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Theoretical part

Estimation of heart rate and blood pressure

Pulse examination used to be an important diagnostic method. Nevertheless since the 20th century blood pressure (BP) examination has become the most common diagnostic method. Both variables, pulse and blood pressure, reflect cardiac activity and vascular condition.

BP represents not only systolic and diastolic blood pressure, but a continual variable, which is a result of the heart's work. Blood is pumped from the heart to the aorta during the ejection phase of the systole. At this moment the blood pressure in aorta rises to its maximal value – systolic blood pressure (SBP). Simultaneously the aorta stretches and contains volume of blood. During diastole, after closing of the aortal valve, the aorta lessens its diameter and shifts blood forward from the heart. Blood pressure decreases to its lowest value – diastolic blood pressure (SBP). Pulse pressure (PP) is the difference between SBP and DBP. This is the way of creating the pressure pulse wave which then spreads through the vascular system. The pressure wave temporarily extends the artery and the palpated pulse is the wave under the place of the palpation.

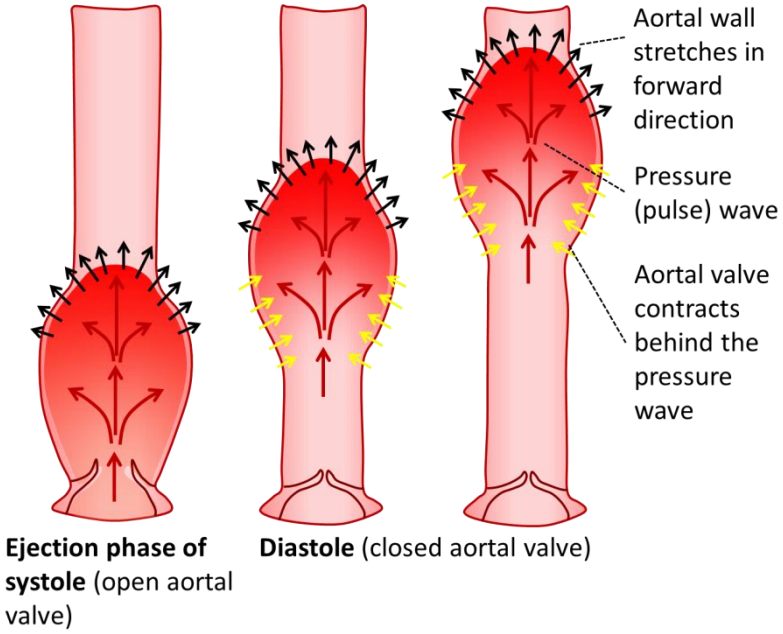

Figure 1: Pulse formation in aorta

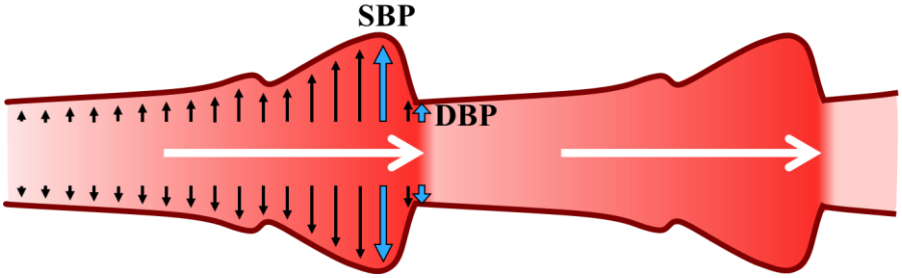

Figure 2: Arterial pulse

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The pulse wave velocity (4 m/s in aorta) is not equal to the velocity of the blood flow (0.18 m/s in aorta).

