MINISTRY OF PUBLIC HEALTH OF UKRAINE
ZAPOROZHE STATE MEDICAL UNIVERSITY
DEPARTMENT OF GENERAL PRACTICE – FAMILY MEDICINE

EMERGENCY IN THE PRACTICE OF FAMILY DOCTOR

THE TEXTBOOK FOR THE PRACTICAL CLASSES AND INDIVIDUAL WORK FOR 6TH-YEARS STUDENTS OF INTERNATIONAL FACULTY (SPECIALITY «GENERAL MEDICINE»)
DISCIPLINE: «GENERAL PRACTICE – FAMILY MEDICINE»

Content module 3

Zaporozhye, 2015

Approved by:
MoH of Ukraine protocol № 2, the 16.06.2015 y.

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Methodical recommendations compiled in accordance with the program of «General practice - family medicine». Guidelines are intended to help students prepare for practical classes and learn the material. Can be used for training of 6th-years students of international faculty, discipline «General practice - family medicine».

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Publishing office of ZSMU

CONTENT

PREFACE

The family doctor has to provide emergency in the case of different life-threatening conditions: cardiac arrest, respiratory standstill, bronchoobstructive syndrome, hypertensic crisis, and pain. The timely diagnostics and ability to provides emergency at the pre-admission stage by family doctor, during transportation of the patient prevents complications, improves the treatment outcomes.

This textbook includes the general principles and algorithms of emergency in the practice of family doctor in the case of pain.

The necessity of this textbook is conditioned by absence of such workbooks, which satisfy requirements of basic parts of academic discipline «General practice – family medicine».

This textbook is recommended for 6^{th}-years students of Institute of higher education III-IV level of accreditation, speciality 7.12010001 «General medicine», training line 1201 «Medicine» studying related topics, interns, general practitioners, other specialists.

THE THEMATIC PLAN OF PRACTICAL CLASSES

Module 1: «The organizational aspects of the system of the primary health care in Ukraine, its role in the development and reforming of the Public health».

№	*Topic*	*Number of hours*
Content module 1. ***Modern approaches to the medico-social and organizational basis of a primary health care***		
1	The place of the family medicine in the structure of a healthcare system and the principles of the family service. The organization of the FD's work. The basis recording documentation of FD in medical institution. The role of information system in FD practice. The basis of information processing of out-patient clinic.	7
Content module 2. ***Medico-social aspects of population's health - the basis of the preventive and curing medicine***		
2	Medico-social aspects of the population's health. The medical examination of the population, and rehabilitation in the family doctor's practice. Medical insurance structure and family doctor activity. The models of medical insurance in the world.	6
3	3.1. The assessment of the risk factors of the main chronic non-epidemic diseases and the preventive measures in case of the cardiovascular, bronchopulmonary, gastrointestinal diseases and some other common syndromes. A role of family doctor in popularization of healthy life style and prophylaxis. The dietotherapy. "The health school". 3.2. The prophylaxis of AIDS.	4 2
4	4.1. The organization of out-of-hospital therapeutic help in case of the most wide-spread diseases. The principles of medico-social expertise. The organization of the day hospital and home care. 4.2. The consultation in the context of HIV-infectious, voluntary testing. 4.3. The consultation in the context of incurable disease and imminent death. The organization of medical care for non-curable patients. The principle of multidisciplinary approach to medical care of non-curable patients and their relatives. Nursing, the methods of palliative care of symptoms and syndromes.	2 2 2
Content module 3. ***The emergency in the family doctor's practice.***		

№	*Topic*	*Number of hours*
5	The emergency in the practice of family doctor. The emergency in the pre-hospital stage in the case of cardiac arrest, acute coronary syndrome, respiratory standstill, arrhythmias, hypertensic crisis, bronchoobstructive syndrome.	6
6	6.1.The emergency in the practice of family doctor in the case of pain syndrome. 6.2.The clinical classification of pain. The mechanism of pain in incurable patient. The principles of treatment of chronic pain syndrome. The emergency in context of incurable diseases and imminent death.	4 2
7	The emergency in the practice of family doctor in the case of seizure, syncope, coma in case of diabetes, acute hepatic failure, alcohol intoxication, renal insufficiency, narcotic abuse.	6
8	The emergency in the practice of family doctor in the case of bite, sting, electrical injury, drowning, frostbite and thermal injury. Final module control.	5 2
	TOTAL	50

THE THEMATIC PLAN OF INDEPENDENT WORK OF STUDENT

№	*Topic*	*Number of hours*	*Control type*
1.	Preparation to the practical classes, the academic level and training of practical skills	20	Current control during practical classes
2.	The implementation and defense of the practical tasks	4	
3.	Filling of the family doctor's documentation	3	
4.	The preparation and writing of the program of treatment in out-patient in the case of most widespread diseases	4	
5.	Drew up the algorithms of the pre-admission emergency in the family doctor's practice	4	
6.	The report at a clinical conference of hospitals.	1	
7.	Preparation for the final module control	4	Final module control
	Total	40	

TOPIC 5

The emergency in the practice of family doctor. The emergency in the pre-hospital stage in the case of cardiac arrest, acute coronary syndrome, respiratory standstill, arrhythmias, hypertensic crisis, bronchoobstructive syndrome.

I. Theme actuality

According to official data, sudden death cases are about 10% among all reasons of death. In developed countries every year dies suddenly a lot of people: in the U.S. – 330 000, in Russia -300 000 persons. According to U.S. statistics, 250 000 cases of sudden death occurring at home, at work, in urban centers and only 80 000 cases - in hospitals. It was found that leading cause of sudden death is ventricular fibrillation.

II. Study purposes: to know clinical presentations of sudden death and the first aid on the pre-admission stage.

III. Concrete purposes of the module:

- To know the organization and content of out-patient clinic;
- To identify diseases that require emergency care;
- To be able to assess the state of patient and provide appropriate medical care in the case of major syndromes which requiring urgent medical assistance of family doctor;
- To be able to perform cardiopulmonary resuscitation: the restoration of patency of airways, to correct arrhythmias, defibrillation.

IV. A student must be able:

- To know the basic diseases that may complicate the development of sudden death;
- To know the principles of restoration of passable respiratory tract;
- To know the technique of closed-chest cardiac massage;
- To explain the mechanism of respiration;
- To know the algorithm of defibrillation.

V. Task for initial independent training

1. At the first defibrillation attempt in adult the electric discharge is:

A. 400 J
B. 250 J
C. 200 J
D. 360 J
E. 300 J

2. The most effective method of urgent ventilation is:

A. Rhythmical chest compression
B. mouth-to-mouth method
C. Sylvester's method
D. Holger-Nielsen's method
E. Ambu's bag

3. After a primary cardiac arrest consciousness disappears through:

A. 10-15 sec
B. 2 min.
C. 15-20 sec
D. 1 min.
E. 30 sec

4. The most common cause of sudden death is:

A. Hypertensive crisis
B. central nervous system lesion
C. Acute hemorrhage
D. Disease of cardiovascular system
E. Cancer

5. What is the primary symptom for diagnose the circulatory arrest?

A. Convulsion
B. Absence of heart sounds by auscultation
C. Absence of carotid pulsation
D. midriatic pupils
E. Absence of spontaneous breathing

6. During cardiopulmonary resuscitation the electrical defibrillation is shown by:

A. izoline
B. irregular waves
C. nodal rhythm
D. Always circulatory arrest
E. sinus bradycardia

7. Asystole is the same as:

A. Absence of systolic blood pressure

B. Collapse

C. complete atrioventricular heart block

D. Cardiac arrest

E. Syncope

8. What is the most frequent complication of resuscitation measures in elderly patients?

A. hepatorrhexis

B. Spine Fractures

C. Gaps lung

D. jawfall

E. Fractures of ribs and sternum

9. More often sudden death occurs as a result:

A. The asystole

B. development of atrioventricular dissociation

C. ventricular fibrillation

D. atrial fibrillation

E. Ventricular tachycardia

10. Identify the share of cases without morphologic signs in heart in the case of sudden death

A. 5-7%

B. 10-15%

C. 20-26%

D. 12%

E. 20%

Answers:

1	2	3	4	5	6	7	8	9	10
C	B	A	D	C	B	D	E	C	D

VI. Basic questions after a theme

Basic principles of emergency providing in the rural and urban area, the role of FD.

The importance of timely medical care to reduce mortality and disability.

The share of sudden death in the mortality structure.

The causes of sudden death. The types of circulatory arrest. Supplemental investigation for determine types of circulatory arrest.

Methodology of emergency care in the case of circulatory arrest, respiratory standstill, traumatic death.

Independent work: preparation to practical studies - 4 hours.

VII. The plan and organizational structure of practical training.

№	Stage	Hours, min	Educational materials		Place of realization of studies
			Facilities of studies	Equipment	
1	Control of initial level	15 min	Tests		Classroom
2	Analysis of theme	90 min	Oral test		Classroom
3	Practical work	115 min	Out-patients case record		Family out-patient's clinic
4	Current control of knowledge	15 min	Situational tasks		Classroom
5	Summation of studies	5 min			Classroom
6	Independent work	4 hours	The individual preparation to practical studies		Classroom

VIII. The content of theme

Cardiovascular collapse, cardiac arrest, and sudden cardiac death

The vast majority of naturally occurring sudden deaths are caused by cardiac disorders. The magnitude of the problem of *cardiac* causes is highlighted by estimates that more than 300,000 sudden cardiac deaths (SCD) occur each year in the U.S., as many as 50% of all cardiac deaths. SCD is a direct consequence of cardiac arrest, which is often reversible if responded to promptly. Since resuscitation techniques and emergency rescue systems are available to save patients who have out-of-hospital

cardiac arrest, which was uniformly fatal in the past, understanding the SCD problem has practical importance.

SCD must be defined carefully. In the context of time, «sudden» is defined, for most clinical and epidemiologic purposes, as 1 h or less between the onset of the terminal clinical event and death. An exception is unwitnessed deaths in which pathologists may expand the definition of time to 24 h after the victim was last seen to be alive and stable [9].

Because of community-based interventions, victims may remain biologically alive for days or weeks after a cardiac arrest that has resulted in irreversible central nervous system damage. Confusion in terms can be avoided by adhering strictly to definitions of death, cardiac arrest, and cardiovascular collapse, as outlined in Table 1. Death is biologically, legally, and literally an absolute and irreversible event. Death may be delayed in a survivor of cardiac arrest, but "survival after sudden death" is contradictory. Currently, the accepted definition of SCD is *natural death due to cardiac causes,* heralded by abrupt loss of consciousness within *1 h* of the onset of acute symptoms, in an individual who may have known *preexisting* heart

- disease but in whom the *time* and *mode* of death are *unexpected.* When biologic death of the cardiac arrest victim is delayed because;

interventions, the relevant pathophysiologic event remains the sudden and unexpected cardiac arrest that leads ultimately to death, even though delayed by artificial methods. The language used should reflect the fact that the index event was a cardiac arrest and that death was due to its delayed consequences [15].

Etiology, initiating events, and clinical epidemiology

Extensive epidemiologic studies have identified populations at high risk for SCD. In addition, a large body of pathologic data provides information on the underlying *structural abnormalities* in victims of SCD, and clinical/physiologic studies have begun to identify a group of *transient functional factors* that may convert a long-standing under-lying structural abnormality from a stable to an unstable state (Table 2). This information is developing into an understanding of the causes and mechanisms of SCD.

Cardiac disorders constitute the most common causes of sudden *natural* death. After an initial peak incidence of sudden death between birth and 6 months of age (the sudden infant death syndrome), the incidence of sudden death declines sharply and remains low through childhood and adolescence. The incidence begins to increase in young adults, reaching a second peak in the age range of 45 to 75 years. Increasing age in this range is a powerful risk factor for sudden *cardiac* death, and the proportion of

cardiac causes among all sudden natural deaths increases dramatically with advancing years. From 1 to 13 years of age, only one of five sudden *natural* deaths is due to cardiac causes. Between 14 and 21 years of age, the proportion increases to 30%, and then to 88% in the middle-aged and elderly [7,10].

Young and middle-aged men and women have very different susceptibilities to SCD, but the gender differences decrease with advancing age.

Table 1

Distinction Between Death, Cardiac Arrest, and Cardiovascular Collapse

Term	Definition	Qualifiers or Exeptoins
Death	Irreversible cessation of all biologic functions	None
Cardiac arrest	Abrupt cessation of cardiac pump function which may be reversible by a prompt intervention but will lead to death in its absence	Rare spontaneous reversions; likelihood of successful interventions; relates to mechanism of arrest and clinical setting
Cardiovascular collapse	A sudden loss of effective blood flow due to cardiac and/or peripheral vascular factors which may reverse spontaneously (e.g., neuro-cardiogenic syncope; vasovagal syncope) or only with interventions (e.g., cardiac arrest)	Nonspecific term which includes cardiac arrest and its consequences and also events which characteristically revert spontaneously

The overall male/female ratio is approximately 4:1, but in 45 to 64-year-old age group, the male SCD excess is nearly 7:1. It falls to approximately 2:1 in the 65- to 74-year-old age group. The difference in risk for SCD parallels the risks for other manifestations of coronary heart disease (CHD) in men and women. As the gap for other manifestations of coronary heart disease closes in the seventh and eighth decades of life, the excess risk of SCD also narrows. Despite the lower incidence in women, the classic coronary risk factors still operate in women – cigarette smoking, diabetes, hyperlipidemia, hypertension – and SDC remains an important clinical and epidemiologic problem [7,8].

Hereditary factors contribute to the risk of SCD, but largely in a nonspecific manner: they represent expressions of the hereditary predisposition to CHD. Except for a few specific syndromes, such as the genetic hyperlipoproteinemias, congenital long

QT interval syndromes, and a number of myopathic and dysplastic syndromes there are no *specific* hereditary risk factors for SCD.

The major categories of structural causes of, and functional factors contributing to, the SCD syndrome are listed in Table 2. Worldwide, and especially in western cultures, coronary atherosclerotic heart disease is the most common structural abnormality associated with SCD. Up to 80% of all SCDs in the U.S. are due to the consequences of coronary atherosclerosis. The cardiomyopathies (dilated and hypertrophic, collectively) account for another 10 to 15% of SCDs, and all the remaining diverse etiologies cause only 5 to 10% of these events. Transient ischemia in the previously scarred or hypertrophied heart, hemodynamic and fluid and electrolyte disturbances, fluctuations in autonomic nervous system activity, and transient electrophysiologic changes caused by drugs or other chemicals (e.g., proarrhythmia) have all been implicated as mechanisms responsible for transition from electrophysiologic stability to instability. In addition, spontaneous reperfusion of ischemic myocardium, caused by vasomotor changes in the coronary vasculature and/ or spontaneous thrombolysis, may cause transient electrophysiologic instability and arrhythmias [16].

Pathology. Data from postmortem examinations of SCD victims parallel the clinical observations on the prevalence of CHD as the major structural etiologic factor. More than 80% of SCD victims have pathologic findings of CHD. The pathologic description often includes a combination of long-standing, extensive atherosclerosis of the epicardial coronary arteries and acute active coronary lesions, which include a combination of fissured or ruptured plaques, platelet aggregates, hemorrhage, and thombosis. In one study, chronic coronary atherosclerosis involving two or more major vessels with >75% stenosis was observed in 75% of the victims. In another study, atherosclerotic plaque assuring, platelet aggregates, and/or acute thrombosis were observed in 95 of 100 individuals who had pathologic studies after SCD. Most of these acute changes were superimposed on preexisting chronic lesions.

As many as 70 to 75% of males who die suddenly have prior myocardial infarctions (Mis), but only 20 to 30% have recent acute Mis. A high incidence of left ventricular (LV) hypertrophy coexists with prior Mis [7,12].

Clinical definition of forms of cardiovascular collapse (Table 1).

Cardiovascular collapse is a general term connoting loss of effective blood flow due to acute dysfunction of the heart and/or peripheral vasculature. Cardiovascular collapse may be caused by vasodepressor syncope (vasovagal syncope, postural

hypotension with syncope, neurocardiogenic syncope), a transient severe bradycardia, or cardiac arrest. The latter is distinguished from the transient forms of cardiovascular collapse in that it usually requires an intervention to achieve resuscitation. In contrast, vasodepressor syncope and many primary bradyarrhythmic syncopal events are transient and non-life-threatening, and the patient will regain consciousness spontaneously [15,16].

The most common electrical mechanism for true cardiac arrest is ventricular fibrillation (VF), which is responsible for 65 to 80% of cardiac arrests. Severe persistent bradyarrhythmias, asystole, and pulseless electrical activity (an organized electrical activity without mechanical response - formerly called electomechanical dissociation) cause another 20 to 30%. Sustained ventricular tachycardia (VT) with hypotension is a less common cause. Acute low cardiac output states, having precipitous onset, also may present clinically as a cardiac arrest. The causes include massive acute pulmonary emboli, internal blood loss from ruptured aortic aneurysm, intense anaphylaxis, cardiac rupture after myocardial infarction, and unexpected fatal arrhythmia due to electrolyte disturbances.

Table 2

Cardiac Arrest and Sudden Cardiac Death

Structural causes

I. Coronary heart disease
  A. Coronary artery abnormalities
    1. Chronic atherosclerotic lesions
    2. Acute (active) lesions
       (plaque assuring, platelet aggregation, acute thrombosis)
    3. Anomalous coronary artery anatomy
  B. Myocardial infarction
    1. Healed
    2. Acute
II. Myocardial hypertrophy
  A. Secondary
  B. Hypertrophic cardiomyopathy
    1. Obstructive
    2. Nonobstructive
III. Dilated cardiomyopathy - primary muscle disease
IV. Inflammatory and infiltrative disorders

A. Myocarditis
B. Noninfectious inflammatory diseases
C. Infiltrative diseases

V. Valvular heart disease

VI. Electrophysiologic abnormalities, structural
A. Anomalous pathways in Wolff-Parkinson-White syndrome
B. Conducting system disease
C. Membrane channel structure (e.g., congenital long QT syndrome)

Functional contributing factors

I. Alterations of coronary blood flow
A. Transient ischemia
B. Reperfusion after ischemia

II. Low cardiac output states
A. Heart failure
1. Chronic
2. Acute decompensation
B. Shock

III. Systemic metabolic abnormalities
A. Electrolyte imbalance (e.g., hypokalemia)

IV. Neurophysiologic disturbances
A. Autonomic fluctuations: central, neural, humoral
B. Receptor function

V. Toxic responses
A. Proarrhythmic drug effects
B. Cardiac toxins (e.g., cocaine, digitalis intoxication)
C. Drug interactions

Clinical characteristics of cardiac arrest

Prodrome, onset, arrest, death. SCD may be presaged by days, weeks, or months of increasing angina, dyspnea, palpitations, easy fatigability, and other nonspecific complaints. However, these *prodromal complaints* are generally predictive of any major cardiac event; they are not specific for predicting SCD [7,17].

The *onset of the terminal event,* leading to cardiac arrest, is defined as an acute change in cardiovascular status preceding cardiac arrest by up to 1 h. When the onset is instantaneous or abrupt, the probability that the arrest is cardiac in origin is >95%.

Continuous ECG recordings, fortuitously obtained at the onset of a cardiac arrest, commonly demonstrate changes in cardiac electrical activity in the minutes or hours before the event. There is a tendency for the heart rate to increase and for advanced grades of premature ventricular contractions (PVCs) to evolve. Most cardiac arrests that occur by the mechanism of VF begin with a run of sustained or nonsustained VT, which then degenerates into VF.

Sudden unexpected loss of effective circulation may be separated into «arrhythmic events» and «circulatory failure». Arrhythmic events are characterized by a high likelihood of patients being awake and active immediately prior to the event, are dominated by VF as the electrical mechanism, and have a short duration of terminal illness (<1 h). In contrast, circulatory failure deaths occur in patients who are inactive or comatose, have a higher incidence of asystole than VF, have a tendency to a longer duration of terminal illness, and are dominated by noncardiac events preceding the terminal illness [9].

The onset of cardiac arrest may be characterized by typical symptoms of an acute cardiac event, such as prolonged angina or the pain of onset of MI, acute dyspnea or orthopnea, or the sudden onset of palpitations, sustained tachycardia, or light-headedness. However, in many patients, the onset is precipitous, without forewarning.

Cardiac arrest is, by definition, abrupt. Mentation may be impaired in patients with sustained VT during the onset of the terminal event. However, complete loss of consciousness is a *sine qua non* in cardiac arrest. Although rare spontaneous reversions occur, it is usual that cardiac arrest progresses to death within minutes (i.e., SCD has occurred) if active interventions are not undertaken promptly.

The ability to resuscitate the victim of cardiac arrest is related to the time from onset to institution of resuscitative efforts, the setting in which the event occurs, the mechanism (VF, VT, pulseless electrical activity, asystole), and the clinical status of the patient prior to the cardiac arrest. Those settings in which it is possible to institute prompt cardiopulmonary resuscitation (CPR) provide a better chance of a successful outcome. The outcome in intensive care units and other in-hospital environments is heavily influenced by the patient's preceding clinical status. The immediate outcome is good for cardiac arrest occurring in the intensive care unit in the presence of an acute cardiac event or transient metabolic disturbance, but the outcome for patients with far-advanced chronic cardiac disease or advanced noncardiac diseases (e.g., renal failure, pneumonia, sepsis, diabetes, cancer) is no more successful in hospital than in the out-of-hospital setting [10].

The success rate for initial resuscitation and ultimate survival from an out-of-hospital cardiac arrest depends in part on the mechanism of the event. When the mechanism is VT, the outcome is best (67%); VF is the next most successful (25%), and asystole and pulseless electrical activity generate dismal outcome statistics. Advanced age also influences adversely the chances of successful resuscitation.

Progression to biologic death is a function of the mechanism of cardiac arrest and the length of the delay before interventions. VF or asystole, without CPR within the first 4 to 6 min, has a poor outcome, and there are few survivors among patients who had no life-support activities for the first 8 min after onset. Outcome statistics are improved considerably by lay bystander intervention (basic life support) prior to definitive interventions (advanced life support - defibrillation) and by early defibrillation. Death during the hospitalization after a successfully resuscitated cardiac arrest relates closely to the severity of central nervous system injury. Anoxic encephalopathy and infections subsequent to prolonged respirator dependence account for 60% of the deaths. Another 30% occur as a consequence of low cardiac output states that fail to respond to interventions. Paradoxically, recurrent arrhythmias are the least common cause of death, accounting for only 10% of in-hospital deaths [8-11].

Among patients who have cardiac arrest in the setting of acute MI, it is important to distinguish between primary and secondary cardiac arrests. *Primary* cardiac arrests refer to those that occur in the absence of hemodynamic instability, and *secondary* cardiac arrest are those that occur in patients in whom abnormal hemodynamics dominate the clinical picture before cardiac arrest. The success rate for immediate resuscitation in primary cardiac arrest during acute MP in a monitored setting should approach 100%. In contrast, as many as 70% of patients with secondary cardiac arrest succumb immediately or during the same hospitalization.

Identification of patients at risk for sudden cardiac death. Primary prevention of cardiac arrest depends on the ability to identify individual patients at high risk. One must view the problem in the context of the total number of events and the population pools from which they are derived. The annual incidence of SCD among an unselected adult population is 1 to 2 per 1000 population, largely reflecting the prevalence of those CHD patients among whom SCD is the first clinically recognized manifestation (20 to 25% of first coronary events are SCD). The incidence (percent per year) increases progressively with addition of identified coronary risk factors to populations free of prior coronary events. The most powerful factors are age, elevated blood pressure, LV hypertrophy, cigarette smoking, elevated serum cholesterol level, obesity, and

nonspecific electrocardiographic abnormalities. These coronary risk factors are not specific for SCD but rather represent increasing risk for all coronary deaths. The proportion of coronary deaths that are sudden remains at approximately 50% in all risk categories. Despite the marked *relative* increased risk of SCD with addition of multiple risk factors (from 1 to 2 per 1000 population per year in an unselected population to as much as 50 to 60 per 1000 in subgroups having multiple risk factors for coronary artery disease), the *absolute* incidence remains relatively low when viewed as the relationship between the number of individuals who have a preventive intervention and the number of events that can be prevented. Specifically, a 50% reduction in annual SCD risk would be a huge *relative* decrease but would require an intervention in up to 200 unselected individuals to prevent one sudden death. These figures highlight the importance of primary prevention of CHD. Control of coronary risk factors may be the only practical method to prevent SCD in major segments of the population, because of the paradox that the majority of events occur in the large unselected subgroups rather than in the specific high-risk subgroups. Under most conditions of higher level of risk, particularly those indexed to a recent major cardiovascular event (e.g., MI, recent onset of heart failure, survival after out-of-hospital cardiac arrest), the highest risk of sudden death occurs within the initial 6 to 18 months and then decreases toward baseline risk of the underlying disease. Accordingly, preventive interventions are most likely to be effective when initiated early [7-9].

For patients with acute or prior clinical manifestations of CHD, high-risk subgroups having a much higher ratio of SCD risk to population base can be identified. The acute, convalescent, and chronic phases of MI provide large population subsets with more highly focused risk. The potential risk of cardiac arrest from the onset through the first 72 h after acute MI (the acute phase) may be as high as 15 to 20%. The highest risk of SCD in relation to MI is found in the subgroup that has experienced sustained VT or VF during the convalescent phase (3 days to 8 weeks) after MI. A greater than 50% mortality in 6 to 12 months has been observed among these patients, when managed with conservative medical therapy, and at least 50% of the deaths are sudden. Since the development of aggressive intervention techniques, the incidence appears to have fallen dramatically.

After the acute phase of MI, long-term risk for total mortality and SCD are predicted by a number of factors. The most important for both SCD and non-SCD is the extent of myocardial damage sustained during the acute event. This is measured by the degree of reduction in the ejection fraction (EF), functional capacity, and/or the

occurrence of heart failure. Increasing *frequency* of postinfarction PVCs, with a plateau above the range of 10 to 30 PVCs per hour on 24-h ambulatory monitor recordings, also indicates increased risk, but advanced *forms* (salvos, nonsustained VT) are probably the more powerful predictor. PVCs interact strongly with decreased left ventricular EF. The combination of frequent PVCs, salvos or nonsustained VT, and an EF<30% identifies patients who have an annual risk of 20%. The risk falls off sharply with decreasing PVC frequency and the absence of advanced forms, as well as with higher EF. Despite the risk implications of postinfarction PVCs, improved outcome as a result of PVC suppression has not been demonstrated.

The extent of underlying disease due to any cause and/or prior clinical expression of risk of SCD (i.e., survival after out-of-hospital cardiac arrest not associated with acute MI) identify patients at very high risk for subsequent (recurrent) cardiac arrest. Survival after out-of-hospital cardiac arrest predicts up to a 30% 1-year recurrent cardiac arrest rate in the absence of specific interventions [10].

A general rule is that the risk of SCD is approximately one-half the total cardiovascular mortality rate. Thus, the SCD risk is approximately 20% per year for patients with advanced CHD or dilated cardiomyopathy severe enough to result in a 40% 1-year total mortality rate. The very high risk subgroups provide more focused population fractions («Percent/Year») for predicting cardiac arrest or SCD; but the impact on the overall population («Events/Yea») is considerably smaller. The requirements for achieving a major population impact are effective prevention of the underlying diseases and/or new epidemiologic probes that will allow better resolution of subgroups within large general populations.

Treatment. The individual who collapses suddenly is managed in four stages:

1) the initial response and basic life support,

2) advanced life support,

3) postresuscitation care,

4) long-term management.

The initial response and basic life support can be carried out by physicians, nurses, paramedical personnel, and trained lay persons. There is a requirement for increasing skills as the patient moves through the stages of advanced life support, postresuscitation care, and long-term management [7,16].

Initial Response and Basic Life Support. The initial response will confirm whether a sudden collapse is indeed due to a cardiac arrest. Observations for respiratory movements, skin color, and the presence or absence of pulses in the carotid or femoral

arteries will promptly determine whether a life-threatening cardiac arrest has occurred. As soon as a cardiac arrest is suspected or confirmed, contacting an emergency rescue system (e.g., 911) should be the immediate priority.

Agonal respiratory movements may persist for a short time after the onset of cardiac arrest, but it is important to observe for severe stridor with a persistent pulse as a clue to aspiration of a foreign body or food. If this is suspected, a prompt Heimlich maneuver may dislodge the obstructing body. A precordial blow, or "thump," delivered firmly by the clenched fist to the junction of the middle and lower third of the sternum may occasionally revert VT or VF, but there is concern about converting VT *to* VF. Therefore, it has been recommended to use precordial thumps as an advanced life support technique when monitoring and defibrillation are available. This conservative application of the technique remains controversial [9].

The third action during the initial response is to clear the airway. The head is tilted back and chin lifted so that the oropharynx can be explored to clear the airway. Dentures or foreign bodies are removed, and the Heimlich maneuver is performed if there is reason to suspect that a foreign body is lodged in the oropharynx. If respiratory arrest precipitating cardiac arrest is suspected, a second precordial thump is delivered after the airway is cleared.

Basic life support, more popularly known as CPR, is intended to maintain organ perfusion until definitive interventions can be instituted. The elements of CPR are the establishment and maintenance of ventilation of the lungs and compression of the chest. Mouth-to-mouth respiration may be used if no specific rescue equipment is immediately available (e.g., plastic oropharyngeal airways, esophageal obturators, masked Ambu bag). Conventional ventilation techniques during CPR require the lungs to be inflated 10 to 12 times per minute, i.e., once every fifth chest compression when two persons are performing the resuscitation and twice in succession every 15 chest compressions when one person is carrying out both ventilation and chest wall compression[8].

Chest compression is based on the assumption that cardiac compression allows the heart to maintain a pump function by sequential filling and emptying of its chambers, with competent valves maintaining forward direction of flow. The palm of one hand is placed over the lower sternum, with the heel of the other resting on the dorsum of the lower hand. The sternum is depressed, with the arms remaining straight, at a rate of approximately 80 to 100 per minute. Sufficient force is applied to depress the sternum 3 to 5 cm, and relaxation is abrupt.

Advanced Life Support is intended to achieve adequate ventilation, control cardiac arrhythmias, stabilize the hemodynamic status (blood pressure and cardiac output), and restore organ perfusion. The activities carried out to achieve these goals include:

1) intubation with an endotracheal tube,

2) defibrillation/cardioversion and/or pacing,

3) insertion of an intravenous line.

Ventilation with O_2 (room air if O_2 is not immediately available) may promptly reverse hypoxemia and acidosis. The speed with which defibrillation/cardioversion is carried out is an important element for successful resuscitation. When possible, immediate defibrillation should precede intubation and insertion of an intravenous line; CPR should be carried out while the defibrillator is being charged. As soon as a diagnosis of VT or VF is obtained, a 200-J shock should be delivered. Additional shocks at higher energies, up to a maximum of 360 J, are tried if the initial shock does not successfully abolish VT or VF. Epinephrine 1 mg intravenously is given after failed defibrillation, and attempts to defibrillate are repeated. The dose of epinephrine may be repeated after intervals of 3 to 5 min [8,9].

If the patient is less than fully conscious upon reversion, or if two or three attempts fail, prompt intubation, ventilation, and arterial blood gas analysis should be carried out. Intravenous $NaHCO_3$, which was formerly used in large quantities, is no longer considered routinely necessary and may be dangerous in larger quantities. However, the patient who is persistently acidotic after successful defibrillation and intubation should be given 1 meq/kg $NaHCO_3$ initially and an additional 50% of the dose repeated every 10 to 15 min.

After initial unsuccessful defibrillation attempts, or with persistent electrical instability, a bolus of 1 mg/kg lidocaine is given intravenously, and the dose is repeated in 2 min in those patients who have persistent ventricular arrhythmias or remain in VF. This is followed by a continuous infusion at a rate of 1 to **4** mg/min. If lidocaine fails to provide control, intravenous procainamid (loading infusion of 100 mg/5 min to a total dose of 500 mg, followed by continuous infusion at 2 to 5 mg/min) or bretylium tosylate (loading dose 5 to 10 mg/kg in 5 min; maintenance dose 0,5 to 2 mg/min) may be tried. Intravenous calcium gluconate is no longer considered safe or necessary for routine administration. It is used only in patients in whom acute hyperkalemia is known to be the triggering event for resistant VF, in the presence of know, hypocalcemia, or in patients who have received toxic doses of calcium channel antagonists [12].

Cardiac arrest secondary to bradyarrhythmias or asystole is mail aged differently. Once it is known that this type of rhythm is presell there is no role for external shock. The patient is promptly intubate CPR is continued, and an attempt is made to control hypoxemia and acidosis. Epinephrine and/or atropine are given intravenously or an intracardiac route. External pacing devices are now available to attempt to establish a regular rhythm, but the prognosis is generally very poor in this form of cardiac arrest. The one exception is bradyrhythmic/asystolic cardiac arrest secondary to airway obstruction This form of cardiac arrest may respond promptly to removal foreign bodies by the Heimlich maneuver or, in hospitalized patients, by intubation and suctioning of obstructing secretions in the airway [14].

Postresuscitation Care is defined by the clinical setting of the cardiac arrest. *Primary VF acute MI* is generally very responsive to life-support, techniques and easily controlled after the initial event. Patients are maintained on a lidocaine infusion at the rate of 2 to 4 mg/min for 24 to 72 h after the event. In the in-hospital setting, respirator support is usually not necessary or is needed for only a short time, and hemodynamics stabilize promptly after defibrillation or cardioversion In *secondary VF in acute MI* (those events in which hemodynamic abnormalities predispose to the potentially fatal arrhythmia), resuscitative efforts are less often successful, and in those patients who are successfully resuscitated, the recurrence rate is high. The clinical picture is dominated by hemodynamic instability. In fact, the outcome is determined more by the ability to control hemodynamic dysfunction than by electrophysiological abnormalities. Bradyarrhythmias, asystole, and pulseless electrical activity are commonly secondary events in hemodynamically unstable patients and are less responsive to interventions.

The outcome after in-hospital cardiac arrest associated with *non-cardiac* diseases is poor, and in the few successfully resuscitated patients, the postresuscitation course is dominated by the nature of the underlying disease. Patients with cancer, renal failure, acute central nervous system disease, and uncontrolled infections, as a group, have a survival rate of less than 10% after in-hospital cardiac arrest. Some major exceptions are patients with transient airway obstruction, electrolyte disturbances, proarrhythmic effects of drugs, and severe metabolic abnormalities, most of whom may have an excellent chance of survival if they can be resuscitated promptly and maintained while the transient abnormalities are being corrected [13].

