

Topic: 2.2 FORENSIC AUTOPSY IN CASE OF SUDDEN DEATH

Substantiation of the Theme: Sudden death occurs in forensic-medical practice rather frequently. The peculiarity of this kind of expert examination includes first of all the fact that by the time of autopsy the expert practically does not have any information on anamnesis, the character of a disease, its course, level of health before death and during the process of dying. Mostly at the expert's disposal there is only an instruction or decision of bodies of law and order and a protocol of examination of a corpse on the place of its detection. Expert examination of such cases requires the knowledge of morphological peculiarities and pathogenesis of a number of diseases that result in sudden death.

It is necessary to keep in mind that the forensic-medical expert has a right to obtain, on request to investigating bodies, the outpatient's card, medical history, and other medical documents, to elucidate circumstances of death and measures on rendering medical aid.

Purpose of the Practical Class: to learn to reveal and diagnose pathological changes of organs and tissues at forensic-medical autopsy; master particular skills of autopsy technique; be able to make a forensic diagnosis and draw a conclusion in case of non-violent death.

Plan of the Practical Class:

1. Control of initial level of knowledge on the topic.
2. Discussion of the key questions.
3. Independent Autopsy with drawing up the introductory and research part of "Protocol of Forensic Autopsy", Conclusion of Expert, Forensic Medical Diagnosis, and Medical Death Certificate.
4. Situational tasks solving.
5. Concluding remarks of the teacher.

Recommended Additional Literature:

1. АБТaHHMJioBT.r. OCHOBHnararioroaHaTOMiOTecKownpaiawii. - M.: PMATIO, 1994.
2. KajiHTeeBCKWM F.I.O. MopqbojionraecKaHawqbd^epemjHairbHaHflwarHOCTHKanaTonorw-^ecKwxnpoueccob. - M.: MefIHU,HHa, 1987.
3. Me^jiyHapoflHaaKnaccwqbwKanjw 6ojie3HeH, TpaBM w nptrowHCMepm M.: MepjmHHa, 1995.
4. Zagorulko A. Short lectures on Pathology. - Simferopol, 2002.

Before the Practical Class, the Student should know:

- > definition of sudden death;
- > aetiology of various diseases that cause sudden death;
- > pathologic physiology and pathologic anatomy of various diseases that cause sudden death;
- > potential of laboratory diagnosis of different pathologic conditions and processes at post-mortem examination in case of sudden death;
- > procedure of collection of samples for laboratory investigations.

During the Practical Class, the Student should acquire the following Skills and be able to:

- > reveal and describe morphological changes in different organs and tissues at post-mortem examination in case of sudden death;
- > carry out an autopsy investigation of the heart and vessels;
- > collect samples for laboratory investigations;
- > fill in the medical death certificate;
- > make a Forensic Medical Diagnosis and draw up a Conclusion in case of sudden death.

Questions for Student's Independent Work:

1. Definition of Sudden Death.
2. The commonest diseases that result in Sudden Death.
3. Clinical and morphological signs of these diseases.
4. The peculiarities of post-mortem examination and registration of documents in case of sudden death.
5. The peculiarities of collecting samples for additional investigations.
6. HIV: the peculiarities of examination.
7. Sudden Infant Death Syndrome.

Block of Information for Student's Independent Work

Sudden and Unexpected Death

In 1964 the group of experts-pathologists of the Department of Cardiovascular diseases of World Health Organization (WHO) for the first time tried to formulate the unified definition of sudden death. According to the criteria of WHO, the death, which took place within 6 hours from the moment of disease in a healthy person or the person, who was ill and was in a satisfactory condition, refers to the category "sudden". The important criterion of sudden death is time of its approach after occurrence of acute symptoms.

The International Committee created in the USA, in 1970 has suggested considering the sudden death like death which has come during 24 hours after occurrence of symptoms of a fatal attack. Both of these criteria are used by various researchers that complicate comparison of the data received by them. The matter is that in case of approach of death within 6 hours after the beginning of a fatal attack the diseases of heart prevail; in case of approach of death during 24 hours the significant rate of died is patients with vascular cerebral diseases, acute and chronic diseases, etc. (Masur N.A, 1985). The problem of sudden death from cardiovascular diseases is traditionally considered in the context of the general definition of sudden death.

However, in forensic practice, most of such deaths occur in minutes or even seconds after the onset of symptoms. A sudden death is not necessarily unexpected and an unexpected death is not necessarily sudden, but very often the two are in combination. No period in life is exempt.

The temporal definition of sudden death strongly influences epidemiological data. Retrospective death certificate studies have demonstrated that a temporal definition of death less than 2 hours after the onset of symptoms results in 12 to 15% of all natural deaths being defined as "sudden" and nearly 90% of all natural sudden deaths being due to cardiac causes. In contrast, the application of a 24-hour definition of sudden death increases the fraction of all natural deaths falling into the sudden category to more than 30 % but reduces the proportion of all sudden natural deaths that are due to cardiac causes to 75 %. A systematic view of the differential diagnosis of the cause of death and a logical choice of the most likely cause of death will help improve the state of mortality statistics, assist the legal authorities and satisfy the bereaved relatives, perhaps by helping them in obtaining insurance and compensation benefits.

According to WHO, the *syndrome of sudden death* is a sudden non-violent death of an infant (sleep apnoea syndrome, sudden infant death), when the ade-

quate causes of death, such as data of anamnesis and post-mortem examination are absent.