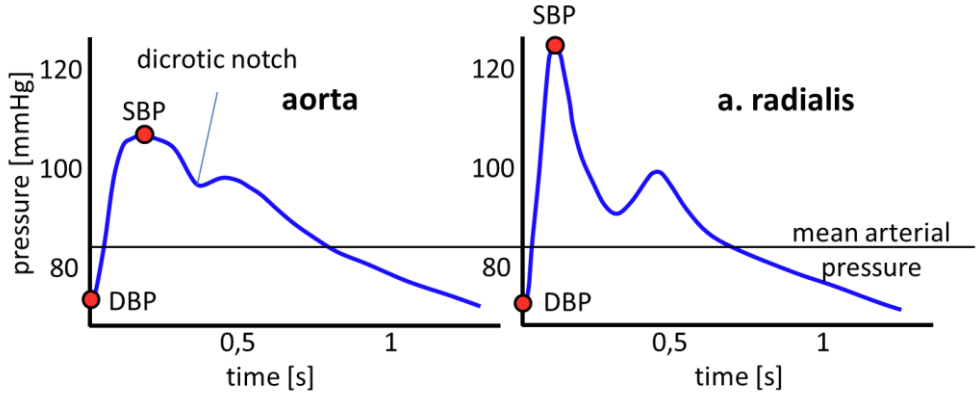

Figure 3: Pulse wave

Distribution of the blood to tissues is the main function of the cardiovascular system. The heart is the pump which creates a pressure gradient between the beginning of the vascular system (mean arterial pressure, MAP) and the end of the system (venous pressure in the vena cava). The pulsatile component of blood pressure is superimposed on the MAP. Pulsation is a result of changes between the ejection phase of systole and non-ejectional phase (diastolic phase and isovolumic systole). MAP is defined by blood volume inflowing into the arterial system and by the volume of blood outflowing from this system. Blood inflow is given by cardiac output (CO, blood volume overdrawn by heart per minute) which consists of stroke volume (SV, blood volume ejected in one cardiac cycle) and heart rate. Blood outflow is given by total peripheral resistance (TPR). TPR is determined by summary vascular diameter. Vasoconstriction increases TPR, vasodilatation decreases TPR. Although CO and TPR influence MAP, SBP is influenced mainly by CO and DBP is influenced mainly by TPR.

$$MAP = SV * HR * TPR$$

Estimation of pulse and heart rate

In pulse estimation we can evaluate its quality, frequency and regularity.

Qualities of peripheral pulse: Some further properties of the pulse may be estimated by palpation. If both the rising and descending phase of the pulse wave are fast, one designates the pulse as fast (pulsus celer). The opposite is a slow pulse (pulsus tardus). If pulsation of the artery can be easily suppressed by pressing it with finger, one speaks of a soft pulse (pulsus mollis), the opposite is a hard pulse (pulsus durus). A soft pulse is found in patients with low blood pressure, a hard pulse in hypertension where also slow pulse is present. A fast pulse is observed in states where the main arteries quickly empty their content (in aortic valve insufficiency or in excessive peripheral vasodilatation). Also the magnitude (amplitude) of the pulse wave is taken into consideration (p. magnus – big pulse, p. parvus– small pulse); very weak, almost impalpable pulse is often designated as a threadlike pulse. It is present in circulatory failure or in hypotension.

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According to its regularity, a person's pulse can be divided into regular and irregular. Irregular pulse can be caused by arrhythmia (for example extrasystols). When observing a healthy person HR changes should correspond to respiration.This HR fluctuation is called respiratory sinus arrhythmia. Bradycardia for an adult person means that the resting HR is lower than 60 bpm (beats per minute).Tachycardia means that HR is higher than 110 bpm. Children have a high resting HR. Bradycardia physiologically occurs in athletes and pathologically in junction rhythm (atrioventricular node is a cardiac pacemaker). Tachycardia can be caused by hyperthyreosis, hypotension, infection, and heart failure. Maximal HR (originating from the sinoatrial node) is 180–200 bpm.