Long-Term Management after Survival of Out-of-Hospital Cardiac Arrest Patients who do not suffer irreversible injury of the central nervous system and who achieve hemodynamic stability should have extensive diagnostic and therapeutic testing

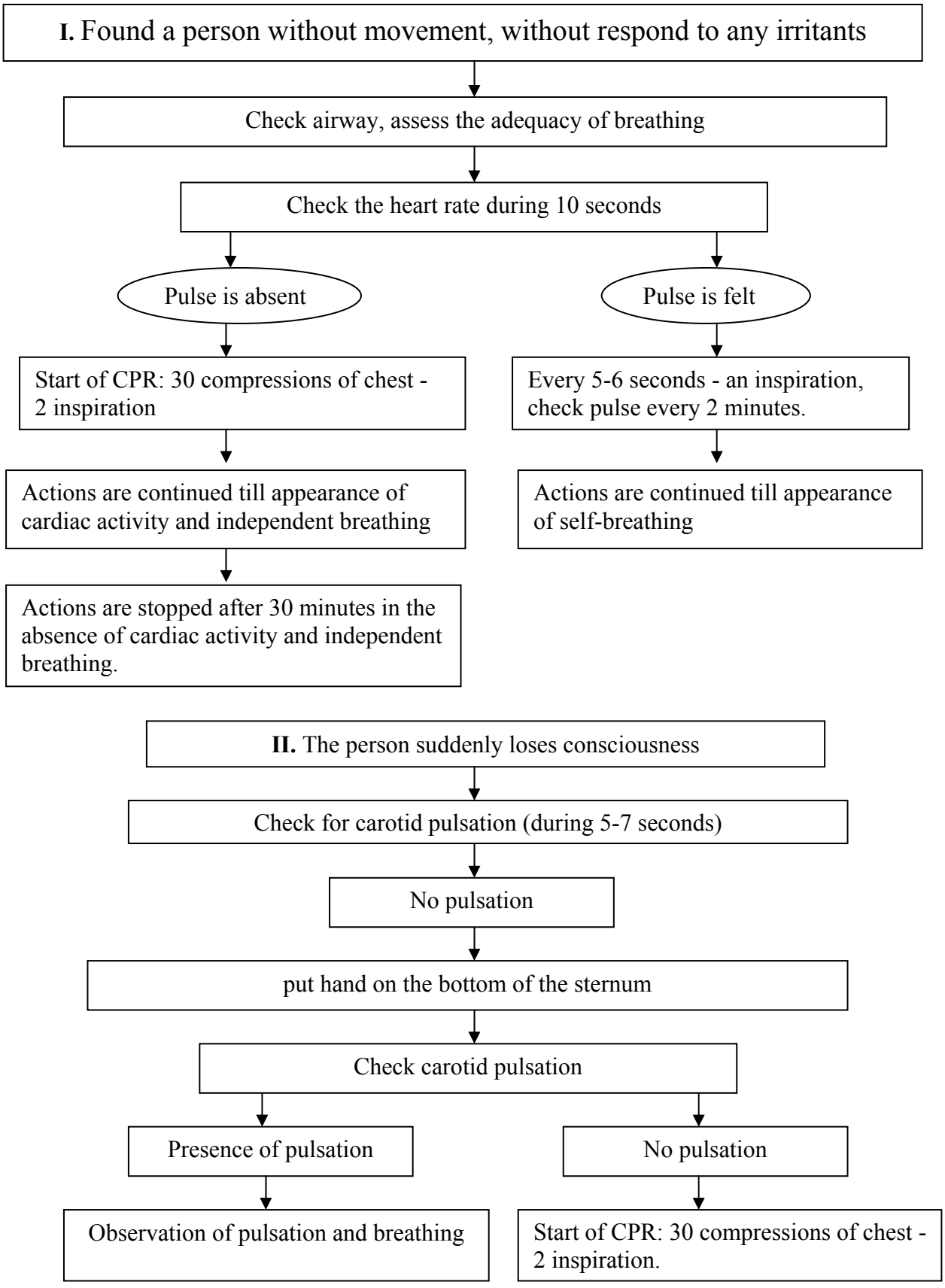
to guide long-term management. This aggressive approach is driven by the fact that statistics from the 1970s indicated survival after out-of- hospital cardiac arrest was followed by a 30% recurrent cardiac arrest rate at 1 year, 45% at 2 years, and a total mortality rate of almost 60% at 2 years. Historical comparisons suggest that these dismal statistics may be significantly improved by newer interventions, but the magnitude of the improvement is unknown because of the lack of concurrently controlled intervention studies [12].

Among those patients in whom an acute transmural MI is the cause of out-of-hospital cardiac arrest, the management is the same as in any other patient who suffers cardiac arrest during the acute phase of a documented MI. For almost all other categories of patients, however, extensive diagnostic studies are carried out to determine etiology, functional impairment, and electrophysiologic instability as guides to future management. In general, patients who have out-of-hospital cardiac arrest due to chronic ischemic heart disease, without an acute MI, are evaluated to determine whether transient ischemia or chronic electrophysiological instability was the more likely cause of the event. If there is reason to suspect an ischemic mechanism, coronary revascularization or drugs, most commonly beta blockers, are used to reduce ischemia. Electrophysiological instability is best identified by the use of programmed electrical stimulation to determine whether sustained VT or VF can be induced. If so, this information can be used as a baseline against which to evaluate drug efficacy for prevention of inducibility or to determine suitability for map-guided antiarrhythmic surgery, or whether an implantable cardiovertor/defibrillator (ICD) might be the best strategy. Using this technique in patients with EF of 30% or more, the recurrent cardiac arrest rate is less than 10% during the first year of follow-up when inducibility is suppressed by a drug. The outcome is not as good for patients with EF under 30% but may be still better than the apparent natural history of survival after cardiac arrest. For patients for whom successful drug therapy cannot be identified by this technique, insertion of an ICD, antiarrhythmic surgery (e.g., coronary bypass surgery, aneurysmectomy, cryoablation), or empiric amiodarone therapy can be considered options. Primary surgical success, defined as surviving the procedure and reverting to a noninducible status without drug therapy, is better than 90% when patients are selected for ability to be mapped in the operating room. However, only a small fraction of patients meet the criteria. In addition, VT/VF *cannot* be induced in a number of survivors of cardiac arrest (30 to 50%), and inducible arrhythmias can be suppressed by drugs in no more than 20 to 30% of those whose arrhythmias can be induced. Because

of these limitations of drug therapy and surgical approaches, ICD therapy has evolved into the most commonly used strategy for cardiac arrest survivors. ICDs have very good success rates for sensing and reverting life-threatening arrhythmias, but improvement in long-term total survival outcomes remains ill defined [15].

The ESVEM study has suggested that ambulatory monitor-based suppression of ambient arrhythmias is equivalent to electrophysiologically guided testing in predicting long-term outcome.

Algorithm of cardiopulmonary resuscitation (CPR) on the pre-admission stage



Cardiovascular Emergencies

– Any condition that lead to respiratory impairment: reducing ability to deliver oxygen

– Severe bleeding: Shock

– Stroke: Reducing cerebral blood flow

– Heart diseases: Reducing of tissue oxygenation

– Heart attack: Can lead to cardiac arrest

– Ventricular fibrillation: Heart muscle flutters rather than pumping blood

Causes of circulatory arrest

<u>Cardiac</u>

- Ischemic heart disease (myocardial infarction, stenocardia)
- Arrhythmias
- Electrolytic disorders
- Valvular disease
- Cardiac tamponade
- Pulmonary embolism
- aortic aneurysm rupture

<u>Extracardiac</u>

- airway obstruction
- acute respiratory failure
- shock
- reflectory cardiac arrest
- embolisms of different origin
- drug overdose
- electric injury
- poisoning

Sequence of actions:

➢ Check responsiveness

➢ Call for help

➢ Correct position of patient and ensure the open airway

➢ Check the presence of spontaneous respiration

➢ Check peripheral pulsation

➢ Start closed-chest massage and artificial ventilation

Main stages of resuscitation:

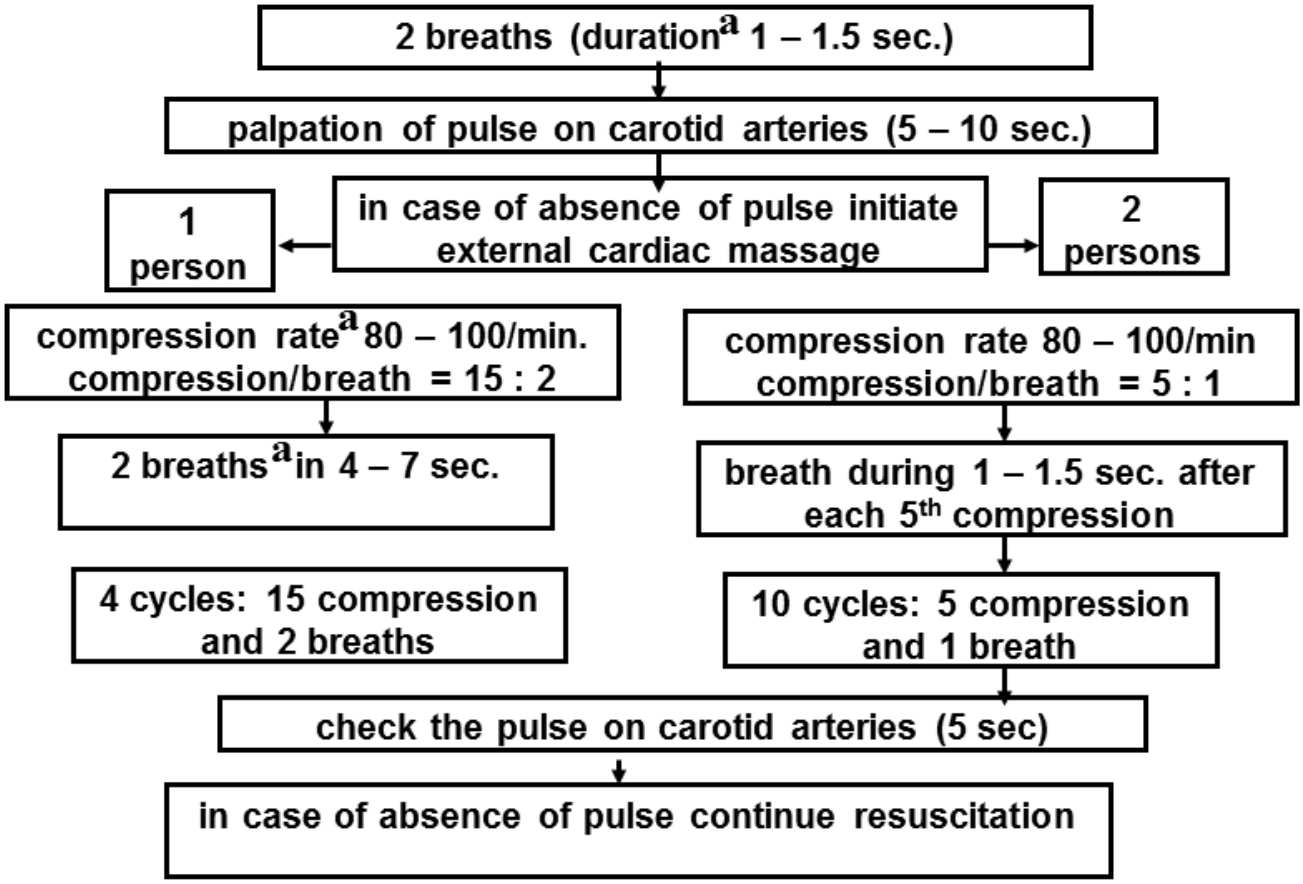
A (Airway) – ensure open airway by preventing the falling back of tongue, tracheal intubation if possible;

B (Breathing) – start artificial lungs ventilation;

C (Circulation) – restore the circulation by closed-chest massage;

D (Differentiation, Drugs, Defibrillation) – quickly perform differential diagnosis of circulatory arrest, use different medication and electric defibrillation in case of ventricular fibrillation.

Algorithm of cardiopulmonary resuscitation



VENTRICULAR FIBRILLATION OR PULSELESS TACHYCARDIA

Witnessed

Precordial thump

Check pulse, if none:

Unwitnessed

- ✓**Begin CPR**
- ✓**Defibrillate with 200 joules**
- ✓**Defibrillate with 200-300 joules**
- ✓**Establish IV access, intubate**
- ✓**Adrenaline 1 mg push**
- ✓**Defibrillate with 360 joules**
- ✓**Lidocaine 1 mg/kg IV, ET**
- ✓**Defibrillate with 360 joules**

IX. Tasks for final control

1. During cardiopulmonary resuscitation should be observed the following rules:
   A. Head should be dropped
   B. Head should be elevated
   C. Compressed abdominal aorta
   D. Whatever surface on which the patient lies
   E. The patient should be ventilate by pure oxygen

2. Which of the following symptoms is the indication for closed-chest massage?
   A. The absence of peripheral arteries pulsation
   B. Midriatic pupils
   C. The absence of carotid pulsation
   D. Miotic pupils
   E. Cyanotic skin

3. The cause of circulatory arrest in diastole can be:
   A. Acidosis
   B. Hypokalemia
   C. Hypercalcemia
   D. Respiratory alkalosis
   E. Hypoglycemia

4. After a primary circulatory arrest spontaneous breathing stops after:
   A. 30 sec
   B. 20 sec
   C. 5 min.
   D. 60 sec
   E. 2 min.

5. In patients with absence of pulse asystole on ECG was diagnosed. Primary remedial measure should be:
   A. Electric defibrillation
   B. Adrenalin injection
   C. Atropine injection

D. Calcium chloride injection

E. Lidocain injection

6. Which of the following medicines cannot be injected endotracheally during cardiopulmonary resuscitation?

A. Lidocain

B. Adrenalin

C. Noradrenaline

D. Atropine sulfate

E. Calcium chloride

7. Define the correct ratio between the number of contractions of chest and the respiration number during cardiopulmonary resuscitation:

A. 5:1

B. 12:2

C. 30:2

D. 24:2

E. 9:1

8. For effective closed-chest massage in adult patients, breast bone drifts toward the spine to:

A. 1 - 2 cm

B. 5 - 6 cm

C. 7 - 8 cm

D. 3 - 4 cm

E. 8 - 9 cm

9. After injection of digoxin 0,75 mg the patient of 55 years old suddenly lost consciousness. Peripheric pulse was absent, agonal breathing, BP didn't determined. What examination do you have to conduct during resuscitation?

A. Heart radiokimography

B. ECG

C. Vektorcardiography

D. Radiography of the chest

E. Heart ultrasonography

10. Adrenaline injection to resume the cardiac function in the case of:

A. Hyperglycemia

B. Alkalosis

C. Acidosis

D. Hypokalemia

E. Acid-base balance plasma does not matter

Answers:

1	2	3	4	5	6	7	8	9	10
E	C	A	D	B	E	C	D	B	C

X. Practical skills

1. 82 years old patient had circulatory arrest and respiratory standstill. Medical history: prolonged heart failure. On the 5th minute of cardiopulmonary resuscitation, which was started on time, there wasn't the cardiac activity. What is the prognosis for further resuscitation?

A. Full restoration is impossible

B. Restoration of beatings with some extrasystoles

C. Full recovery

D. Full recovery with following deterioration

E. Possible restore of sinus rhythm

2. There was an attack of rhythm disturbance in the patient: atrial fibrillation, paroxysm of ventricular tachycardia. Which universal antiarrhythmic is most reasonable for patient?

A. Pananhin

B. Procainamide hydrochloride

C. Lidokain

D. Verapamil

E. Digoxin

3. On the fourth postoperational day the 68 years old patient had acute bradicardia with asystole, no consciousness, coarse breathing. Resuscitation was started. What is the criterion of effectiveness of resuscitation?

A. xerophthalmus

B. Appearance of breath

C. Reduction of cyanosis

D. The carotid pulsation
E. Myotic pupils

4. In-hospital 50 year old patient with arterial hypertension, ischemic heart disease suddenly fainted. Duty doctor diagnosed the circulatory arrest and respiratory standstill. The cardiopulmonary resuscitation was started. A set of false teeth was extracted. What will be the following actions?

A. Intracardiac injection of adrenalin
B. Intracardiac injection of atropine
C. Closed-chest massage
D. Proceed to ventilation
E. Throw back patient's head, lift chin, pull and fix tongue

5. Man was injured as a result of falling from a height. Physical examination: no response to natural irritants, unconscious, no breathing, thready pulse, left foot was unnatural turn to the other side, numerous wounds and scratches on skin. Which are primary measures in this case?

A. Applying of sterile bandages on wounds
B. Immobilization of fractures
C. Transfusion-infusion therapy
D. Respiratory restoration
E. Anesthesia

6. The 75-years-old man was unconscious in the street with no pulsation on main arteries, mydriatic pupils. The clinical death was diagnosed. Which are primary measures in this case?

A. emergency call
B. Start a closed-chest cardiac massage
C. Start the cardiopulmonary resuscitation
D. Elevate the lower limbs
E. Start artificial respiration

7. Resuscitation of elderly patient was not effective: wave of artificial carotid pulsation was not determined; the closed-chest cardiac massage was performed with displacement of the lower half of the sternum on 2-3 cm toward the spine because of chest stiffness. When the heart massage will be effective?

A. Increasing the displacement of sternum to 4-5 cm

B. Start the open-chest cardiac massage
C. Perform chest compression in left parasternal region
D. 10 ml 10% calcium chloride solution injection
E. Increasing displacement of sternum to 6-7 cm

8. The patient suddenly turned pale, unconscious. The skin was cyanotic, no photoreaction, no carotid pulsation, asystole on ECG. Which are primary measures in this case?

A. Precardiac blow
B. Artificial respiration
C. The closed-chest cardiac massage
D. Droperidol injection
E. Adrenaline injection

9. You perform the resuscitation measures for patient - closed-chest cardiac massage, mouth-to-mouth ventilation. Which will be duration of these actions in the absence of recovery of cardiac and central nervous system activity?

A. 45 minutes
B. Before the emergency arrival
C. 60 minutes
D. 30 minutes
E. 15 minutes

10. During diagnostic endoscopy the 45-years-old patient was asystole on ECG. Which will be your primary action?

A. Electrical cardiac acceleration
B. Sodium bicarbonate injection
C. Atropine injection
D. defibrillation.
E. closed-chest cardiac massage, artificial ventilation

Answers:

1	2	3	4	5	6	7	8	9	10
C	B	C	E	C	C	A	B	D	E

TOPIC 6

The emergency in the practice of family doctor in the case of pain syndrome. The clinical classification of pain. The mechanism of pain in incurable patient. The principles of treatment of chronic pain syndrome. The emergency in context of incurable diseases and imminent death.

I. Theme actuality

The task of medicine is to preserve and restore health and to relieve suffering. Understanding pain is essential to both these goals. Because pain is universally understood as a signal of disease, it is the most common symptom that brings a patient to a physician's attention. The function of the pain sensory system is to detect, localize, and identify tissue-damaging processes. Since different diseases produce characteristic patterns of tissue damage, the quality, time course, and location of a patient's pain complaint and the location of tenderness provide important diagnostic clues and are used to evaluate the response to treatment.

II. Study purposes: to learn the basic organizational, diagnostic and therapeutic features of surgical patients (obstetrics and gynecology, surgery, traumatology and orthopedics, urology, proctology, oncology, ophthalmology, otolaryngology, dentistry, resuscitation and intensive therapy) in practice of FD; to diagnose the medical condition of pain in the chest, abdomen during initial contact with patient and determine the indications for urgent hospitalization in the pre-admission stage in the case of pain.

III. Concrete purposes of the module:

- to know the features inter-sectorial interrelation of FD in outpatient clinic;
- to evaluate the benefits of the FD in the proceedings of preventive measures;
- to identify diseases and conditions that require emergency care;
- to assess the patient's condition and provide appropriate medical care in emergency case in FD practice;
- to know dosage, indications and contraindications for drug in emergency case;
- to identify emergency cases in FD practice.

IV. A student must be able:

• to know the clinical presentations of disease with abdominal pain: acute appendicitis, pancreatitis, cholecystitis, peptic ulcer and its complications, acute gynecological diseases, ectopic pregnancy, renal colic, and inflammatory diseases of the kidneys and urinary tract.

• to know the clinical presentations of disease with chest pain: various forms of CHD, pericarditis, myocarditis, pulmonary embolism, dissecting aortic aneurysm, pneumothorax, dry pleurisy, lesions of peripheral nerves, spine and muscles.

• to identify the acute coronary syndrome with ST segment elevation, ST-segment elevation without clinical characteristics of acute coronary syndrome, therapeutic approach of pre-admission and emergency care.

• to know the characteristics of pain in the extremities and clinical signs of acute lesions of veins and arteries.

V. Task for initial independent training

1. The patient complaints for pain in the left upper quadrant of abdomen, weakness, vomiting, dizziness, which appeared 5 hours ago. Hypotension, tachycardia. Medical history: 10 days ago blunt trauma of abdomen. What is the working diagnosis?

A. Rupture of the spleen;
B. Rupture of the liver;
C. Rupture of intestine;
D. Peritonitis;
E. Perforated ulcer.

2. 2 days ago the patient felt a sharp pain in the right upper quadrant of abdomen with irradiation under the right scapula. Next day he had vomiting. The right half of the abdomen is tense. Positive symptoms of Ortner, Musset. What is the working diagnosis?

A. Acute pancreatitis;
B. Mesenteric ischemia;
C. Acute intestinal obstruction;
D. Biliary colic;
E. Acute cholecystitis.

3. In the case of angina the typical pain is:

A. Pressing, squeezing, localized behind the breastbone.
B. Burning heart pain.
C. Barbed heart pain associated with movements or breathing.
D. Feeling of discomfort in the pericardial region during physical or emotional stress.
E. Feeling heterogonous object under the sternum.

4. The 21 years old patient admitted to the doctor. She complained for lower abdomen pain spreading to the anus, weakness, which came on the 12th day of the menstrual cycle. BP 70/40 mm Hg, HR 120. What is the working diagnosis?

A. ectopic pregnancy;
B. Torsion of ovarian cyst;
C. Acute appendicitis;
D. Acute pelvioperitonit;
E. Ovarian apoplexy.

5. The 43 years old patient complains for right lumbar pain spreading to the lower abdomen. Patient was motionless, groaning. HR 100, BP 130/70 mm Hg. The muscles tension in the right iliac region. Negative Lassega, Schetkina, Ortner signs. Positive Pasternatsky sign. What is the working diagnosis?

A. perforated duodenal ulcer;
B. Renal colic;
C. Radiculitis;
D. Acute appendicitis;
E. Acute cholecystitis.

6. Dangerous electric current for human health is:

A. Above 50 volts.
B. Above 150 volts.
C. Above 100 volts.
D. Above 75 volts.
E. Above 200 volts.

7. The sign of severe overheating of the body is:

A. Body temperature of 40,5 C.
B. Heart rate 130 per min.
C. Reduced muscle tone.
D. Body temperature 39,5 C.
E. Heart rate 120 per min.

8. The skin lesions in the case of thermal most often determined by:

A. «Rule of Donald».

B. «Rule of Nine».

C. «Frank Rule».

D. «Rule palm».

E. Rule of thermal shock.

9. The sign of deep frostbite is:

A. Pale skin.

B. Cyanotic skin.

C. Pale skin, soft tissue lying below.

D. Heart rate 110/hv.

E. None of the above.

10. The victim of hypothermia was admitted to the hospital. Rectal temperature was 32,0 C. What was degree of hypothermia?

A. Severe.

B. Light.

C. Moderate.

D. Deep.

E. Coma.

Answers:

1	2	3	4	5	6	7	8	9	10
A	E	A	E	B	C	A	B	E	C

VI. Basic questions after theme

• Surgical diseases, symptoms and syndromes, the prevention, diagnosis and treatment of patients by a family doctor.

• Surgical diseases, symptoms and syndromes, which are the indication for refer to specialists for making final diagnosis, differential diagnosis, planning a therapeutic approach.

• Surgical diseases, symptoms and syndromes, for which the FD may prescribe treatment and rehabilitation for patients according to the recommendations of specialists, may conduct clinical supervision under patients.

• Surgical diseases, symptoms and syndromes, which require FD and medical specialist's supervision for implementation of working and final diagnosis, treatment, rehabilitation of patients.

• Surgical diseases that require treatment and clinical supervision of doctors.

• Surgical diseases that need emergency or planned hospitalization of patients.

• Differential diagnosis of acute and chronic pain, somatic and psychosomatic disorders.

• Diagnosis of pain in various clinical situations and treatment.

• Method of patient's help in the case of acute coronary syndrome at the pre-admission stage.

• The therapeutic approach for patient with acute arterial occlusion.

• Pathophysiological changes in the case of insects stinging, serpents, emergency.

VII. Practical skills: The examination of patients in the surgical clinic by ophthalmologist, otolaryngologist, urologist, gynecologist or family doctor. In the case of pain propose the algorithms and differential diagnosis of medical care in the pre-admission stage.

Independent work: compiling algorithms of emergency during practical classes - 6 hours.

VIII. The content of theme

The pain sensory system. Pain is an unpleasant sensation localized to a part of the body.

Chest discomfort and palpitation

Chest discomfort is one of the most frequent complaints for which patients seek medical attention; the potential benefit (or harm) resulting from the proper (or improper) assessment and management of the patient with this complaint is enormous. Failure to recognize a serious disorder, such as ischemic heart disease, may result in the dangerous delay of much-needed treatment, while an incorrect diagnosis of a potentially hazardous condition such as angina pectoris is likely to have harmful psychological and economic consequences and may lead to unnecessary cardiac catheterization. There is little relation between the severity of chest discomfort and the gravity of its cause. Therefore, a frequent problem in patients who complain of chest discomfort or pain is distinguishing trivial complaints from coronary artery disease and other serious disorders (Table 1) [7].

Causes of chest discomfort. Discomfort due to Myocardial Ischemia. Discomfort due to myocardial ischemia occurs when the oxygen supply to the heart is deficient in relation to the oxygen need. The blood flow through the coronary arteries is directly proportional to the pressure gradient between the aorta and the ventricular myocardium (during systole) or the ventricular cavity (during diastole). However, in the presence of critical stenosis, it is also proportional to the fourth power of the radius of the coronary arteries. A relatively slight alteration in coronary luminal diameter below a critical level can produce a large decrease in coronary flow, provided that other factors remain constant. Coronary blood flow occurs primarily during diastole, when it is unopposed by systolic myocardial compression of the coronary vessels.

Table 1

Some Causes of Chest Discomfort and the Types of Discomfort associated with Them

Cause	New, acute, often ongoing	Recurrent, episodic	Persistent, even for days
CARDIAC			
Coronary artery disease	+	+	-
Aortic stenosis	-	+	-
Hypertropic cardiomyopathy	-	+	-
Pericarditis	+	+	+
VASCULAR			
Aortic dissection	+	-	-
Pulmonary embolism	+	+	-
Pulmonary hypertension	+	+	-
Right ventricular strain	+	+	-
PULMONARY			
Pleuritis or pneumonia	+	+	+
Tracheobronchitis	+	+	+
Pneumothorax	+	-	+
Tumor	-	-	+
Mediastinitis or mediastinal emphysema	+	-	+
GASTROINTESTINAL			
Esophogeal reflux	+	+	+
Esophogeal spasm	+	+	+
Mallory-Weiss tear	+	-	-
Peptic ulcer disease	+	+	-
Biliary disease	+	+	-
Pancreatitis		+	+

Cause	New, acute, often ongoing	Recurrent, episodic	Persistent, even for days
MUSCULOSKELETAL			
Cervical disk disease	-	+	+
Arthritis of the shoulder or spine	-	+	+
Costochondritis	+	+	+
Intercostal muscle cramps	+	+	+
Interscalene or hyperabduction syndromes	-	+	+
Subacromial bursitis	+	+	+
OTHER			
Disorders of the breast	-	+	+
Chest wall tumors	-	-	+
Herpes zoster	+	-	+
Emotional	+	+	+

When the epicardial coronary arteries are narrowed critically (>70% stenosis of the luminal diameter), the intramyocardial coronary arterioles dilate in an effort to maintain total flow at a level that will avert myocardial ischemia at rest. The further dilation that would normally occur during exercise is, therefore, not possible. Hence, any condition in which increased heart rate, arterial pressure, or myocardial contractility occurs in the presence of coronary obstruction tends to precipitate anginal attacks by increasing myocardial oxygen needs in the face of a fixed oxygen supply.

By far the most frequent underlying cause of myocardial ischemia is organic narrowing of the coronary arteries secondary to coronary atherosclerosis. Acute thrombosis superimposed on an atherosclerotic plaque is frequently the cause of unstable angina and acute myocardial infarction [19].

Aside from conditions that narrow the lumen of the coronary arteries, the only other frequent causes of myocardial ischemia are disorders such as valvular aortic stenosis or hypertrophic cardiomyopathy, which cause a marked disproportion between the coronary perfusion pressure and the heart's oxygen requirements. Under such conditions, the rise in left ventricular systolic pressure is not, as in hypertensive states, balanced by a corresponding elevation of aortic perfusion pressure. The chest pain is no more common in patients with mitral valve prolapse than in those without it.

The chest discomfort of myocardial ischemia, most commonly from coronary artery disease but also occasionally from the other causes of ischemia noted above, is

angina pectoris. Myocardial ischemia secondary to coronary atherosclerosis is more common in adults, especially the elderly who have hypercholesterolemia, diabetes mellitus, hypertension, obesity, or who smoke cigarettes. Toxins, including cocaine ingestion or withdrawal of chronic exposure to nitroglycerin, can cause sufficient coronary vasoconstriction to result in myocardial ischemia, and cocaine also can cause myocardial infarction.

Angina pectoris is usually described as a heaviness, pressure, squeezing, or sensation of strangling or constriction in the chest, but it also may be described as an aching or burning pain, or even as indigestion. Some patients steadfastly deny pain but will admit to a discomfort or unusual feeling or may complain of difficulty in breathing.

Typically, angina pectoris develops gradually during exertion, after heavy meals, and with anger, excitement, frustration, and other emotional states; it is not precipitated by coughing, respiratory movements, or other motion. When angina is induced by walking, it often forces the patient to stop or to reduce speed. Anginal pain typically resolves within 5 to 30 min. More prolonged myocardial ischemia often represents a myocardial infarction, while more prolonged pain without other evidence for myocardial ischemia suggests a noncoronary etiology [18].

The correct diagnosis of angina pectoris may be aided by noting that the pain disappears more rapidly (usually within 5 min) and more completely when sublingual nitroglycerin is used. The demonstration that the time required for a given exercise to produce pain is consistently and considerably longer when it is undertaken within a few minutes after a sublingual nitroglycerin pill than after a placebo may, in some instances, be powerful clinical evidence for the diagnosis of angina pectoris. Angina is rarely relieved within a few seconds of lying down. It is not precipitated by stooping forward or by chest palpation, deep breathing, or simple changes in position.

Angina occurs most typically in the substernal region, anteriorly across the midthorax; it may radiate to or rarely occur alone in the intercapsular region, arms, shoulders, teeth, or abdomen. It rarely radiates to below the umbilicus, to the back of the neck, or to the occiput. Although the radiation of chest discomfort to the left arm increases the likelihood that myocardial ischemia or infarction is present, impulses from the skin and from visceral structures, such as the esophagus and heart, converge on a common pool of neurons in the posterior horn of the spinal cord, and their origin may be confused by the cerebral cortex [20].

An increase in heart rate is especially harmful in patients with coronary atherosclerosis or with aortic stenosis, because it both increases myocardial oxygen

need and shortens diastole more than systole, thereby decreasing the total available perfusion time per minute. Tachycardia, a decline in arterial pressure, thyrotoxicosis, and diminution in arterial oxygen content (such as occurs in anemia or arterial hypoxia) are precipitating and aggravating factors rather than underlying causes of angina.

Patients with marked ***right ventricular hypertension*** may have exertional pain that is quite similar to that of angina. This discomfort probably results from relative ischemia of the right ventricle brought about by the increased oxygen needs and by the elevated intramural resistance, with reduction of the normally large systolic pressure gradient that perfuses this chamber [24].

Myocardial infarction is usually associated with a discomfort that is similar in quality and distribution to that of angina but of longer duration (usually 30 min) and usually of greater intensity. In contrast to angina, the pain of myocardial infarction is not rapidly relieved by rest or by coronary dilator drugs and may require large doses of narcotics. It may be accompanied by diaphoresis, nausea, and hypotension.

The results of ***physical examination*** may be normal in patients with myocardial ischemia caused by coronary atherosclerosis. However, myocardial ischemia can cause a third or fourth heart sound because of an impairment of myocardial contraction or relaxation. Ischemic papillary muscle dysfunction can cause transient mitral regurgitation and its associated murmur. Myocardial infarction and, less commonly, severe and generalized ischemia can cause congestive heart failure.

The chest discomfort from myocardial ischemia that is caused by aortic stenosis, hypertrophic cardiomyopathy, and non-atherosclerotic causes of coronary artery disease is generally similar to that of angina pectoris from coronary atherosclerosis. However, in these conditions the physical examination will usually reveal classic findings of an aortic systolic murmur in patients with aortic stenosis (and will reveal dynamic outflow obstruction in many patients with hypertrophic cardiomyopathy [23,24].

Chest Pain due to Pericarditis. The visceral surface of the pericardium ordinarily is insensitive to pain, as is the parietal surface, except in its lower portion, which has a relatively small number of pain fibers carried in the phrenic nerves. The pain associated with pericarditis is believed to be due to inflammation of the adjacent parietal pleura. These observations explain why noninfectious pericarditis (e.g., that associated with uremia and with myocardial infarction) and cardiac tamponade with relatively mild inflammation are usually painless or accompanied by only mild pain, whereas infectious pericarditis, being nearly always more intense and spreading to the neighboring pleura, is usually associated with pain.

Pericarditis can cause pain in several locations. Since the central part of the diaphragm receives its sensory supply from the phrenic nerve (which arises from the third to fifth cervical segments of the spinal cord), pain arising from the lower parietal pericardium and central tendon of the diaphragm is felt characteristically at the tip of the shoulder, the adjoining trapezius ridge, and the neck. Involvement of the more lateral part of the diaphragmatic pleura, supplied by branches from the sixth to ninth intercostal nerves, causes pain not only in the anterior part of the chest but also in the upper part of the abdomen or corresponding region of the back, sometimes simulating the pain of acute cholecystitis or pancreatitis.

Pericardial pain commonly has a pleuritic component; i.e., it is related to respiratory movements and aggravated by cough and/or deep inspiration, because of pleural irritation. It is sometimes brought on by swallowing, because the esophagus lies just behind the posterior portion of the heart, and is often altered by a change of body position, becoming sharper and more left-sided in the supine position and milder when the patient sits upright, leaning forward [19].

In some patients, however, pericardial pain may be described as a steady substernal discomfort that can mimic the pain of acute myocardial infarction. The mechanism of this steady substernal pain is not certain, but it may arise from marked inflammation of the relatively insensitive inner parietal surface of the pericardium or from irritated afferent cardiac nerve fibers lying in the periadventitial layers of the superficial coronary arteries. Occasionally, both pleuritic and steady pain may be present simultaneously.

Vascular Causes of Chest Pain. *Aortic dissection* develops as a result of a subintimal hematoma, which may start either because a tear has developed in the intimae of the aorta or because of bleeding into the vasa vasorum. Antegrade movement of this hematoma can compromise major branches off the aorta, while retrograde spread can occlude a coronary artery, disrupt the aortic valve annulus, or rupture into the pericardial space [21].

The pain due to *acute dissection of the aorta* or to an expanding aortic aneurysm results from stimulation of nerve endings in the adventitia. The pain usually begins abruptly, reaches an extremely severe peak rapidly, is felt in the center of the chest and/or in the back depending on the site of the dissection, lasts for hours, and requires unusually large amounts of analgesics for relief. Patients commonly describe a true pain rather than the vague discomfort that is sometimes described with myocardial ischemia. The pain is not aggravated by changes in position or respiration.

Chest Pain due to Pulmonary Embolism. The acute pain from massive *pulmonary emboli* is thought to be related to pulmonary hypertension and to distention of the pulmonary artery. Infarction of a segment of the lung that is adjacent to the pleura commonly irritates the pleural surface and causes chest discomfort hours or even days later. The pain resulting from pulmonary embolism may resemble that of acute myocardial infarction, and in cases of massive embolism it is located substernally. In patients with smaller emboli, the pain is caused by focal pulmonary infarction and is usually located more laterally, is pleuritic in nature, and sometimes is associated with hemoptysis [22].

Other Pulmonary Causes of Chest Discomfort. A variety of diseases of the lung can cause chest discomfort. Pleural pain, which is usually a brief, sharp, knifelike pain that is precipitated by inspiration or coughing, is very common and generally results from stretching of a parietal pleura that is inflamed by fibrinous pleurisy or any pneumonic process.

Gastrointestinal Causes of Chest Discomfort. *Esophageal pain* commonly presents as a deep thoracic burning discomfort, which is the hallmark of acid-induced pain. Ingestion of aspirin, alcohol, or certain foods typically exacerbates this burning discomfort, and the discomfort may be relieved promptly by antacids or even by one or two swallows of food or water. Patients may have accompanying dysphagia, regurgitation of undigested food, or weight loss. The symptoms of a hiatus hernia tend to be exacerbated by lying down, and all forms of acidpeptic disease may be worse in the early morning, when acidic secretions are not neutralized by food. Esophageal spasm, which may be induced by reflux of gastric acid into an esophagus in which the mucosa has been previously irritated, can cause a squeezing pain that may be indistinguishable from that of myocardial ischemia and that may even have a similar pattern of radiation. Pain resulting from injury of the esophagus, such as in the case of a Mallory-Weiss tear that is caused by severe vomiting, can cause severe acute chest pain. Occasionally, other gastrointestinal diseases, including *peptic ulcer disease, biliary disease,* and *pancreatitis,* may present with chest discomfort as well as abdominal discomfort. Pain resulting from gastric or duodenal ulcer is epigastric or substernal; usually commences about 1 to 1,5 h after meals, and is usually relieved in several minutes by antacids or milk [7,22].