Sequence of stages of Examination at Sudden Death (preliminary points of actions)

1. Studying of official documents (including medical ones).
2. Plan of Autopsy.
3. Autopsy.
4. Performance of laboratorial and special researches.
5. Studying of the special literature.
6. Research of additional medical documents.
7. Formulation of the Diagnosis and Conclusions.

Causes of Sudden and Unexpected Death I. Diseases of Cardiovascular System (45 to 50%)

- 1) Coronary artery disease (narrowing and obliteration of the lumen by atherosclerosis).
- 2) Coronary atherosclerosis with coronary thrombosis.
- 3) Coronary atherosclerosis with haemorrhage in the wall causing occlusion of the lumen.
- 4) Coronary artery embolism.
- 5) Occlusion of the ostium of the coronary artery associated with atherosclerosis or syphilitic aortitis.
- 6) Rupture of a fresh myocardial infarct.
- 7) Cardiomyopathies.
- 8) Acute endocarditis, myocarditis or pericarditis.
- 9) Congenital heart disease in the newborn.
- 10) Lesions of the conducting system: fibrosis, necrosis.
- 11) Valvular lesions: aortic stenosis, aortic regurgitation, mitral stenosis, rupture of the chordae, ball-valve thrombus.
- 12) Angina pectoris.
- 13) Arterial hypertension with atherosclerosis.
- 14) Spontaneous rupture of aorta.
- 15) Rupture of aortic or other aneurysm.
- 16) Systemic embolism occurring in bacterial endocarditis.

II. Respiratory System (15 to 23%)

- 1) Lobar pneumonia.
- 2) Bronchitis and bronchopneumonia.
- 3) Lung abscess.
- 4) Massive collapse of the lung.
- 5) Acute oedema of the lungs.
- 6) Influenza.
- 7) Diphtheria.
- 8) Acute oedema of the glottis.
- 9) Neoplasm of the bronchus.
- 10) Impaction of foreign body in the larynx and regurgitation of stomach contents into air-passages and bronchioles.
- 11) Bronchial asthma.
- 12) Pleural effusion.
- 13) Pneumothorax.

- 14) Rupture of blood vessel in pulmonary tuberculosis.
- 15) Pulmonary embolism and infarction.
- 16) Air embolism.

III. Central Nervous System (10 to 18%)

- 1) Cerebral haemorrhage.
- 2) Cerebellar haemorrhage.
- 3) Pontine haemorrhage.
- 4) Subarachnoid haemorrhage.
- 5) Brain abscess.
- 6) Brain tumour.
- 7) Meningitis.
- 8) Acute polioencephalitis.
- 9) Cysts of third or fourth ventricle.
- 10) Epilepsy.
- 11) Cerebral thrombosis and embolism.
- 12) Carotid artery thrombosis.

IV. Alimentary System (6 to 8%)

- 1) Haemorrhage into the gastrointestinal tract from peptic ulcer, oesophageal varicose, cancer oesophagus, etc.
- 2) Perforation of ulcers.
- 3) Twisting and intussusceptions of the bowel.
- 4) Paralytic ileus.
- 5) Intestinal obstruction.
- 6) Strangulated hernia.
- 7) Appendicitis.
- 8) Bursting of the liver abscess.
- 9) Rupture of enlarged spleen.
- 10) Obstructive cholecystitis.
- 11) Acute hemorrhagic pancreatitis.

V. Genito-urinary System (3 to 5%)

- 1) Chronic nephritis.
- 2) Nephrolithiasis.
- 3) Obstructive hydronephrosis and pyonephrosis.
- 4) Tuberculosis of the kidney.
- 5) Tumours of the kidney or bladder.
- 6) Uterine haemorrhage due to fibroids.
- 7) Twisting of ovary, ovarian cyst or fibroid tumour.
- 8) Cancer vulva eroding femoral vessel.

VI. Miscellaneous (5 to 10%)

- 1) Diabetes mellitus.
- 2) Hyperthyroidism.
- 3) Addison's disease.
- 4) Blood dyscrasia.
- 5) Mismatched blood transfusion.
- 6) Haemochromatosis.
- 7) Cerebral malaria.
- 8) Shock due to emotional excitement.

9) Reflex vagal inhibition.

10) Anaphylaxis due to drugs.

At a number of diseases, sudden death comes only in adverse conditions.

Risk factors that may lead to sudden death

- Adverse meteorological conditions (sharp change of atmospheric pressure and/or air temperature);
- Physical overstrain (even insignificant) in patients having Ischaemic Heart Disease;
- Psychological emotional exposure, especially if unexpected.
- However, the most frequent risk factor is alcoholic intoxication and drinking, even in small doses, of ethanol. This, along with smoking, can lead to a spasm of the arteries of heart. For small toxic concentrations of alcohol, it is necessary to carry out differential diagnostics between sudden death from a disease with alcohol as the promoting factor, and poisoning with ethanol as an independent cause of a violent death.

Sudden deaths attributable to natural diseases comprise the bulk of the cases seen by forensic pathologists. A number of such deaths are considered for three main systems: cardiovascular, respiratory and central nervous system. In case of any suspicious or unnatural death, the significance of and any contribution from natural disease must be assessed.

Cardiovascular System

Coronary Artery Disease: Stenosis of the coronary arterial tree by atheroma is extremely common. The dangerous consequences are due to the effect of reduced blood flow to the myocardium, which can lead to sudden death in different ways.