Respiratory sinus arrhythmia (RSA)

HR increases when breathing in and decreases when breathing out. Sinus arrhythmia is called such because cardiac electrical activity begins in the sinoatrial node. This physiological arrhythmia occurs in a resting condition, mostly in the young, and it is a manifestation of vagal activity. RSA is highlighted in deep slow breathing and disappears during fast shallow breathing.
There are several mechanisms explaining RSA. The central mechanism includes transfer of action potentials from respiratory to cardioinibitory centres in medulla. An other mechanism includes baroreflex. Changes in pressure in the chest during breathing influences venous return, cardiac filling, stroke volume and therefore blood pressure. Blood pressure changes are registered by baroreceptor, and therefore HR changes in correlation with breathing. Oscillations of pCO_2, pO_2 and pH during breathing also influence HR. Also, a denervated heart can change HR: a light mechanical prolongation of the sinoatrial node when breathing in increases the creation of an action potential in the node.

Blood pressure changes in the vascular system

Blood pressure changes through the vascular system. MAP in the arterial system (from the aorta to small arteries) is almost unchanged (approx. 90–100 mmHg). The pulsatile component (120/80 mmHg) is maintained in arterial blood pressure from aorta up to small arteries or arterioles, but pressure amplitude changes: pulse pressure slightly increases from the aorta to small arteries, mainly due to SBP rise. At the level of small arteries and arterioles, blood pressure rapidly decreases and the pulsatile compound disappears (SBP and DBP value become closer).These vessels have smooth muscles in arterial wall, and therefore small arteries and arterioles are able to change their diameter and vascular resistance and are called resistant vessels.

Blood pressure in the capillary system decreases from 25 mmHg in the arteriolar end to 15 mmHg in the venous end and blood pressure pulsation is not present. Capillaries are only vessels enabling transfer of substances between blood and tissues. The capillary system has the greatest summary surface and one capillary has the smallest diameter and capillary wall, which maximizes communication between blood and tissues.
In the venous system, blood pressure decreases from 15 mmHg in venules to 4–5 mmHg in the vena cava. The venous system can absorb almost half of the blood volume because of the high compliance of venous wall. Therefore, veins are considered to be capacitance vessels. Blood pressure is also given by position relative to the heart. Blood pressure increases by 0.77 mmHg/cm away from the heart level above and vice versa.

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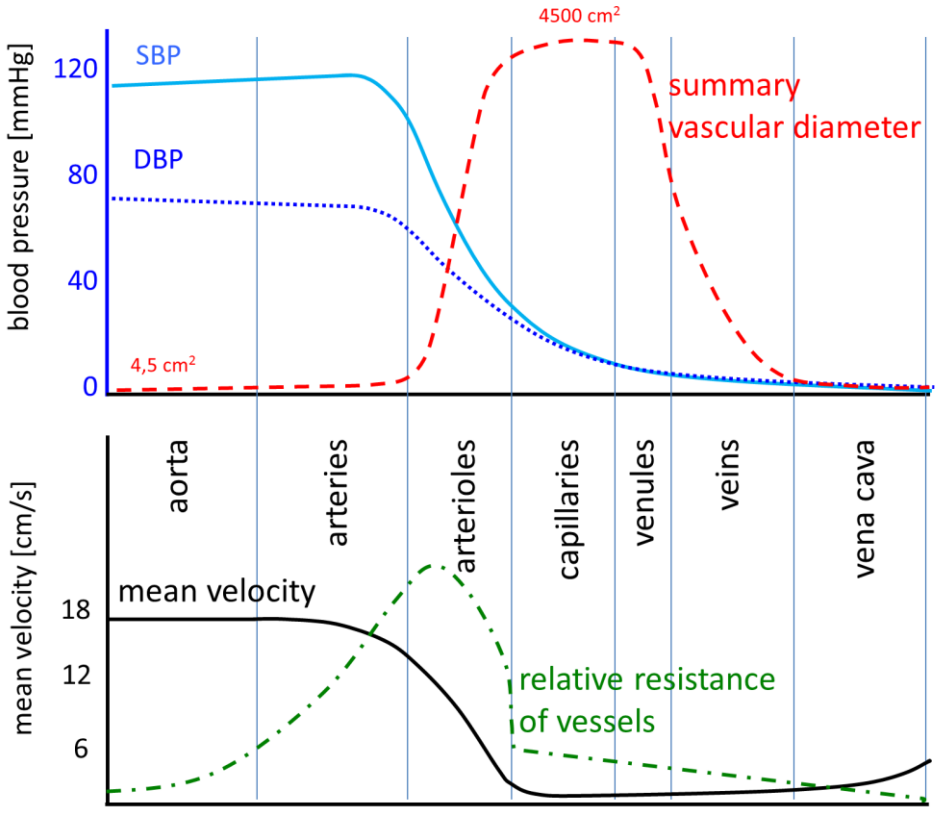