The discomfort caused by acute cholecystitis is more commonly described as an ache, which may be epigastric or substernal. It most commonly occurs an hour or so after meals and not in relation to exertion. The presence of an abdominal disorder, such

as a hiatus hernia or a duodenal ulcer, does not constitute proof that the patient's chest pain is related to it. Such disorders are frequently asymptomatic and are not at all uncommon in patients who also have myocardial ischemia [24].

Neuromusculoskeletal Causes of Chest Discomfort. Neuromusculoskeletal chest discomfort can be caused by *cervical disk disease* because of compression of nerve roots, by *arthritis of the shoulder or spine,* or by *costochondritis,* which is an inflammation of the costochondral junctions. Inflammation of the subacromial bursa or, less commonly, the supraspinatus or deltoid tendon may cause pain that radiates to the chest. *Intercostal muscle cramps* may occur throughout the chest. Anterior scalene and hyperabduction syndromes also can cause chest discomfort.

The *costochondral and chondrosternal articulations* are the most common sites of anterior musculoskeletal chest pain. Objective signs in the form of swelling (Tietze's syndrome), redness, and heat are rare, but sharply localized tenderness is common. The pain may be darting and last for only a few seconds or may be a dull ache enduring for hours or days. An associated feeling of tightness due to muscle spasm is frequent. *Pressure on the chondrosternal and costochondral junctions and on the pectoralis muscles is an essential part of the examination of every patient with chest pain* and will reproduce the pain arising from these tissues [23].

Emotional Causes of Chest Pain. Emotional disorders are commonly associated with chest pain. Usually, the discomfort is experienced as a sense of "tightness," sometimes called "aching," and occasionally it may be designated as a pain of considerable magnitude. Since the discomfort may be described as a tightness or constriction and is often localized at least in part beneath the sternum, it is not surprising that this type of discomfort is frequently confused with that of myocardial ischemia. Ordinarily, it lasts for a half hour or more, is unrelated to exertion, and fluctuates slowly in intensity. The association with fatigue or emotional strain is usually clear, although this may not be volunteered by the patient. Associated hyperventilation can cause innocent changes in the T waves and ST segments, which can be confused with coronary artery disease. Alternatively, chest pain associated with emotional disorders may be sharp and very brief and located near the left nipple.

Differential diagnosis of chest discomfort. The key issue in the evaluation of the patient with chest discomfort is to distinguish potentially life-threatening conditions such as coronary artery disease, aortic dissection, or pulmonary embolism from other causes of chest discomfort. Even patients who have brief episodes of pain and are otherwise in apparently excellent health may have intermittent myocardial ischemia or

recurrent pulmonary emboli. One useful approach to the patient with chest pain is to determine whether the syndrome represents new, acute and often ongoing pain; recurrent, episodic pain; or pain that is persistent, perhaps for days. The various causes of chest pain (Table 1) can be distinguished by their likelihood to present in these three different ways [21].

New, Acute, often Ongoing Pain. In a patient who presents with this syndrome, the physician must immediately distinguish whether the pain represents a condition for which acute circulatory or respiratory insufficiency is a substantial risk. Emergent stabilization and treatment must precede detailed diagnostic evaluation.

In the patient with acute chest discomfort, the diagnostic evaluation begins with a focused history and physical examination that is designed to evaluate the likelihood of conditions, such as acute myocardial infarction, aortic dissection, or pulmonary embolism that could be life-threatening even in a patient who currently appears to be stable. Accumulated data from large numbers of patients who have presented to emergency departments with acute chest pain can aid in the assessment of the probability that an individual is experiencing an acute myocardial infarction. In this setting, an emergent electrocardiogram is the single best diagnostic test. In patients in whom the ECG shows Q waves of at least 0,04 s duration or ST- segment elevation that is not known to be old in two more leads, the probability of acute myocardial infarction is 75%. In patients in whom the ECG shows evidence of ST-segment depression of 1 mm or more or T-wave inversion that is not known to be old in two or more leads, the probability of acute myocardial infarction is about 20%. The patients with either of these ECG changes and a clinical syndrome suspicious for acute myocardial ischemia require urgent admission to an intensive care unit, partly for further evaluation but mainly because of the substantial short-term risk of developing life-threatening complications. For patients with ST-segment elevation, the likelihood of occlusion of an infarct-related artery mandates urgent reperfusion unless otherwise contraindicated or unless a diagnosis other than acute myocardial infarction is being considered very seriously. For patients with changes suggesting ischemia but without ST elevation, treatment is usually for presumed unstable angina [23].

For patients without ECG changes of new (or presumably new) ischemia or infarction, further observation with serial ECG and cardiac enzymes is useful if the clinical presentation nevertheless raises a reasonable likelihood of acute myocardial infarction or acute myocardial ischemia. In this assessment, multiple factors must be taken into account, especially in patients who do not have evident changes on the initial

ECG. For example, a patient is more likely to be having acute myocardial ischemia if the pain is clearly similar to prior known angina pectoris but somehow worse in terms of intensity, duration, or failure to respond to usual measures. In patients with no prior history of ischemic heart disease, multiple factors, including the patient's age and gender and the location and description of pain, influence whether further observation is required. New approaches to these patients include admission to chest pain observation units in which patients are watched for up to 6 to 12 h with serial testing. After this observation period, further cardiac testing with a standard exercise ECG, a myocardial perfusion scintiscan, or a stress echocardiogram may aid in establishing the diagnosis. In patients in whom no myocardial cause is evident, a musculoskeletal and/or gastrointestinal evaluation may be helpful, but such testing can usually be performed on an outpatient basis [7,19].

Acute aortic dissection may be suggested by the sudden onset of symptoms, the finding of asymmetric pulses, and a history of hypertension or Marfan's syndrome. The routine chest radiograph may show a dilated aortic root to suggest or support this diagnosis, which may then be established by transesophageal echocardiography, computed tomography, or magnetic resonance imaging. Aortography has traditionally been the definitive test, but, because of its invasiveness, it is often now replaced by magnetic resonance imaging in hemodynamically stable patients and preceded by a screening transesophageal echocardiogram in unstable patients.

Acute pulmonary embolism may be suggested on the basis of respiratory symptoms, hemoptysis, pleurisy chest discomfort, or a history of deep venous thrombosis or coagulation abnormalities. The evaluation usually requires a lung scan and/or pulmonary arteriogram to evaluate pulmonary perfusion.

Of the various potential gastrointestinal causes of chest discomfort, the most worrisome include a Mallory-Weiss esophageal tear, acute cholecystitis, pancreatitis, and a perforating gastric or duodenal ulcer. For peptic ulcer disease, cholecystitis, and pancreatitis, the abdominal examination usually is critical to the diagnosis. Mallory-Weiss tears are commonly associated with hematemesis. In the acute setting, esophageal disease may be a diagnosis of exclusion in patients who have no evidence for cardiac, pulmonary, or vascular causes. Upper gastrointestinal endoscopy or an upper gastrointestinal roentgenogram can diagnose esophageal or peptic ulcer disease. Esophageal manometry and measurement of lower esophageal sphincter pressure are useful in identifying esophageal spasm. The Bernstein acid perfusion test, in which an

attempt is made to reproduce the pain by infusion of hydrochloric acid into the esophagus, can help establish acid reflux as the cause of chest pain.

Recurrent Episodic Pain. In the patient with recurrent, episodic pain, the diagnostic and therapeutic tempo is different than in the patient with new, acute, and often ongoing pain. Although life-threatening conditions such as acute myocardial infarction, recurrent pulmonary emboli, or even aortic dissection can sometimes present in this way, patients with recurrent, episodic pain are more likely to have a variety of less critical diagnoses (Table 1).

In this setting, a detailed and meticulous history of the behavior of the pain is the cornerstone of the evaluation. One useful approach is to divide recurrent, episodic pain into those syndromes that represent a high likelihood for angina, those that are atypical but possibly represent angina, and those that are very unlikely to be caused by angina or myocardial ischemia. The history, physical examination, and subsequent diagnostic testing can be guided by this approach [22-24].

The *history* should focus on the behavior of the pain as the cornerstone of the evaluation. The location, radiation, quality, intensity, and duration of the episodes are important. Even more so is the story of the aggravating and alleviating factors. A history of intense aggravation by breathing, coughing, or other respiratory movements will usually point toward the pleura and pericardium or mediastinum as the site, although chest wall pain is likewise affected by respiratory motion. Similarly, a pain that regularly appears on rapid walking, or with other exertion such as sexual activity, and vanishes a few minutes after stopping suggests the diagnosis of angina pectoris, although a similar story will occasionally be obtained from patients with skeletal disorders.

A thorough *physical examination* can provide important clues to the cause of chest discomfort. Blood pressure should be checked in both arms if aortic dissection is being considered. Examination of the skin may reveal cyanosis, which suggests hypoxemia from either diminished cardiac output or impaired respiratory function, or xanthelasthma, which would suggest hyperlipidemia and associated coronary disease. The finding of lymphadenopathy suggests a tumor. The examination of the chest wall should include both inspection and palpation to search for costochondritis and other musculoskeletal abnormalities. Lung examination may reveal a pleural rub, signs of pneumonic consolidation, or evidence of congestive heart failure. The physical examination may be totally normal in persons with severe myocardial ischemia, but it also may demonstrate abnormalities of vital signs, a third or fourth heart sound, or

mitral regurgitation from papillary muscle dysfunction. Aortic stenosis will be accompanied by its typical murmur. The cardiac examination also should search for an increased pulmonic second sound that may indicate elevated pulmonary artery pressure, such as is found in pulmonary embolism, and the pericardial friction rub that strongly suggests pericarditis. A careful upper abdominal examination may be the first clue to peptic ulcer disease or cholecystitis [18-20].

Critical information can often be obtained by attempts to produce or alleviate the pain, such as with nitroglycerin. Careful palpation of the chest wall, subacromial bursa, deltoid tendon, abdomen, and other structures may be very helpful if it reproduces the chest discomfort. Shoulder and arm motion commonly reproduces pain related to these structures. Evaluation of the patient at the time of a spontaneous episode, such as with an electrocardiogram during pain, is also extremely helpful.

Persistent Chest Discomfort. Pain that persists unabated for many hours or days is more likely to represent a noncoronary cause, such as pericarditis, a musculoskeletal condition, a pulmonary abnormality, or one of several gastrointestinal conditions. Once again, a complete history and physical examination is mandatory, and further testing is guided by the findings (Table 2). If pericarditis is suspected, an ECG, chest radiograph, and echocardiogram may establish or exclude the diagnosis. Potential pulmonary conditions are commonly evaluated with a chest radiograph. Suspected musculoskeletal abnormalities may be confirmed, if necessary, by radiographic studies and/or the response to targeted therapies, including a local injection of lidocaine. Potential gastrointestinal causes may be evaluated with a variety of tests as described above. Although acute myocardial infarction, pulmonary embolism, and aortic dissection rarely present as persistent pain, these diagnoses must also be considered in patients in whom the more common causes of persistent pain are not found [20].

In considering diagnostic tests for all the various causes of chest discomfort, the clinician must remember that a useful test result is commonly one that moves the likelihood of a diagnostic possibility across a threshold, so the test result would lead to a change in management, either by influencing the decision to order additional tests or by causing a change in treatment. In the case of chest discomfort, the decisions cannot be based on a 50% threshold: Probabilities of coronary artery disease, pulmonary embolism, or aortic dissection that are well below 50% may still demand further evaluation because of the dire consequences of missing one of these important diagnoses. The physician must be prepared to embark on an appropriate evaluation when the history and physical examination do not exclude these diagnoses with a

reasonable degree of certainty. The degree of certainty must be determined for the individual condition and patient at hand, typically after an appropriately full and frank discussion between the patient and the physician.

Weighing the Evidence. The assessment of the probability of the various causes of chest pain requires the integration of multiple pieces of data, because no single clinical feature can be considered decisive. Each of the conditions that can cause chest discomfort can have varied presentations, and the diagnostic tests upon which physicians often rely can also have false-positive or false-negative results. Thus the principles of clinical reasoning should be applied to the evaluation of the patient with chest discomfort.

The information obtained from a careful medical history and physical examination can be used to develop a differential diagnosis of the causes of chest discomfort in an individual patient, to rank these diagnostic possibilities, and often to assign approximate percent probabilities to them. Although the various causes of chest discomfort have typical characteristics, these characteristics must be interpreted in light of the prior probability that a person with a given age and sex and with a particular past medical history would have such a cause of chest discomfort. For example, the possibility of angina pectoris as a cause of precordial or substernal discomfort must be seriously considered in a middle-aged man with coronary risk factors such as hypercholesterolemia and smoking, even if the description of the discomfort is not perfectly typical for angina pectoris. Conversely, when a 20-year-old woman describes the onset of new discomfort in a way that is seemingly classic for angina pectoris, such a diagnosis is relatively unlikely because the prior probability of ischemic heart disease, given her age and sex, is so low [22].

Although it is not always possible to assign numerical probabilities to the various causes of chest discomfort in an individual patient, experienced clinicians either implicitly or explicitly assess the relative likelihoods of various potential explanations for any chest discomfort syndrome to help guide their future diagnostic evaluations and therapy. For example, a middle-aged or elderly man with typical characteristics for angina pectoris has about an 85% probability of having hemodynamically significant coronary artery disease. By comparison, the same man with a history of chest discomfort that has some characteristics that are typical for angina pectoris but other characteristics that are atypical has a probability of important coronary disease ranging from about 30 to 60%. Even persons with chest pain that is decidedly unlikely to represent coronary disease still have some finite possibility of coronary disease, with a

likelihood ranging from exceedingly small in a young woman to somewhere in the 10% range in a middle-aged man with many coronary risk factors.

Diagnostic tests. Although myocardial ischemia commonly is associated with ECG changes, many patients have normal tracings between attacks, and in some the ECG may even be normal during an episode of pain. However, depression of the ST segments, caused by myocardial ischemia, typically occurs during exertion and is accompanied by anginal discomfort; moreover, ECG evidence of myocardial ischemia may occur at rest and with or without accompanying chest discomfort. The finding of fiat or down-sloping ST-segment depressions of 0.1 mV or greater during an attack of pain substantially increases the likelihood that the pain is anginal in origin. Exercise ECG will show ischemic changes in about 50 to 80% of persons with symptomatic coronary disease but also in about 10 to 15% of patients who do not have coronary disease. The accuracy of ambulatory ischemia monitoring in the general population is less clear [7,18].

Although exercise ECG and exercise thallium scintigraphy are of value in distinguishing between cardiac and non-cardiac causes of chest discomfort, the results must be interpreted in light of the prior probability of coronary artery disease, which is the probability that the patient has coronary disease based on the presenting clinical characteristics, age, and sex. Since exercise perfusion scintigraphy appears to provide information that is correlated with the standard exercise ECG no more than would be expected by chance, it can provide additional independent information and further change the probability of coronary artery disease. If absolute diagnostic knowledge is required, cardiac catheterization with coronary angiography serves as the gold standard - i.e., the test that is considered definitive regarding the presence or absence of coronary disease - even though the presence of anatomic disease does not guarantee that the coronary stenosis are causing the chest discomfort.

A sequence of consistently negative cardiologic test results reduces the probability of coronary artery disease to below 10% in patients with atypical chest discomfort. However, even after a normal exercise ECG and exercise perfusion scintigram, the probability of coronary disease will still be about 30% in a middle-aged or elderly patient with a typical history of angina pectoris. By recognizing the potential change in probabilities that can be obtained with positive and negative results of the diagnostic tests that are planned, the physician can decide whether these potential changes in probability are sufficient to warrant the test. For example, the physician should commonly decide that a patient with typical angina pectoris and a positive

exercise ECG does not require an exercise thallium scintigram to *diagnose* coronary disease, although under some circumstances this examination might aid in the estimation of the patient's subsequent prognosis [7].

Table 2

Approach to the Patient with Persistent Pain (Lasting Perhaps for Days)

1. Complete history and physical examination
2. Testing guided by data may include:
   - ECG
   - Chest radiograph
   - Computed tomography of chest
   - Gastrointestinal evaluation
   - Spine, shoulder, or rib radiographs
   - Echocardiogram

Palpitations are a common, disagreeable symptom that may be defined as an awareness of the beating of the heart, an awareness most commonly brought about by a change in the heart's rhythm or rate or by an augmentation of its contractility. Palpitations are not pathognomonic of any particular group of disorders; indeed, often they signify not a primary physical disorder but rather a psychological disturbance. Even when they occur as a more or less prominent complaint, the diagnosis of the underlying disease is made largely on the basis of other associated symptoms and data. Nevertheless, palpitations are frequently of considerable importance in the minds of patients, who fear they may indicate heart disease. Concern is all the more pronounced in patients who have been told that they *may* have heart disease; to them palpitations may seem to be an omen of impending disaster. Since the resulting anxiety may be associated with increased activity of the autonomic nervous system, with consequent increases in the cardiac rate and rhythm and the vigor of contraction, the patient's awareness of these changes may then lead to a vicious cycle, which may ultimately be responsible for incapacitation [18].

Palpitations may be described by the patient in various terms, such as «pounding», "fluttering", "flopping", and "skipping", and in most cases it will be obvious that the complaint is of a sensation of disturbed heartbeat. The sensitivity to alterations in cardiac activity among different individuals varies widely. Some patients seem to be unaware of the most serious and chaotic dysrhythmias; others are seriously troubled by an occasional extrasystole. Patients with anxiety states often exhibit a lowered threshold at which disorders of rate and rhythm result in palpitation. The

awareness of the heartbeat also tends to be more common at night and during introspective moments than during activity. Patients with organic heart disease and chronic disorders of cardiac rate, rhythm, or stroke volume tend to accommodate to these abnormalities and are often less sensitive than normal persons to such events. Persistent tachycardia and/or atrial fibrillation may not be accompanied by continuous palpitations, in contrast to a sudden, brief alteration in cardiac rate or rhythm, which often causes considerable subjective discomfort. Palpitations are particularly prominent when the precipitating cause for increased heart rate or contractility or arrhythmia is recent, transient, and episodic. Conversely, in emotionally well- adjusted individuals, palpitations commonly become progressively less disconcerting as they become more chronic [21].

Differential diagnosis. The patient's description of palpitations represents the most important clue to their etiology. Every effort should be made to ask the patient to check the radial pulse during episodes of palpitation to assist in the diagnosis. If the rhythm is steady and regular and the rate is normal, the patient may be aware of an abnormal stroke volume from a condition such as aortic regurgitation but more likely is reporting a heightened awareness of normal cardiac function, sometimes in response to routine stress. Conversely, if the rhythm is steady and regular but the rate is clearly increased, especially above 120 beats per minute at a time when activity or other stresses should not result in tachycardia, the palpitations may well represent a supraventricular or sometimes even a ventricular tachycardia.

An irregular pulse noted during an episode of palpitation virtually always represents a true arrhythmia. The arrhythmia may be as simple as an occasional extrasystole. The premature contraction and post- premature beat after an extrasystole are often described as a "flopping", or the patient may say that it feels as if "the heart were turning over". The pause following the premature contraction may be felt as an actual cessation of the heart beat. The first ventricular contraction after the pause may be felt as an unusually vigorous beat and often be described as "pounding" or "thudding". By the patient's account alone, numerous extrasystoles may be difficult to distinguish from atrial fibrillation. Bursts of beats may represent atrial fibrillation or brief episodes of a supraventricular or ventricular tachycardia [23].

Ectopic tachycardia commonly begins instantaneously and stops suddenly, sometimes followed by a pause before sinus rhythm resumes. Patients are usually acutely aware of both the onset and ending of the arrhythmia. A variety of vagal

maneuvers may already have been used by the patient or may be suggested by the physician to aid in cessation of the ectopic arrhythmia.

All of the various arrhythmic causes of palpitations may be more common in the setting of thyrotoxicosis, hypoglycemia, pheochromocytoma, fever, and certain drugs. These arrhythmias may also be precipitated by tobacco, coffee, tea, alcohol, epinephrine, ephedrine, aminophylline, atropine, or thyroid medication. The physical examination should include a search for evidence of valvular heart disease, hyperthyroidism, and pulmonary disease.

Associated symptoms. While palpitations are extremely common in healthy individuals, regardless of whether they are caused by a true arrhythmia or not, the presence of associated signs, symptoms, or other conditions helps to determine the potential seriousness of palpitations. Palpitations associated with symptoms of dizziness, shortness of breath, or chest discomfort, or occurring in a patient with an underlying history of cardiac disease, often require further evaluation. In such individuals, palpitations may be a manifestation of important arrhythmias that influence prognosis and, in some situations, require treatment [7,18].

Approach to the Patient. The most common initial test for a patient with palpitations is a continuous Holter monitor, especially if the palpitations occur on a daily basis. For individuals with less frequent symptoms, a variety of loop recorders can be worn for days and activated by the patient at the time that the palpitations are experienced. It should be remembered that many patients have asymptomatic irregularities of the heart rhythm.

In addition to ambulatory ECG monitoring, exercise testing is sometimes of value in reassuring both the patient and the physician that vigorous exercise does not produce a worrisome arrhythmia. In general, arrhythmias that disappear with exercise are more likely to be more benign than those that are precipitated by it.

The management of palpitations focuses on the treatment of specifically diagnosed arrhythmias, on the elimination of precipitating factors (medications, tobacco, coffee, tea, alcohol), or on the recognition of associated syndromes, including hyperthyroidism, pheochromocytoma, hypoglycemia, menopausal symptoms, or an anxiety state.

As a rule, palpitations themselves often produce anxiety and fear out of proportion to the seriousness to the underlying condition. When the cause has been accurately determined and the significance is explained to the patient, concern is often ameliorated or eliminated. In patients with no important cause, evaluation and

appropriate reassurance may be critical to helping the patient regain confidence and return to a normal quality of life [7,19].

Abdominal pain. The correct interpretation of acute abdominal pain is one of the most challenging demands made of any physician. Since proper therapy may require urgent action, the luxury of the leisurely approach suitable for the study of other conditions is sometimes denied. Few other clinical situations demand greater experience and judgment, because the most catastrophic of events may be forecast by the subtlest of symptoms and signs. Nowhere in medicine is a meticulously executed, detailed history and physical examination of greater importance. The etiologic classification in Table 3, although not complete, forms a useful frame of reference for the evaluation of patients with abdominal pain [25].

The diagnosis of "acute or surgical abdomen" so often heard in emergency wards is not an acceptable one because of its often misleading and erroneous connotation. The most obvious of "acute abdomens" may not require operative intervention, and the mildest of abdominal pains may herald the onset of an urgently correctable lesion. Any patient with abdominal pain of recent onset requires early and thorough evaluation with specific attempts at accurate diagnosis.

Some mechanisms of pain originating in the abdomen Inflammation of the Parietal Peritoneum. The pain of parietal peritoneal inflammation is steady and aching in character and is located directly over the inflamed area, its exact reference being possible because it is transmitted by somatic nerves supplying the parietal peritoneum. The intensity of the pain is dependent on the type and amount of foreign substance to which the peritoneal surfaces are exposed in a given period of time. For example, the sudden release into the peritoneal cavity of a small quantity of *sterile* acid gastric juice causes much more pain than the same amount of grossly contaminated neutral fecal material. Enzymatically active pancreatic juice incites more pain and inflammation than does the same amount of sterile bile containing no potent enzymes. Blood and urine are often so bland as to go undetected if exposure of the peritoneum has not been sudden and massive. In the case of bacterial contamination, such as in pelvic inflammatory disease, the pain is frequently of low intensity early in the illness until bacterial multiplication has caused the elaboration of irritating substances [31].

So important is the rate at which the irritating material is applied to the peritoneum that cases of perforated peptic ulcer may be associated with entirely different clinical pictures dependent only on the rapidity with which the gastric juice enters the peritoneal cavity.

The pain of peritoneal inflammation is invariably accentuated by pressure or changes in tension of the peritoneum, whether produced by palpation or by movement, as in coughing or sneezing. The patient with peritonitis lies quietly in bed, preferring to avoid motion, in contrast to the patient with colic, who may writhe incessantly.

Another characteristic feature of peritoneal irritation is tonic reflex spasm of the abdominal musculature, localized to the involved body segment. The intensity of the tonic muscle spasm accompanying peritoneal inflammation is dependent on the location of the inflammatory process, the rate at which it develops, and the integrity of the nervous system. Spasm over a perforated retrocecal appendix or perforated ulcer into the lesser peritoneal sac may be minimal or absent because of the protective effect of overlying viscera. A slowly developing process also often greatly attenuates the degree of muscle spasm. Catastrophic abdominal emergencies such as a perforated ulcer have been repeatedly associated with minimal or occasionally no detectable pain or muscle spasm in obtunded, seriously ill, debilitated elderly patients or in psychotic patients [30].

Obstruction of Hollow Viscera The pain of obstruction of hollow abdominal viscera is classically described as intermittent, or colicky. Yet the lack of a truly cramping character should not be misleading, because distention of a hollow viscus may produce steady pain with only very occasional exacerbations. Although not nearly as well localized as the pain of parietal peritoneal inflammation, some useful generalities can be made concerning its distribution.

Table 3

Some Important Causes of Abdominal Pain

PAIN ORIGINATING IN THE ABDOMEN

1. Parietal peritoneal inflammation
    a. Bacterial contamination, e.g., perforated appendix, pelvic inflammatory disease
    b. Chemical irritation, e.g., perforated ulcer, pancreatitis, mittelschmerz
2. Mechanical obstruction of hollow viscera
    a. Obstruction of the small or large intestine
    b. Obstruction of the biliary tree
    c. Obstruction of the ureter
3. Vascular disturbances
    a. Embolism or thrombosis
    b. Vascular rupture
    c. Pressure or torsional occlusion
    d. Sickle cell anemia

4. Abdominal wall
    a. Distortion or traction of mesentery
    b. Trauma or infection of muscles
5. Distention of visceral surfaces, e.g., hepatic or renal capsules

PAIN REFERRED FROM INTRAABDOMINAL SOURCE

1. Thorax, e.g., pneumonia, referred pain from coronary occlusion
2. Spine, e.g., radiculitis from arthritis
3. Genitalia, e.g., torsion of the testicle

METABOLIC CAUSES

1. Exogenous
    a. Black widow spider bite
    b. Lead poisoning and others
2. Endogenous
    a. Uremia
    b. Diabetic ketoacidosis
    c. Porphyria
    d. Allergic factors (Cl-esterase inhibitor deficiency)

NEUROGENIC CAUSES

1. Organic
    a. Tabes dorsalis
    b. Herpes zoster
    c. Causalgia and others
2. Functional

The colicky pain of obstruction of the small intestine is usually periumbilical or supraumbilical and is poorly localized. As the intestine becomes progressively dilated with loss of muscular tone, the colicky nature of the pain may become less apparent. With superimposed strangulating obstruction, pain may spread to the lower lumbar region if there is traction on the root of the mesentery. The colicky pain of colonic obstruction is of lesser intensity than that of the small intestine and is often located in the infraumbilical area. Lumbar radiation of pain is common in colonic obstruction [7].

Sudden distention of the biliary tree produces a steady rather than colicky type of pain; hence the term *biliary colic* is misleading. Acute distention of the gallbladder usually causes pain in the right upper quadrant with radiation to the right posterior region of the thorax or to the tip of the right scapula, and distention of the common bile duct is often associated with pain in the epigastrium radiating to the upper part of the lumbar region. Considerable variation is common, however, so that differentiation between these may be impossible. The typical subscapular pain or lumbar radiation is frequently absent. Gradual dilatation of the biliary tree, as in carcinoma of the head of the pancreas, may cause no pain or only a mild aching sensation in the epigastrium or

right upper quadrant. The pain of distention of the pancreatic ducts is similar to that described for distention of the common bile duct but, in addition, is very frequently accentuated by recumbency and relieved by the upright position.

Obstruction of the urinary bladder results in dull suprapubic pain, usually low in intensity. Restlessness without specific complaint of pain may be the only sign of a distended bladder in an obtunded patient. In contrast, acute obstruction of the intravesicular portion of the ureter is characterized by severe suprapubic and flank pain which radiates to the penis, scrotum, or inner aspect of the upper region of the thigh. Obstruction of the ureteropelvic junction is felt as pain in the costovertebral angle, whereas obstruction of the remainder of the ureter is associated with flank pain that often extends into the corresponding side of the abdomen [27].

Vascular Disturbances. A frequent misconception, despite abundant experience to the contrary, is that pain associated with intraabdominal vascular disturbances is sudden and catastrophic in nature. The pain of embolism or thrombosis of the superior mesenteric artery or that of impending rupture of an abdominal aortic aneurysm certainly may be severe and diffuse. Yet, just as frequently, the patient with occlusion of the superior mesenteric artery has only mild continuous diffuse pain for 2 or 3 days before vascular collapse or findings of peritoneal inflammation appear. The early, seemingly insignificant discomfort is caused by hyperperistalsis rather than peritoneal inflammation. Indeed, absence of tenderness and rigidity in the presence of continuous, diffuse pain in a patient likely to have vascular disease is quite characteristic of occlusion of the superior mesenteric artery. Abdominal pain with radiation to the sacral region, flank, or genitalia should always signal the possible presence of a rupturing abdominal aortic aneurysm. This pain may persist over a period of several days before rupture and collapse occur [29].

Abdominal Wall. Pain arising from the abdominal wall is usually constant and aching. Movement, prolonged standing, and pressure accentuate the discomfort and muscle spasm. In the case of hematoma of the rectus sheath, now most frequently encountered in association with anticoagulant therapy, a mass may be present in the lower quadrants of the abdomen. Simultaneous involvement of muscles in other parts of the body usually serves to differentiate myositis of the abdominal wall from an intraabdominal process that might cause pain in the same region.

Referred pain in abdominal diseases. Pain referred to the abdomen from the thorax, spine, or genitalia may prove a vexing diagnostic problem, because diseases of the upper part of the abdominal cavity such as acute cholecystitis or perforated ulcer are

frequently associated with intrathoracic complications. A most important, yet often forgotten dictum is that the possibility of intrathoracic disease must be considered in every patient with abdominal pain, especially if the pain is in the upper part of the abdomen. Systematic questioning and examination directed toward detecting the presence or absence of myocardial or pulmonary infarction, pneumonia, pericarditis, or esophageal disease (the intrathoracic diseases that most often masquerade as abdominal emergencies) will often provide sufficient clues to establish the proper diagnosis. Diaphragmatic pleurisies resulting from pneumonia or pulmonary infarction may cause pain in the right upper quadrant and pain in the supraclavicular area, the latter radiation to be sharply distinguished from the referred subscapular pain caused by acute distention of the extrahepatic biliary tree. The ultimate decision as to the origin of abdominal pain may require deliberate and planned observation over a period of several hours, during which time repeated questioning and examination will provide the proper explanation [30,31].

Referred pain of thoracic origin is often accompanied by splinting of the involved hemithorax with respiratory lag and decrease in excursion more marked than that seen in the presence of intraabdominal disease. In addition, apparent abdominal muscle spasm caused by referred pain will diminish during the inspiratory phase of respiration, whereas it is persistent throughout both respiratory phases if it is of abdominal origin. Palpation over the area of referred pain in the abdomen also does not usually accentuate the pain and in many instances actually seems to relieve it. The frequent coexistence of thoracic and abdominal disease may be misleading and confusing, so differentiation may be difficult or impossible. For example, the patient with known biliary tract disease often has epigastric pain during myocardial infarction, or biliary colic may be referred to the precordium or left shoulder in a patient who has suffered previously from angina pectoris.

Referred pain from the spine, which usually involves compression or irritation of nerve roots, is characteristically intensified by certain motions such as cough, sneeze, or strain and is associated with hyperesthesia over the involved dermatomes. Pain referred to the abdomen from the testicles or seminal vesicles is generally accentuated by the slightest pressure on either of these organs. The abdominal discomfort is of dull aching character and is poorly localized [26].

Metabolic abdominal crises. Pain of metabolic origin may simulate almost any other type of intraabdominal disease. Here several mechanisms may be at work. In certain instances, such as hyperlipidemia, the metabolic disease itself may be

accompanied by an intraabdominal process such as pancreatitis, which can lead to unnecessary laparotomy unless recognized. Cl-esterase deficiency associated with angioneurotic edema is also often associated with episodes of severe abdominal pain. Whenever the cause of abdominal pain is obscure, a metabolic origin always must be considered. Abdominal pain is also the hallmark of familial Mediterranean fever.

The problem of differential diagnosis is often not readily resolved. The pain of porphyria and of lead colic usually is difficult to distinguish from that of intestinal obstruction, because severe hyperperistalsis is a prominent feature of both. The pain of uremia or diabetes is nonspecific, and the pain and tenderness frequently shift in location and intensity. Diabetic acidosis may be precipitated by acute appendicitis or intestinal obstruction, so if prompt resolution of the abdominal pain does not result from correction of the metabolic abnormalities, an underlying organic problem should be strongly suspected. Black widow spider bites produce intense pain and rigidity of the abdominal muscles and of the back, an area infrequently involved in disease of intraabdominal origin [29].

Neurogenic causes. Causalgic pain may occur in diseases that injure nerves of sensory type. It has a burning character and is usually limited to the distribution of a given peripheral nerve. Normal stimuli such as touch or change in temperature may be transformed into this type of pain, which is also frequently present in a patient at rest. A helpful finding is the demonstration that cutaneous pain spots are irregularly spaced, and this may be the only indication of an old nerve lesion underlying causalgic pain. Even though the pain may be precipitated by gentle palpation, rigidity of the abdominal muscles is absent, and the respirations are not disturbed. Distention of the abdomen is uncommon, and the pain has no relationship to the intake of food.

Pain arising from spinal nerves or roots comes and goes suddenly and is of a lancinating type. It may be caused by herpes zoster, impingement by arthritis, tumors, herniated nucleus pulposus, diabetes, or syphilis. Again, it is not associated with food intake, abdominal distention, or changes in respiration. Severe muscle spasm, as in the gastric crises of tabes dorsalis, is common but is either relieved or is not accentuated by abdominal palpation. The pain is made worse by movement of the spine and is usually confined to a few dermatome segments. Hyperesthesia is very common [25].

Psychogenic pain conforms to none of the aforementioned patterns of disease. Here the mechanism is hard to define. The most common problem is the hysterical adolescent or young person who develops abdominal pain and who frequently loses an appendix or other organs because of it. Ovulation or some other natural event that

causes brief mild abdominal discomfort may sometimes be experienced as an abdominal catastrophe.

Psychogenic pain varies enormously in type and location but usually has no relation to meals. It is often at its onset markedly accentuated during the night. Nausea and vomiting are rarely observed, although occasionally the patient reports these symptoms. Spasm is seldom induced in the abdominal musculature and, if present, does not persist, especially if the attention of the patient can be distracted. Persistent localized tenderness is rare, and if found, the muscle spasm in the area is inconsistent and often absent. Restriction of the depth of respiration is the most common respiratory abnormality, but this is in the nature of a smothering or choking sensation and is part of an anxiety state. It occurs in the absence of thoracic splinting or change in the respiratory rate [28].

Approach to the Patient. There are few abdominal conditions that require such urgent operative intervention that an orderly approach need be abandoned, no matter how ill the patient. Only those patients with exsanguinating hemorrhage must be rushed to the operating room immediately, but in such instances, only a few minutes are required to assess the critical nature of the problem. Under these circumstances, all obstacles must be swept aside, adequate access for intravenous fluid replacement obtained, and the operation begun. Many patients of this type have died in the radiology department or the emergency room while awaiting such unnecessary examinations as electrocardiograms or films of the abdomen. *There are no contraindications to operation when massive hemorrhage is present.* Although exceedingly important, this situation fortunately is relatively rare.