Coronary insufficiency from narrowing of the lumen of major vessels may lead to chronic ischaemia and hypoxia of the muscle distal to the stenosis (Colour fig. 26). Hypoxic myocardium is electrically unstable and liable to arrhythmias and ventricular fibrillation, especially at moments of sudden stress such as exercise or adrenaline response to anger or another emotion. Ischaemia, without being severe enough, may produce a myocardial infarct, and if the ischaemic region includes one of the pace-making nodes or a major branch of the conducting system, the liability to rhythm abnormalities increases.

Complications of atheroma may worsen the coronary stenosis and subsequent myocardial ischaemia. Ulcerative atheromatous plaques may rupture, filling the vessel partially or completely with cholesterol, fat and fibrous debris. This may wash downstream in the coronary artery and impact distally at bifurcations, causing multiple mini-infarcts. The endothelial cap of a ruptured plaque may act as a valve within the vessel and cause a complete obstruction.

Mural thrombus on a plaque may further reduce the vessel lumen, without fully blocking the vessel.

Another common complication of atheroma is the sub-intimal haemorrhage, where bleeding occurs into a plaque, expanding it suddenly and often reducing or blocking the lumen.

Coronary thrombosis is over-diagnosed by clinicians as a cause of sudden death. Less than one third of sudden cardiac deaths reveal a coronary thrombus at autopsy, as pure stenosis and complications of atheroma are much more common. However, thrombi are still frequent and are often associated with a myocardial infarct. Multiple thrombi also occur, some being post-infarction, due to a stagnant circulation.

Myocardial Ischaemia is one of the commonest causes of death worldwide and is a direct result of impaired supply of oxygenated blood to the myocardium.

Myocardial Infarction occurs when a severe stenosis or a complete occlusion occurs in a coronary artery, if the collateral circulation is insufficient to maintain the muscle. Not every thrombus or complete occlusion leads to an infarction if blood can still reach all the myocardium by other routes, but if 70% or more of the lumen of a major branch is blocked, an infarct commonly occurs.

Myocardial infarction is the necrosis of myocardium caused by an inadequate supply of oxygenated blood to the myocardium. It is a consequence of sustained myocardial ischaemia.

Most myocardial infarctions occur in hearts supplied by arteries demonstrating severe complicated atheroma. Angiographic studies have indicated that thrombus developing on ulcerated atheromatous plaque is important in producing an acute diminution of blood supply in a coronary artery. Eccentric atheromatous plaques with abundant lipid are prone to rupture and, apart from overlying thrombus, they may demonstrate haemorrhage into the plaque. In most coronial deaths due to coronary artery atheroma (this term being synonymous with coronary atherosclerosis), recent myocardial infarction is not found and death is presumed to be due to an arrhythmia consequent upon myocardial ischaemia.

In sudden cardiac death, examination of the coronary arteries may reveal severe stenosis or thrombosis in a coronary artery. Infrequently, coronary artery dissection is found.

Lesions in the cardiac conducting system have been studied intensively in recent years, especially in relation to sudden death. Many abnormalities have been found, varying from extensive fibrosis to haemorrhage, tumours and infective lesions. However, it is always difficult to know whether such lesions are the cause of the fatal arrhythmia or merely an incidental finding.

The effect of a large infarction is either to reduce cardiac function because of "pump failure", as the dead muscle cannot contract; or to lead to arrhythmias and ventricular fibrillation, both because of its presence and because the adjacent muscle is inevitably ischaemic.

A visible infarction does not occur at the time of sudden death, as it takes many hours after coronary occlusion to become apparent. Neither does an infarction occur during exercise, as a coronary thrombosis usually develops during a period of slow, stagnant circulation. However, the fatal effects of an infarction may appear at any time after the muscle has become ischaemic.

A *ruptured myocardial infarction* may cause sudden death from a haemo-pericardium and cardiac tamponade. This is most common in old women, who have a soft, senile myocardium, but can occur in anyone. It tends to take place two or three days after the onset of the infarction, when the necrotic muscle is becoming soft. The rupture occasionally occurs through the interventricular septum, causing a left-right shunt. Over the inner surface of a myocardial infarction, mural thrombus may develop on the endocardium. Parts of this may break off, causing emboli in the systemic circulation, which in turn may cause infarctions in kidney, spleen and brain.

Myocardial fibrosis develops when a myocardial infarction heals (Colour fig. 24), as myocardial fibres cannot proliferate. Large plaques on the endocardium or in the wall of the ventricle or septum may later interfere with cardiac function or with the conduction system. Anyone with an appreciable fibrotic area is a candidate for sudden death. A large fibrotic area on the free wall of the left

ventricle may later swell due to the high pressure during systole, forming a "cardiac aneurysm", which may protrude into the pericardium and fill with laminated blood clot. These aneurysms rarely rupture, as they are tough and fibrotic.

Papillary muscle rupture can occur due to infarction and necrosis. This allows part of the mitral valve to prolapse, with signs of valve insufficiency and perhaps sudden death.

Aneurysm: An aneurysm is a localized area of ballooning in the wall of a vessel. Ruptured atheromatous aneurysms are a common cause of death in those parts of the world where ischaemic heart disease occurs. Atheromatous aneurysms usually occur below the diaphragm but infrequently they can be in the thorax, in its site they should be distinguished from syphilitic aneurysms.