Figure 4: Changes of blood pressure, blood velocity, vascular diameter and resistance in the vascular system

Blood pressure regulation

Stable sufficient blood pressure is necessary for homeostasis. Impairment of BP regulation leads to damage to the cardiovascular system and target organs. Low BP (hypotension) leads to low perfusion of target organs. High BP (hypertension) damages vessels and exhausts the heart. MAP is defined by HR, SV and TPR, so mechanism of BP regulation changes these variables. Mechanism of blood pressure regulation can be divided according to several criteria. Dividing according to the duration of effect, we distinguish short-term, intermediate and long-term regulation. Dividing according to the mechanism of BP regulation, we distinguish neural regulation, hormonal regulation and autoregulation.

Neural control of blood pressure

Blood pressure is controlled by two branches of the autonomic nervous system (ANS): sympathetic and parasympathetic systems. The sympathetic branch usually has an excitatory influence on the cardiovascular system: it increases heart rate, heart contractility and blood pressure. The parasympathetic branch has a contrary effect.
An efferent neural pathway begins in the medulla, which are under the influence of the higher nervous centres. BP changes under different mental and emotional conditions. Acetylcholine is a parasympathetic neuromediator. Noradrenalin (norepinephrine) and adrenalin (epinephrine) are sympathetic neuromediators. All neuromediators are also hormones, and therefore neural control cannot be divided from hormonal control. Both ANS branches control cardiac function, but vascular tonus is controlled by the sympathetic branch. The vascular

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walls in each organ have a different concentration of receptors for adrenalin and noradrenalin depending on the particular function of the organ.

- Sympathetic branch – blood pressure increase
    - Cardiac effect – increase of heart rate and contractility
    - Vascular effect
        - β receptors (higher affinity to adrenalin, less to noradrenalin)
            - vasodilatation of arteries and arterioles in skeletal muscles and liver
        - α receptors (higher affinity to noradrenalin, less to adrenalin)
            - vasoconstriction of most arteries and arterioles (except for skeletal muscles and liver) – increase of total peripheral resistance
            - venoconstriction – redistribution of blood volume
- Parasympathetic branch – blood pressure decrease
    - Cardiac effect – decrease of heart rate and contractility
    - Neuromediator – acetylcholine

Baroreflex

Baroreflex is the fastest mechanism of blood pressure regulation. Baroreflex regulates BP fluctuation though changes in the heart rate and peripheral resistance. Baroreceptors are placed in the carotids and aortic arch and they register the extension of aortic and carotid wall during increased BP. Information from the baroreceptors are transferred by the parasympathetic nerves (n. vagus and n. glosopharingeus) to cardiovascular centres in the medulla. Baroreflex has cardiac and peripheral branches. Cardiac efferent pathways (n. vagus) change the heart rate. Vascular efferent pathways (sympathetic nerves) change the peripheral resistance through changing the vascular diameter.

Example of baroreflex regulation during orthostatic stress

During a change of position from a lying position to a supine one (orthostatic stress) blood falls downward following gravity – the arterial blood pressure in the upper half of the body and venous return decreases. The perfusion of the brain would be insufficient and conscious could be impaired were the decreased arterial BP not regulated. Decreased BP is detected by baroreceptors and information is sent to the medulla. The medulla increases sympathetic efferent activity and inhibits vagal efferent activity.