Nothing will supplant an orderly, painstakingly *detailed history,* which is far more valuable than any laboratory or roentgenologic examination. This kind of history is laborious and time-consuming, making it not especially popular, even though a reasonably accurate diagnosis can be made on the basis of the history alone in the majority of cases. Recent studies of computer-aided diagnosis of abdominal pain indicate that this technique provides no advantage over clinical assessment alone. In cases of *acute* abdominal pain, a diagnosis is readily established in most instances, whereas success is not so frequently achieved in patients with *chronic* pain. Since the irritable bowel syndrome is one of the most common causes of abdominal pain, the possibility of this diagnosis must always be kept in mind. The *chronological sequence of events* in the patient's history is often more important than emphasis on the location of pain. If the examiner is sufficiently open-minded and unhurried, asks the proper

questions, and listens, the patient will usually provide the diagnosis. Careful attention should be paid to the extraabdominal regions that may be responsible for abdominal pain. An accurate menstrual history in a female patient is essential. Narcotics or analgesics should be withheld until a definitive diagnosis or a definitive plan has been formulated, because these agents often make it more difficult to secure and to interpret the history and physical findings [28-30].

In the examination, simple critical inspection of the patient, e.g., of facies, position in bed, and respiratory activity, may provide valuable clues. The amount of information to be gleaned is directly proportional to the *gentleness* and thoroughness of the examiner. Once a patient with peritoneal inflammation has been examined brusquely, accurate assessment by the next examiner becomes almost impossible. For example, eliciting rebound tenderness by sudden release of a deeply palpating hand in a patient with suspected peritonitis is cruel and unnecessary. The same information can be obtained by gentle percussion of the abdomen (rebound tenderness on a miniature scale), a maneuver that can be far more precise and localizing. Asking the patient to cough will elicit true rebound tenderness without the need for placing a hand on the abdomen. Furthermore, the forceful demonstration of rebound tenderness will startle and induce protective spasm in a nervous or worried patient in whom true rebound tenderness is not present. A palpable gallbladder will be missed if palpation is so brusque that voluntary muscle spasm becomes superimposed on involuntary muscular rigidity.

As in history taking, there is no substitute for sufficient time spent in the examination. It is important to remember that abdominal signs may be minimal but nevertheless, if accompanied by consistent symptoms, may be exceptionally meaningful. Signs may be virtually or actually totally absent in cases of pelvic peritonitis, so careful *pelvic and rectal examinations are mandatory in every patient with abdominal pain.* The presence of tenderness on pelvic or rectal examination in the absence of other abdominal signs must lead the examiner to consider such important operative indications as perforated appendicitis, diverticulitis, twisted ovarian cyst, and many others [25].

Much attention has been paid to the presence or absence of peristaltic sounds, their quality, and their frequency. Auscultation of the abdomen is probably one of the least rewarding aspects of the physical examination of a patient with abdominal pain. Severe catastrophes, such as strangulating small intestinal obstruction or perforated appendicitis, may occur in the presence of normal peristalsis. Conversely, when the

proximal part of the intestine above an obstruction becomes markedly distended and edematous, peristaltic sounds may lose the characteristics of borborygmi and become weak or absent even when peritonitis is not present. It is usually the severe chemical peritonitis of sudden onset that is associated with the truly silent abdomen. Assessment of the patient's state of hydration is important. The hematocrit and urinalysis permit an accurate estimate of the severity of dehydration so that adequate replacement can be carried out.

Laboratory examinations may be of enormous value in assessment of the patient with abdominal pain, yet with but a few exceptions they rarely establish a diagnosis. Leukocytosis should never be the single deciding factor as to whether or not operation is indicated. A white blood cell count greater than 20 000xL may be observed with perforation of a viscus, but pancreatitis, acute cholecystitis, pelvic inflammatory disease, and intestinal infarction may be associated with marked leukocytosis. A normal white blood cell count is by no means rare in cases of perforation of abdominal viscera. The diagnosis of anemia may be more helpful than the white blood cell count, especially when combined with the history [27,28].

The urinalysis is also of great value in indicating to some degree the state of hydration or to rule out severe renal disease, diabetes, or urinary infection. Determination of the blood urea nitrogen, blood sugar, and serum bilirubin levels also may be helpful. The serum amylase determination is overrated. Since many diseases other than pancreatitis, e.g., perforated ulcer, strangulating intestinal obstruction, and acute cholecystitis, may be associated with very marked increase in the serum amylase, great care must be exercised in denying an operation to a patient solely on the basis of an elevated serum amylase level. The determination of the serum lipase may have a somewhat greater accuracy than the serum amylase.

Plain and upright or lateral decubitus roentgenograms of the abdomen may be of the greatest value. They are usually unnecessary in patients with acute appendicitis or strangulated external hernias. However, in cases of intestinal obstruction, perforated ulcer, and a variety of other conditions, films may be diagnostic. In rare instances, barium or water-soluble medium examination of the upper part of the gastrointestinal tract may demonstrate partial intestinal obstruction that may elude diagnosis by other means. If there is any question of obstruction of the colon, oral administration of barium sulfate should be avoided. On the other hand, barium enema is of inestimable value in cases of colonic obstruction and should be used with greater frequency where the possibility of perforation does not exist [29].

Peritoneal lavage is a safe and effective diagnostic maneuver in patients with acute abdominal pain. It is of special value in patients with blunt trauma to the abdomen, in whom evaluation of the abdomen may be difficult because of other multiple injuries to the spine, pelvis, or ribs and in whom blood in the peritoneal cavity produces only a very mild peritoneal reaction. In the absence of trauma, peritoneal lavage has been replaced by ultrasound, computed tomography (CT), and laparoscopy. Ultrasonography has proved to be useful in detecting an enlarged gallbladder or pancreas, the presence of gallstones, an enlarged ovary, or a tubal pregnancy. Laparoscopy is especially helpful in diagnosing pelvic conditions, such as ovarian cysts, tubal pregnancies or salpingitis, and acute appendicitis. Radioisotopic scans (HIDA) may help differentiate acute cholecystitis from acute pancreatitis. A CT scan may demonstrate an enlarged pancreas, ruptured spleen, or thickened colonic wall and streaking of the mesocolon characteristic of diverticulitis [31].

Sometimes, even under the best of circumstances with all available auxiliary aids and with the greatest of clinical skill, a definitive diagnosis cannot be established at the time of the initial examination. Nevertheless, despite lack of a clear anatomic diagnosis, it may be abundantly clear to an experienced and thoughtful physician and surgeon that on clinical grounds alone operation is indicated. Should that decision be questionable, watchful waiting with repeated questioning and examination will often elucidate the true nature of the illness and indicate the proper course of action.

Differential diagnosis of chest pain:

- Chest wall pain
- Pulmonary causes
- Cardiac causes
- Vascular causes
- GI causes
- Other (psychogenic causes).

I. Chest wall pain

- **Herpes zoster**

- Reactivation of Herpes zoster
- Immunocompromised patients with risk of reactivation.
- Herpes zoster infections involve the trunk (in 60%)
- Rash may precede pain

- Clusters of vesicles (with clear or purulent fluid) on erythematous base. Lesions with rupture and crust.

- distribution in dermatome.

- Usually unilateral involvement.

Treatment:

- Antivirals: reduce duration of symptoms. May also reduce incidence of postherpatic neuralgia.
- +/- corticosteroids: May reduce inflammation
- Analgesia

- **Post herpetic neuralgia:**

* May follow course of acute Herpes zoster
* Shooting intensive pain.
* Hyperesthesia in involved dermatomes
* Treatment: analgesics, antidepressants, gabapentin.

- **Musculoskeletal Pain**

- Usually localized, acute, positional

- Pain often reproducible by palpation

- pain reproduced by turning or arm movement

- May elicit history of repetitive or unaccustomed activity involving trunk/arms

- Rheumatic diseases will cause musculoskeletal pain via thoracic joint involvement.

DIAGNOSIS	CLINICAL FEATURES
COSTOCHONDRITIS →	Inflammation of costal cartilages +/- sternal articulations. No swelling
TIETZE SYNDROME →	Painful swelling in one or more upper costal cartilages.
XIPHODYNIA →	Discomfort over xyphoid reproduced by palpation
PRECORDIAL CATCH SYNDROME →	Sharp pain lasting for 1-2 min episodes near the cardiac apex and associated with inspiration, poor posture, and inactivity
RIB FRACTURE →	Pain over involved rib

II. Pulmonary causes of chest pain

- **Pulmonary embolism**

Risk factors:

- Virchow's triad

- Hypercoagulation

*Malignancy

*Pregnancy, Early Postpartum, OCPs, HRT

*Genetic Mutations: Factor V Leiden, Prothrombin, Protein C or S deficiencies, antiphospholipid Ab, etc

- Venous Stasis

* Long distance travel

* Prolonged bed rest or hospitalization

* Cast

- Venous Injury: Recent surgery or Trauma

Clinical features

- Hypopnoe

- Chest pain: often pleurisy

- Tachycardia, tachypnea, hypoxemia

- Hemoptysis, Cough

- Consider diagnosis - A fib

- Look for asymmetric leg swelling (deep vein thrombosis signs) as risk factor for PE

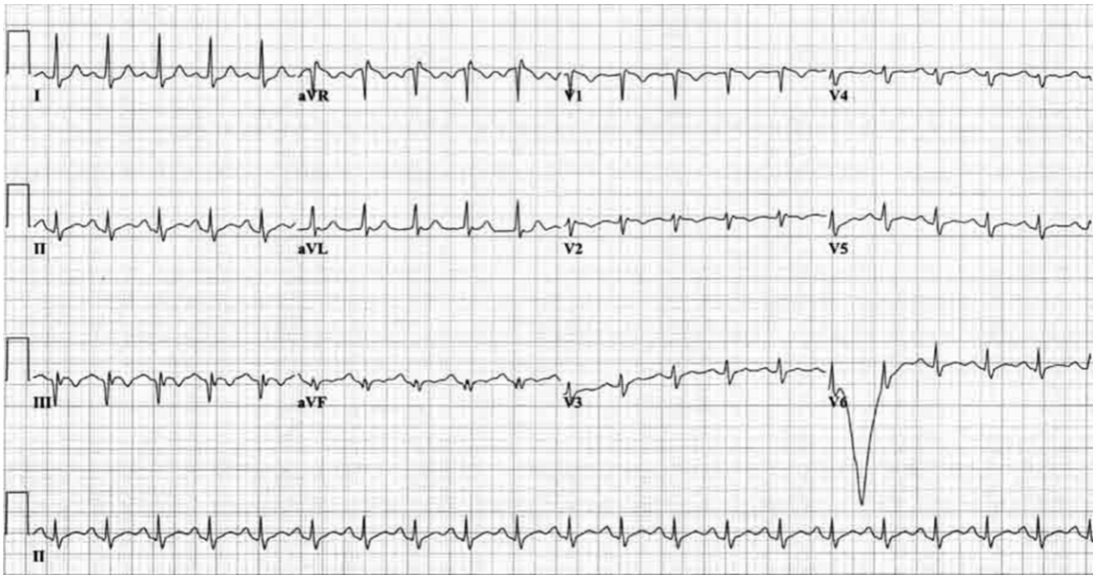
- If massive PE, may present with hypotension, unstable vital signs, and acute cor pulmonale. Also may present with cardiac arrest (PEA >> asystole).

ECG:

- most common sinus tachycardia;

- Often see nonspecific abnormalities;

- Look for S1-Q3-T3 (S wave in lead I, Q wave in lead III, inverted T wave in lead III).



Chest x-ray:

- Normal in 25% of cases;
- Often nonspecific findings;
- Look for Hampton's Hump (triangular pleural based density with apex pointed towards hilum): sign of pulmonary infarction;

-Look for Westermark's sign: Dilation of pulmonary vessels proximal to embolism and collapse of distal.

ABG: *Look for abnormal PaO2 or A-a gradient.

D-Dimer:

*Often elevated in PE.

* Useful test in low probability patients.

*May be abnormally high in various conditions: Malignancy, Pregnancy, sepsis, surgery.

Ventilation-Perfusion scan- use in setting of renal insufficiency.

Helical CT scan with IV contrast.

Pulmonary angiography: Gold Standard !

Treatment: Initiate Heparin.

* Unfractionated Heparin: 80 Units/kg bolus IV, then 18 units/kg/hr.

* Fractionated Heparin: 1mg/kg SubQ BID.

* If high pre-test probability for PE, initiate empiric heparin while waiting for imaging.

* Make sure no intraparenchymal brain hemorrhage or GI hemorrhage prior to initiating heparin.

- Consider Fibrinolytic Therapy:

* Especially if PE + hypotension.

▪ **Pneumonia.** Clinical features.

- Cough +/- sputum production.

- Fevers/chills.

- Pleurisy chest pain.

- hypopnoe.

- May be preceded by viral URI symptoms.

- Weakness/malaise/ myalgias.

- If severe: tachycardia, tachypnea, hypotension.

- Decreased Sats.

-Abnormal findings on pulmonary auscultation: (rale, decreased breath sounds, wheezing, rhonchi).

Treatment. Community- acquired.

- Outpatient:

* Doxycycline: Low cost option.

* Macrolide.

* Newer fluoroquinolone: Moxifloxacin, Levofloxacin, Gatifloxacin.

- Inpatient:

* Second or third generation cephalosporin +macrolide.

* Fluoroquinolone: Avelox.

- Nursing Home.

* Zosyn + Erythromcyin.

* Clindamycin + Cipro.

▪ **Spontaneous pneumothorax (PTX)**

Risk factors:

- Primary:

* No underlying lung disease.

* Young male with greater height to weight ratio.

* Smoking: 20:1 relative risk compared to nonsmokers.

- Secondary:

* COPD (chronic obstructive pulmonary disease).

* Cystic Fibrosis.

* AIDS/PCP.

* Neoplasms.

Clinical features.

- Acute pleuritic chest pain (95%).
- Usually pain localized to side of PTX.
- Dyspnea.
- May see tachycardia or tachypnea.
- Decreased breath sounds on side of PTX.
- Hyperresonance on side of PTX.
- If tension PTX, will have above findings + tracheal deviation + unstable vital signs. This is rare complication with spontaneous PTX.

- **Tension Pneumothorax**
  - Trachea deviates to contralateral side.
  - Mediastinum shifts to contralateral side.
  - Decreased breath sounds and hyperresonance on affected side.
  - Treatment: Emergent needle decompression followed by chest tube insertion.
- **Pleurisy / Serositis:**

Inflammation of pleura that covers lung. Pleuritic chest pain.

Causes:

- Viral etiology;
- SLE;
- Rheumatoid Arthritis;
- Drugs causing lupus-like reaction: Procainamide, Hydralazine, Isoniazid.

Clinical features:

- Decreased O2 saturations;
- Hypopnoe;
- May be chest pain;
- Decreased breath sounds, wheezing, or prolonged expiratory phase;
- Look for accessory muscle use (nasal flaring, tracheal tugging, retractions).

Order CXR to r/o associated complications: PTX, pneumonia that may have led to exacerbation.

- **COPD/asthma exacerbations**

Clinical features:

- Decreased O2 saturations;
- hypopnoe;
- May see chest pain;
- Decreased breath sounds, wheezing, or prolonged expiratory phase on exam;

- Look for accessory muscle use (nasal flaring, tracheal tugging, retractions).

Order CXR to r/o associated complications: PTX, pneumonia that may have led to exacerbation

Treatment

- Oxygen: Must prevent hypoxemia. Watch for hypercapnia with O2 therapy.
- β2 agonist (albuterol).
- Anticholinergic (atrovent).
- Corticosteroids.
- Consider Abx if: change in sputum or fever.
- If patient is tiring out, not oxygenating well despite O2, developing worsening respiratory acidosis or mental status changes, then intubate.

III. Cardiac causes of chest pain

▪ Ischemic chest pain

Clinical features

- Chest pain: often described as constricting, heaviness, gripping, squeezing.
- Pain usually substernal or in left chest.
- Pain spreading to neck, jaw, and arm.
- Associated symptoms: nausea, vomiting, diaphoresis, hypopnoe, palpitations.
- In appropriate setting, consider above associated symptoms, as well as neck/jaw/arm pain, and epigastric pain as ischemic equivalents.
- Pain may be associated with activity.
- Symptoms may improve with rest.
- exertional angina
  * brief episodes brought on by exertion and relieved by rest.
- Unstable angina
  * new onset;
  * change of frequency/severity;
  * pain at rest: AMI;
  * severe persistent symptoms;
  * elevated troponin level.

12 lead ECG:

- Look for ST segment elevation (at leas 1mm in two contiguous leads).
- Look for ST segment depression.

- Look for T wave inversions.
- Look for Q waves.
- Look for new LBBB.
- Always compare with previous ECGs.

ACUTE MYOCARDIAL INFARCTION

TERRITORY	CORONARY ARTERY	EKG
INFERIOR	RCA	II, III, AVF
ANTERIOR	LAD	V2-4
LATERAL	CIRCUMFLEX	V5-6, I, AVL
POSTERIOR	VARIABLE	TALL R WAVE IN V1/2 OR ST SEGMENT DEPRESSION

Cardiac enzymes

- Myoglobin: will rise within 3 hrs, peak within 4-9 hrs, and return to baseline within 24 hrs.
- CKMB: will rise within 4 hrs, peak within 12- 24 hrs and return to baseline in 2-3 days.
- TROPONIN I: will rise within 6 hrs, peak in 12 hrs and return to baseline in 3-4 days.

Ischemic heart disease treatment: acute ST segment elevation MI

- Oxygen;
- aspirin - IV nitroglycerin:
  * Hold for SBP <100;
  * Use cautiously in inferior wall MI. Some of these patients have Right ventricular involvement which is volume/preload dependent.
- Beta blockers:
  * Hold for SBP <100 or HR <60;
  * Hold if wheezing;
  * Hold if cocaine use (unopposed alpha).
- Morphine.
- Heparin: Before starting:
  * Rectal exam;

* Check CXR: to r/o dissection - cath lab vs tpa.

Ischemic heart disease treatment: unstable angina:

- Oxygen;
- Aspirin;
- Nitroglycerin:
  * Hold for SBP <100;
  * Use cautiously in myocardial inferior wall. Some of these patients have Right ventricular involvement which is volume/preload dependent.
- Plavix.
- Beta blockers:
  * Hold for SBP <100 or HR <60;
  * Hold if wheezing;
  * Hold if cocaine use (unopposed alpha).
- Morphine.
- Heparin: Before starting:
  * Check rectal exam;
  * Check CXR: to r/o dissection.

- **Valvular heart disease**

Aortic stenosis: Classic triad: dyspnea, chest pain, and syncope.

* Harsh systolic ejection murmur at right 2nd intercostal space radiating towards carotids.

* Carotid pulse: slow rate of increase.

* Brachioradial delay: Delay in pulses between right brachial and right radial arteries.

* Try to avoid nitrates: Theses patients are preload dependent.

Mitral valve prolapse: symptoms include atypical chest pain, palpitations, fatigue, dyspnea.

* Often hear mid-systolic click.

* Patients with chest pain or palpitations often respond to beta blockers.

- **Acute pericarditis**

Clinical features:

- Acute stabbing chest pain.
- Pleuritic chest pain.
- Pain often spreading to left trapezial ridge.

- Pain more severe when supine.
- Pain often relieved when sitting up and leaning forward.
- Listen for pericardial friction rub.

Common causes:

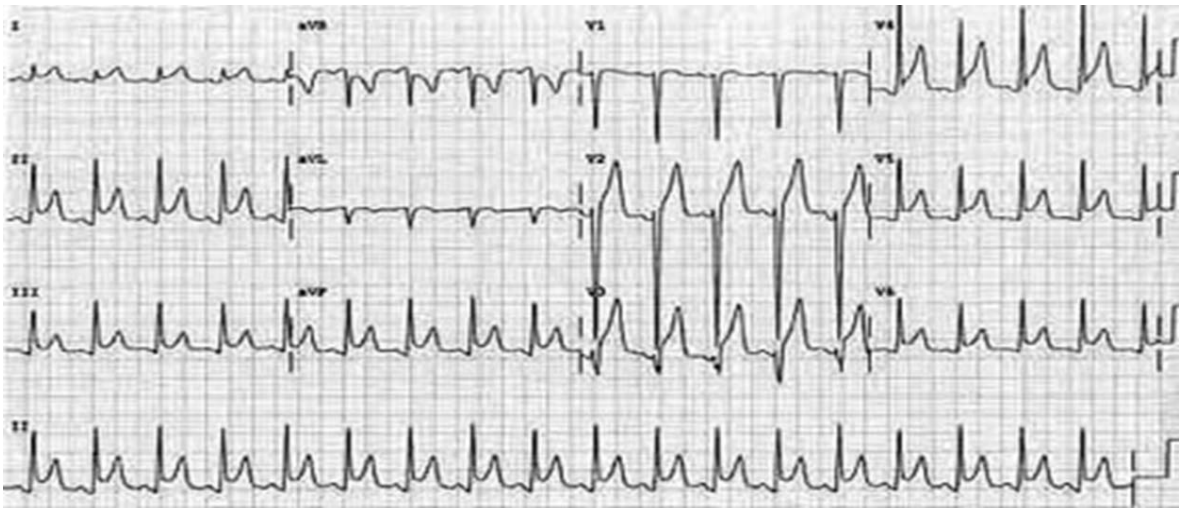
* Idiopathic.
* Infectious.
* Malignancy.
* Uremia.
* Radiation induced.
* after MI (Dressler syndrome).
* Myxedema.
* Drug induced.
* systemic rheumatic diseases.

ECG: look for diffuse ST segment elevation and PR depression.

* If large pericardial effusion/tamponade, may see low voltage and electrical alternans

CXR: limited value. Look at size of cardiac silhouette.

ECHO: to look for pericardial effusion.



- Diffuse ST segment elevation.

▪ **Myocarditis:**

- Inflammation of heart muscle.
- Frequently accompanied by pericarditis.
- Fever.
- Tachycardia in the case of fever.
- mild signs of pericarditis +fevers, myalgia, rigor, headache.

- If severe, will also see signs of heart failure.
- May see elevated cardiac enzymes.
- Treatment: Largely supportive.

- **Aortic dissection**

Risk factors:

- Uncontrolled hypertension.
- Congenital heart disease.
- Connective tissue disease.
- Pregnancy.
- Iatrogenic (s/p aortic catheterization or cardiac surgery).

Clinical features:

* Abrupt onset of chest pain or pain between scapulae.
* Tearing or ripping pain.
* Pain often worst a symptom onset.
* As other vessels become affected, will see.
  - Stroke symptoms: carotid artery involvement.
  - Tamponade: Ascending dissection into aortic root.
  - New onset Aortic Regurgitation.
  - Abdominal/Flank pain/Limb Ischemia: Dissection into abdominal aorta, renal arteries, iliac arteries.
  - AMI.
* Decreased pulsations in radial, femoral, carotid arteries.
* Significant blood pressure differences between extremities.
* Usually hypertension (but if tamponade, hypotension).

CXR: look for widened mediastinum.

CT SCAN.

Angiography.

TEE.

** Suspected dissections must be confirmed radiological prior to operative repair.

Treatment:

- Antihypertensive therapy.
* Start with beta blockers (esmolol, labetalol).

* Can add vasodilators (nitroprusside) if further BP control is needed. ONLY after achieved HR control with beta blockers.

- If ascending dissection: OR.
- If descending: May be able to medically manage.

IV Gastrointestinal causes of chest pain

- **GERD**

Risk factors:

* High fat food.
* Caffeine.
* Nicotine, alcohol.
* Medicines: CCB, nitrates, Anticholinergics.
* Pregnancy.
* DM.
* Scleroderma.

Clinical features:

* Burning pain.
* Association with sour taste in mouth, nausea/vomiting.
* May be relieved by antacids.
* May find association with food.
* May mimic ischemic disease and visa versa.

Treatment:

* Can try GI coctail in ED (30cc Mylanta, 10 cc lidocaine).
* H2-blockers and PPI.
* Behavior modification:
  - Avoid alcohol, nicotine, caffeine, fatty foods.
  - Avoiding eating prior to sleep.
  - Sleep with Head of Bed elevated.

- **Esophagitis.**

Clinical features: Chest pain +Odynophagia (pain with swallowing)

Causes:

*Inflammatory process: GERD or med related.

*Infectious process: Candida or HSV (often seen in immunocompromised patients).

Diagnosis: Endoscopy with biopsy and culture.

Treatment: Address underlying pathology.

IX. Tasks for final control

1. Physician's tactics in the detection of patients with acute coronary syndrome:

A. Immediate hospitalization in specialized intensive care unit of cardiology department.

B. Confinement to bed and outpatient treatment.

C. Planned hospitalization in therapeutic department.

D. Tactics determined after routine examination of the patient.

E. Only patients with ECG changes are hospitalized.

2. Patient of 72 years old complained of severe pain in the right lower limb, inability to walk because of the pain. He was sick during 2 days. Physical examination: right lower extremity was cold by touch, pale skin, all kinds of sensitivity was reduced. No pulsation of arteries of the right lower limb, the left lower limb - weakened. Diagnosis: obliterate atherosclerosis of the lower extremities during 15 years. Your preliminary diagnosis is?

A. Acute ileofemoral venous thrombosis

B. Right-side Lerishe syndrome;

C. Acute ileofemoral arterial thrombosis

D. Ileofemoral abdominal aortic aneurysm.

E. lymphostasis

3. The healthy man of 28 years felt acute pain in the left half of the chest, hypopnoea, heart pain, palpitations, dry hoarse cough. Physical examination: acrocyanosis, restriction of respiratory excursions, percussion - left tympanic, auscultation - diminished breath sounds. What is most informative diagnostic method?

A. Thoracoscopy;

B. Bronchoscopy;

C. Computer tomography;

D. Angiography;

E. Plain radiography of the chest.

4. The family doctor was appealed to the 12 week's pregnant woman. She complains to stabbing abdominal pain, intensive uterine bleeding. Which doctor should be appealed?

A. from gynecological department;

B. call the obstetrician - gynecologist;

C. hospitalized to the maternity hospital;

D. hospitalized to the surgical department;

E. haemostatic therapy.

5. Man of 30 years old, with a total overheating and heat stroke after high temperature working feels vomiting. Which solution should be administrated intravenous?

A. Hypotonic glucose.

B. Atropine sulfate solution.

C. Hypertensive sodium chloride.

D. Solution Reglan 4,0 ml.

E. Polarizing mixture.

6. After the snake bite the patient was hospitalized immediately. Moderately severe general condition. In the city he feels pain and heartburn. HR 100 per min, BP 100/60 mm Hg. consciousness is not impaired. What treatment should be prescribed immediately?

A. glucocorticoid.

B. Water-salt solutions.

C. Cardiotonic drugs.

D. Specific serum against snake.

E. Narcotic painkillers.

7. After wasps bite there appeared itchy skin, hoarseness of voice, "barking cough", anxiety. Physical examination: lips and eyelids swelling, cyanosis. What is your action?

A. Diclofenac sodium.

B. Seduxen.

C. Adrenaline.

D. Laziks.

E. Prednison.

8. Family doctor was appealed to the patient. 2 days ago the 46 years old man became too cold when he was drunk. He complains of burning pain, itch, paresthesias in the toes of both feet. Physical examination: cyanosis of toes of both feet, moderate swollen, some vesicles with clear yellow fluid. What is working diagnosis?

A. III degree frostbitten.

B. Crash syndrome.

C. I degree frostbitten.

D. IV degree frostbitten.

E. II degree frostbitten.

9. Worker during some minutes was under the impact of electric current. Physical examination: unconscious, seizures, self-breathing is absent, no carotid pulsation, mydriatic pupil, no photoreaction. What will be your action?

A. intracardiac injection of adrenalin.

B. Introduction of anticonvulsant.

C. Closed cardiac massage and artificial ventilation.

D. Intravenous respiratory analeptics.

E. Hit on the chest.

10. High diving a young boy hit his head on the bottom. He was brought to the sea side in a state of clinical death with signs of cervical spine injury. What is the resuscitation volume in this situation?

A. Do not throw back the head of the victim.

B. Do not open the mouth of the victim, conduct CPR mouth to nose.

C. Artificial respiration by Hohera-Nielsen method.

D. Do not print the victim's jaw up and forward.

E. Artificial respiration conduct by Sylvester method.

Answers:

1	2	3	4	5	6	7	8	9	10
A	C	E	A	C	D	E	E	C	A

TOPIC 7

The emergency in the practice of family doctor in the case of seizure, syncope, coma in case of diabetes, acute hepatic failure, alcohol intoxication, renal insufficiency, narcotic abuse

I. Theme actuality. First aid for children and adults has great importance for their further treatment and recovering diagnosis at pre-admission stage. And at the same time family doctor always faces a problem: what is better – either to give maximum necessary scope of emergency on the site of the incident or to take the patient to the nearest hospital as soon as possible. According to expert data there is only one decision of this problem. This means give maximum necessary scope of emergency in a short-run and then to admit to specialized hospital. Stabilization of patient's vital functions is the criteria of emergency scope in the place of the incident. Fundamental factors in this process are timeliness of emergency on the site of the incident, vocational training of a specialist and sufficient medical provision.

II. Study purposes: to be able to detect the first signs of loss of consciousness, to propose a plan of examination and treatment of patients with seizure and loss of consciousness.

III. Concrete purposes of the module: diagnostics and emergency in the case of seizure or loss of consciousness.

IV. A student must be able:

- to estimate general state of the patient;
- to acquire skills of clinical examination of the patients with seizure and loss of consciousness;
- to diagnose the state of the patient on the basis of patient complaints, medical history, degree of consciousness impairement and data of clinical examination;
- to define a plan for examination of patient with loss of consciousness;
- to estimate vital functions;
- to integrate data of clinical examination and laboratory data.

V. Task for initial independent training

1. The status of patient when his speech and thoughts become slow, his attention is distracted, there is fatigue, drowsiness and lack of perception and evaluation of what is happening:

A. Clear consciousness.

B. Obtundation.

C. Sopor.

D. Coma.

E. Syncope.

2. The status of patient when his mental state is depressed. After repeated appeal to the patient he opens his eyes but there is no contact with him:

A. Clear consciousness.

B. Obtundation.

C. Sopor.

D. Coma.

E. Syncope.

3. The status of patient when a dead faint and non-responsiveness to external irritants are observed:

A. Clear consciousness.

B. Obtundation.

C. Sopor.

D. Coma.

E. Syncope.

4. Short-time loss of consciousness accompanied by loss of postural tone and caused by temporary inadequate blood supply to brain is:

A. Clear consciousness.

B. Obtundation.

C. Sopor.

D. Coma.

E. Unconsciousness (Syncope).

5. Most important criteria of coma severity is:

A. Dead faint.

B. Non-responsiveness to external irritants.

C. Two-sided fixed mydriasis.

D. Areflexia.

E. Reduced muscle tone.

6. Types of syncopes are:

A. Neurogenic.

B. Orthostatic.

C. Cardiogenic.

D. Cerebrovascular.

E. All of mentioned above.

7. The paroxysm which starts with vertigo, loss of consciousness and fall of patient, with further tonoclonic spasms, ends by loss of consciousness with further sleep or psychomotor agitation:

A. Absence.

B. Unconsciousness.

C. Generalized tonoclonic attack.

D. Myoclonic attack.

E. Focal attack.

8. The status of patient when there is sudden short-time loss of consciousness with physical inactivity of the patient:

A. Absence.

B. Unconsciousness

C. Atonic attack.

D. Myoclonic attack .

E. Focal attack.

9. Generalized clonic seizure is typical for:

A. Epilepsy.

B. Alcoholic abstinence.

C. Fever, infectious brain diseases.

D. Metabolic disorders.

E. All of mentioned above.

10. Which metabolic disorders lead to coma:

A. Uremia.

B. Pancreatic (insular) diabetes.

C. Hypoglycemia.

D. Hepatic coma.

E. All of mentioned above.

Anwsers:

1	2	3	4	5	6	7	8	9	10
B	C	D	E	C	E	C	A	E	E

VI. Basic questions after theme

Convulsions and emergency in the pre-admission stage. Classification of convulsions.

Emergency in the case of generalized and focal convulsions.

Emergency in case of loss of consciousness and its causes.

VII. Practical skills: – types and tasks

Independent work: drawing algorithms of emergency.

VIII. Algorithms and the logical structure of theme

Causes of convulsions:

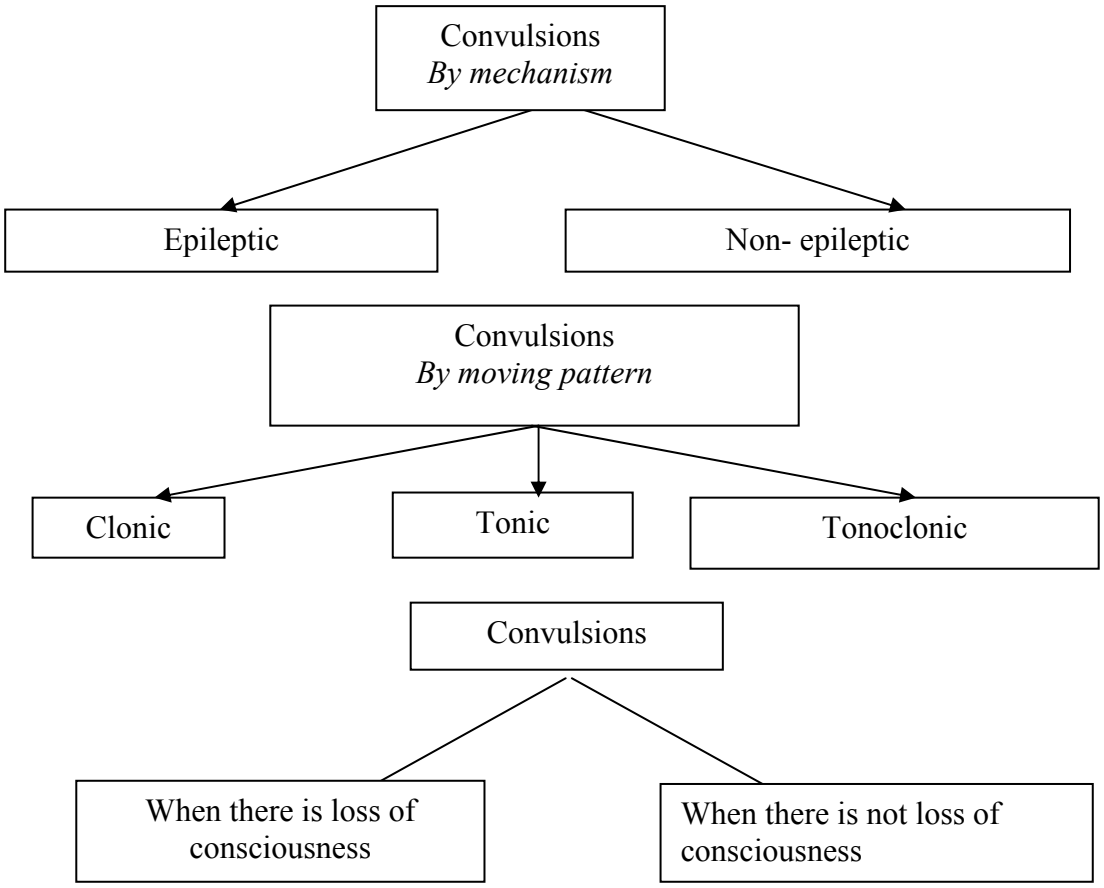
1. Idiopathic epilepsy.
2. Infectious diseases of brain.
3. Intoxications.
4. Fever (febrile convulsions).
5. Alcohol abstinence.
6. Metabolic disorders.
7. Drugs adverse reaction (neuroleptics, metoclopramide etc.).
8. Physical factors (electrical injury, overheating).
9. Affective respiratory attacks among children.
10. Unconsciousness.
11. Psychogenic (hysteric) attacks.
12. Stem convulsions (hormetonic, decorticate, decerebrate spasms).
13. Multifocal myoclonia.
14. Syndrome of rigidity.
15. Tetanus.
16. Tetany (spasmophilia).
17. Metabolic myopathy.