Hypertensive Heart Disease: This condition may lead to sudden cardiac death from left ventricular hypertrophy. In hypertension, the myocardium has to work against increasing external pressure and enlarges to deliver more thrust. The upper limit of normal heart weight of about 400 g may increase to 600 g or more ("bovine heart") and the muscle mass thus outgrows its coronary supply, even if the coronary arteries are healthy. Atheroma is often associated with hypertension, so that the enlarged muscle mass is further deprived of even a normal flow and becomes ischaemic. In histochemical studies, a wide zone of the inner part of the hypertrophied left ventricular wall can be seen to be deficient in dehydrogenase enzymes, an indicator of ischaemic/hypoxic damage. Such muscle is unstable and irritable and easily jumps into arrhythmias and fibrillation.

Aortic Stenosis: This is similar to the last condition, in that it leads to left ventricular hypertrophy. This may be even more marked than in hypertension, with heart weights up to 700-1000 g. The cause is usually a calcific stenosis of the aortic valve, of uncertain aetiology, but probably related to atherosclerosis. It classically affects males over 60, but may be seen in younger people who have a congenital bicuspid aortic valve. The older victims most often have normal tricuspid valves.

Not only does the very large ventricle require more blood to supply its muscle but, in aortic stenosis, the problem is worsened by the narrow valve, which causes a low perfusion pressure at the coronary ostia, further reducing the coronary flow. Sudden death is common in such patients.

Myocarditis: This is much less common than the degenerative conditions described above. Myocarditis occurs in many infective diseases, such as diphtheria and virus infections, including influenza. Disseminated sarcoidosis can also affect the myocardium. They are usually the cause of death in such conditions. In sudden death pathology, a myocarditis of unknown aetiology is sometimes discovered on histology of autopsy tissues, known as "isolated Fiedler's or Saphir's" myocarditis. This condition is occasionally blamed for the death, usually in young adults, but some doubt has been cast on the reality of this cause, as control studies in the victims of traffic accidents where the cause of the accidents was known, also had a similar incidence of "myocarditis". It may therefore be an incidental finding in many cases.

A more definite intrinsic cardiac condition is the group known as the "cardiomyopathies", where a large heart shows certain abnormal histological characteristics. Some are due to metabolic defects, but the others are "idiopathic". Huge hearts of over 1000 g may be seen; massive thickening of the ventricular walls, sometimes asymmetric, can be seen in the hypertrophic type and dilatation of the chambers in the congestive type. All the myocardial conditions may be associated with sudden unexpected death.

Endocarditis: Microorganisms in the bloodstream may infect the heart valve to form vegetations. These friable vegetations may then break off and embolize to various sites, commonly brain, spleen or kidney. Endocarditis is an important complication of drug abuse.

Carcinomatous pericarditis. In such cases, the pericardial fluid is usually haemorrhagic.

Respiratory System

Asthma: In recent years, concern has been expressed about the high mortality due to asthma, particularly since many deaths might be avoided. Asthma is a disease characterized by an abrupt onset of narrowing of the bronchial airways. This narrowing can be caused by irritant substances being inhaled, by exposure to an allergen or by emotional stress. Asthma deaths are not uncommonly associated with drug abuse, especially when materials are smoked.

Pulmonary thrombo-embolism: This term describes occlusive blood clot in the pulmonary arteries, the blood clot usually embolic in origin. A deep venous thrombosis of the lower limbs is a common source of such emboli. Deep venous thrombosis seldom occurs without some predisposing event or underlying condition, such as myocardial infarction or immobility.

Infection: Infections range from an acute epiglottitis to a florid bronchopneumonia but may all kill quickly. An important public health exercise of the autopsy is to establish the identity of the causative organisms, although this may be difficult in the case of fragile organisms such as the meningococcus and also in the presence of decomposition.

Pneumothorax: This term describes the accumulation of gas, usually air, in the thoracic cavity. It is a complication of many respiratory diseases and also of chest trauma. The condition is easily missed at autopsy and is demonstrable by puncturing the thorax under a pocket of water and checking for the escape of bubbles.

In transportation accidents, there may be treatment of pneumothorax by the insertion of chest drains at hospital. In fatal cases, it is important not to confuse the drain wounds with stab wounds.

Central Nervous System

Subarachnoidalhaemorrhage: Non-traumatic subarachnoidalhaemorrhage is typically produced by rupture of an aneurysm, usually of berry type or, in older patients, of atheromatous origin. It is advisable to remove the blood from the fresh brain to aid identification of the aneurysm. Failure to find an aneurysm should prompt a search for evidence of a traumatic cause for the subarachnoidalhaemorrhage. This search should include examination of the neck in the region of the mastoid process and angiography or dissection of the vertebral arteries.

Epilepsy: Epilepsy is characterized by a low threshold for seizures. Hypoxic changes associated with seizures over a long period of time may be detected histologically in the hippocampi of the brain. Post-mortem examination may yield evidence of a bitten tongue and neuropathological abnormalities, but, not infrequently, the diagnosis may have to be made by exclusion of other causes of death. Histological examination of the tongue can effectively demonstrate recent bruising caused by biting and may also reveal evidence of previous such trauma. The tongue is sometimes clamped between the teeth in rigor mortis and this appearance should not be interpreted as evidence of epilepsy.

Cerebral haemorrhage: Such haemorrhages are usually immediately obvious in the fresh brain at autopsy, especially when the haemorrhage is in an important area such as the pons. Problems may arise in motor vehicle accidents when the haemorrhage may have preceded a fatal crash with severe head injury. Fixation of the brain and dissection by a neuropathologist may resolve some of these problems.