The response of cardiac baroreflex branch is faster, with a latency of 1 or 2 cardiac cycles. Inhibition of cardiac vagal control and excitation of cardiac sympathetic control increases heart rate. Sympathetic vascular control with a latency of approx. 6 seconds increases peripheral resistance. Increased heart rate is less efficient in long-term preservation of blood pressure, but it quickly helps to prevent a drop in blood pressure in the first phase of orthostatic stress and it helps to maintain brain perfusion. After a few seconds the heart rate can decrease, because blood pressure is held by increased vascular peripheral resistance as a more efficient long-term mechanism.

The reaction to a decreased blood pressure during clinosthasis (from supine to lying position) is contrary to an orthostatic response.

Hormonal regulation of blood pressure

Hormonal blood pressure regulation lasts from tens of seconds to days. Adrenalin and noradrenalin are hormones with the fastest effect on blood pressure. Renin-angiotensin-aldosterone system (RAAS) is another important intermediate mechanism of controlling blood pressure. When the plasma sodium concentration is lower than normal or the renal

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blood flow in kidneys is reduced, the juxtaglomerular cells in the kidneys convert prorenin (an intracellular protein) into renin, which is then secreted directly into the circulation. Renin in plasma converts angiotensinogen to angiotensin I. Angiotensin I is then converted by the angiotensin-converting enzyme (ACE) to angiotensin II. ACE is mostly found in the lung capillaries. Angiotensin II is a potent vaso-active peptide that causes constriction of arterioles, resulting in increased arterial blood pressure. Angiotensin II also stimulates the secretion of the hormone aldosterone from the adrenal cortex, the antidiuretic hormone (ADH, vasopresin) from neurohypophysis and supports sympathetic activity.
Aldosterone causes the tubular epithelial cells of the kidneys to increase the reabsorption of sodium ions from the tubular fluid back into the blood. It causes them at the same time to excrete potassium ions into the tubular fluid. Sodium reabsorption by the blood is followed by water, and therefore blood volume increases. ADH increases reabsorption of water in the kidneys and in high concentrations it is a strong vasoconstrictor. Blood pressure can also be influenced by corticoids, somatotropin and thyroid hormones.

Autoregulation of blood pressure and blood flow

Blood pressure is regulated on the level of the organs by metabolic autoregulation, myogenic autoregulation and by endothelial vasoactive substances. The target effect is a blood flow change rather than blood pressure change.
When high pressure stretches the arterial wall in an organ, the smooth muscles in the wall contract. This myogenic vasoconstriction reduces blood pressure in the artery supplying the organ and protects the organ from damage. Myogenic autoregulation is well developed in the brain and kidneys.
Tissue with increased activity has higher metabolic entitlements – higher consumption of nutrients and production metabolites. The decreased pO2, increased pCO2, temperature, osmolality and increased concentration of adenosine, potassium, H+, lactic acid causes local vasodilatation. This metabolic autoregulation dominates in working muscles or in the gastrointestinal tract during digestion of food.
Endothelium also products vasoactive substances, such as prostaglandins (tromboxan, PG2 – dilatation), endothelins (endothelin 1 – constriction), kinins (bradykinin – dilatation), histamine (dilatation) and serotonin (constriction). Most of these substances act through creation or inhibition of NO, which relaxes the smooth muscles in the arterial wall.