Classification of convulsions

1. Convulsions which are nonspecific reaction of brain for irritators: injuries, infections, intoxications and so on. These are encephalitic or episodic epileptic reactions.

2. Symptomatic convulsions or symptomatic epilepsy on the base of active cerebral process (neoplastic, inflammatory process, etc.).

3. Epilepsy (attacks of convulsions on the base of organic CNS lesions).



CONVULSIONS

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Diagnostic criteria
1. There is a disease in medical history.
2. There are hallucinations, aura and paraesthesia.
3. The onset.
4. Convulsions, their type.
5. Breathe.
6. Reflexes.
7. Pupillary reaction.
8. Involuntary urination.
9. Tongue bite.

↓

Volume of medical aid
- to avoid further injuries
- to avoid tongue bite
- to provide patency of airways
- oxygen inhalation and ALV if necessary
- ECG control
- vein feedback– isotonic solution of sodium chloride
- glucose 40% - 60 ml (in the case of diabetes)
- diazepam (seduxen) 0,5% - 2 ml (separately up to 6 ml) after stopping of convulsions
- cardiac glycosides – strophanthine 0,05% - 0,5-1 ml intravenously
- fight against brain edema (see correspondent protocol)

Volume of medical aid for children:
physical methods of cooling in the case of hyperthermia
- cleansing enema
- antipyretic agents – analgin 50% - 0,1 ml per 1 year of life (no more than 1 ml)
- magnesium sulfate 25% intramuscularly 0,2 ml per 1 year of life (no more than 10 ml)
- diazepam (seduxen) 0,55 – 0,3 mg per 1kg of weight

↓

Hospitalization: In accordance with indications for hospitalization to the neurological, infectious or resuscitation departments

IX. The content of theme

Syncope is defined as transient loss of consciousness with postural collapse caused by an acute decrease in cerebral blood flow. Although often preceded by faintness or light-headedness (presyncope), it may not be when caused by cardiac asystole or ventricular tachycardia. Presyncope symptoms may be manifest as "dizziness", always without true vertigo, or may be mimicked by a warning (aura) prior to a seizure. The sequence of symptoms is reasonably stereotyped and includes increasing lightheadedness, visual blurring proceeding to blindness, diaphoresis, and heaviness in the lower limbs progressing to postural sway. These symptoms increase in severity until consciousness is lost or the ischemia is corrected, often by assuming the recumbent position. The differentiation of syncope from seizure is an important, sometimes difficult, diagnostic problem [42].

At the beginning of a syncopal attack, the patient is nearly always in the upright position, either sitting or standing. A cardiac etiology, such as a Stokes-Adams attack, is exceptional in this respect. The patient is warned of the impending faint by a sense of "feeling bad", of giddiness, and of movement or swaying of the floor or surrounding objects. The patient becomes confused and may yawn, visual spots and dimming may occur, and the ears may ring. Nausea and sometimes vomiting accompany these symptoms. There is a striking pallor or ashen gray color of the face, and generalized perspiration ensues. In some patients, a deliberate onset with presyncopal symptoms may allow time for protection against injury; in others, the syncope is sudden and without warning. The onset varies from instantaneous to 10 to 30 s, rarely longer.

The depth and duration of unconsciousness vary. Sometimes the patient remains partly aware of the surroundings, or there may be profound coma. The patient may remain in this state for seconds to minutes or even as long as half an hour. Usually the patient lies motionless with skeletal muscles relaxed, but a few clonic jerks of the limbs and face may occur shortly after the beginning of the unconsciousness. In some situations there may be a brief tonic-clonic seizure. Sphincter control is usually maintained. The pulse is feeble or apparently absent, the blood pressure may be low to undetectable, and breathing may be almost imperceptible. Once the patient is in a horizontal position, gravity no longer hinders the flow of blood to the brain. The strength of the pulse may then improve, color begins to return to the face, breathing becomes quicker and deeper, and consciousness is regained. There is usually an immediate recovery of consciousness. Some patients may be keenly aware of physical weakness, and rising too soon may precipitate another faint. In other patients,

particularly those with transient tachyarrhythmias, there may be no residual symptoms following the initial syncope. Headache and drowsiness, which, with mental confusion, are the usual sequelae of a convulsion, do not follow a syncopal attack [44].

Etiology. The list of causes in Table 1 is based on established or assumed physiologic mechanisms. The more common types of syncope result from a sudden impairment of brain metabolism usually brought about by hypotension with reduction of cerebral blood flow.

Several mechanisms subserve circulatory adjustments to the upright posture. Approximately 3/4 of the systemic blood volume is contained in the venous bed, and any interference with venous return may lead to a reduction in cardiac output. Cerebral blood flow may still be maintained, as long as systemic arterial vasoconstriction occurs, but when this adjustment fails, serious hypotension with resultant cerebral underperfusion to less than half normal results in syncope. Normally, the pooling of blood in the lower parts of the body is prevented by:

1) pressor reflexes that induce constriction of peripheral arterioles and venules,

2) reflex acceleration of the heart by means of aortic and carotid reflexes,

3) improvement of venous return to the heart by activity of the muscles of the limbs. Placing a normal person on a tilt table to relax the muscles and tilting upright slightly diminishes cardiac output and allows the blood to accumulate in the legs to a slight degree; this may then be followed by a slight transitory fall in systolic arterial pressure and, in patients with defective vasomotor reflexes, may produce faints [43].

Table 1

Causes of Faintness and Disturbances of Consciousness

I. Circulatory (reduced cerebral blood flow).

A. Inadequate vasoconstrictor mechanisms:

1. Vasovagal (vasodepressor);
2. Postural hypotension;
3. Primary autonomic insufficiency;
4. Sympathectomy (pharmacologic, due to antihypertensive medications such as methyldopa and hydralazine, or surgical);
5. Diseases of central and peripheral nervous systems, including autonomic nerves;
6. Carotid sinus syncope (Bradyarrhythmias);
7. Hyperbradykininemia.

B. Hypovolemia:

1. Blood loss - gastrointestinal hemorrhage;
2. Addison's disease.

C. Mechanical reduction of venous return:

1. Valsalva maneuver;
2. Cough;
3. Micturition;
4. Atrial myxoma, ball valve thrombus.

D. Reduced cardiac output:

1. Obstruction to left ventricular outflow: aortic stenosis, hypertrophic subaortic stenosis;
2. Obstruction to pulmonary flow: pulmonic stenosis, primary pulmonary hypertension, pulmonary embolism;
3. Myocardial: massive myocardial infarction with pump failure;
4. Pericardial: cardiac tamponade.

E. Arrhythmias:

1. Bradyarrhythmias:
    a. Atrioventricular (AV) block (II and III degree), with Stokes-Adams attacks;
    b. Ventricular asystole;
    c. Sinus bradycardia, sinoatrial block, sinus arrest, sick-sinus syndrome4;
    d. Carotid sinus syncope;
    e. Glossopharyngeal neuralgia (and other painful states).
2. Tachyarrhythmias:
    a. Episodic ventricular tachycardia with or without associated bradyarrhythmias;
    b. Supraventricular tachycardia without AV block.

II. Other causes of disturbances of consciousness

A. Altered state of blood to the brain

c. Hypoxia
d. Anemia
e. Diminished carbon dioxide due to hyperventilation (faintness common, syncope seldom occurs)

f. Hypoglycemia (episodic weakness common, faintness occasional, syncope rare)

B. Cerebral

1. Cerebrovascular disturbances (cerebral ischemic attacks)
    a. Extracranial vascular insufficiency (vertebral-basilar, carotid)
    b. Diffuse spasm of cerebral arterioles (hypertensive encephalopathy)
2. Emotional disturbances, anxiety attacks, and hysterical seizures

Types of syncope. Vasodepressor (Vasovagal) or Neurocardiogenic Syncope. This form of syncope is the common faint that may be experienced by normal persons and accounts for about 50% of all instances of syncope. It is frequently recurrent and commonly precipitated by emotional stress (especially in a warm, crowded room), fear (e.g., in a dentist's chair), extreme fatigue, injury, or pain. Many episodes occur without obvious antecedent cause. Vasodepressor syncope comprises a constellation of symptoms including hypotension, bradycardia, nausea, pallor, and diaphoresis. Syncope typically occurs in the setting of diminished venous return that leads to reduced stroke volume and a reflex increase in sympathetic activity. In susceptible individuals, this increase in sympathetic activity causes cardiac hypercontractility and excessive stimulation of ventricular mechanoreceptors (afferent vagal C-fibers), which, in turn, leads to sympathetic withdrawal and activation of the parasympathetic nervous system via a centrally mediated vasomotor reflex. The net result is a vicious cycle of *inappropriate peripheral vasodilatation* and *relative bradycardia* leading to progressive hypotension and syncope that can be reversed by assumption of supine posture and elevation of the legs. Orthostatic stress induced by prolonged upright tilt testing at 60-80 degrees is a sensitive technique for reproducing syncope in many patients with this syndrome. Vasodepressor syncope may occur with sudden severe pain, particularly if it originates in the viscera, and may also rarely accompany a severe migraine headache [42-44].

Postural (Orthostatic) Hypotension with Syncope affects persons who have a chronic defect or variable instability of vasomotor reflexes. The fall in blood pressure on assumption of upright posture is due to a loss of vasoconstriction reflexes in resistance and capacitance vessels of the lower extremities. Although the syncopal attack differs little from vasodepressor syncope, the effect of posture is critical. Sudden arising from a recumbent position or standing quietly are precipitating circumstances. Postural syncope may occur in the following conditions:

1. In otherwise normal persons who have defective postural reflexes. In such individuals, fainting may occur when they are tilted on a table. Under such circumstances the blood pressure at first diminishes slightly and then stabilizes at a lower level. Shortly thereafter, the compensatory reflexes suddenly fail and the arterial pressure falls precipitously. The condition is often familial.

2. In *primary autonomic insufficiency* and in the *dysantonomias. Acute or subacute dysautonomia* is a rare condition. An otherwise healthy adult or child develops, over a period of a few days or weeks, a partial or complete paralysis of the parasympathetic and sympathetic nervous systems. Pupillary reflexes are lost, and lacrimation, salivation, and sweating are absent or diminished. Impotence, paresis of bladder and bowel musculature, and orthostatic hypotension are present. The cerebrospinal fluid (CSF) protein is often increased. Sensory and motor nerves are intact, but unmyelinated autonomic nerves degenerate. The disease appears to represent a variant of Guillain-Barre syndrome, and recovery usually occurs within a few months.

Chronic postganglionic autonomic insufficiency is a disease of the middle-aged and elderly who gradually develop chronic orthostatic hypotension, sometimes in conjunction with impotence and sphincter disturbances. Typically, after standing for 5 to 10 min, the blood pressure falls by at least 35 mmHg, the pulse pressure narrows, and there is no compensatory tachycardia, pallor, or nausea. Men are more often affected than women [47].

Chronic preganglionic autonomic insufficiency is a condition, when orthostatic hypotension with anhidrosis, impotence, and sphincter disturbances is combined with a disorder of the central nervous system (CNS). These disorders, designated *multisystem atrophies,* include syndromes characterized by:

1) tremor, extrapyramidal rigidity, and akinesia (Shy-Drager syndrome);

2) progressive cerebellar degeneration, some instances of which are familial;

3) a more variable extrapyramidal and cerebellar disorder (striatonigral degeneration). These syndromes lead to severe disability and often death within a few years.

The differentiation of chronic peripheral postganglionic and central preganglionic insufficiency is based on pathologic and pharmacologic evidence. In the postganglionic type, neurons of the sympathetic ganglia degenerate, whereas in the central type, intermediolateral horn preganglionic cells of the thoracic spinal cord degenerate. In the postganglionic peripheral type, resting levels of norepinephrine are subnormal because of failure to release norepinephrine from postganglionic endings, and there is

hypersensitivity to injected norepinephrine. In the central type, resting levels of norepinephrine are normal. On standing, there is little, if any, rise in norepinephrine levels in either type. And in both types, the levels of plasma dopamine p-hydroxylase (the enzyme that converts dopamine to norepinephrine) are subnormal [48].

Other causes of postural syncope:

1) After physical deconditioning (e.g., after prolonged illness with recumbency, especially in elderly with reduced muscle tone, or after prolonged weightlessness, as in space flight).

2) After a sympathectomy that has abolished vasopressor reflexes.

3) In diabetic, alcoholic, beri-beri, amyloid and other neuropathies. The most common neurogenic orthostatic hypotension accompanies diseases of the peripheral nervous system. Usually the orthostatic hypotension is associated with disturbances in sweating, impotence, and sphincter difficulties. Presumably the lesion involves postganglionic, unmyelinated fibers in peripheral nerves.

4) In patients receiving antihypertensive and vasodilator drugs as well as those who may be hypovolemic because of diuretics, excessive sweating, or adrenal insufficiency.

Micturition syncope is a condition usually seen in elderly men during or after urination, particularly after arising from sleep, is probably a special type of vasodepressor syncope. Release of intravesicular pressure may trigger vasodilatation and vagally mediated bradycardia [42].

Cardiac Syncope results from a sudden reduction in cardiac output, caused most commonly by a cardiac arrhythmia. In normal individuals, slow ventricular rates (above 35 to 40 beats per minute) and fast rates not exceeding 180 beats per minute do not reduce cerebral blood flow, especially if the person is in the supine position. Changes in pulse rate outside these limits may impair cerebral circulation and function. Upright posture, cerebrovascular disease, anemia, and coronary, myocardial, or valvular disease all reduce the tolerance to alterations in rate.

High-degree *atrioventricular (AV) block* is commonly associated with fainting *(Stokes-Adams-Morgagni syndrome).* In patients with these attacks, the block may be persistent or intermittent. When the block is high-grade or complete and the pacemaker below the block fails to function, or functions at too slow a rate, syncope occurs. Stokes-Adams attacks occur precipitously, usually without warning symptoms. Should cardiac asystole persist more than 8-10 s, the patient turns pale, falls unconscious, and may exhibit a few clonic jerks. With longer periods of asystole, cyanosis ensues,

breathing is irregular, and fixed pupils, incontinence, and bilateral Babinski signs may be present. While recovery following a Stokes-Adams attack is usually prompt and complete, prolonged confusion and neurologic signs due to cerebral ischemia may occur in some patients, and permanent impairment of mental function may occasionally result, although focal neurologic signs are rare. The patient usually does not recall presyncopal symptoms. Cardiac faints of this type may recur several times a day. Commonly the heart block is transitory, and the ECG may not show any arrhythmia. Ventricular tachycardia or fibrillation may follow a period of asystole, resulting in prolonged coma or sudden death [43].

Disorders of sinus node automaticity or sinoatrial conduction may result in asystole or bradycardia of sufficient severity to cause presyncope or syncope. This disorder is most frequently detected with ambulatory ECG monitoring. Findings consistent with a diagnosis of sinus node dysfunction include symptomatic sinus pauses (>3s) resulting from sinus arrest or sinoatrial block and severe unexplained sinus bradycardia (<40 beats per min). The *bradycardia-tachycardia syndrome* is a common form of sinus node dysfunction in which syncope occurs as a result of marked sinus pauses following termination of paroxysmal supraventricular tachycardia. Electrophysiologic testing is sometimes helpful in patients with syncope and suspected sinus node dysfunction in whom the diagnosis is not established by ambulatory ECG recording [46].

Tachyarrythmias. Recurrent paroxysmal tachyarrhythmias also may cause presyncope and syncope as a result of a sudden reduction in cardiac output. The magnitude of tachycardia-induced hypotension is dependent on the interaction of several variables, including the rate and mechanism of the tachycardia, the type and severity of underlying cardiac disease, the patient's posture and activity level at the onset of the tachycardia, the sensitivity of the tachycardia to endogenous catecholamines, and the integrity of compensatory autonomic reflexes. *Supraventricular tachyarrhythmias* are not commonly associated with syncope. However, even in the absence of structural heart disease, the extremely high heart rates may impair cardiac filling and output sufficiently to cause loss of consciousness. These tachycardias result most commonly from the occurrence of paroxysmal atrial flutter, atrial fibrillation, or reentry involving the AV node or accessory pathways that bypass part or all of the AV conduction system. Patients with the Wolff-Parkinson-White syndrome are susceptible to several forms of supraventricular tachycardia, the most dangerous of which is atrial fibrillation with rapid antegrade conduction to the

ventricles over an accessory AV connection that may result in syncope and, in rare instances, sudden death. When abnormal conduction over an accessory AV connection or an AV nodal reentry rhythm is suspected as a cause of syncope, electrophysiologic testing is indicated to define the mechanism and pathway of the tachycardia and to facilitate the selection of an effective therapeutic intervention [45].

Paroxysmal ventricular tachycardia is a relatively common cause of syncope, particularly in patients with structural heart disease. Typically, the tachycardias are rapid and associated with abrupt loss of consciousness without premonitory symptoms. Usually the patient is unaware of palpitations, and recovery following an episode is prompt and complete without residual neurologic or cardiac sequelae. Unexplained syncope in a patient with structural heart disease is a potentially ominous finding and merits careful evaluation. The presence of pathologic Q-waves on the ECG, indicative of a prior transmural myocardial infarction, is strongly associated with ventricular tachycardia as a cause of syncope in patients with ischemic heart disease. Other forms of heart disease (hypertrophic and dilated cardiomyopathy, right ventricular dysplasia, long-QT-interval syndromes) are also frequently associated with paroxysmal ventricular tachycardia and syncope.

Reflexive heart block (due to irritation of the vagus nerves) is another form of cardiac syncope. E.g., in patients with esophageal diverticula, mediastinal tumors, gallbladder disease, carotid sinus disease, glossopharyngeal neuralgia, and pleural and pulmonary irritation. In these conditions, reflex bradycardia is more commonly of the sinoatrial than the atrioventricular type [44].

Other causes. Cardiac syncope also may result from *acute massive myocardial infarction*, particularly when associated with cardiogenic shock. *Aortic stenosis* often sets the stage for exertional syncope, most commonly by limiting cardiac output in the face of peripheral vasodilatation, with resultant myocardial and cerebral ischemia and occasionally arrhythmias. *Idiopathic hypertrophic subaortic stenosis* also may lead to exertional syncope because of intensified obstruction, ventricular arrhythmias, or both. In *primary pulmonary hypertension*, a relatively fixed cardiac output and bouts of acute right ventricular failure may be associated with syncope. However, vagal reflexes may be involved in this condition as well as in the syncope that occurs with *pulmonary embolism.* Ball-valve thrombus in the left atrium, left atrial myxoma, or thrombosis (malfunction) of a prosthetic valve may produce sudden mechanical obstruction of the circulation and syncope. *Tetralogy of Fallot* is the congenital cardiac malformation most commonly responsible for syncope. In this condition, systemic vasodilatation,

associated with infundibular spasm, greatly increases the right-to-left shunt and produces arterial hypoxia, which leads to syncope.

Carotid Sinus Syncope. The carotid sinus is normally sensitive to stretch and gives rise to sensory impulses carried via the nerve of Hering, a branch of the glossopharyngeal nerve, to the medulla oblongata. Massage of one or both of the carotid sinuses (in elderly persons) causes:

1) a reflex cardiac slowing (sinus bradycardia, sinus arrest, or even AV block), the so-called vagal type of response,

2) a fall of arterial pressure without cardiac slowing, the so-called depressor type of response. Both types of carotid sinus response may coexist [42].

Syncope due to carotid sinus sensitivity is extremely uncommon and said to be initiated by turning of the head to one side, by a tight collar, or by shaving over the region of the sinus. In a patient displaying faintness on compression of one carotid sinus, it is important to distinguish between the benign disorder (carotid sinus hypersensitivity) and a much more serious condition such as carotid artery stenosis on the opposite side. Thus, carotid compression is a risky maneuver that may lead to cerebral ischemia.

Glossopharyngeal Neuralgia is painful disorder which may induce reflex fainting. The sequence is always pain, followed by syncope. The pain is localized to the jaw, base of the tongue, pharynx or larynx, tonsillar area, and ear. The cardiovascular effects are attributable to excitation of the dorsal motor nucleus of the vagus via collateral fibers from the nucleus of the tractus solitarius. Treatment of the neuralgia with carbamazepine is often effective for the syncope, as well as for the pain.

Cough Syncope is a rare condition that follows paroxysm of coughing (e.g., chronic bronchitis). After forceful coughing, the patient suddenly becomes weak and loses consciousness momentarily. The coughing increases intrathoracic pressure and the elevated pressure is transmitted via the great veins to the intracranial compartment, causing increased intracranial pressure and secondarily decreased cerebral blood flow. At a critically low blood flow, syncope ensues [43].

Stretch Syncope is a rare condition that occurs in otherwise normal adolescents. The provoking maneuver is simultaneous neck extension and upper limb stretching. The mechanism seems to be compression of the vertebral arteries in the neck.

Differential diagnosis. Anxiety Attacks and the Hyperventilation Syndrome. Anxiety, such as occurs in panic attacks, is frequently interpreted as a feeling of faintness or dizziness without loss of consciousness. The symptoms are not

accompanied by facial pallor and are not relieved by recumbency. The diagnosis is made on the basis of the associated symptoms, and the attack can be reproduced by hyperventilation. Hyperventilation results in hypocapnia, alkalosis, increased cerebrovascular resistance, and decreased cerebral blood flow. The release of epinephrine in anxiety states also contributes to the symptoms.

Hypoglycemia is usually traceable to a serious disease such as a tumor of the islets of Langerhans; advanced adrenal, pituitary, or hepatic disease; or to excessive administration of insulin; it leads to confusion or loss of consciousness. Mild hypoglycemia, often reactive type and occurring 2 to 5 h after eating, is not usually associated with a disturbance of consciousness [42-44].

Acute Hemorrhage, usually within the gastrointestinal tract, is an occasional cause of syncope. In the absence of pain and hematemesis, the cause of the weakness, faintness, or even unconsciousness may remain obscure until the passage of a black stool.

Cerebral Transient Ischemic Attacks (TIAs) occur in patients with atherosclerotic narrowing, occlusion, or emboli to the major arteries of the brain. The symptoms are manifold. Sudden drop attacks may mimic syncope. Isolated loss of consciousness is rare.

Hysterical Fainting is usually unattended by an outward display of anxiety. Lack of change in pulse and blood pressure or color of the skin and mucous membranes distinguishes it from the vasodepressor faint.

Approach to the Patient

Type of Onset may assist in determining the cause. Syncope that begins over a period of a few seconds is most likely due to postural hypotension, sudden AV block, asystole, or ventricular tachycardia. When symptoms develop gradually during a period of several minutes, hyperventilation or hypoglycemia should be considered. Onset of syncope during or immediately after exertion suggests aortic stenosis, idiopathic hypertrophic subaortic stenosis or excessive bradycardia, and, in elderly subjects, postural hypotension. Exertional syncope is seen occasionally in persons with aortic insufficiency. In patients with ventricular standstill or ventricular fibrillation, loss of consciousness occurs 8 to 10 s later and then often by brief clonic muscle contractions.

Position at Onset of Attack. Epilepsy and syncopal attacks due to hypoglycemia, hyperventilation, or heart block are likely to be independent of posture. Faintness associated with a drop in blood pressure and with ectopic tachycardia usually occurs only in the sitting or standing position, whereas faintness resulting from orthostatic

hypotension is apt to begin shortly after change from the recumbent to the standing position [43].

Associated Symptoms such as palpitation may be present when the attack is due to anxiety or hyperventilation, ectopic tachycardia, or hypoglycemia. Numbness and tingling in the hands and face are frequent accompaniments of hyperventilation. Genuine convulsions during the attack may occasionally occur with heart block, asystole, or ventricular tachycardia.

In patients with recurrent syncope an attempt to reproduce an attack may assist in diagnosis.

Symptoms induced by hyperventilation can be reproduced readily by having the subject breathe rapidly and deeply for 2 to 3 min. Anxiety attacks induced by hyperventilation tend to be lessened when the patient learns that the symptoms can be produced and alleviated at will simply by controlling breathing.

Other conditions in which the diagnosis is commonly clarified by reproducing the attacks are orthostatic hypotension and orthostatic tachycardia (observations of pulse rate, blood pressure, and symptoms in the recumbent and standing positions), and cough syncope (by inducing the Valsalva maneuver). In each instance, the crucial point is not whether symptoms are produced (the procedures mentioned frequently induce symptoms in healthy persons) but whether the exact pattern of symptoms that occurs in the spontaneous attacks is reproduced in the artificial ones. Continuous ambulatory ECG monitoring is essential to identify arrhythmias responsible for syncope, particularly in patients with frequently recurring symptoms. Monitoring is diagnostic if it shows episodes of asystole, extreme bradycardia, or tachyarrhythmia [47].

In cases of recurrent syncope of unknown cause in which ambulatory ECG monitoring is unrevealing and there is underlying heart disease, particularly ischemic and prior myocardial infarction, the use of intracardiac electrophysiologic techniques with programmed stimulation can be helpful in detecting cardiac rhythm abnormalities and in establishing effective treatment. During stimulation, up to two-thirds of such patients can be shown to have rapid ventricular tachycardia. Occasionally, intracardiac electrophysiologic studies are helpful in identifying significant His bundle conduction delays or sick-sinus syndrome. The diagnostic yield of intracardiac electrophysiologic testing is lower in patients with nonischemic heart disease and in patients with structurally normal hearts than in patients with ischemic heart disease. Recently, the signal-averaged ECG has proved to be useful in identifying patients with unexplained

syncope who are likely to have ventricular tachycardia induced by electrophysiologic study.

Head-up tilt testing is a useful provocative technique for the diagnosis of vasodepressor syncope. Upright tilt to a maximum of 60 to 70 degrees usually precipitates symptomatic hypotension or syncope within 10 to 30 min in patients with this syndrome. In normal subjects, passive tilting to 60 degrees causes a small decrease in systolic blood pressure and an increase in diastolic blood pressure and heart rate. Recently, tilt testing has been used in conjunction with electrophysiologic testing to assess the efficacy of prophylactic pacing in selected patients with vasodepressor syncope and to evaluate the impact of posture on the hemodynamic consequences of some tachyarrhythmias. Sublingual nitroglycerine administered during head-up tilt testing may unmask certain vasovagal-induced causes of syncope [47,48].

Syncope should be distinguished from disturbances of cerebral function caused by a seizure. A seizure may occur day or night, regardless of the position of the patient; syncope rarely appears when the patient is recumbent, the only common exception being the Stokes-Adams attack. The patient's color may not change in seizures, though there may be cyanosis; pallor is an early and invariable finding in all types of syncope, except chronic orthostatic hypotension and hysteria, and it precedes unconsciousness. Seizures are often heralded by an aura, which is caused by a focal seizure discharge and hence has brain- localizing significance. The aura is usually followed by rapid return to normal or by loss of consciousness. The onset of syncope is usually more deliberate and without aura. Injury from falling is frequent in a seizure and rare in syncope for the reason that only in seizures are protective reflexes abolished instantaneously. Tonic-convulsive movements are a feature of seizures and usually do not occur with syncope, although, as stated above, brief tonic clonic seizure-like activity can accompany fainting episodes. Upward deviation of the eyes occurs with both conditions. The period of unconsciousness tends to be longer in seizures than in syncope. Urinary incontinence is frequent in seizures and rare in syncope. The return of consciousness is prompt in syncope, slow after a seizure. Mental confusion, headache, and drowsiness are eommon sequelae of seizures; physical weakness with a clear sensorium characterizes the postsyncopal state. Repeated spells of unconsciousness in a young person at a rate of several per day or month are more suggestive of epilepsy than of syncope [42].

The EEG may be helpful in differentiating syncope from seizures. In the interval between epileptic seizures, it may show some degree of abnormality in up to 80% of patients. In the interval between syncopal attacks, the EEG should be normal.

Treatment. In most instances, fainting is relatively benign. In dealing with patients who have fainted, the physician should think first of those causes of fainting that constitute a therapeutic emergency. Among them are massive internal hemorrhage and myocardial infarction, which may be painless, and cardiac arrhythmias. In elderly persons, a sudden faint, without obvious cause, should arouse the suspicion of complete heart block or a tachyarrhythmia, even though all findings are negative when the patient is seen.

Patients encountered during the preliminary stages of fainting, or after they have lost consciousness, should be placed in a position which permits maximal cerebral blood flow, i.e., with head lowered between the knees, if sitting, or, preferably, in the supine position. All tight clothing and other constrictions should be loosened, and the head turned so that the tongue does not fall back into the throat, blocking the airway. Peripheral irritation, such as dashing cold water on the face and neck or the application of cold moist towels, is helpful. If the temperature is subnormal, the body should be covered with a warm blanket. Since emesis is frequent, aspiration should be prevented. The head should be turned to the side and nothing given by mouth until the patient has regained consciousness. Patients should not be permitted to rise until the sense of physical weakness has passed and should be watched carefully for a few minutes after rising [42,43].

Because of the critical role of β-adrenergic stimulation and hypercontractility in the vasovagal syncope syndrome, effective prophylaxis can usually be achieved with the use of β -adrenergic receptor blocking agents or disopyramide. Since vasovagal syncope usually occurs in patients with normal left ventricular systolic function, the use of these agents is usually well tolerated. Other pharmacologic agents that have been used to treat this type of syncope include theophylline, scopolamine, and ephedrine. Cardiac pacing alone is rarely indicated or effective in the prevention of this type of syncope but may be necessary in a small minority of patients in whom profound bradycardia or asystole predominates over peripheral vasodilatation as the primary mechanism of syncope.

The *prevention* of fainting depends on the mechanisms involved. In the usual vasovagal faint of adolescents, which tends to occur in periods of,emotional excitement, fatigue, hunger, etc., it is enough to advise the patient to avoid such circumstances.

In postural hypotension, patients should be cautioned against arising suddenly from bed. Instead, they should first exercise their legs for a few seconds and then sit on the edge of the bed and make certain they are not lightheaded or dizzy before starting to

walk. Sleeping with the headposts of the bed elevated on wooden blocks 8 to 12 in. high and wearing a snug elastic abdominal binder and elastic stockings are often helpful. The distinction made between the various types of orthostatic hypotension has therapeutic significance. In the peripheral postganglionic type, the most effective treatment is 9a-fluorohydrocortisone (oral dose 0,1 to 0,2 mg/d) and salt loading to increase blood volume, supplemented by mechanical devices to prevent pooling of blood in the legs and lower trunk. However, salt together with mineralocorticoids may induce serious supine hypertension, and the dose of the drug must be adjusted for this. For the central preganglionic type, there has been greater success with use of a sympathomimetic amine such as tyramine (which releases norepinephrine from intact postganglionic endings) supplemented by a monoamine oxidase inhibitor (to prevent destruction of the amine) and possibly propranolol. Levodopa has been effective in some cases. In the postganglionic type, judicious use of phenylephrine or ephedrine may be beneficial if they do not cause insomnia. If there are no contraindications, a high intake of sodium chloride, which expands the extracellular fluid volume, may be tried [45].

The treatment of carotid sinus syncope involves first of all instructing the patient in measures that minimize the hazards of a fall. Loose collars should be worn, and the patient should learn to turn the whole body, rather than the head alone, when looking to one side. Atropine or the ephedrine group of drugs should be used in patients with pronounced bradycardia or hypotension during attacks.

The chief hazard of a faint in most elderly persons is not the underlying disease but fracture or other trauma due to the fall. Therefore, patients subject to recurrent syncope should cover the bathroom floor and bathtub with rubber mats and should have as much of their home carpeted as is feasible. Especially important is the floor space between the bed and the bathroom, because faints are common in elderly persons when walking from bed to toilet. Outdoor walking should be on soft ground rather than hard surfaces and the patient should avoid standing still, which is more likely than walking to induce an attack [45].

Acute confusional states and coma. Confusional states and coma are among the most common problems in general medicine. Over 5% of admissions to the emergency ward of large municipal hospitals are due to diseases that cause a disorder of consciousness. Because a clouding of consciousness (confusion) cannot easily be separated from a diminished level of consciousness (drowsiness, stupor, and coma) and the two are produced by many of the same medical disorders, these conditions are presented here.

Although the interpretation of consciousness is a psychological and philosophical matter, the distinction between *level* of consciousness, or wakefulness, and *content* of consciousness, or awareness, has neurologic significance. Wakefulness-alertness is maintained by a system of upper brainstem and thalamic neurons, the reticular activating system (RAS), and its broad connections to the cerebral hemispheres. Therefore, reduced wakefulness results from depression of the neuronal activity in either the cerebral hemispheres or in the RAS. *Awareness* and *thinking* are dependent on integrated and organized thoughts, subjective experiences, emotions, and mental processes, each of which resides to some extent in anatomically defined regions of the brain. Self-awareness requires that the organism senses this personal stream of thoughts and emotional experiences. The inability to maintain a coherent sequence of thoughts, accompanied usually by inattention and disorientation, is the best definition of *confusion* and is a disorder of the content of consciousness [46].

States of reduced alertness. The unnatural condition of reduced alertness and lessened responsiveness is a continuum that in extreme form characterizes the deep sleeplike state from which the patient cannot be aroused, called *coma. Drowsiness* is a disorder that simulates light sleep from which the patient can be easily aroused by touch or noise and can maintain alertness for some time. *Stupor* defines a state in which the patient can be awakened only by vigorous stimuli, and an effort to avoid uncomfortable or aggravating stimulation is displayed. As already indicated, both drowsiness and stupor are usually attended by some degree of mental confusion. Verbal responses in these states are therefore incorrect, slow, or absent during periods of arousal. *Coma* indicates a state from which the patient cannot be aroused by stimulation, and no purposeful attempt is made to avoid painful stimuli.

In clinical practice these terms must be supplemented by a narrative description of the behavioral state of the patient and of responses evoked by various stimuli precisely as they are observed at the bedside. Such descriptions are preferable to ambiguous summary terms such as semicoma or obtundation, the definitions of which differ between observers.

The confusional state is a behavioral state of reduced mental clarity, coherence, comprehension, and reasoning. *Inattention and disorientation are the main early signs;* however, as an acute confusional state worsens there is deterioration in memory, perception, comprehension, problem solving, language, praxis, visuo-spatial function, and various aspects of emotional behavior that are each identified with particular regions of the brain. Early in the process it is difficult to know if these complex mental

functions are reduced solely as a result of the pervasive defect in attention, but global cortical dysfunction is expected from the metabolic diseases and pharmacologic agents that are the most common sources of the acute confusional state. When there is in addition to confusion an element of drowsiness, the patient is said to have an *encephalopathy.*

Confusion may be a feature of a dementing illness, in which case the chronicity of the process and often a disproportionate effect on memory distinguish it from acute confusion. The confusional state may also derive from a single cortical deficit in higher mental function such as impaired language comprehension, loss of memory, or lack of appreciation of space, in which case each state is defined by the dominant behavioral change (namely, aphasia, dementia, agnosia) rather than characterizing the state as confusion [47].

The drowsiness caused by systemic metabolic changes or by brain lesions is typically accompanied by confusion (encephalopathy). In these instances the primary problem that is causing a diminished level of consciousness should be addressed. A difficult circumstance arises when a process that ultimately leads to drowsiness or stupor begins with confusion or delirium in a fully awake patient.

The confused patient is usually subdued, not inclined to speak, and is inactive physically. In certain cases confusion is accompanied by illusions (misperceptions of environmental sight, sound, or touch) or hallucinations (spontaneous endogenous perceptions). While psychiatrists use the term *delirium* interchangeably with confusion, neurologists prefer to reserve it as a description for an agitated, hypersympathotonic, hallucinatory state most often due to alcohol or drug withdrawal or to hallucinogenic drugs.