Colloid cyst of the third ventricle: This is a rare cause of sudden death which may be heralded by headaches. The cyst is an example of how a simple, non-malignant structure can kill because of its location in a crucial site. The cyst may be missed if the brain is sectioned in the fresh state.

Alimentary System

Once again, causes of sudden death may be vascular, in that very severe bleeding from a gastric or duodenal peptic ulcer can be fatal in a short time, even though most are moderate enough to allow medical or surgical treatment. Mesenteric thrombosis and embolism, leading to infarction of the gut are not sudden, but may be rapid and remain undiagnosed.

Perforation of a peptic ulcer can be fatal in hours if not treated and intestinal gangrene due to strangulated hernias and torsion due to peritoneal adhesions can also be fulminating and fatal conditions.

Genito-urinary System

A ruptured ectopic gestation, usually tubal in position, is another grave emergency that can end in death from intraperitoneal bleeding, unless rapidly treated by surgical intervention.

Human Immunodeficiency Virus

The human immunodeficiency virus (HIV) may be transmitted sexually or by contact with infected blood. About one-third of those infected develop symptoms in the early phase, often a fever with or without enlarged lymph nodes and a rash. There then follows a prolonged symptom-free stage. Most infected individuals advance to full-blown acquired immunodeficiency syndrome (AIDS) within 10 years but the range of time required to develop AIDS may be between 2 and 15 years. The immune devastation caused by the HIV virus is a direct consequence of its evolution in the body. The viral level rises gradually in parallel with a decline in the T-helper lymphocyte population.

The forensic pathologist considers all corpses to be potentially infective. Testing for HIV antibodies in our institution is limited to those bodies where consideration of the circumstances of death suggests that HTV-positivity is likely. These circumstances may also suggest the possibility of hepatitis viruses and other infections potentially hazardous to the pathologist.

Body fluids considered to transmit HIV include blood, semen, vaginal secretions, and cerebrospinal, peritoneal, amniotic, pericardial and synovial fluids. Other fluids are not implicated in the transmission of HIV unless they contain visible blood.

Sudden death in HIV infection is becoming increasingly recognized, especially in persons who have kept their infection secret. A knowledge of the huge range of clinical features demonstrated in HIV and AIDS may alert the pathologist to the diagnosis. In those people newly diagnosed as being HIV-positive, suicides have occurred where counselling services have been absent.

About 10% of patients with AIDS present because of neurological symptoms, but up to three-quarters of them will have evidence of neurological disease. This disease may be caused by a cryptococcal infection of the brain, by a direct effect of HIV on the brain or from other infective causes such as toxoplasmosis. Cryptococcus has a particular affinity for the meninges, where it causes gelatinous meningitis. Such brains tend to be very slippery when handled at post-mortem examination although the meningitis itself is very difficult to see macroscopically. Histological examination shows the organisms within mucoid pools. Cryptococcosis is believed to be the commonest type of cerebral mycosis in AIDS sufferers.

Autopsy in cases of AIDS and highly infectious diseases Highly infectious diseases transmitted by direct contact or contact with infected materials are tuberculosis, hepatitis B, C, diphtheria, meningitis, smallpox, cholera, rabies, tetanus, poliomyelitis, mumps, typhoid equine encephalomyelitis, HIV, etc. Ethically, doctor cannot refuse to handle such bodies on ground of risk involved.

There is a risk of transmission of HIV through needle prick injury during collection of blood and other body fluids, and mucosal splashes and skin contact with superficial injury during autopsy on a HTV infected dead body. Body fluids considered to transmit HTV include blood, semen, vaginal secretions, peritoneal, pericardial, amniotic and synovial fluids. HIV and hepatitis viruses are not associated with air-borne transmission.

Universal precautions do not apply to faeces, nasal secretions, sputum, sweat, tears, urine and vomitus unless they contain visible blood.

If the recommended guidelines are adhered to there is no risk to the staff conducting autopsies on AIDS patients. *Laboratory analyses*

1) *Histologically* pieces of various internal organs are examined routinely.

2) *Bacteriologic Examination:* All specimens must be collected under sterile conditions. Blood for culture must be obtained before organs are disturbed. After opening the pericardial sac, the anterior surface of the right ventricle is seared with heated knife, and 10 ml of blood aspirated using a sterile needle and syringe. The same technique may be used to remove material from other organs. The sample should be placed in sterile containers. The subarachnoid space is opened with a sterile scalpel and a specimen is taken from the space by sterile swabs from which smears and cultures may be made. The samples should be placed in sterile containers.

3) *Virological examination:* A piece of appropriate tissue is collected under sterile conditions and the sample is frozen or preserved in 80% glycerol in buffered saline. The specimen should be placed in a sterile container and sealed tightly.

Sudden Infant Death Syndrome (SIDS)

Also known as *Cot Death* or *Crib Death*, it is defined as the sudden and unexplained death of any infant who was either well, or almost well prior to death, and whose death remains unexplained even after a thorough autopsy including an investigation, and laboratorial examination if necessary.

Death usually occurs between the ages of two weeks to two years with peak around two months to four months, strikes boys somewhat more often than girls, is more common among low birth weight of child and among low income families, among children whose mothers smoke, or are drug addicts.

and is commonly associated with seasonal upper respiratory diseases, The incidence is about three fold among twins, most twins being premature and of low birth weight. About half of the victims have some symptoms of a cold during the week prior to death, and a few have a history of bowel upset.