Example of blood pressure regulation during and after physical activity

Working skeletal muscles need more nutrients and produce more metabolites. This leads to the following changes in the cardiovascular system.
Arteries and arterioles in working muscles dilate due to metabolic autoregulation. Blood pressure in the early phase of physical activity decreases. The blood pressure decrease is detected by the baroreceptors and a change in partial pressure of respiratory gases is detected by the chemoreceptors. The reflexes activate the sympathetic nervous system including catecholamine release. Catecholamines cause vasodilatation if they are bound to βreceptors, or they causes vasoconstriction if they are bound to α receptors. Skeletal muscles have more β□receptors than α receptors, therefore metabolic vasodilatation in working muscles is supported by catecholamines. On the other hand most vessels, especially in the skin, kidney and splanchnic organs have more α receptors in the wall. Vasoconstriction in these organs enables redistribution of blood to working muscles and prevents drops in blood pressure. Vasoconstriction in the skin during the early phase of physical work is later replaced by prevailing thermoregulatory mechanisms causing vasodilatation.
The pumping action of contracting skeletal muscle improves venous return, but the venous return during static work can remain unchanged. As a result, cardiac filling increases and, according to the Starling mechanism, the result should be an increase in stroke volume. Simultaneously, the heart rate increases and therefore cardiac output rises.

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Physical work leads to a drop in the peripheral resistance in working muscle and an increase in the peripheral resistance in most of other vessels (except the brain or liver). Peripheral resistance influence mainly diastolic blood pressure, and thus diastolic pressure can remain unchanged, slightly increase or decrease depending on the type, level and duration of physical activity. During weight-lifting diastolic pressure will be higher than during fast walking. Systolic blood pressure positively increases, because it is primarily influenced by cardiac output.

The effect of rhythmic physical activity on blood pressure is that systolic blood pressure increases, diastolic blood pressure changes only slightly and mean arterial pressure rises, but usually not to an extreme degree. However, the aim of these cardiovascular changes is not an increase of blood pressure, but an increase of blood flow to cover the needs of working muscles.

Principles of non-invasive methods of measuring blood pressure

Palpatory (Riva-Rocci) and auscultatory (Korotkow) methods

Indirect measurement of BP is possible thanks to the compliance of the arterial wall. If a variable outer pressure acts upon a region with an artery, complete compression of the artery and interruption of the blood flow occurs at the moment when the outer pressure just exceeds the systolic pressure (SBP). This moment and the corresponding value of pressure may be estimated by palpation of the pulse distally from the compression area (Palpatory method). If the outer pressure is lower than SBP but higher than diastolic pressure (DBP), the blood flow during each pulse is interrupted and renewed alternately. Sudden changes of blood pressure and flow arising in this way cause turbulent flow and vibrations in the acoustic frequency range. The vibrations (the sound) can be detected by hearing, e.g. by a stethoscope applied to the artery distally from the compressed area. If the outer pressure is below DBP, no periodic closing of the artery occurs and no sound is heard. Thus, the sounds are audible at all values of outer pressure lying between SBP and DBP. The auscultatory method measures the SBP as well as the DBP.

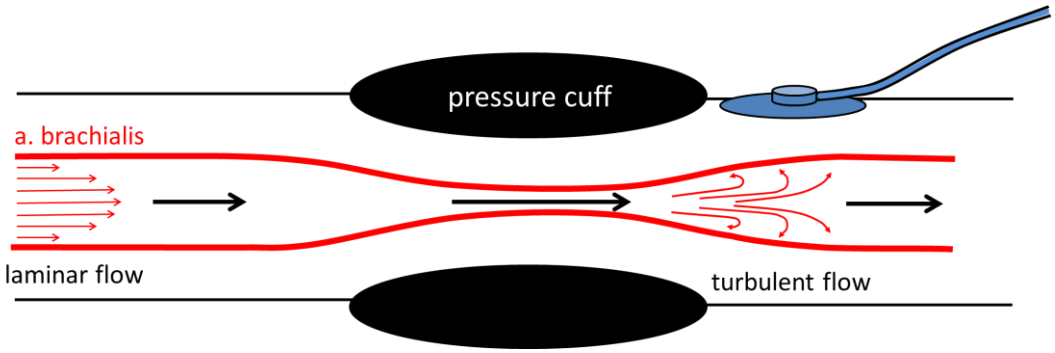

Figure: Formation of sounds in auscultatory blood pressure measurement

Oscillometric method

This method is based on recording small pressure changes in the measuring cuff, caused by volume changes of the brachial artery when the mean pressure in the cuff is lower than systolic and higher than diastolic pressure. At the beginning of the measurement cuff pressure is increased over systolic pressure. This stops circulation in the forearm and empties the brachial artery. Then, the cuff pressure is slowly decreased. At the moment when the cuff pressure is lower than systolic pressure, the brachial artery starts to change its volume during each cardiac cycle – it is empty during the diastole and is filled with blood during a short