Comalike syndromes and related states. Coma is characterized by complete unarousability. Several other syndromes render patients apparently unresponsive or insensate but are considered separately because of their special significance. The *vegetative state,* an unfortunate term, describes patients who were earlier comatose but whose eyelids have after a time opened, giving the appearance of wakefulness. There may be yawning, grunting, and random limb and head movements, but there is an absolute absence of response to commands and an inability to communicate - in essence, an "awake coma". There are accompanying signs of extensive damage to both cerebral hemispheres, i.e., Babinski signs, decerebrate or decorticate limb posturing, and absent response to visual stimuli. Autonomic nervous system functions such as cardiovascular, thermoregulatory, and neuroendocrine control are preserved and may be

subject to periods of overactivity. *Akinetic mutism,* refers to a partially or fully awake patient who when unstimulated remains immobile and silent. The state may result from hydrocephalus, from masses in the region of the third ventricle, or from large bilateral lesions in the cingulate gyrus or other portions of both frontal lobes. Lesions in the periaqueductal or low diencephalic regions may cause a similar state. *Abulia* can be viewed as a mild form of akinetic mutism with the same anatomic origins. The abulic patient is hypokinetic and slow to respond but generally gives correct answers. It is typical to halt while reciting numbers or sequential calculations and, with a delay, to resume correctly. The *locked-in state* describes a pseudocoma in which patients are awake but deefferented, i.e., have no means of producing speech or limb, face, or pharyngeal movements. Infarction or hemorrhage of the ventral pons, which transects all descending corticospinal and corticobulbar pathways are the usual causes. The RAS arousal system, vertical eye movements, and lid elevation remain unimpaired. Such eye movements can be used by the patient to signal to the examiner. A similar awake state simulating unresponsiveness may occur as a result of total paralysis of limb, ocular, and oropharyngeal musculature in severe cases of acute Guillain-Barre syndrome. Unlike brainstem stroke, vertical eye movements are not selectively spared [49].

Certain psychiatric states can mimic coma by producing an apparent unresponsiveness. *Catatonia* is a peculiar hypomobile syndrome associated with major psychosis. In the typical form patients appear awake with eyes open but make no voluntary or responsive movements, although they blink spontaneously and may not appear distressed. It is characteristic but not invariable to have a "waxy flexibility", in which limbs maintain their posture when lifted by the examiner. Upon recovery, such patients have some memory of events that occurred during their catatonic stupor. Patients with *hysterical* or *conversion pseudocoma* show signs that indicate voluntary attempts to appear comatose, though it may take some ingenuity on the part of the examiner to demonstrate these. Eyelid elevation is actively resisted, blinking occurs to a visual threat when the lids are held open, and the eyes move concomitantly with head rotation, all signs belying brain damage [50].

Anatomic correlates of consciousness. A normal level of consciousness (wakefulness) depends upon activation of the cerebral hemispheres by neurons located in the brainstem RAS. Both of these components and the connections between them must be preserved for normal consciousness to be maintained. The principal causes of coma are therefore:

1) widespread damage in both hemispheres from ischemia, trauma, or other less common brain diseases;

2) suppression of cerebral function by extrinsic drugs, toxins, or hypoxia or by internal metabolic derangements such as hypoglycemia, azotemia, hepatic failure, or hypercalcemia; and

3) brainstem lesions that cause proximate damage to the RAS.

The RAS is a physiologic system contained within the rostral portion of the reticular formation; it consists of neurons located bilaterally in the medial tegmental gray matter of the brainstem that extends from the medulla to the diencephalon. Animal experiments and human clinicopathologic observations have established that the region of the reticular formation that is of critical importance for maintaining wakefulness extends from the rostral pons to the caudal diencephalon. A practical consideration follows: *Destructive lesions that produce coma also affect adjacent brainstem structures of the upper pons, midbrain, and diencephalon that are concerned with pupillary function and eye movements.* Abnormalities in these systems provide convenient, albeit indirect evidence of direct brainstem damage as the source of coma. Lesions confined to the cerebral hemispheres do not immediately affect the brainstem RAS, although secondary dysfunction of the upper brainstem often results from compression by a mass in a cerebral hemisphere [52].

Brainstem RAS neurons project rostrally to the cortex, primarily via thalamic relay nuclei that exert a tonic influence on the activity of the cerebral cortex. Experimental work in primates suggests that the brainstem RAS affects the level of consciousness by suppressing the activity of the nonspecific nuclei that have a predominantly inhibitory effect on the cortex, but this is an oversimplification. It is believed that high-frequency (30 to 40 Hz) rhythms synchronize cortical and thalamic neurons during wakefulness. The basis of behavioral arousal by environmental stimuli (somesthetic, auditory, and visual) is related to the rich innervation that the RAS receives from these sensory systems.

The relays between the RAS and the thalamic and cortical areas are accomplished by neurotransmitters. Of these, the influences on arousal of acetylcholine and biogenic amines have been studied most extensively. Cholinergic fibers connect the midbrain to other areas of the upper brainstem, thalamus, and cortex. Serotonin and norepinephrine also subserve important functions in the regulation of the sleep-wake cycle. Their roles in arousal and coma have not been clearly established, although the alerting effects of amphetamines are likely to be mediated by catecholamine release [51].

A reduction in alertness is related in a semiquantitative way to the total mass of damaged cortex or RAS and is not focally represented in any region of the hemispheres, with the exception that large, acute, and purely unilateral hemispheral lesions, particularly on the left, may cause transient drowsiness even in the absence of damage to the opposite hemisphere or RAS. *Hemispheral lesions in most instances cause coma indirectly when a large mass in one or both hemispheres secondarily compresses the upper brainstem and diencephalic RAS.* This is most typical of cerebral hemorrhages and rapidly expanding tumors. The magnitude of decrease in alertness is also related to the rapidity of onset of the cortical dysfunction or RAS compression [49].

This secondary compressive effect has led to a concept of *transtentorial herniation* with progressive brainstem dysfunction to explain the neurologic signs that accompany coma from supratentorial mass lesions. Herniation refers to displacement of brain tissue away from a mass, past a less mobile structure such as the dura, and into a space that it normally does not occupy. The common herniations seen at postmortem examinations are transfalcial (displacement of the cingu- late gyrus under the falx in the anterior midline), transtentorial (medial temporal lobe displacement into the tentorial opening), and foraminal (the cerebellar tonsils forced into the foramen magnum). Uncal transtentorial herniation, or impaction of the anterior medial temporal gyrus mto the anterior portion of the tentorial opening, causes compression of the third nerve with pupillary dilation. Subsequent coma may be due to midbrain compression by the parahippocampal gyrus. Central transtentorial herniation denotes symmetric downward movement of ttie upper diencephalon (thalamic region) through the tentorial opening in the midline and is heralded by miotic pupils and drowsiness. These shifts in brain are thought to cause a progression of rostral to caudal brainstem compression of first the midbrain, then the pons, and finally the medulla, leading to the sequential appearance of neurologic signs corresponding to the level damaged and to progressively diminished alertness. However, many patients with supratentorial masses do not follow these stereotypic patterns; for example, an orderly progression of signs from midbrain to medulla is often bypassed in catastrophic esions where all brainstem functions are lost almost simultaneously, urthermore, drowsiness and stupor typically occur with moderate ateral shifts at the level of the diencephalon when there is only minimal vertical displacement of structures near the tentorial opening and well before downward herniation is evident on computed tomogaphy (CT) scan or magnetic resonance imaging (MRI) [7,50].

Pathophysiology of coma and confusion. Coma of metabolic origin is produced by interruption of energy substrate delivery (hypoxia, ischemia, hypoglycemia) or by alteration of the neurophysiology responses of neuronal membranes (drug or alcohol intoxication, toxic endogenous metabolites, anesthesia, or epilepsy). These same metabolic abnormalites can cause widespread neuronal dysfunction in the cortex that reduces all aspects of mentation and results in an acute confusional state. In this way, acute confusion and coma can be viewed as a continuum in metabolic encephalopathy.

The large group of *drugs* that depress the CNS, anesthetics, and some endogenous toxins appear to produce coma by suppression of both the RAS and the cerebral cortex. For this reason, combinations of cortical and brainstem signs occur in drug overdose and some other metabolic comas, which may lead to a specious diagnosis of structural brainstem damage [50].

Although all metabolic derangements alter neuronal electrophysiology, the only primary disturbance of brain electrical activity encountered in clinical practice is *epilepsy*. Continuous, generalized electrical discharges of the cortex (seizures) are associated with coma even in the absence of epileptic motor activity (convulsions). Coma following seizures, termed the *postictal state*, may be due to exhaustion of energy metabolites or be secondary to locally toxic molecules produced during the seizures. Recovery from postictal unresponsiveness occurs when neuronal metabolic balance is restored. The postictal state produces a pattern of continuous, generalized slowing of the background EEG activity similar to that of metabolic encephalopathy [52].

Approach to the Patient

In Coma. The diagnosis and acute management of coma depend on knowledge of its main causes, an interpretation of certain clinical signs, notably the brainstem reflexes, and the efficient use of diagnostic tests. It is common knowledge that acute respiratory and cardiovascular problems should be attended to prior to neurologic diagnosis. A complete medical evaluation, except for the vital signs, funduscopy, and examination for nuchal rigidity, may be deferred until the neurologic evaluation has established the severity and nature of coma.

History. In many cases, the cause of coma is immediately evident (e.g., trauma, cardiac arrest, or known drug ingestion). In the remainder, historical information about the onset of coma is often sparse. The most useful historical points are:

1) the circumstances and temporal profile of the onset of neurologic symptoms;

2) the precise details of preceding neurologic symptoms (confusion, weakness, headache, seizures, dizziness, diplopia, or vomiting);

3) the use of medications, illicit drugs, or alcohol;

4) a history of liver, kidney, lung, heart, or other medical disease. Telephone calls to family and observers on the scene are an important part of the initial evaluation. Ambulance attendants often provide the best information in an enigmatic case.

Physical examination and general observations. The temperature, pulse, respiratory rate and pattern, and blood pressure should be measured. Fever suggests systemic infection, bacterial meningitis, encephalitis, or a brain lesion that has disturbed the temperature-regulating centers. High body temperature (42° to 44°C) associated with dry skin should arouse the suspicion of heat stroke or anticholinergic drug intoxication. Hypothermia is observed with bodily exposure to lowered environmental temperature; alcoholic, barbiturate, sedative, or phenothiazine intoxication; hypoglycemia; peripheral circulatory failure; or hypothyroidism. Hypothermia itself causes coma only when the temperature is below 31°C. Aberrant respiratory patterns that may reflect brainstem disorders are discussed below. A change of pulse rate combined with hyperventilation and hypertension may signal an increase in intracranial pressure. Marked hypertension is a very helpful signature of hypertensive encephalopathy, cerebral hemorrhage, or hydrocephalus and occurs acutely, but to lesser degree, after head trauma. Hypotension is characteristic of coma from alcohol or barbiturate intoxication, internal hemorrhage, myocardial infarction, septicemia, and Addisonian crisis. The funduscopic examination is used to detect subarachnoid hemorrhage (subhyaloid hemorrhages), hypertensive encephalopathy (exudates, hemorrhages, vessel-crossing changes), and increased intracranial pressure (papilledema). Generalized cutaneous petechiae suggest thrombotic thrombocytopenic purpura or bleeding diathesis associated with intracerebral hemorrhage [53].

General neurologic assessment. An exact description of spontaneous and elicited movements is of great value in establishing the level of neurologic dysfunction. The patient's state should be observed first without examiner intervention. The nature of respirations and spontaneous movements are noted. Patients who toss about, reach up toward the face, cross their legs, yawn, swallow, cough, or moan are closest to being awake. The only sign of seizures may be small excursion twitching of a foot, finger, or facial muscle. An outturned leg at rest or lack of restless movements on one side suggests a hemiparesis.

The terms *decorticate* and *decerebrate rigidity,* or «posturing», describe stereotyped arm and leg movements occurring spontaneously or elicited by sensory stimulation. Flexion of the elbows and wrists and arm supination (decortication) suggest

severe bilateral damage in the hemispheres above the midbrain, whereas extension of the elbows and wrists with pronation (decerebration) suggests damage to the corticospinal tracts in the midbrain or caudal diencephalon. Arm extension with minimal leg flexion or flaccid legs has been associated with lesions in the low pons. These terms, however, have been adapted from animal work and cannot be applied with the same precision to coma in humans. Acute lesions of any type frequently cause limb extension regardless of location, and almost all extensor posturing becomes flexion as time passes, so posturing alone cannot be utilized to make an anatomic localization. Metabolic coma, especially after acute hypoxia, also may produce vigorous spontaneous extensor (decerebrate) rigidity. Posturing may coexist with purposeful limb movements, usually reflecting subtotal damage to the motor system. Multifocal myoclonus is almost always an indication of a metabolic disorder, particularly azotemia, anoxia, or drug ingestion. In a drowsy and confused patient bilateral asterixis is a certain sign of metabolic encephalopathy or drug ingestion [49-51].

Elicited movements and level of arousal If the patient is not aroused by conversational voice, a sequence of increasingly intense stimuli is used to determine the patient's best level of arousal and the optimal motor response of each limb. It should be recognized that the results of this testing may vary from minute to minute and that serial examinations are most useful. Nasal tickle with a cotton wisp is a strong arousal stimulus. Pressure on the knuckles or bony prominences a is the preferred and humane form of noxious stimulus. Pinching the skin over the face, chest, or limbs causes unsightly ecchymoses is not necessary.

Responses to noxious stimuli should be appraised critically. Abduction avoidance movement of a limb is usually purposeful and denotes an intact corticospinal system to that limb. Stereotyped posturing following stimulation of a limb indicates severe dysfunction of the corticospinal system. Adduction and flexion of the stimulated limbs may be reflex movements and imply corticospinal system damage. Brief clonic or twitching limb movements occur at the end of extensor posturing excursions and should not be mistaken for convulsions.

Brainstem reflexes are a key to localization of the lesion in coma. As a rule, coma associated with normal brainstem function indicates widespread and bilateral hemi-spheral disease or dysfunction. The brainstem reflexes that allow convenient examination are pupillary light responses, eye movements, both spontaneous and elicited, and respiratory pattern.

Pupillary reaction is examined with a bright, diffuse light and, if the response is absent, confirmed with a magnifying lens. Light reaction in pupils smaller than 2 mm is often difficult to appreciate, and excessive room lighting mutes pupillary reactivity. Symmetrically reactive round pupils (2,5 to 5 mm in diameter) usually exclude midbrain damage as the cause of coma. One enlarged (greater than 5 mm) and unreactive or poorly reactive pupil results either from an intrinsic midbrain lesion (on the same side) or, far more commonly, is secondary to compression or stretching of the III nerve by the secondary effects of a mass. Unilateral pupillary enlargement usually denotes an ipsilateral mass, but this sign occasionally occurs contralateral possibly by compression of the midbrain or III nerve against the opposite tentorial margin. Oval and slightly eccentric pupils accompany early midbrain III nerve compression. Bilaterally dilated and unreactive pupils indicate severe midbrain damage, usually from secondary compression by transtentorial herniation or from ingestion of drugs with anticholinergic activity. The use of mydriatic eye drop by a previous examiner, self-administration by the patient, or direct ocular trauma may cause misleading pupillary enlargement. Reactive and bilaterally small but not pinpoint pupils (1 to 2,5 mm) are most commonly seen in metabolic encephalopathy or after deep bilateral hemispheral lesions such as hydrocephalus or thalamic hemorrhage. This has been attributed to dysfunction of sympathetic nervous system efferents emerging from the posterior hypothalamus. Very small but reactive pupils (less than 1 mm) characterize narcotic or barbiturate overdose but also occur with acute, extensive bilateral pontine damage, usually from hemorrhage. The response to naloxone and the presence of reflex eye movements distinguish these. The unilaterally small pupil of a Horner's syndrome is detected by failure of the pupil to enlarge in the dark. It is rare in coma but may occur ipsilateral to a large cerebral hemorrhage that affects the thalamus. Lid tone, tested by lifting the eyelids, palpating resistance to opening, and speed of closure, is reduced progressively as coma deepens [50].

Eye movements are the second foundation of physical diagnosis in coma because their examination permits an analysis of a large portion of the brainstem. The eyes are first observed by elevating the lids and noting the resting position and spontaneous movements of the globes. Horizontal divergence of the eyes at rest is normally observed in drowsiness. As patients either awaken or coma deepens, the ocular axes become parallel again. An adducted eye at rest indicates lateral rectus paresis due to a VI nerve lesion, and when bilateral, it is often a sign of increased intracranial pressure. An abducted eye at rest, often accompanied by ipsilateral pupillary enlargement, indicates

medial rectus paresis due to III nerve dysfunction. With few exceptions, vertical separation of the ocular axes, or *skew deviation*, results from pontine or cerebellar lesions.

Spontaneous eye movements in coma generally take the form of conjugate horizontal roving. This motion exonerates the midbrain and pons and has the same meaning as normal reflex eye movements. Cyclic vertical downward movements are seen in specific circumstances. «Ocular bobbing» describes a brisk downward and slow upward movement of the globes associated with loss of horizontal eye movements and is diagnostic of bilateral pontine damage. «Ocular dipping» is a slower, arrhythmic downward movement followed by a faster upward movement in patients with normal reflex horizontal gaze and denotes diffuse anoxic damage to the cerebral cortex. The eyes may turn down and inward in thalamic and upper midbrain lesions [49].

«Doll's-eye», or *oculocephalic,* responses are reflex movements tested by moving the head from side to side or vertically, first slowly then briskly; eye movements are evoked in the opposite direction to head movement. These responses are generated by brainstem mechanisms originating in the labyrinths and cervical proprioceptors. They are normally suppressed by visual fixation mediated by the cerebral hemispheres in awake patients but appear as the hemispheres become suppressed or inactive. The neuronal pathways for reflex horizontal eye movements require integrity of the region surrounding the VI nerve nucleus and are yoked to the contralateral III nerve via the medial longitudinal fasciculus (MLF). Two disparate pieces of information are obtained from the reflex eye movements. First, in coma resulting from bihemispheral disease or metabolic or drug depression, the eyes move easily or «loosely» from side to side in a direction opposite to the direction of head turning. The ease with which the globes move toward the opposite side is a reflection of disinhibition of brainstem reflexes by damaged cerebral hemispheres. Second, conjugate oculocephalic movements demonstrate the integrity of brainstem pathways extending from the high cervical spinal cord and medulla, where vestibular and proprioceptive input from head turning originates, to the midbrain, at the level of the III nerve. Thus full and conjugate eye movements that are induced by the oculocephalic maneuver demonstrate the intactness of a large segment of brainstem and virtually exclude a primary lesion of the brainstem as the cause of coma [53].

Incomplete ocular adduction indicates an ipsilateral midbrain (III nerve) lesion or damage to the pathways mediating reflex eye movements in the MLF (i.e., internuclear ophthalmoplegia). III nerve damage is usually associated with an enlarged pupil and

horizontal ocular divergence at rest, whereas MLF lesions are unrelated to pupillary function and leave the globe in the primary position. Adduction of the globes is by nature more difficult to obtain than abduction, and subtle abnormalities in the doll's-eye maneuver should be interpreted with caution.

Caloric stimulation of the vestibular apparatus (*oculovestibular* response) is an adjunct to the oculocephalic test, acting as a stronger stimulus to reflex eye movements but giving fundamentally the same information. Irrigation of the external auditory canal with cool water causes convection currents in the endolymph of the labyrinths of the inner ear. An intact brainstem pathway from the labyrinths to the oculomotor nuclei of the midbrain is indicated, with brief latency, by tonic deviation of both eyes (lasting 30-120 s) to the side of cool-water irrigation. Bilateral conjugate eye movements therefore have similar significance as full oculocephalic responses. If the cerebral hemispheres are functioning properly, as in hysterical coma, an obligate rapid corrective movement is generated away from the side of tonic deviation. The absence of this nystagmus-like quick phase signifies that the cerebral hemispheres are damaged or suppressed [49].

Conjugate horizontal ocular deviation at rest or incomplete conjugate eye movements with head turning indicate damage in the pons on the side of the gaze paresis or frontal lobe damage on the opposite side. This phenomenon may be summarized by the following aphorism: *The eyes look toward a hemispheral lesion and away from a brainstem lesion.* It is usually possible to overcome the ocular deviation associated with frontal lobe damage by oculocephalic testing. Seizures also may cause aversive opposite eye deviation with rhythmic, jerky movements to the side of gaze. On rare occasions, the eyes may turn paradoxically away from the side of a deep hemispheral lesion («wrong-way eyes»). In hydrocephalus with dilatation of the III ventricle, the globes frequently rest below the horizontal meridian. Two types of rapid rhythmic eye movements may occur in stupor or coma. *Ocular myoclonus* is a rapid horizontal oscillatory nystagmus usually associated with a similar movement of the palate and due to damage to the central tegmental fasciculus, a longitudinal tract in the brainstem. *Opsoclonus* is an irregular, jerky, saccadic movement varying in direction those results from cerebellar lesions [51].

A major pitfall in coma diagnosis may occur when reflex eye movements are suppressed by drugs. The eyes then move with the head as it is turned as if the globes locked in place, thus spuriously suggesting brainstem damage. Overdoses of phenytoin, tricyclic antidepressants, and barbiturates are commonly implicated as well as, alcohol,

phenothiazines, diazepam, and neuromuscular blockers such as pancuronium. The presence of normal pupillary size and light reaction will distinguish most drug-induced comas from brainstem damage. Small to midposition, 1-3 mm nonreactive pupils may occur with very high serum levels of barbiturates or secondary to hydrocephalus.

The *corneal reflexes* are rarely useful alone, they may corroborate eye movement abnormalities because they also depend on the integrity of pontine pathways. By touching the cornea with a wisp of cotton, a response consisting of brief bilateral lid closure may be observed. The corneal response may be lost if the reflex connections between the V and VII cranial nerves within the pons are damaged. The normal efferent response is bilateral, with closure of both eyelids. CNS depressant drugs diminish or eliminate the corneal responses soon after the reflex eye movements become paralyzed but before the pupils become unreactive to light.

Respiration. Respiratory patterns have received much attention in coma diagnosis but are of inconsistent localizing value. Shallow, slow, but well-timed regular breathing suggests metabolic or drug depression. Rapid, deep (Kussmaul) breathing usually implies metabolic acidosis but also may occur with pontomesencephalic lesions. Cheyne-Stokes respiration in its classic cyclic form, ending with a brief apneic period, signifies mild bihemispheral damage or metabolic suppression and commonly accompanies light coma. Agonal gasps reflect bilateral lower brainstem damage and are well known as the terminal respiratory pattern of severe brain damage. In brain-dead patients, shallow respiratory-like movements with irregular, nonrepetitive back arching may be produced by hypoxia and are probably generated by the surviving cervical spinal cord and lower medulla. Other cyclic breathing variations are not usually diagnostic of specific local lesions [50,51].

Approach to the Patient

Acute Confusion is characterized by difficulty in maintaining a coherent stream of thinking and mental performance. This is manifested most obviously by inattention and disorientation, which in turn may generate difficulty with memory and all mental activities. Attention may be gauged by the clarity of and speed of response while the history is being taken but should also be examined by having the patient repeat strings of numbers (most adults easily retain seven digits forward and four backward) or perform serial calculations that require holding the result of one calculation in a working memory in order to pursue the next step - the serial 3-from-30 subtraction test is the usual paradigm. Orientation and memory are tested by asking the patient in a forthright manner the date, inclusive of month, day, year, and day of week; the precise

place; and some items of generally acknowledged and universally known information (the name of the President, a recent national catastrophe, the state capital). Further probing may be necessary to reveal a defect - why is the patient in the hospital; what is his or her address, zip code, telephone number, social security number? Problems of increasing complexity may be pursued but they give little more practical information once a confusional state has been established.

Evidence of drug ingestion should be sought on general physical examination. Other salient neurologic findings are the level of alertness, which typically fluctuates in acute cases; indications of focal damage of the cerebrum such as hemiparesis, hemianopia, and particularly aphasia; adventitious movements of myoclonus; or convulsions. The most pertinent sign of a metabolic encephalopathy is *asterixis,* which is an arrhythmic flapping tremor that is typically elicited by asking the patient to hold the arm out straight with the wrist fully extended. After a few seconds, a large jerking lapse in the posture of the hand occurs and then a rapid return to the original position. The same can be appreciated in any tonically held posture, even of the tongue, and in extreme form the movements may intrude on voluntary limb motion. Bilateral asterixis always signifies a metabolic encephalopathy, for example, from hepatic failure or from drug ingestion, especially with anticonvulsants. Myoclonic jerking and tremor in an awake patient are typical of uremic encephalopathy or antipsychotic (butyrophone) drug ingestion [52].

The language of the confused patient may be disorganized and rambling, even to the extent of incorporating paraphasic words. These features, along with impaired comprehension that is due to inattention, may be mistaken for aphasia.

Distinguishing dementia from confusion is a great problem. The memory loss of dementia necessarily engenders a confusional state that varies in severity from hour to hour and day to day. Poor mental performance is derived mainly from incomplete recollection, inadequate access to names and ideas, and on the inability to retain new information, thus affecting orientation and factual knowledge; attention is preserved in the early stages of the process. Depending upon the nature of the dementing disease, there may be added specific deficits of language, praxis, visual-spatial performance, or a slowed frontal lobe state. Eventually dementia produces a chronic confusion with breakdown of all types of mental performance, and the distinction from confusion depends simply on the chronic nature of the condition.

Laboratory examination for acute confusion and coma. 4 laboratory tests are used most frequently in the diagnosis of confusion and coma: chemical-toxicologic analysis of blood and urine, CT or MRI, EEG, and CSF examination.

Chemical blood determinations are made routinely to investigate metabolic, toxic, or drug-induced encephalopathies. The major metabolic aberrations are those of electrolytes, calcium, blood urea nitrogen (BUN), glucose, plasma osmolarity, and hepatic dysfunction (NH_3). Toxicologic analysis is of great value in any case of coma where the diagnosis is not immediately clear. However, the presence of exogenous drugs or toxins, especially alcohol, does not ensure that other factors, particularly head trauma, may not also contribute to the clinical state. Ethanol levels in non-habituated patients of 200 mg/dL generally cause confusion and impaired mental activity and above 300 mg/dL are associated with stupor. The development of tolerance may allow the chronic alcoholic to remain awake at levels over 400 mg/dL [7,49].

The increased availability of CT and MRI has focused attention on causes of coma that are radiologically detectable (e.g., hemorrhages, tumors, or hydrocephalus). This approach, although at times expedient, is imprudent because most cases of confusion and coma are metabolic or toxic in origin. The notion that a normal CT scan excludes anatomic lesions as the cause of coma is also erroneous. Early bilateral hemisphere infarction, small brainstem lesions, encephalitis, meningitis, mechanical shearing of axons as a result of closed head trauma, absent cerebral perfusion associated with brain death, sagittal sinus thrombosis, and subdural hematomas that are isodense to adjacent brain are some of the lesions that may be overlooked by CT. Even MRI may fail to demonstrate these processes early in their evolution. Nevertheless, in coma of unknown etiology, a CT or MRI scan should be obtained. In those cases in which the etiology is clinically apparent, these provide verification and define the extent of the lesion. With acute mass lesions, 3 to 5 mm of horizontal displacement of the pineal body from the midline generally corresponds to drowsiness; 5 to 8 mm corresponds to, stupor, and greater than 8 mm corresponds to coma. As a supratentorial mass enlarges, the opposite perimesencephalic cistern is first compressed from lateral movement of the brainstem, the ipsilateral cistern is widened, and finally, both are compressed from the lateral mass effect. The lateralventricle opposite the mass becomes enlarged as the third ventricle is compressed. These radiologic features of tissue shifts near the tentorial opening are helpful in correlating the clinical state with the progress of a mass lesion on scans. For technical reasons, MRI is difficult to perform in comatose patients, and it also does not demonstrate hemorrhages as well as CT.

The EEG is useful in metabolic or drug-induced confusional states but is rarely diagnostic in coma, with the exception of comas due to clinically unrecognized seizures, herpes virus encephalitis, and Creutzfeldt-Jakob disease. The amount of background slowing of the EEG is a useful gauge of the severity of any diffuse encephalopathy. Predominant high-voltage slowing δ-waves in the frontal regions is typical of metabolic coma, as from hepatic failure and widespread fast β-activity implicates the effects of sedative drugs. A pattern of «α-coma» is defined by widespread, invariant 8-12-Hz activity superficially resembling the normal α-rhythm of waking but unresponsive to environmental stimuli. A-coma results from either high pontine or diffuse cortical damage and is associated with a poor prognosis. Coma due to persistent epileptic discharges that are not clinically manifested may be revealed by EEG recordings. Normal α-activity on the EEG also may alert the clinician to the locked-in syndrome or a hysterical case. Computed on-line EEG analysis and evoked potential recordings (auditory and somatosensory) are useful additional methods for coma diagnosis and monitoring.

Lumbar puncture is now used more judiciously in cases of coma or confusion because the CT scan excludes intracerebral hemorrhages and most subarachnoid hemorrhages. The use of lumbar puncture in coma is limited to diagnosis of meningitis or encephalitis and instances of suspected subarachnoid hemorrhage in which the CT is normal. Lumbar puncture should not be deferred if meningitis is a strong clinical possibility. Xanthochromia indicates preexisting blood in the CSF (or very high protein levels) and permits exclusion of a traumatic puncture. Knowing the pressure within the subarachnoid space is of further help in interpreting abnormalities of the cell count and protein content of the CSF [7,50].

Differential diagnosis of confusion and coma. In most instances, confusion and coma are part of an obvious medical problem such as known drug ingestion, hypoxia, stroke, trauma, or liver or kidney failure. Attention is then appropriately focused on the primary illness. A complete listing of all diseases that cause confusion and coma would serve little purpose, since it would not aid diagnosis. Some general rules, however, are helpful. Illnesses that cause sudden or acute coma are due to drug ingestion or to one of the catastrophic brain lesions - hemorrhage, trauma, hypoxia, or, rarely, acute basilar artery occlusion. Coma that appears subacutely is usually related to preceding medical or neurologic problems, including the secondary brain swelling that surrounds a preexisting lesion. Coma diagnosis, therefore, requires familiarity with the common intracerebral catastrophes. These may be summarized as follows:

1) basal ganglia and thalamic hemorrhage (acute but not instantaneous onset, vomiting, headache, hemiplegia, and characteristic eye signs);

2) subarachnoid hemorrhage (instantaneous onset, severe headache, neck stiffness, vomiting, III or VI nerve lesions, transient loss of consciousness, or sudden coma with vigorous extensor posturing);

3) pontine hemorrhage (sudden onset, pinpoint pupils, loss of reflex eye movements and corneal responses, ocular bobbing, posturing, hyperventilation, and sweating);

4) cerebellar hemorrhage (occipital headache, vomiting, gaze paresis, and inability to stand);

5) basilar artery thrombosis (neurologic prodrome or warning spells, diplopia, dysarthria, vomiting, eye movement and corneal response abnormalities, and asymmetric limb paresis). The most common stroke, namely, infarction in the territory of the middle cerebral artery, does not cause coma acutely. The syndrome of acute hydrocephalus causing coma may accompany many intracranial catastrophes, particularly subarachnoid hemorrhage. Acute symmetric enlargement of both lateral ventricles causes headache and sometimes vomiting followed by drowsiness that may progress quickly to coma, with extensor posturing of the limbs, bilateral Babinski signs, small nonreactive pupils, and impaired vertical oculocephalic movements.

If the history and examination are not typical for any neurologic diagnosis and metabolic or drug causes are excluded, then information obtained from CT or MRI may be used as outlined in Table 2. The CT scan is useful to focus the differential diagnosis, and because of its accuracy and general availability. The majority of medical causes of coma are established without a CT or with the study being normal [49-51].

Coma after head trauma. Concussion is a common form of transient coma that results from torsion of the hemispheres about the midbrain-diencephalic junction with brief interruption of RAS function. Persistent coma after head trauma presents a more complex and serious problem. The main causes are subdural or epidural hemorrhage, deep cerebral hemorrhage, bilateral frontotemporal contusions, and extensive white matter damage.

Coma with ischemic-anoxic brain damage. There are widespread and complex changes in the CNS following cardiac arrest, profound hypotension, or anoxia. Some of these are physiologic and mediated by alterations in electrical and neurotransmitter function, and others may result from endogenously released neurotoxins that ultimately lead to neuronal death. Several clinically recognizable patterns emerge that occur usually in pure form but that may coexist:

1) a deep coma with preserved brainstem function that evolves to the vegetative state or to a dementia, reflecting damage to neurons throughout the cortex-brainstem

function may be suppressed in the first hours, thus emulating brain death, and the limbs may be either flaccid or show vigorous extensor posturing or myoclonic jerks;

2) syndromes of proximal bibrachial and paraparetic weakness or of cortical blindness that are due to bilateral infarctions of the watershed regions between major cortical vessel territories from diminished blood flow;

3) Korsakoff-amnestic state that indicates the selective vulnerability of neurons in the hippocampal cortex;

4) a cerebellar syndrome [49].

Treatment. The immediate goal in acute coma is the prevention of further nervous system damage. Hypotension, hypoglycemia, hypercalcemia, hypoxia, hypercapnia, and hyperthermia should be corrected rapidly and assiduously. An oropharyngeal airway is adequate to keep the pharynx open in drowsy patients who are breathing normally. Tracheal intubation is indicated if there is apnea, upper airway obstruction, hypoventilation, or emesis, or if the patient is liable to aspirate. Mechanical ventilation is required if there is hypoventilation or if there is an intracranial mass and induced hypocapnia is necessary. Intravenous access is established, and naloxone and dextrose are administered if narcotic overdose or hypoglycemia is even remote possibilities. Thiamine is administered with glucose in order to avoid eliciting Wernicke's encephalopathy in malnourished patients. The veins of intravenous drug abusers may be difficult to cannulate; in such cases, naloxone can be injected sublingually through a small-guage needle. In cases of suspected basilar thrombosis with brainstem ischemia, intravenous heparin or a thrombolytic agent is administered after obtaining a CT scan, keeping in mind that cerebellar and pontine hemorrhages resemble the syndrome of basilar artery occlusion. Physostigmine, when used by experienced nhvsicians with careful monitoring, may awaken patients with anti-cholinergic-type drug overdose,,but many physicians believe that this is justified only to treat cardiac arrhythmias resulting from these overdoses. The use of benzodiazepine antagonists is promising for treatment of overdoses and has transient benefit in hepatic encephalopathy. Intravenous administration of water should be monitored carefully in any serious acute CNS illness because of the potential for exacerbati ng brain swelling. Neck injuries must not be overlooked, particularly prior to attempting intubation or eliciting oculocephalic responses. Headache accompanied by fever and meningismus indicates an urgent need for examination of the CSF to diagnose meningitis, and *lumbar puncture should not be delayed while awaiting a CTscan* [52].