Two clinical features of SIDS are nearly universal, viz., the babies die during sleep, and the death is silent. Most cases of so-called overlaying are actually victims of SIDS.

The diagnosis, by definition, is retrospective. The role of virus infection is obscure. Other possible causes, such as milk-allergy, parathyroid inadequacy, and adrenal insufficiency have not been substantiated, and the role of laryngospasm and cardiac arrhythmia has not been fully worked out.

The fact that the baby does not make a sound possibly indicates that laryngospasm may be the terminal event. Gentle nasal obstruction due to respiratory infection leads in some infants to cessation of respiration (apnoea) with no attempt at breathing through the mouth. This observation leads to the belief that quite trivial respiratory infection may trigger SIDS in an infant aged 2 months to 4 months.

At Autopsy milk or a bloodstained froth is sometimes seen on the child's mouth or bedding. The post-mortem findings are completely negative. Some pathological condition may be found, such as acute pneumonia, congenital heart disease, Down's syndrome or a tracheobronchitis. The only constant signs are multiple petechial haemorrhages on the visceral surfaces of the heart, lungs and thymus, which are agonal in nature, perhaps from terminal respiratory efforts against closed glottis. A small amount of milky vomit in the trachea and main bronchi and shedding of individual tracheobronchial epithelial cells are commonly found. Many infants show froth in the air passages and facial pallor. The lungs show patchy or uniform purplish discolouration of the surface and are firm in consistency with congestion, oedema and increase in weight.

Theories: There is no single cause for cot death, and death may result from a number of causes. The commonly accepted hypothesis suggests that some infants have prolonged "sleep apnoea" (a periodic failure to breathe during sleep), which makes them susceptible to hypoxia, which leads to bradycardia and cardiac arrest. Respiratory infection may produce a viraemia, which adds to the sleep depression of the respiratory centres. Nasal oedema and mucus secretion may further narrow the small upper respiratory passages and in some hypotonic babies, a flaccid pharynx and even neck posture may further reduce the airway. An element of laryngeal spasm has also been suggested. Other causes of death, which have been proposed, are: conduction system anomalies, hypersensitivity to cow's milk, parathyroid deficiency, selenium deficiency, vitamin E deficiency, antibody deficiency, suffocation by bed clothes and pillows, bacterial infection, neurogenic shock, hypogammaglobulinaemia, metabolic disorders, anaphylactic shock, etc.

Exercises for Student's Independent Work

TASK. Study circuits of research of medical documents for the typical pattern of "Protocol of Forensic Examination of a corpse". **Data from a medical card of the inpatient are researched in the following order:**

- date and time of hospitalization;
- conditions of transportation;

- medical manipulations before hospitalization;
- common condition of organism at hospitalization;
- initial description of injuries;
- dynamics of clinical symptoms;
- operations: time of carrying out, the description of character of injuries and operative intervention;
- objective manifestations and time of their appearance;
- character of conservative treatment: character, type and scope of blood-induced therapy, total scope of hormonal and antibacterial therapy;
- results of laboratorial researches in dynamics;
- dates of researches and results;
- rate and character of dying with the indication of character of loss of the basic vital functions (cardiovascular, respiratory, central nervous system, etc.);
- character of resuscitation actions;
- time of death;
- the final clinical diagnosis (in case of doubts in correctness and timeliness of diagnostics at the description of data from the case record, the full text and date of all diagnoses are researched; necessity of entering other data from the case record is conditioned by the purposes and tasks of concrete examination and concrete questions of the inspector).

Independent Work in Practical Class under Teacher's Supervision

Task 1. Post-mortem examination at mortuary. Students under supervision of a teacher carry out Autopsy in case of non-violent death: they must (1) study the resolution of inspector and other materials presented for expert examination (Protocol of inspection of a Scene of Incident, accompanying leaflet of ambulance, medical history, etc.), (2) clear up the problems that remained unsolved for inspector, (3) draw up a plan of autopsy, (4) carry out the external and internal examination of a corpse, (5) collect samples for additional investigations.

Task 2. After examination of the corpse and data collection, the students get acquainted with peculiarities of pathological conditions and changes of organs and tissues on macropreparations of the Department's museum.

On description of macropreparation, it is necessary to designate:

- name of a tissue or an organ;
- its appearance (colour, size, and consistency);
- character of its surface at section;
- presence of pathologic changes, deposits, haemorrhages, and other features.

Having finished the description of preparation, it is necessary to draw a brief conclusion about the character of revealed changes.

Group analysis of Task 2.

Situational Tasks

Instructions: study attentively the task content, make Forensic Medical Diagnosis, and draw up "Medical Death Certificate". **TASK1** On May 2 citizen P. aged 67 was found dead in his flat by his flatmate. It is known that on May 1, P. and his flatmate drank 0.5 litres of beer during supper, at about 19.00, and parted with each other.