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interval when the cuff pressure is lower than the arterial pressure during the systole. Thus, the moment of systolic pressure can be determined. The volume oscillations in the brachial artery and thus also the pressure oscillations are the highest at the moment when the cuff pressure corresponds to the mean arterial pressure in the brachial artery. The processor in the measuring device can calculate the diastolic blood pressure using the value of systolic blood pressure and the mean blood pressure. Some devices measure only mean arterial pressure, SBP and DBP are counted. Both the systolic and diastolic values of blood pressure are displayed on the screen of the blood pressure monitor.

24-hour blood pressure monitoring

In some cases ambulatory blood pressure measurement is not sufficient for diagnosis assessment. 24-hour blood pressure monitoring is used, for example, when white-coat hypertension (the result of fear of doctors) is suspected, in the case of a seizure of hypertension or when we want to estimate the difference between daily and nightly blood pressure. The advantage of this kind of measurement is that blood pressure is monitored in the patient's natural environment. The measuring device is usually based on an oscillometric method and it automatically measures each time interval (15, 20, 30 or 60 minutes). A series of SBP, DBP, MAP and mean heart rate values are the output from this measurement.

Photoplethysmographic method of continual blood pressure measurement (Volume-clamp or Peňáz's method)

The photoplethysmographic method is the only method of measuring blood pressure non-invasively and continually. The above-mentioned methods provide only SBP, DBP and MAP values. A pressure cuff is placed on a finger and it has a built-in light source and a photosensor. Light passing through tissue detects blood volume changes under the cuff during each pulse (i.e. as the translucence of tissue changes). The device aims to change the cuff pressure to make blood volume constant. Under these conditions, the pressure in the cuff is equal to blood pressure.