Table 2

Approach to the Differential Diagnosis of Coma

NORMAL BRAINSTEM REFLEXES, NO LATERAL1ZING SIGNS

A. Bilateral hemispheral dysfunction without mass lesion *(CT* ***or*** *MRI* ***normal; primary test used for diagnosis is indicated in parentheses)***

1. Drug-toxin ingestion (toxicologic analysis)
2. Endogenous metabolic encephalopathy (glucose, ammonia, calcium, osmolality, Poj, Pccv urea, sodium)
3. Shock, hypertensive encephalopathy
4. Meningitis (CSF analysis)
5. Nonherpetic viral encephalitis (CSF analysis)
6. Epilepsy (EEG)
7. Reye's syndrome (ammonia, increased intracranial pressure)
8. Fat embolism
9. Subarachnoid hemorrhage with normal CT (CSF analysis)
10. Creutzfeldt-Jakob disease (EEG)
11. Hysterical coma or catatonia

B. Anatomic lesions of hemisphere found by CT or MRI

1. Hydrocephalus
2. Bilateral subdural hematomas
3. Bilateral contusions, edema, or axonal shearing of hemispheres due to closed head trauma
4. Subarachnoid hemorrhage
5. Acute disseminated encephalomyelitis (CSF analysis)

NORMAL BRAINSTEM REFLEXES (WITH/WITHOUT UNILATERAL THIRD NERVE PALSY), LATERALIZING MOTOR SIGNS
(CT or MRI ABNORMAL)

A. Unilateral mass lesion

1. Cerebral hemorrhage (basal ganglia, thalamus)
2. Large infarction with surrounding brain edema
3. Herpes virus encephalitis (temporal lobe lesion)
4. Subdural or epidural hematoma

5. Tumor with edema
6. Brain abscess with edema
7. Vasculitis with multiple infarctions
8. Metabolic encephalopathy superimposed on preexisting focal lesions (i.e., stroke with hyperglycemia, hyponatremia, etc.)
9. Pituitary apoplexy

B. Asymmetric signs accompanied by diffuse hemispheral dysfunction

1. Metabolic encephalopathies with asymmetric signs (blood chemical determinations)
2. Isodense subdural hematoma (MRI, CT with contrast)
3. Thrombotic thrombocytopenic purpura (blood smear, platelet count)
4. Epilepsy with focal seizures or postictal state (EEG)

MULTIPLE BRAINSTEM REFLEX ABNORMALITIES

A. Anatomic lesions in brainstem

1. Pontine, midbrain hemorrhage
2. Cerebellar hemorrhage, tumor, abscess
3. Cerebellar infarction with brainstem compression
4. Mass in hemisphere causing advanced upper brainstem compression
5. Primary brainstem tumor, demyelination, or abscess
6. Traumatic brainstem contusion-hemorrhage

B. Brainstem dysfunction without mass lesion

1. Basilar artery thrombosis causing brainstem infarction (clinical signs, angiogram)
2. Severe drug overdose (toxicologic analysis)
3. Brainstem encephalitis
4. Basilar artery migraine

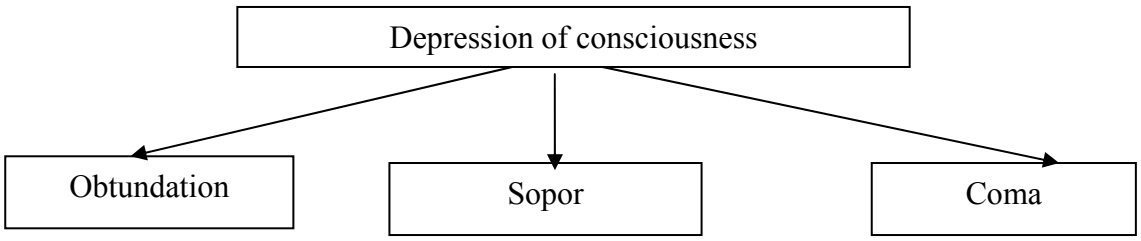
Enlargement of one pupil usually indicates secondary midbrain compression by a hemispherical mass and demands immediate reduction of intracranial pressure (ICP). Surgical evacuation of the mass may be appropriate. Medical management to reduce intracranial pressure consists of intravenous fluid normal saline (the safest fluid because it is slightly hyperosmolar in most patients). Therapeutic hyperventilation may be used to achieve an arterial P_{C02} of 3,7 to 4,2 kPa (28 to 32 mmHg), but its effects are brief. Hyperosmolar therapy with mannitol or an equivalent is the mainstay of ICP reduction.

It may be used simultaneously with hyperventilation in critical cases. A ventricular puncture is necessary to decompress hydrocephalus if medical measures fail to improve alertness. The use of high-dose barbiturates and other neuronal sparing agents soon after cardiac arrest has not been shown in clinical studies to be beneficial and corticosteroids have no proven value except in cases of brain tumor.

Prognosis of coma and the vegetative state. Interest in predicting the outcome of coma is oriented toward allocating medical resources and limiting the support of hopeless cases. To date, no collection of clinical signs except those of brain death assuredly predicts outcome of coma, but certain constellations have prognostic value. Children and young adults may have ominous early clinical findings such as abnormal brainstem reflexes and yet recover. All schemes for prognosis should be taken as only approximate indicators, and medical judgments must be tempered by other factors such as age, underlying disease, and general medical condition. In an attempt to collect prognostic information from large numbers of patients with head injury, the Glasgow Coma Scale was devised; empirically it has predictive value in cases of brain trauma. Major points include a 95% death rate in patients whose pupillary reaction or reflex eye movements are absent 6 h after onset of coma, and a 91% death rate if the pupils are unreactive at 24 h (although roughly 5% make a good recovery).

Prognostication of nontraumatic coma is difficult because of the heterogeneity of contributing diseases. Metabolic coma generally has a more favorable prognosis than anoxic or traumatic coma. Unfavorable signs in the first hours after admission are the absence of any two of pupillary reaction, corneal reflex, or the oculovestibular response. One day after the onset of coma, the preceding signs, in addition to absence of eye opening and muscle tone, predict death or severe disability, and the same signs at 3 days strengthen the prediction of a poor outcome. In many patients, precise combinations of predictive signs do not occur, and coma scales lose their value. The use of evoked potentials aids prognostication in head-injured and post-cardiac arrest patients. Bilateral absence of cortical somatosensory evoked potentials is associated with death or a vegetative state in most cases. Medical practitioners are becoming less reluctant to withdraw support from non-brain-dead but severely neurologically injured patients as predictions become more reliable and resources more limited.

The prognosis for regaining full mental faculties once the vegetative state has supervened is almost nil. Most instances of dramatic recovery, when investigated carefully, yield to the usual rules for prognosis, but it must be acknowledged that rare instances of awakening to a condition of dementia or paralysis after months or years in this state have been documented [7,49].



Glasgow coma scale

Response	Score
Eye opening	
- does not open eyes in response to any stimulation	1
- open eyes in response to painful stimulation	2
- open eyes in response to speech	3
- open eyes spontaneously	4
Motor response	
- no motor response	1
- extends upper extremities in response to pain	2
- flexes upper extremities in response to pain	3
- withdrawal from pain	4
- localized movement in response to painful stimulation	5
- obeying commands	6
Verbal response	
- no verbal response	1
- incomprehensible sounds	2
- inappropriate words	3
- confused speech	4
- orientated	5

Summarized score due to Glasgow coma scale	Traditional terms
15	Clear consciousness
13-14	Obtundation
9-12	Sopor
4-8	Coma
3	Brain death

COMA OF UNKNOWN ETIOLOGY
R 40

Diagnostics criteria:
1. Onset of disease.
2. Obtaining the history: information received from outsiders, medical documents data and other available information.
3. Level of conciousness (Glasgow coma scale).
4. Skin and mucous membranes.
5. Characteristics of breathing, respiratory rate
6. Muscular and eye-bulbe tonus.
7. Pathological reflexes.
8. Offensive breath.
9. Convulsions.
10. Hemodynamics (pulse, blood pressure),ECG.

Volume of medical aid

To provide patency of airways - artificial airway,
S-shape tube.
Oxygen inhalation.
In case of inappropriate ventilation – ALV, intubation of trachea

To provide venous access - isotonic solution of sodium chloride 250-500 ml
Glucose injection 40% 20-40 ml
Naloxone injection 0,04% -1 ml

Maintenance of blood circulation in accordance to pulse indications, blood pressure and ECG → Strophanthine 0,05% - 0,5 ml
Dopamine 4% - 5 ml drop-by drop intravenously

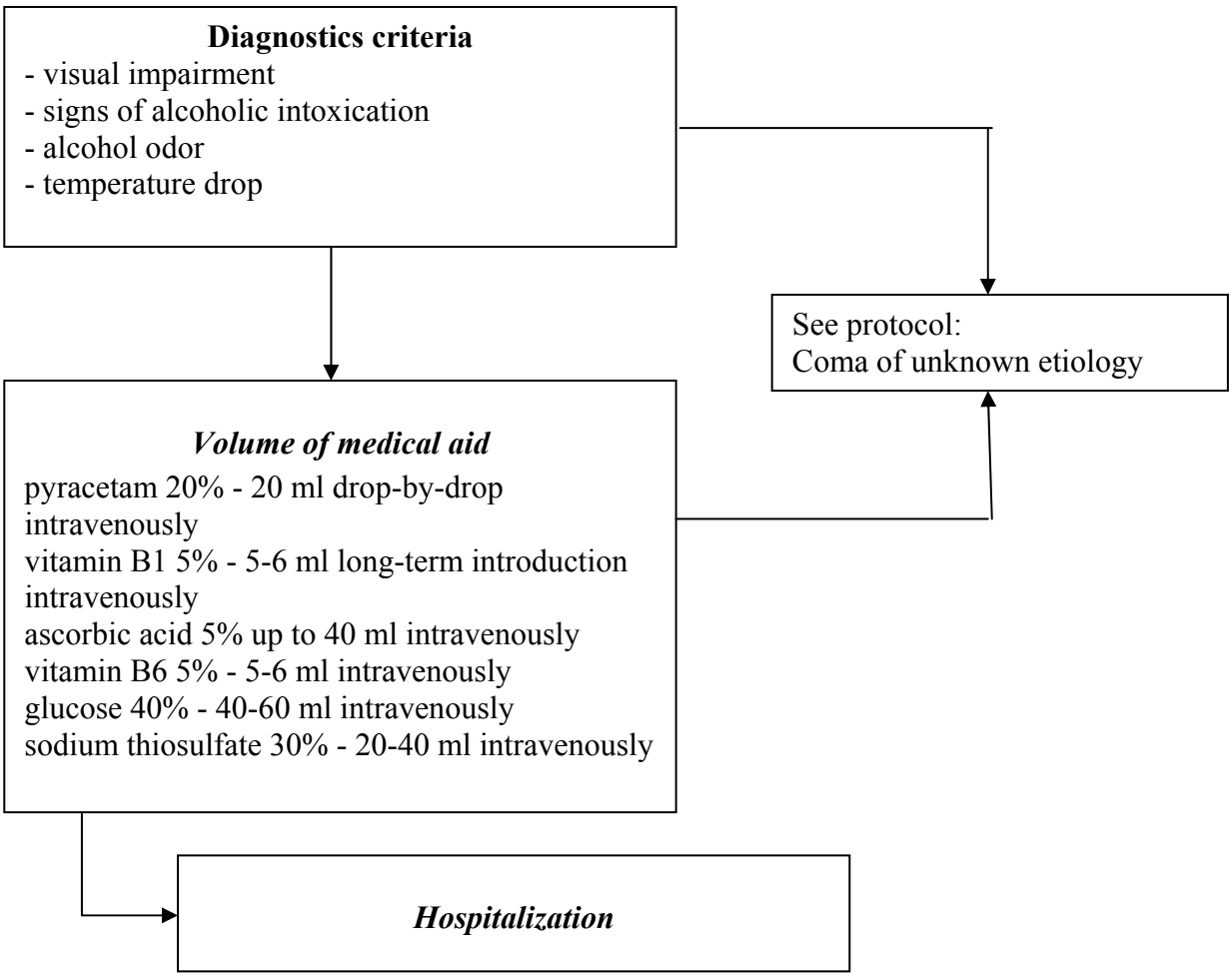
In case of arrhythmia – see correspondent protocol

Elimination of brain edema → Dexamethasone 0,4% - 8-10 mg intravenously
Natrii oxybutiras 20% - 10-12 ml intravenously
Furosemide 1% - 2 ml intravenously, intramusculary

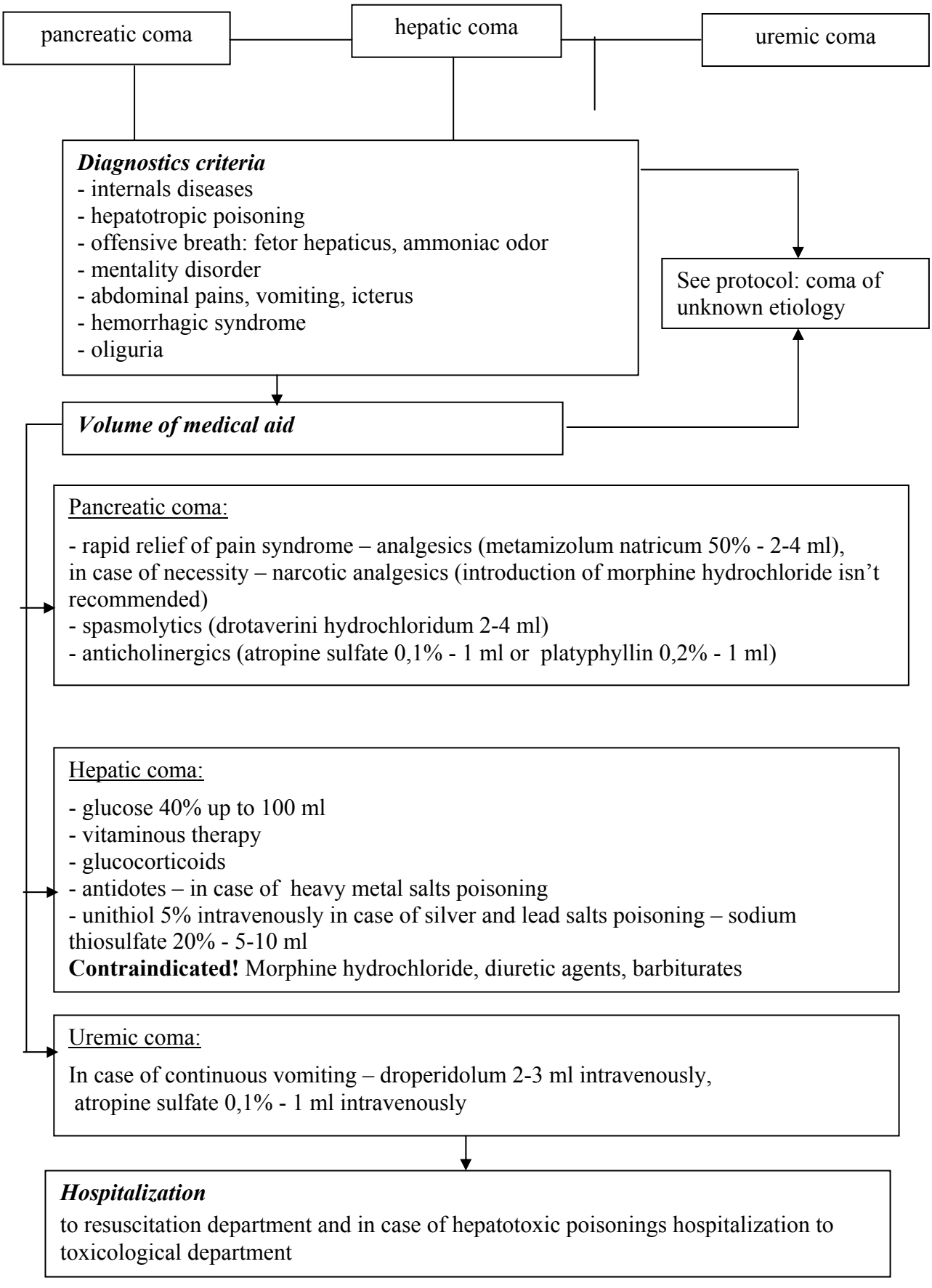
In case of convulsions → Diazepam [seduxen] 0,5% - 2 ml
In case of ineffectiveness provide intermittent introduction until the patient's condition is better – maximum dose – 6 ml

Urgent hospitalization to the toxicological (resuscitation) department

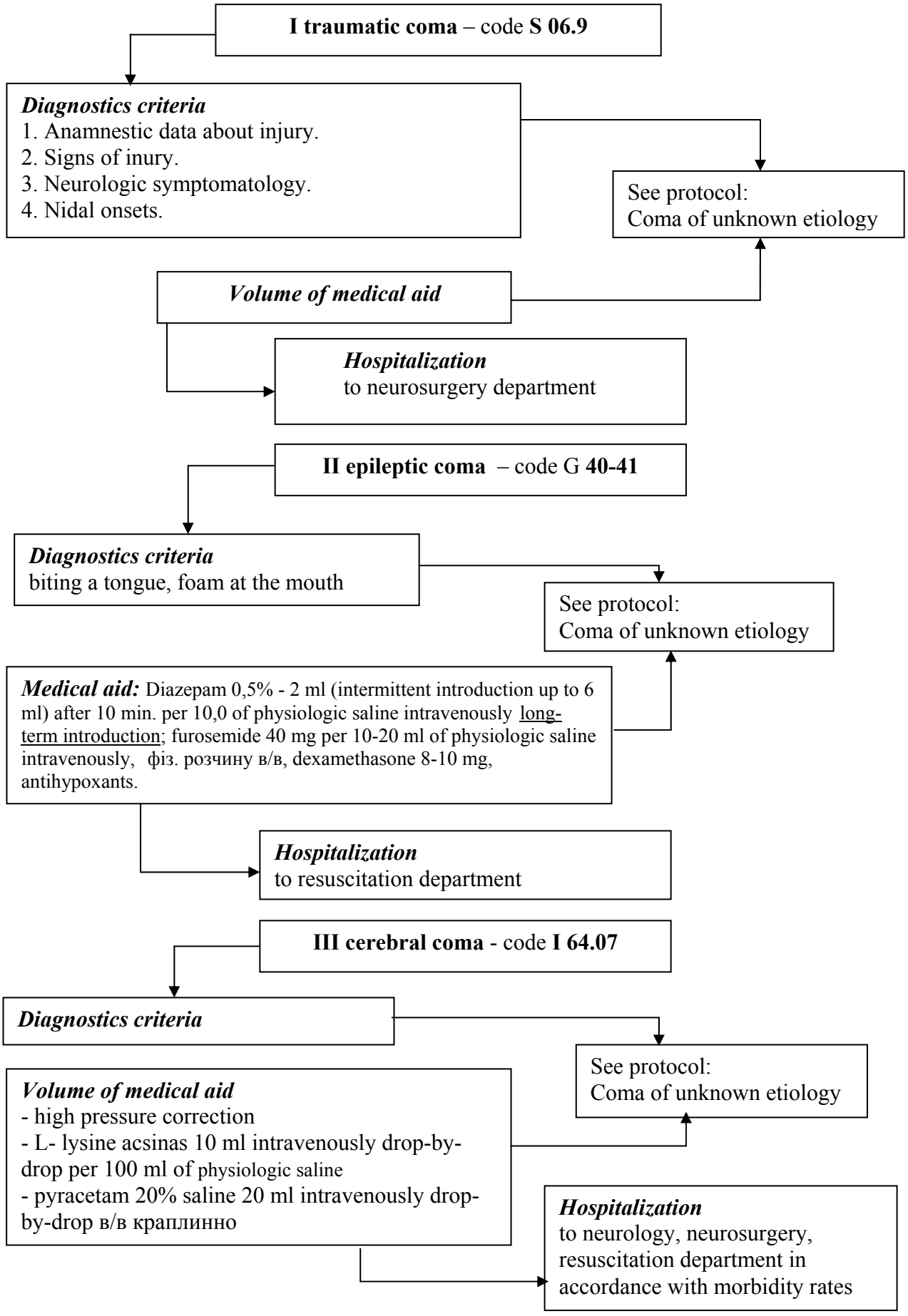
ALCOHOLIC COMA
F 10.0



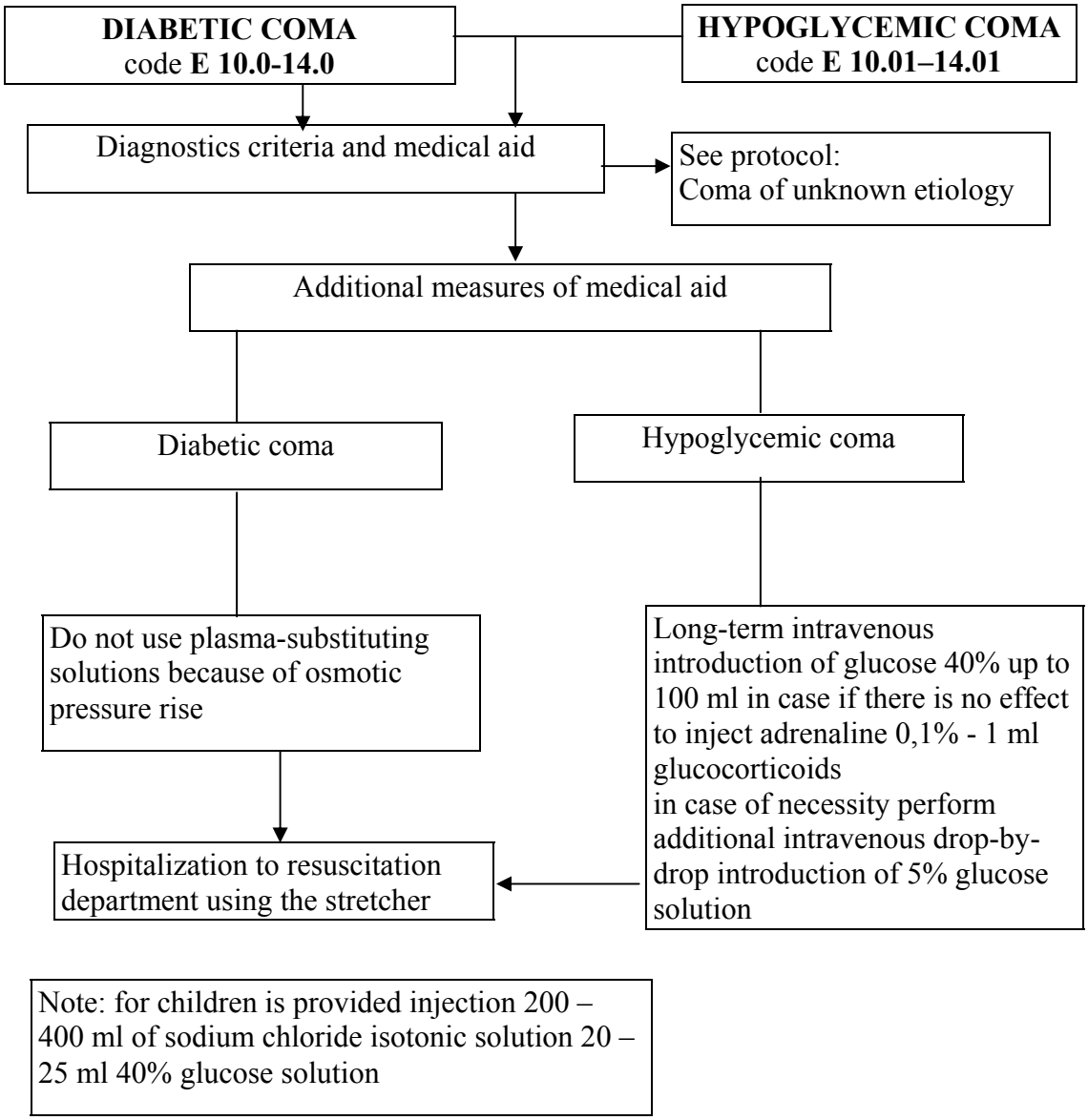
COMAS AS A RESULT OF INTERNAL DISEASES
R 40



COMAS AS A RESULT OF NEUROLOGIC DISEASES

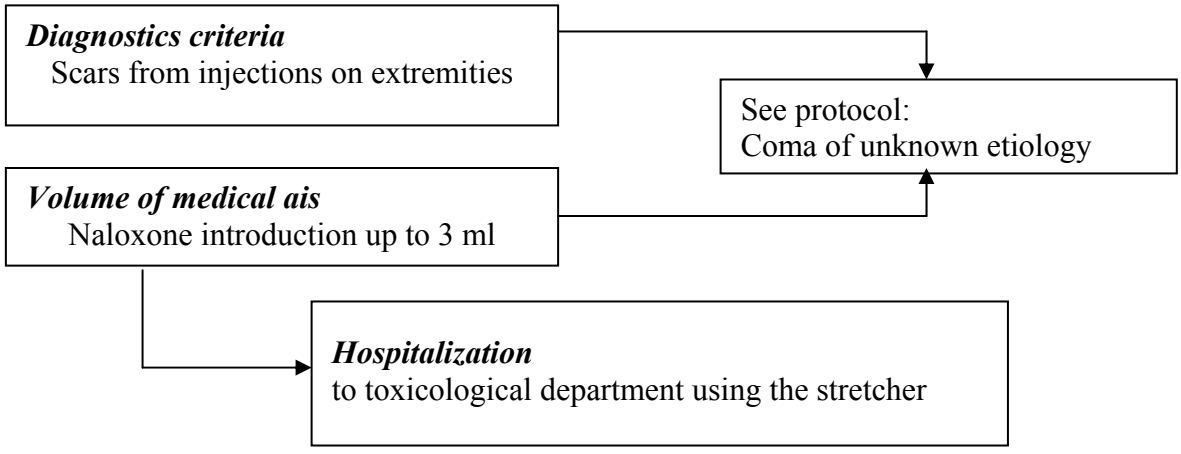


COMAS AS A RESULT OF PANCREATIC DIABETES



Note: for children is provided injection 200 – 400 ml of sodium chloride isotonic solution 20 – 25 ml 40% glucose solution

NARCOMA
F 11 - 14



X. Tasks for final control

1. Which of the following are not referred to the focal epileptic attacks:
   A. Jacksonian sensory.
   B. Jacksonian motor.
   C. Secondary generalized convulsive attacks with aura.
   D. Kojewnikoff's epilepsy.
   E. Absences.

2. Which of the following diseases could be taken into account if convulsive attack arose as a result of rise of temperature?
   A. Epilepsy
   B. Acute inflammatory cerebral diseases.
   C. Alcoholism.
   D. Acute hypertensive encephalopathy.
   E. Brain infarction.

3. First aid in case of generalized tonoclonic seizure is:
   A. Prevent further injury risk.
   B. Prevent tongue bite.
   C. Provide a patency of airways.
   D. Diazepam 0,5% - 2 ml (fractionally up to 6 ml) after 10 min until the cessation of convulsions.
   E. All of mentioned above.

4. First aid in the case of febrile convulsions:
   A. Physical methods of cooling and hyperthermia.
   B. Cleansing enema.
   C. Antipyretic agents – ibuprofen 5-10mg/kg oral intake (for children older than 3 months), paracetamol 10-15 mg/kg, analgin 50% 0,1 ml per 1 year of life, intramuscularly, but no more than 1 ml.
   D. Magnesium sulfate 25% intramuscularly 0,2 ml per 1 year of life but no more than 10 ml, diazepam 0,55 – 0,3 mg per 1 kg.
   E. All of mentioned above.

5. While patient was in a stuffy room he felt sick, lazy eyesight, ringing in the ears, paleness and impairment of consciousness during 1 minute. What is working diagnosis?
   A. Loss of consciousness.
   B. Absence.

C. Torpor.
D. Sopor.
E. Attack without convulsions.

6. Emergency team delivered unconscious patient of 43 years old to admission department. After patient examination it was defined that patient opened his eyes and withdrew his hand in response to painful stimulation, and that his replies were with inappropriate words. Estimate the conscious level by Glasgow coma scale.

A. Clear consciousness.
B. Torpor.
C. Sopor.
D. Coma.
E. Brain death.

7. On the third day of acute respiratory viral infection 20-year-old patient had a headache, vomiting, tonoclonic colvunsions, spoor, oculogyric impairments, hemiparesis; trivial pleocytosis, reduced protein content. What is working diagnosis?

A. Meningitis.
B. Encephalitis.
C. Epilepsy.
D. Cerebral stroke.
E. Migrainous stroke.

8. First aid in the case of hepatic coma is:

A. Glucose 40% - 100 ml, vitamin, glucocorticoids, antidotes.
B. Glucose 40% - 100 ml, morphine hydrochloride, diuretics, barbiturates.
C. Glucocorticoids, antidotes, diuretics, barbiturates.
D. Glucose 40% - 100 ml, vitamin, diuretics, barbiturates.
E. Vitamin, glucocorticoids, antidotes, diuretics.

9. Untidy 50-year-old patient is in a coma. He has miotic pupils, hypersalivation, alcohol odor, muscular hyper tonus of extremities. Body temperature is $35,7^0$C. Blood pressure is 90/60. Define the type of coma:

A. Narcoma.
B. Diabetic coma.
C. Hypoglycemic coma.
D. Uremic coma.
E. Alcoholic coma.

10. 50-year-old patient is unconscious. He does not open eyes to painful stimulation but flexes upper extremities in response to pain. No verbal response. Estimate the level of conscious by Glasgow coma scale:

A. Clear consciousness .
B. Torpor.
C. Sopor.
D. Coma.
E. Brain death.

11. 20-year-old woman suddenly felt unwell while she was taking exercising in sports hall. She felt acute "strike" in her head which was accompanied with severe headache, sickness, multiple vomiting with further impairment of consciousness. The neurological status: somnolentia, tendon reflexes S=D, double-sided pathological Babinskii reflex, Bare test is neganive. Acute symptoms: stiffness of occiput muscles, double-sided Kernig sign, Brudzinski sign. What is working diagnosis?

A. Subarachnoid hemorrhage.
B. Parenchimatous hemorrhage.
C. Cerebellar hemorrhage.
D. Migrainous stroke.
E. Thromboembolic ischemic stroke.

12. 60-year-old patient with malignant course of arterial hypertension and with high blood pressure 210/130 felt diffuse intensive headache, sickness, vomiting, and impairment of consciousness, generalized tonic-clonic seizure. Neurological status: positive meningeal symptoms, no focal neurological symptoms. The eye grounds: double-sided edema of optic nerve disks. After blood pressure correction and brain edema, the described symptoms had been retrogressed after 72 hours. What is working diagnosis?

A. Acute hypertensive encephalopathy.
B. Subarachnoid hemorrhage.
C. Intraventricular hemorrhage.
D. Epilepsy.
E. Cardioembolic ischemic stroke.

13. 55-year-old patient felt sudden headache. He also had vomiting, hyperemia of face and psychomotor agitation. These symptoms arouse on the basis of arterial hypertension and after emotional stress. After 10 minutes there was impairment of consciousness and central superior paraplegia. In 3 hours meningeal symptom arose. What is working diagnosis?

A. Intracerebral bleeding
B. Subarachnoid hemorrhage.
C. Cerebellar hemorrhage.
D. Cardioembolic ischemic stroke.
E. Acute hypertensive encephalopathy.

14. After emotional stress the patient with previous myocardial infarction has coma. There was impairment of vital functions, hemodynamics reduction and respiratory impairment. Primary inspection: miotic pupils, flabby photoreaction, absence of tendon and pathological reflexes. What is working diagnosis?

A. Brainstem hemodynamic stroke.
B. Brainstem cardioembolic stroke.
C. Intracerebral bleeding.
D. Recurrent myocardial infarction.
E. Cardiogenic unconsciousness.

15. After athletic overexertion and alcohol intake 45-year-old patient has coma. Primary inspection: pale skin, hyperhidrosis, mydriasis, blood pressure 100/70 mm Hg, body temperature $36,7^0$C, clonic convulsions, overactive tendon reflexes. Define the type of coma.

A. After epileptic seizure.
B. Diabetic coma.
C. Hypoglycemic coma.
D. Coma as a result of stroke.
E. Alcoholic coma.

16. Emergency team delivered unconscious patient of 18 years old to admission department. Primary inspection: coma, cyanosis of face, injection marks extremities, miosis, Cheyne-Stokes respiration, BP 80/50 mm Hg, heart rate 48 beats per min. Define the type of coma:

A. Narcoma.
B. Diabetic coma.
C. Hypoglycemic coma.
D. Alcoholic coma.
E. Traumatic coma.

17. 50-year old woman was unconscious. Primary inspection: pale face, swelling, dry skin and mucous membranes, urine odor, BP 190/120 mmm Hg, epileptiform activity, meningeal syndrome. Define the type of coma:

A. Hepatic coma.
B. Diabetic coma.
C. Hypoglycemic coma.
D. Uremic coma.
E. Alcoholic coma.

18. 59-year old patient was in coma. Primary inspection: icteric skin and mucous membranes, nosebleed, mydriasis, absence of photoreaction, raw meat odor, periodic clonic convulsions, Cheyne-Stokes respiration, body temperature 38,2^{0}C, BP 80/60 mm Hg, heart rate 120 beats per minute, muffled heart sounds, anuria. Define the type of coma:

A. Hepatic coma.
B. Diabetic coma.
C. Hypoglycemic coma.
D. Uremic coma.
E. Alcoholic coma.

19. 60-year-old woman was unconscious. Primary inspection: dry skin and mucous membranes, cold loose skin, soft eye-bulbs by touch, nosebleed, mydriasis, Kussmaul's respiration, acetone odor, body temperature 36,2^{0}C, BP 70/40 mm Hg, hart rate 120 beats per minute, irregular heart rhythm, muffled heart sounds, thready pulse, abdominal distension, oliguria. Define the type of coma.

A. Hepatic coma.
B. Diabetic coma.
C. Hypoglycemic coma.
D. Uremic coma.
E. Alcoholic coma

20. 40-year old patient suddenly fainted. Primary inspection: unconsciousness, pale skin, generalized tonoclonic convulsion with involuntary urination, cyanosis of face, BP isn’t defined, heart rate 36 beats per minute, ECG: atrioventricular heart block with rare ventricular complexes. What is working diagnosis?

A. Epileptic seizure.
B. Morganya -Adam’s and Stock’s attack.
C. His' bundle peduncles block.
D. Orthostatic syncope.
E. Neurogenic unconsciousness.

21. Among the patients older than 65 years old the causes of convulsive attacks are:

A. Brain-growth.
B. Cerebrovascular accidents.
C. Epilepsy.
D. Metabolic disorders.
E. Infections.

22. 45-year old patient had sudden generalized tonic-clonic convulsion. Patient was in psychic excitement, general trembling. Life history: during 3 days there was alcohol abuse. What is working diagnosis?

A. Epileptic seizure.
B. Abstinent attack.
C. Psychotogenous attack.
D. Unconsciousness.
E. Convulsive attack in case of metabolic disorders.

Answer:

1	2	3	4	5	6	7	8	9	10
E	B	E	E	A	B	B	A	E	D
11	12	13	14	15	16	17	18	19	20
A	A	A	B	E	A	D	A	B	B
21	22								
B	B								

XI. Basic questions after theme

1. Classification of convulsions.
2. The first aid in case of generalized convulsions.
3. Factors which cause loss of consciousness.
4. First aid in the case of loss of consciousness.

TOPIC 8

The emergency in the practice of family doctor in the case of bite, sting, electrical injury, drowning, frostbite and thermal injury

I. Theme actuality. The need of emergency most often occurs in case of wasps, bees, and hornet's bites. This is due to effect of venom in body of victim. In these cases may develop local allergic reaction, Quincke's edema, acute broncho-obstructive syndrome, anaphylactic shock, which require emergency. Drowning is on the 3rd place among death reasons because of accidents. Timely emergency care saves the lives of many victims.

The high temperatures exposure may occur in the case of: a) total body overheating, which is characterized by severe degree of heat stroke, b) thermal skin damage (burns). The most frequent thermal burns as a consequence of the flame, hot liquid in contact with hot objects. The burns are distinguished by depth of skin lesions area.

Electric current voltage 50 W has more heat and causes electrolytic action, voltage above 100 W is dangerous and more than 500 watts - always fatal. Electric shock is the result of domestic and industrial accident. The result of this lesion may occur 1-2 days after injury.

II. Study purposes: to know the clinical signs and the algorithm of emergency in case of bites, electric injuries, drowning and low and high temperatures exposure.

III. Concrete purposes of the module: to be able to provide emergency in case of bites, electric injuries, drowning and low and high temperatures exposure.

IV. A student must know:

- pathophysiological changes in case of bites (insects, snakes), their clinical manifestation;
- the clinical manifestation of bites;
- pathophysiological changes in case of electric injuries, clinical manifestation;
- pathophysiological changes in case of drowning, types of drowning;
- the classification and clinical manifestations of frostbite;
- the classification and clinical manifestations of thermal burns.

V. Task for initial independent training

1. Manifestation voltage which is dangerous for human health:

A. Above 50 volts
B. Above 150 volts
C. Above 100 volts
D. Above 75 volts
E. Above 200 volts

2. The sign of severe degree of overheating is:

A. Body temperature $40,5^0$ C

B. Heart rate more that 130 / min.

C. Reduced tonus of muscles

D. Body temperature $39,5^0$C

E. Heart rate 120/min.

3. Burn shock will develop if the index of lesion is:

A. 30 units

B. 20 units

C. 10 units

D. 35 units

E. 15 units

4. The mechanism of toxic effect of Ukrainian snakes belongs to:

A. hemorrhagic action

B. neurotoxic action

neurotoxic and hemorrhagic actions

D. allergic action

E. Allergic and neurotoxic actions

5. The domestic dog bites the man for his shin. There was shallow wound, bleeding was absent. What is necessary to provide as emergency?