On examination of the corpse at 12.00 on May 2, the investigator and the legal expert revealed that the corpse of a plump man, lying on a sofa, had no damages, the eyes were half-open, a small amount of foamed fluid of pale-rose colour was discharged from the nose and the mouth; discharges from the other natural openings were absent; livores mortis were of dark-blue colour, well-defined, after pressing with a thumb they disappeared and restored their colour in 6 minutes; rigor mortis was well pronounced in all the groups of muscles,

Autopsy has revealed (only Limited information is given) a small amount of atherosclerosis calcific plaques, some of them having pasty mass; in the left coronary artery at the distance of 2 cm from its point of origin there is a plaque that narrows the lumen by 75%, with dark-red colour deposits on the surface, not washed when water is applied; the myocardium has pale areas and areas of whitish colour of dense consistency; the thickness of the left ventricle is 1.8 cm, that of the right one is 0.6 cm; on the surface of the kidneys there are non-uniform[^] shaped scars of whitish dense tissue. Histological examination has revealed marked blood supply irregularity in myocardium, an extensive area of anuclear cardiac hystiocytes, areas of cardiac hystiocyte fragmentation and their crimp passage with contractures; a mural thrombus on an atherosclerosis plaque in the coronary artery that has features of petrification, the lumen of the artery is closed completely by a thrombus; a pronounced hyperaemia is observed in the lungs; hydropic fluid in combination with erythrocytes and brown pigment in the alveoli; a transparent fluid in the passages of the bronchial tubes; moderate vascular hyperaemia in the brain; perivascular and pericellular spaces are considerably enlarged. Forensic-toxicological examination of blood has revealed ethanol concentration of 0.3‰; urine does not contain alcohol.

TASK 2

On February 28, woman K. aged 25 gave birth to her baby (the 4-th pregnancy, the first labour; three previous pregnancies came to an end by spontaneous abortions). On March 3, K. felt general weakness, dizziness, experienced loss of consciousness, became pale, and perspired. Her relatives called an ambulance. An emergency doctor verified sudden death before arrival of the ambulance.

On March, 3, at 19.00, during post-mortem examination the inspector and the legal expert revealed a corpse of a well-nourished woman lying on a sofa, without visible damages; skin integuments were pale; the eyes were closed; discharges from natural openings were absent; livores mortis were ill-defined, with bluish tint, they disappeared after moderate thumb pressure and reappeared in 2 minutes.

Autopsy has revealed (only the most significant information is given) that the corpse of the woman is of proportional constitution and of good degree of nourishment; the hair growth on the abdomen is of virilizing type; the internal organs are pale; the large blood vessels are bloodless; up to 1000 ml of blood is accumulated in the abdominal cavity; the uterus is slightly enlarged; the right ovary is considerably enlarged in size (6 x 7 x 4 cm), there is an opening with diameter of up to 0.3 cm on its anterior surface;- a section of the ovary demonstrates a cavity filled with fluid and clotted blood. Histological examination has reveal haemorrhages in the stroma and the yellow body of the ovary that has not been subjected to involution.

TASK 3

On examination of a corpse of a 63-year man, who had suddenly died at his place of residence with the signs of impairment of respiration and heartbeat, the signs of fast death are found: plentiful livores mortis, cyanosis of the face, venous

hyperaemia of the internal organs, the pericardium filled with blood from a slit-like opening on the lateral wall of the left ventricle of heart, irregular blood filling in the cardiac muscle that spreads over the area around the rupture, the atherosclerotic plaques narrowing up to 2/3 the lumens of the coronary arteries, and multiple plaques with calcification and atheromatosis in the intima of aorta. The alcohol content in blood is 2.6 ‰; other poisons are not revealed.

TASK 4

Woman aged 25, a librarian, was found dead by her colleagues at the book depository at 13.00 on June 10. An emergency doctor verified death.

During post-mortem examination at 16.00 on June 10, the inspector and the legal expert revealed that a corpse of the woman was lying between the bookshelves; above the right eyebrow there was an abrasion of 5 x 2 cm in size with linear scratches located vertically. The woman's colleagues said that she had been registered at the polyclinic for a heart disease.

Autopsy has revealed (only limited information is given) that livores mortis of dark-blue colour are on the posterior part of the body; rigor mortis is present in all groups of muscles. Oedema of the brain and lungs is observed place. The heart is of a slightly rounded shape; its mass is 350 g; the thickness of the walls of the left ventricle is 1.5 cm, that of the right one is 0.3 cm; the thickness of interventricular septum is 2.0 cm; the volume of the heart cavity is diminished; musculus papillaris anterior is shifted upwards; endocardium is thickened under the aortic valve. Histological examination has revealed impairment of mutual orientation of the muscular fibrils in the cardiac muscle; the fibrils themselves are hypertrophic; their nuclei have changed and have a perinuclear aureole; their length is diminished as a result of marginal fibrosis. Pathologic changes in the vessels are absent.

TASK 5

Citizen I. aged 53, suddenly died at his place of residence.

Autopsy has revealed that the heart is of spherical shape 15 x 14 x 12 cm in size; the right and the left venous foramina can pass three fingers. On section, the cardiac muscle is non-uniformly filled with blood, with multiple small whitish interlayers; the coronary arteries are wide with atherosclerotic plaques; the consistence of the cardiac muscle is compact. The fibres of the heart valves have changed; the tendinous fibres are contracted. The thickness of the cardiac muscle of the right ventricle is 0.8 cm that of the left one is 1.6 cm. The kidneys are of 9 x 5 x 4 cm in size; the fibrous capsule can be easily dismantled; the surface of the kidneys is fine-grained with multitude star-shaped retractions.

