : a randomized controlled trial. CMAJ 2009 jun 23; 180 (13): 1283-4
- 68. Song YM, Cho HJ. Risk of Stroke and myocardial infarction after reduction or cessation of cigarette smoking. Stroke 2008; 39: 2432-2438
- 69. Song, Rhayun, and Haejung Lee. 2001. "Managing health habits for myocardial infarction (MI) patients ". International Journal of Nursing Studies 38: 375-380
- 70. Stack NM .: Smoking cessation : an overview of treatment options with focus on varenicline . Pharmacother . 2007 Nov; 27 (11): 1550-7 ..
- 71. Stead LF, Perera R, Lancaster T: Telephone counseling for smoking cessation . Cochrane database Syst.Rev . 2006 Jul . 19; 3: CD002850
- 72. Stead LF., Bergson G. Lancaster T.: Phisician advice for smoking cessation . Cochrane Database Syst . Rev. 2008 Apr 16; (2): CD000165 and the National Health Service: perspectives and commitments 2008. www.iss.it/ofad/umo / cont. php? Id = 289 & lang = 1 & type = 3 (accessed 08.07.2008).
- 73. Thomas D. Cardiovascular benefits of smoking cessation . Med presses Apr 2009
- 74. University of Michigan. Smoking cessation 2006. www.med.umich.edu/mfit/tobacco/ PDF / smoking.pdf (accessed on 04.07.2008). Wiggers LC, Smets EM, de Haes JC, Peters RJ, Legemate DA: Smoking cessation intervenes
- 75. Wu P, Wilson K, Dimoulas P, et Al: Effectiveness of smoking cessation therapies : a systematic review and metaanalysis . BMC Public Health 2006; 6: 300