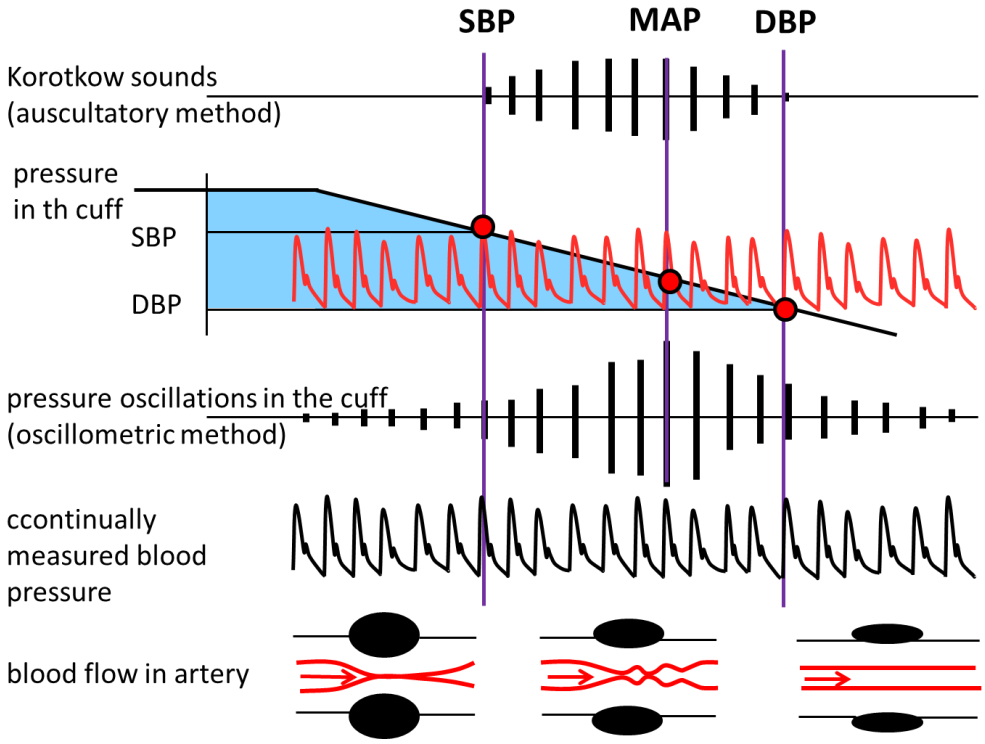

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Figure: Differences between methods of blood pressure measurement

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Method	Advantages	Disadvantages	Measured value
auscultatory	• More accurate estimation of SBP/DBP • Simple, do not need electric power, cheap	• Subjective, requires experience • SBP/DBP are not counted from the same cardiac cycle	SBP and DBP
Oscillometric	• Accurate estimation of MAP • Automatic, quick, cheap, suitable for laymen (home measurement)	• SBP/DBP are counted (dependence on computational model) • SBP/DBP are not counted from the same cardiac cycle • Cannot be used in cardiac arrhythmia	MAP, in some devices also SBP
24-hour blood pressure measurement	• Blood pressure recorded during the whole day • White-coat hypertension can be ruled out	• Disruptive effect of inflating the cuff (during sleeping)	Blood pressure values every 15–60 minutes
Photoplethysmo-graphic method	• Continual recording of blood pressure • Possibility of SBP and DBP evaluation beat to beat	• Measured at the finger, need to calculate brachial blood pressure • Expensive device	Continual blood pressure

Table 1: advatnages and disadvantages of methods of measuring blood pressure

Hypertension

High blood pressure (hypertension) is a risk factor for cardiovascular diseases like heart failure, cardiac ischemia, myocardial infarct, and bleeding in CNS (Table 2). To diagnose hypertension, several blood pressure measurements have to be done and prove high blood pressure.

According to the origin of hypertension we can distinguish hyperresistant hypertension caused by an increase in cardiac output and hyperdynamic hypertension caused by an increase in total peripheral resistance. Hypertension can be caused by increased sympathetic activity, higher sensitivity to catecholamines, increased blood volume or a higher level of angiotensin II. Although the cause of hypertension may be temporary, long-term hypertension leads to permanence by reinforcement of the smooth muscles in the arterial wall.

Hypertension can be divided into primary (essential) and secondary hypertension. The origin of primary hypertension is unknown or it is a set of possible causes. Secondary hypertension is formed as a result of another disease. Secondary hypertension is often caused by diseases of

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glands producing a hormone that influences blood pressure, such as Cushing syndrome (cortisol), hyperthyreosis (T3, T4), acromegaly (somatotropin), hyperaldosteronism, pheochromocytoma (catecholamines), etc. Ischemia of kidneys (e.g. due to obstruction of the renal artery) leads to renal hypertension. Hypertension can be caused by pregnancy (preeclampsia), by medications or neurologically (encephalitis, brain edema, tumours). Isolated systolic hypertension is typical for increased systolic pressure while diastolic pressure is unchanged or even decreased. Systolic hypertension is caused by lowered aortal compliance.

	Blood pressure	**SBP [mmHg]**	**DBP [mmHg]**	**Possible complications**
hypotension	low	< 90	< 60	nausea, impaired consciousness
normal	Optimal	<120	<80	
	Normal	120–129	80–84	
	upper normal	130–139	85–90	
hypertension	1st degree	140–159	90–99	without organ changes
	2nd degree	160–179	100–109	Left ventricular hypertrophy, proteinuria, angiopathy, etc.
	3rd degree	> 180	> 110	morphological and functional changes of some organs, retinopathy, heart and renal insufficiency, ischemia of CNS, bleeding in CNS, etc.
isolated systolic hypertension		> 140	<90	

Table 2: Classification of blood pressure

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