Histological examination reveals the following: non-uniform filling with blood in the myocardium; moderate excrescence of connective tissue is noted around the vessels; oedema of the stroma; albuminous degeneration of the myocardium; cardiac hystiocytes are hypertrophic; the nuclei are hyperchromic; marginal fibrosis; median fragmentation of the muscular fibres; arterio-arteriolonephrosclerosis. Any other changes are not revealed.

TASK 6

A corpse of citizen M. aged 70, who had suddenly died at his place of residence, was subjected to autopsy.

External and internal examinations do not reveal any peculiarities, except for the following: the heart is of spherical shape, 12 x 12 x 9 cm in size; the right and left venous foramina can pass three fingers. On section, the cardiac muscle is non-uniformly filled with blood, with multiple small whitish interlayers. The coronary arteries of the heart are narrowed in the proximal portions by 2/3.

The cardiac muscle is of flaccid consistence. The fibres of the heart valves have changed. The thickness of the cardiac muscle of the left ventricle is 1.3 cm that of the right one is 0.4 cm. The aorta is sclerosed.

Histological examination of the cardiac muscle reveals non-uniform filling with blood in the myocardium with predominance of venous hyperaemia; intramural arteries of the heart are sclerosed. Oedema of the stroma; albuminous degeneration of the myocardium; irregular hypertrophy and fragmentation of cardiac myocytes are observed.

TASK 7

A corpse of citizen L. aged 30, who had been found dead in his place of residence, was subjected to autopsy.

External and internal examinations have not revealed any peculiarities, except for the following: the lungs are of pasty consistence; there are multiple small haemorrhages of red colour; on section the pulmonary tissue is plethoric; liquid dark blood of foamy character flows from the surface of section.

The heart is conical in shape, of 12 x 10 x 8 cm in size; the cardiac muscle on sections is plethoric, of dark-brown colour, with solitary small whitish interlayer of compact consistency. On section the coronary arteries gape; there are flat small plaques in the lumen, which do not narrow it. The thickness of the cardiac muscle of the right ventricle is 0.5 cm that of the left one is 1.3 cm. The valves have not changed. The aorta contains lipomatous sites.

Histological examination reveals vesicular emphysema in the lungs; marked hyperaemia, alveolar oedema, non-uniform filling with blood with predominance of venous hyperaemia, oedema of the stroma, moderate excrescence of connective tissue is noted around the vessels; albuminous degeneration of the myocardium is found in the heart.

TASK 8

A woman suffering from thrombophlebitis of the deep veins of the crus suddenly died in a shop. In the main trunk and at the point of bifurcation of the pulmonary artery a medico-legal expert has revealed freely lying red loose masses with lustreless corrugated surface,.

TASK 9

On medico-legal examination of the corpse of a man aged 63, in the myocardium of the anterior wall of the left ventricle of the heart a focus with distinct bounds of fibrous structure, of irregular shape, grey in colour, 5 x 4 cm in size, is revealed.

TASK 10

A man aged 64, who had survived infarction, died 3 weeks later with the diagnosis of acute cardiac insufficiency. Post-mortem examination revealed infarction at the stage of formation, and fresh infarction.

TASK 11

Medico-legal examination of a corpse has revealed an enlargement of the entire right lung; its thickness is of that of the liver; on section the tissue is of grey colour, a cloudy fluid is seen on its surface; fibrin deposits are observed on the pleura.

TASK 12

A man, who suffered with urolithiasis, has died due to uraemia. Autopsy reveals an enlargement of the right kidney, its parenchyma has been thinned; the pelvis and calices have extended, filled with fluid. In the orifice of ureter there is a calculus.

TASK 13

A 7 year-old child died suddenly at home. On autopsy, the pathologist has found in the both lungs oedema, small foci of emphysema, atelectasis, haemorrhages, and inflammation without pus ("mottled lungs").

TASK 14

A 52-year-old man died because of pulmonary-cardiac insufficiency. On autopsy, the pathologist has found croupous pneumonia in the lower lobe of the left lung, 400 ml of greenish-yellow fluid in the left pleural cavity. Microscopically, it contains many neutrophils.

TASK 15

A 49 year-old man died because of pulmonary-cardiac insufficiency. On autopsy, the pathologist has found that the upper lobe of the right lung is red and dense as the liver, white fibrin is observed on pleura. Microscopic investigation reveals exudate of fibrin, a lot of erythrocytes and a few leukocytes.

TASK 16

On December 23 at 14.00 infant Eduard N. aged 6 months was found dead by his mother in the bed in 2 hours after she had fed him and had put him to bed. An emergency doctor verified death. From medical documents it follows that: The child was born from the first pregnancy. During the second half of pregnancy moderate gestosis and intranatal foetal hypoxia took place. Medical examination of the pregnant woman revealed an enlargement of Q-T interval in electrocardiogram. It was on December 12, when the pediatrician examined the infant and considered him to be in good health. During autopsy the forensic expert revealed that the corpse of the infant was without any signs of injuries, with marked livores mortis on the back, with marked signs of rigor mortis in all groups of muscles. What is the supposed cause of death?

TASK 17

The corpse of an infant at the age of 3 months was found in his bed; a pillow lied beside. Livores mortis of blue-violet colour are located on the posteriolateral surfaces of the body; punctuated haemorrhages to the palpebral conjunctiva; haemorrhages of dark-red colour to the mucous membrane of the upper and lower lips. Internal examination has revealed hyperaemia and oedema of the vocal cords, punctuated haemorrhages to the larynx; the signs of asphyxia death. What is the supposed cause of death?