A. the immobilization of limb

B. to give painkillers

C. Toilet of wound, to impose aseptic bandage

D. Toilet of wound, to impose aseptic bandage, appoint to traumotology center

E. appoint to surgical hospital

6. The severity of thermal burns of the skin most often determined by:

A. Rule of Donald

B. rule of nine

C. Frank's Rule

D. rule of palm

E. Rule of thermal shock

7. Indication of deep frostbite is:

A. Pale skin

B. Cyanotic skin

C. pale skin, soft underlying tissues
D. Heart rate 110/min.
E. No correct option

8. After a snake bite (Schytomordnykiv species) on the skin there is:
A. Two deep stab wound
B. edema and hyperemia
C. Swelling and bruising
D. Two-deep stab wound, bruises petechial hemorrhagic
E. The two deep stab wound, swelling

9. A child of 10-years-old dropped in cold water. He was extended after 10 minutes. Clinical examination: pale skin, upper respiratory tract with foam liquid. What is the most likely diagnosis?
A. «Dry» drowning
B. Syncope type of drowning
C. Asphyctic type of drowning
D. Genuine drowning
E. Blue" drowning

10. Frostbite victim got to the hospital. Rectal temperature is 32^0C. What is the degree of frostbite of man?
A. Hard
B. Light
C. Moderate
D Deep
E. Coma

11. The victim's skin of waist, buttocks, thighs, perineum and external genital organs was affected. Identify an area of injury:
A. 28%
B. 21%
C. 19%
D. 36%
E. 18%

Answers:

1	2	3	4	5	6	7	8	9	10	11
C	A	E	A	D	B	E	D	B	C	A

VI. The content of theme

The algorithm of emergency in the case of snake bites

1. Not apply tourniquet.
2. In the first 1-2 min after the sting, squeeze out the first drops of blood.
3. Incision wipe with a damp cloth.
4. To appease the victim.
5. Immobilize limb, elevated position.
6. As soon as possible admit to the hospital.

The algorithm of emergency in the case of spider's bite

1. Not apply tourniquet.
2. Incision wipe with a damp cloth.
3. To appease the victim.
4. Immobilize limb, elevated position.
5. 10 ml - 10% calcium chloride injection in the case of extensive muscles injury.
6. 10 ml - 25% magnesium sulfate injection in the case of hypertension.
7. As soon as possible admit to the hospital.

The algorithm of emergency in the case of bee bites

1. to draw the sting in the first 2-3 minutes after bite.
2. To apply cold on the place of injury
3. To appease the victim.
4. To give a nonsteroid anti-inflammatory drug (Rofika, Paracetamol).
5. In case of Quincke's edema, obstructive syndrome - 90 mg of prednisolone or 12-16 mg of Dexamethasone injection.
6. As soon as possible admit to the hospital.

The algorithm of emergency in the case of burns

1. Determination of the thermal agent (to carry out a victim from burning building, to put out his clothes, to throw water or impose any tissue).
2. Cooling burnt surfaces.
3. Carefully take off the clothing. Do not tear off his clothes from the affected areas of skin; it is necessary cut them.
4. Inhibitory action in the accident site is manipulation with burn wounds.
5. On the burning wound impose protective (aseptic) bandages.
6. Give a warm tea, coffee, alkaline drinks (better mineral water).
7. Give painkillers.
8. As soon as possible admit to the hospital in supine position.

The algorithm of emergency in the case of electric injury

1. The victim is in the street. Examine the site: if there are fallen wires or other sources of electric current.
2. Approach to the victim by small steps or jumping on feet when there are the wires.
3. In the case of absence of consciousness check airway, breathing and heartbeat.
4. In the case of absence of independent breathing and heartbeat resuscitation should be started.
5. As soon as possible admit to the hospital.

The algorithm of emergency in the case of supercooling

1. Remove wet clothes from the victim.
2. Remove victim to warm, dry, isolated material, such as sleeping bag.
3. For reducing heat loss it is better to put something under the patient than to cover him superiors.
4. Eliminate any physical activity.
5. Prescribe hot drinks.
6. In case of serious exposure to give the victim the drink 50.0 ml of solution every 3-5 min. (1 liter of water, 2 tablespoons of sugar, ½ tablespoon of salt and ½ teaspoon of baking soda).
7. Do not massage cold limbs, rub them with snow.
8. In case of frostbite of limbs lay the tire for immobilization.
9. Patients with severe hypothermia should be moved with caution.

DROWNING AND NEAR-DROWNING

It is an unexpected tragedy when a previously healthy person dies or is exposed to severe cerebral hypoxia and suffers permanent brain damage. For many years, drowning was considered a "fight for survival": Arms flailing and screaming for help, a person who could not swim struggled to remain on the surface of the water to reach safety. This situation, however, is rarely reported by persons at the scene of aquatic emergencies. Furthermore, no single set of circumstances comprises drowning or near-drowning. It may be a secondary event following such precursors as head or spinal trauma, hypoxia-induced unconsciousness, or unconsciousness due to preexisting cardiovascular disease, sudden cardiac death, or myocardial infarction. The initiating event is usually unknown, so the drowned or near-drowned victim must be treated based on probable physiologic effects of the near-drowning itself. If survival with normal brain function is to occur, a thorough understanding of the pathophysiology of drowning and an organized approach to therapy are imperative [54].

Pathophysiology of drowning. Approximately 90% of drowning victims aspirate fluid into their lungs. In those who do not aspirate fluid, hypoxemia results simply from apnea. In those who do aspirate, the volume and the composition of the fluid determine the physiologic basis of the hypoxemia. Freshwater aspiration alters the surface tension properties of pulmonary surfactant arid makes alveoli unstable, which causes a decreased ventilation/perfusion ratio. Some alveoli collapse and become atelectatic, which produces a true or absolute intrapulmonary shunt, while others are poorly ventilated and produce a relative shunt; in either case, significant pulmonary venous admixture occurs. Fresh water in the alveoli is hypotonic and is rapidly absorbed and redistributed throughout the body. While some have proposed that water continues to enter the lungs after death, at autopsy, the lungs of victims who died in the water frequently contain little water. These findings support the premise that active respiration determines the volume of water aspirated.

Hypertonic seawater pulls additional fluid from the plasma into the lungs, and thus the alveoli are fluid-filled but perfused, which causes substantial pulmonary venous admixture. With both types of water, pulmonary edema may occur secondary to events such as fluid shifts, a change in capillary permeability, or cerebral hypoxia, which causes neurogenic pulmonary edema. Regardless of the cause, pulmonary edema adds to the ventilation/perfusion abnormality [56].

Water that is grossly contaminated with bacteria or that contains particulate matter may complicate the picture. Particulate matter can obstruct the smaller bronchi and respiratory bronchioles. Grossly contaminated water increases the risk of severe pulmonary infection. Neither problem is sufficiently common, however, to justify recommending specific therapy routinely for all victims.

At least 85% of near-drowned victims are thought to aspirate 22 mL/kg of water or less, which does not significantly affect blood volume or serum electrolyte concentrations. After resuscitation, by the time blood is analyzed, serum electrolyte concentrations usually are normal or close to normal. Significant changes are documented in only approximately 15% of those who cannot be resuscitated and only rarely in those who are resuscitated. These findings suggest that either a small amount of water was aspirated, fluid was rapidly redistributed, or both. T'herefore, electrolyte disturbance rarely needs treatment. When a large quantity of water is aspirated, seawater causes hypovolemia, which concentrates extracellular electrolytes, and fresh water causes acute hypervolemia. If enough water is aspirated that plasma becomes severely hypotonic and the patient is hypoxemic, red cell membranes can rapture, and plasma hemoglobin and serum potassium concentrations increase significantly. However, this development has been reported only rarely. With rapid redistribution of

fluid and development of pulmonary edema, even freshwater victims frequently demonstrate hypovolemia by the time they reach the hospital.

Hypercarbia, which is associated with apnea and/or hypoventilation, is less often documented by blood gas analysis than is hypoxemia. While hypoxemia due to pulmonary venous admixture persists in all near-drowned victims who aspirate water, hypercarbia is usually corrected sooner with artificial mechanical ventilation and improved minute ventilation and, thus, is reported in only a small percentage of victims evaluated at the hospital. Besides hypoxemia, metabolic acidosis also persists in most patients. Abnormal cardiovascular function, usually ascribed to hypoxemia, is brief with effective, timely therapy. Abnormality in renal function is uncommon, but when it does occur, it too is secondary to hypoxemia, altered renal perfusion, or, in extremely rare circumstances, significant hemoglobinuria [53-56].

Treatment of near-drowning. Retrieve the victim from the water, and, if necessary, performing artificial ventilation and circulation.

An abdominal thrust not be used routinely in victims of submersion (Heimlich manoeuvre - a technique in first aid to dislodge a foreign body in a person's windpipe by applying sudden upward pressure on the upper abdomen). In these patients, an abdominal thrust may lead to regurgitation of gastric contents and, thus, to aspiration of the vomitus. Further, an abdominal thrust may delay ventilatory or circulatory resuscitation. Therefore, an abdominal thrust should only be used when the airway is obstructed with a foreign body or when the victim fails to respond to mouth-to-mouth ventilation.

Central nervous system depression presents the major therapeutic challenge in near-drowning. Some factors that adversely influence survival are prolonged submersion, delay in initiation of effective cardiopulmonary resuscitation, severe metabolic acidosis (pH < 7,1), asystole upon arrival to a medical facility, fixed dilated pupils, and a low Glasgow coma score (< 5). None of these predictors is absolute, however, and normal survivors have been reported in all of the above categories. Absence of cortical evoked potentials does indicate irreversibility of the cerebral hypoxic lesion; this test, however, cannot be done in the field to guide rescuers [55].

Hypothermia appears to be protective, but only if it occurs early, at the time of the accident, in which case it increases the victim's chance of cerebral salvage after relatively long periods of acute hypoxia and cardiac arrest. While hypothermia prolongs tolerance to hypoxia, it also can precipitate fatal cardiac arrhythmia: thus, its occurrence can be helpful on the one hand and harmful on the other. The diving reflex produces bradycardia, breath-holding, and circulatory redistribution when the face is

submerged in cold water. However, the effect of the diving reflex in explaining cerebral recovery after prolonged immersion has not been specifically documented.

Significant pulmonary venous admixture usually persists even after successful resuscitation; therefore, supplemental oxygen should be administered until arterial blood gas analysis confirms that oxygen is no longer needed. Intravenous access should be established as soon as possible. The trachea should be intubated if necessary for airway maintenance or to facilitate mechanical ventilatory support. ECG monitoring will facilitate prompt treatment of cardiac arrhythmia.

Victims should be transported to a hospital for definitive testing of the adequacy of ventilation and blood gas exchange, cardiac activity, and effective circulating blood volume. Other variables, such as serum electrolyte concentrations, renal function, and cerebral status, should be analyzed as indicated.

The single most effective treatment for hypoxemia, regardless of cause, is mechanical ventilatory support including continuous positive airway pressure (CPAP). After freshwater aspiration, improvement in ventilation/perfusion matching is more consistent when CPAP is combined with mechanical inflation of the lung than with spontaneous respiration. The question of whether CPAP should be combined with spontaneous respiration or with mechanical ventilation should be decided by whether the specific patient can perform the necessary work of breathing, adequately eliminate carbon dioxide, and adequately match ventilation/perfusion ratios. Positive airway pressure should be withdrawn gradually as the lungs stabilize and ventilation/perfusion ratio returns toward normal[58,59].

The pH in near-drowned victims is commonly significantly acidotic, which, in turn, can depress cardiac function. The metabolic component of the acidosis, if it results in a pH < 7.20, should be corrected pharmacologically, although there is some disagreement on this point. With cardiovascular instability, cannulation of the pulmonary artery with a Swan-Ganz catheter or evaluation by transesophageal echocardiography is indicated. Many patients will be hypovolemic from loss of fluid into the lung as pulmonary edema or from decreased venous return secondary to increased intrathoracic pressure during mechanical ventilatory support.

Because recovery after long periods of submersion under frigid conditions has been reported, body temperature should be taken into account before a decision is made to terminate therapy. The body temperature of victims depends not only on the temperature of the water from which they are retrieved but also on how well they were insulated by clothing. The volume of water actually aspirated is also important, because a large volume, if distributed before cardiac arrest occurs, can produce rapid central cooling. Thus, cold water can be protective when it produces total-body hypothermia, which decreases metabolic oxygen requirement. On the other hand, cold water may also

contribute to the accident if hypothermia occurs before total submersion, and severe, or even fatal, cardiac arrhythmia results. Several methods of rewarming hypothermic victims have been advocated, but any technique that increases oxygen utilization, such as shivering, should be avoided [55].

Regardless of the conditions surrounding a drowning or near-drowning, treatment should adhere to the following sequence of priorities:

1. Remove the victim from the water as soon as possible and stabilize the patient's head and neck if trauma is suspected.

2. Immediately follow the ABCs of cardiopulmonary resuscitation - even in the water if this does not endanger the rescuer.

3. If the patient is unconscious, protect the airway as needed with endotracheal intubation.

4. Establish venous access as soon as possible.

5. Provide supplemental oxygen and ventilatory support until each is no longer needed. This can be judged from analysis of arterial blood for oxygen tension, carbon dioxide tension, and pH.

6. Monitor cardiac rhythm with an electrocardioscope as soon as possible.

7. Monitor body temperature and restore it to normal.

8. If the patient has persistent respiratory insufficiency, provide intensive pulmonary support with CPAP and mechanical ventilation therapy as necessary.

9. If the patient has cardiovascular instability, evaluate cardiac output and effective circulatory volume by invasive monitoring, and measure serum electrolyte concentrations.

10. Evaluateand treat renal function and cerebral status as indicated.

Glucocorticoid therapy, prophylactic antibiotic therapy, and monitoring of intracranial pressure are no longer recommended.

ACCIDENT PREVENTION. For those victims in whom the accident is secondary to a medical condition, as in persons susceptible to syncope or seizure, the only way to prevent the accident is to identify those who ought to avoid the water or to encourage them to use the buddy system. For young children, early swimming lessons, vigilant caretakers, and stringent laws governing pool enclosures are needed. Those who teach parenting classes should routinely warn parents about the risk of toddlers' drowning in such household fixtures as toilets, buckets of water, and even washing machines. Preventing accidents during boating, athletics and other water-related recreational activities requires public education. Rules associated with these activities to maximize safety and judicious, responsible behavior should be portrayed as life-saving measures. Similarly, drinking alcohol, a "ubiquitous catalyst" to drowning, should be portrayed as life-threatening whenever water is nearby[57].

ELECTRICAL INJURIES

EPIDEMIOLOGY. The exact incidence of electrical injury is unknown. The prevalence of electrical technology in modern society has resulted in more people experiencing electrical injury. The current treatment of electrical injury has resulted in a low mortality, ranging 3–15%, but the amputation rate remains high, and disfigurement due to extensive soft tissue destruction is frequent. More than 60% of the electricity-related fatalities occur in males, with the highest incidence among those 20 to 34 years of age. Approximately one-third of *high-voltage* injuries occur in electrical workers, one-third in construction workers, and the remainder from non-work-related events. One-half of *low-voltage* injuries occur at home, most of them to young children. Bum center referral is usually necessary for electrical burns.

PATHOPHYSIOLOGY. Electrical burns result from the conversion of electrical energy into heat. Factors that determine the severity and distribution of injury include the type of current (direct or alternating), the quantity of the current (amperage), the potential of the current (voltage), the resistance offered by the body, the pathway of the current, and the duration of contact. These variables are interrelated, and their interactions produce the varied spectrum of injury seen clinically.

Direct and alternating current have different effects. At low voltages (<1000 V), the low-frequency (40-150 Hz) alternating current range, which is used almost exclusively for incandescent lighting and appliances, is three times more dangerous than direct current. Immediate death can result from ventricular fibrillation, central respiratory arrest, or asphyxia due to tetanic respiratory muscle contractions. Tetanic muscle spasms, which freeze the contact point to the power source, tend to increase the flow of current and the severity of injury. Cutaneous burns may be entirely absent or minimal. In contrast, at high voltages, high-frequency alternating current and direct current are equally lethal [60].

Survivals from electric shock of greater than 100 000 V and deaths from as little as 50 V have occurred, which underscores the interplay of the variables already noted. Clinically, the severity of injury relates primarily to the voltage. Low-voltage contact, while potentially lethal, does not result in the magnitude of tissue necrosis seen with high-voltage injury. Tissue resistance is an important factor in determining both the initiation of current flow and its subsequent path. A person completing a circuit between two contact points has a resistance that is the sum of the skin resistances at both contact points plus the internal body resistance. If skin resistance is high, there will be considerable local tissue destruction. Conversely, if skin resistance is low, systemic effects, such as those on the heart and brain, predominate. Skin resistance varies widely according to the thickness, cleanliness, and wetness of the skin. The resistance of skin in water is only about 0.1% of the resistance of dry skin. The extent of tissue damage can be explained by the differing resistances of various tissues. Listed in the order of

increasing magnitude of resistance are nerves, blood vessels, muscle, skin, tendon, fat, and bone.

All tissues and organs can be affected by electrical injury. The cutaneous burns are often limited, with variable deep tissue damage. Appreciation of this special property of electrical wounds is critical in their management. Skin wounds are typically leathery or charred areas of full-thickness skin loss. The entry and exit sites are usually depressed; giving the appearance that current exploded the tissues. The arc burn is produced by current coursing external to the body from contact to ground, favoring a path of least resistance. The flexor surfaces of the wrist, elbow, and axilla are most often involved, because the hand is the most commonly involved body part. After several days, the demarcation between viable and nonviable tissue becomes more obvious. Flame burns of the skin may result from ignition of clothing by electrical arcing and may be full-thickness burns because of the prolonged exposure of the dazed victim to the flame [63].

The most severe cardiopulmonary manifestations occur at the time of the injury. These include anoxia and ventricular fibrillation, which may cause immediate death due to respiratory or cardiac arrest. Major electrical injury is accompanied by a 3-15% incidence of acute renal failure, which is greater than the incidence after thermal burns.

Nervous tissue is highly susceptible to electrical injury because of its low resistance. Neurologic deficits can be seen initially or up to 3 years later, and neurologic aberrations are the most frequent nonfatal sequelae of electrical injury. An important diagnostic point is to ask the patient if the details of the accident are remembered. An inability to recall recent events indicates that electricity entered the body, erasing recent memory. Lesions of the central nervous system may cause varying levels of consciousness and respiratory and motor paralysis, which are usually transient; recovery is the rule. If the effects are permanent, they often assume the character of cortical encephalopathy or hemiplegia with or without aphasia. Spinal cord damage is the most common permanent sequela of electrical injury and is seldom complete. Many deficits seen initially resolve spontaneously; others may not develop until 6 to 9 months after the electrical injury. Permanent deficits may not be seen for days to months, are of gradual onset, and progress slowly. Often these disturbances are not noted until the rehabilitative phase of recovery, when gait abnormalities become evident. Peripheral nerves may be burned directly or may be compressed by surrounding edema or scar tissue. Neuropathies also can develop in unbumed limbs. Autonomic nervous system dysfunction may be seen in both the acute and recovery phases. Reflex sympathetic dystrophy or causalgia can occur. Late onset of burning pain, frequently associated with vasomotor, trophic, and dermal changes, is characteristic [62].

Cataracts characteristically occur following high-voltage injuries. The incidence of these lesions, which are usually bilateral, may be as great as 30% when electrical

contact is made above the clavicles, particularly when the entry wound is on the head. The latent period between the accident and the onset of blurred vision averages 6 months (ranges from a few weeks to 3 years).

Direct vascular injury is more common after electrical injury than after any other type of burn. The blood flow in large arteries and veins is usually sufficient to dissipate the heat generated by the electric current. However, smaller vessels may experience significant heat- related damage, resulting in thrombosis. Direct vascular injury probably contributes to the high amputation rate after high-voltage electrical injury. Delayed hemorrhage from mural necrosis of large blood vessels also may occur.

Associated injury in electrical accidents can be due to falls of considerable distance or to the explosive effects of the current. Fractures of the vertebrae and long bones and dislocations also may result from the violent titanic muscle contractions [65].

Treatment. At the scene of the accident, the patient must be separated immediately from the electric current, but rescuers must not touch or approach the patient until the current has been shut off to avoid injury to themselves. Flames should then be extinguished.

Cardiopulmonary support must be initiated if necessary and maintained during transport. Aggressive life support is essential, because victims of high-voltage electrical injury may be resuscitated successfully without permanent neurologic damage even after a prolonged cessation of vital functions. Because blunt trauma and skeletal injury may both coexist, a thorough history and careful physical examination are essential. Accurate assessment of the extent and nature of the burns is essential to the determination of subsequent therapy. Neurologic status must be evaluated repeatedly during convalescence because it changes frequently.

Fluid replacement is essential in the initial management. Hypovolemia results from the rapid loss of fluid into damaged tissues. Small entry and exit wounds may lead to underestimation of the underlying injury, so fluid requirements may be grossly underestimated. Ringer's lactate should be infused rapidly (0,5 to 1 mL/kg per hour) as necessary to correct hypovolemia and to maintain urinary output. Large volumes of fluid are necessary in high-voltage injury because the fluid and electrolyte needs are much greater than in patients with thermal burns of equivalent surface area. The adequacy of resuscitation is monitored, with urinary output being the single most reliable indicator of circulatory status. The presence of urinary hemoglobin and myoglobin necessitates treatment with mannitol, and the urine should be made alkaline to prevent precipitation of these pigments in the kidney. When hemoglobinuria and/or myoglobinuria is present, the rate and volume of the crystalloid infusion must be sufficient to maintain a minimum urine output of 100 mL/h. This infusion is continued

until the urine is grossly clear of pigment. Red blood cell transfusion, plasma, dextran, or other plasma expanders are unnecessary during the acute resuscitative phase.

Low-Voltage Injury related to household appliances, are usually small and limited to the area of contact. These burns frequently involve the hands, feet, or, in children, the corners of the mouth, lips, and tongue. The evolution of tissue injury and vascular necrosis from the current itself are complete within 7 to 10 days. During this time the wounds are allowed to slough and heal by contracture. Small but deep contact injuries on the trunk and extremities may be excised and grafted as necessary when the extent of the slough is evident. Early excision and local flap repair are rarely indicated. Delayed bleeding from the lip, seen in one-quarter of these injuries, is usually readily controlled by direct pressure [60-62].

High-Voltage Injury with devitalized skin, fat, and muscle are fundamentally surgical problems. They usually involve a limited amount of the total body surface, have upper-extremity contact points, and require amputation or other surgical procedures. They may affect any organ system. The ultimate treatment goals are stabilization of the patient, salvage of the limb, debridement of devitalized tissue, wound coverage, and rehabilitation. Prolonged expectant nonsurgical therapy only increases the risk of invasive infection.

The timing of surgical debridement and its aggressiveness are controversial issues. Opinions range from early total excision with primary wound closure to the now outmoded expectant non-surgical approach. Most surgeons favor an intermediate approach, individualizing according to the amount of tissue destruction and the location and type of the injury. Major amputations and surgical debridement are usually performed 2 to 4 days following injury, when the extent of necrosis is reasonably well defined but the risk of significant infection is small. During this interval, neurologic and cardiopulmonary abnormalities usually stabilize or resolve, and the demarcation between viable and nonviable tissues becomes more evident. Because these wounds are prone to anaerobic infection, particularly myonecrosis with Clostridia, aqueous penicillin is given prophylactically from the time of admission until debridement is complete. Antibiotic administration should be further guided by identification of infecting organisms. Local wound care is started immediately and includes mechanical cleansing followed by the application of topical antibacterial agents and cotton gauze dressings. Silver sulfadiazine cream has excellent antibacterial activity but penetrates only a few millimeters into the tissues. Sulfamylon, a burn cream containing mafenid (a carbonic anhydrase inhibitor that readily penetrates soft tissue), may be advantageous in deep injuries and, therefore, is the topical agent of choice in the prevention and/or treatment of deep-seated infections. However, when absorbed, this drug causes a bicarbonate diuresis resulting in systemic acidosis [64].

High-voltage electrical burns frequently produce muscle compartment syndromes requiring fasciotomy. Circumferential deep limb burns due to associated flame or arc burns also may require escharotomy or fasciotomy. Indications for surgical decompression include loss of distal pulses, impaired capillary filling, paresthesia, and rigid muscle compartments. Decompression of the carpal tunnel is especially important owing to the high incidence of electrical injury to hands. If done expeditiously, this procedure may save a hand.

Coverage of open wounds usually requires skin grafting. Deep electrical injuries frequently result in complex soft tissue wounds, which may require skin flaps for coverage. Nutritional support is essential and should be instituted early. As with other types of burns, appropriate splints should be fashioned and applied, and an aggressive program of physical therapy should be formulated and instituted. Psychological counseling is usually beneficial for patients who require a major amputation or other mutilating surgical procedure [62].

LIGHTNING INJURY

When the difference in potential between the undersurface of a cloud that becomes progressively more negative and the surface of the earth, which is positive, exceeds the insulator strength of air, electrical energy discharges as lightning. There are four mechanisms of lightning strike: direct strike, flashover phenomenon, side flash, and stride potential. A direct strike consists of a major current flow directly through the victim. It is the most serious type of strike and is facilitated by metal objects such as golf clubs, umbrellas, or too is carried during a thunder storm. In flashover, the lightning travels on the outside of the body; this type of strike is facilitated by wet garments and sweat. Side flash occurs when the current splashes from a building, tree, or other person and then travels to the victim. Stride potential occurs when the lightning strikes the ground close to a victim with one foot touching the ground closer to the point of the lightning strike than the other foot. In this position, there will be a potential electrical difference between the legs called *stride potential.* The lightning current may enter one leg, pass up and through the victim's body, and exit through the other leg. Stride potential and side-flash strikes can involve several individuals at once and may contribute to the multiple casualties often associated with lightning strike [63].

The pathophysiology of lightning injury is similar to that described for other electrical injuries. Lightning is direct current. The voltage of lightning may range from 3 million to 200 million volts and may carry a current of between 2000 and 3000 A. Lightning victims have a very short exposure to current because of the brief duration of strike, which usually lasts only 1 to 100 ms. Most of the nay flash over the outside of the body. In contrast, contact with current from high-voltage electrical injuries may be prolonged, since the victim may become frozen to the current source. The injuries

resulting from lightning strikes are similar to those from direct current and alternating current, as described above. Thus, treatment is essentially the same as for other types of electrical injury. The best form of treatment is prevention, consisting of avoidance of dangerous situations during rainstorms and electrical storms, such as standing near tall structures and metal poles or beneath metal shelters. It is erroneous to assume that once lightning strikes it is safe to venture into the open; another strike may occur in the same location. Many churches have sustained multiple strikes [64].

ANIMAL BITES

Domestic pets are the cause of most animal bites. Dogs are more likely to bite than cats. However, cat bites are more likely to cause infection. For these reasons, the best treatment for an animal bite is prevention.

Teach your child from earliest years to avoid strange animals. If an animal bites you or another person, report about it to the local authorities. Animals that habitually bite should be constantly restrained or destroyed. The vast majority of animal bites are inflicted by household pets, but strays and wild animals such as skunks, raccoons, bats, and others also bite thousands of people each year. Animals living in the wild are especially dangerous because they may carry rabies, but any animal that bites a human should be impounded and checked for rabies.

Minor Bites. Treat a minor bite (one in which the skin is broken but not torn, and bleeding is limited) as you would any minor wound. Wash the wound thoroughly with soap and water, and apply an antibiotic cream to prevent infection

Establish whether the person who was bitten has had tetanus shot within the past 10 years; if not, seek medical care from your physician or local emergency room.

Serious Bites. If the bite results in a deep puncture wound, if the skin in the bitten area is badly torn, or if bleeding persists, apply pressure to stop the bleeding.

Then seek emergency medical assistance. Your physician will examine, wash, and treat the wound; he or she also may give a tetanus shot [7,68].

ARTHROPOD BITES AND STINGS

Spider bites. Of the more than 30000 recognized species of spider, only about 100 defend themselves aggressively and have fangs sufficiently long to penetrate human skin. The venom that spiders use to immobilize and digest their prey can cause necrosis of skin and systemic toxicity. While the bites of most spiders are painful but not harmful, envenomations of the brown or fiddle spiders (*Loxosceles*species), widow spiders (*Latrodectus* species), and other species may be life-threatening. Identification of the offending spider should be attempted, since specific treatments exist for bites of

widow and brown recluse spiders and since injuries attributed to spiders are frequently due to other causes [7,75].

Recluse Spider Bites and Necrotic Arachnidism Severe necrosis of skin and subcutaneous tissue follows envenomation by*loxoscelesreclusa,* the brown recluse spider, and by at least four other species *of Loxosceles* in the southern and midwestern U.S. Other spiders that produce necrotic ulceration include the hobo spider *(Tegenariaagrestis)* in the Pacific Northwest, the sac spiders (*Chiracanthium* species) throughout the U.S. and abroad, the South American brown spider *Loxosceleslaeta* in Central, and South America, and other *Loxosceles* species in Africa and the Middle East. AH these spiders measure 7 to 15 mm in body length and 2 to 4 cm in leg span. Recluse spiders are brown and have a dark violin-shaped spot on their dorsal surface; hobo spiders are brown with gray markings; and sac spiders may be pale yellow, green, or brown.

These spiders are not aggressive toward human beings and bite only if threatened or pressed against the skin. They hide under rocks and logs or in caves and animal burrows, and they emerge at night to hunt other spiders and insects. They invade homes, particularly in the fall, and seek dark and undisturbed hiding spots in closets, in folds of clothing, or under furniture and rubbish in storage rooms, garages, and attics. Bites often occur while the victim is dressing and are sustained primarily to the arms, neck, and lower abdomen [77].

The clear viscous venoms of these spiders contain an esterase, alkaline phosphatase, protease, and other enzymes that produce tissue necrosis and hemolysis. Sphingomyelinase B, the most important dermonecrotic factor, binds cell membranes and promotes chemotaxis of neutrophils, leading to vascular thrombosis and an Arthus-like reaction. Initially, the bite is painless or produces a stinging sensation. Within the next few hours, the site becomes painful and pruritic, with central induration surrounded by a pale zone of ischemia and a zone of erythema. In most cases, the lesion resolves without treatment over 2 to 3 days. In severe cases, the erythema spreads, and the center of the lesion becomes hemorrhagic and necrotic with an overlying bulla. A black eschar forms and sloughs several weeks later, leaving an ulcer that may be ≥25 cm in diameter and eventually a depressed scar. Healing usually takes place within 3 to 6 months but may take as long as 3 years if adipose tissue is involved. Local complications include injury to nerves and secondary infection. Fever, chills, weakness, headache, nausea, vomiting, myalgia, arthralgia, maculopapular rash, and leukocytosis may develop within 72 h of the bite. In rare instances, acute complications such as hemolytic anemia, hemoglobinuria, and renal failure are fatal.

Initial management includes local cleansing, application of sterile dressings and cold compresses, and elevation and loose immobilization of the affected limb. Analgesics, antihistamines, antibiotics, and tetanus prophylaxis should be administered

if indicated. Within the first 48 to 72 h, the administration of dapsone, a leukocyte inhibitor, may halt the progression of lesions that are becoming necrotic. Dapsone is given in oral doses of 50 to 100 mg twice daily after glucoses-phosphate dehydrogenase deficiency has been ruled out. The efficacy of locally or systemically administered glucocorticoids has not been demonstrated, and a potentially useful *Loxosceles*-specific antivenin has not been approved for use in the U.S. Debridement and later skin grafting may be necessary after signs of acute inflammation have subsided, but immediate surgical excision of the wound is detrimental. Patients should be monitored closely for signs of hemolysis, renal failure, and other systemic complications [71].

Widow Spider Bites. The bite of the female widow spider is notorious for the effect of its potent neurotoxin. *Latrodectusmactans,* the black widow, has been found in every state of the U.S. except Alaska and is most abundant in the southeast. It measures up to 1 cm in body length and 5 cm in leg span, is shiny black, and has a red hourglass marking on the ventral abdomen. Other dangerous North American *Latrodectus* species include *L. geometricus* (the brown widow), *L. bishopi* (the red widow), *L. variolus,* and *L. hesperus,* and there are related species in other temperate and subtropical parts of the world.

Widow spiders spin their webs under stones, logs, plants, or rock piles or in dark spaces in barns, garages, and outhouses. Bites are most common in the summer and early autumn and occur when the web is disturbed or when the spider is trapped or provoked. The buttocks or genitals are sites of bites incurred by humans while sitting in an outdoor privy.

The initial bite goes unnoticed or is perceived as a sharp pinprick. Two small red marks, mild erythema, and edema develop at the fang entrance site. The oily yellow venom that is injected does not produce local necrosis, and some persons experience no other symptoms. However, alpha-latrotoxin, the most active component of the venom, binds irreversibly to nerves and causes release and eventual depletion of acetylcholine, norepinephrine, and other neurotransmitters from presynaptic terminals. Within 30 to 60 min, painful cramps spread from the bite site to large muscles of the extremities and the trunk. Extreme rigidity of the abdominal muscles and excruciating pain may suggest peritonitis, but the abdomen is not tender on palpation. Other features include salivation, diaphoresis, vomiting, hypertension, tachycardia, labored breathing, anxiety, headache, weakness, fasciculations, paresthesia, hyperreflexia, urinary retention, uterine contractions, and premature labor. Rhabdomyolysis and renal failure have been reported, and respiratory arrest, cerebral hemorrhage, or cardiac failure may end fatally, especially in very young, elderly, or debilitated persons. The pain begins to subside during the first 12 h but may recur during several days or weeks before resolving spontaneously [72-75].

Treatment consists of local cleansing, application of ice packs, and tetanus prophylaxis. Hypertension that does not respond to analgesics and antispasmodics, such as benzodiazepines or methocarbamol, requires specific antihypertensive medication. Intravenous administration of one or two vials of a widely available equine antivenin rapidly relieves pain and can be life-saving. Because of the risk of anaphylaxis and serum sickness, antivenin should be reserved for severe cases involving respiratory arrest, uncontrollable hypertension, seizures, or pregnancy.

Envenomations by Tarantulas and Other Spiders. Tarantulas are long-lived, hairy spiders of which 30 species are found in the U.S., primarily in the southwest. The tarantulas that have become popular household pets are usually imported species with bright colors and a leg span of up to 25 cm. Tarantulas bite only when threatened and cause no more harm than a bee sting, but the venom occasionally provokes deep pain and swelling. Several species are covered with urticating hairs that are launched in the thousands when a threatened spider rubs its hind legs across the dorsal abdomen. These hairs penetrate human skin and produce pruritic papules that last for weeks. Treatment of bites includes local washing and elevation of the bitten area, tetanus prophylaxis, and analgesic administration. Antihistamines and topical or systemic glucocorticoids are given for exposure to urticating hairs.

Atraxrobustus, the Sydney funnel-web spider of Australia, and *Phoneutria* species, the South American banana spiders, are among the most dangerous spiders in the world because of their aggressive behavior and potent neurotoxins. Envenomation by *A. robustus* causes a rapidly progressive neuromotor syndrome that can be fatal within 2 h. The bite of the banana spiders causes severe local pain followed by profound systemic symptoms and respiratory paralysis that can lead to death within 2 to 6 h. Specific antivenins for envenomation by each of these spiders are available. *Lycosa* species (wolf spiders) are found throughout the world and may produce painful bites and transient local inflammation [71].

Scorpion stings. Scorpions are crablike arachnids that feed on ground-dwelling arthropods and small lizards, which they grasp with a pair of frontal pinchers and paralyze by injecting venom from a stinger on the tip of the tail. Painful but relatively harmless scorpion stings need to be distinguished from the potentially lethal envenomations that are produced by about 30 of the approximately 1000 known species and cause more than 5000 deaths worldwide each year. Scorpions feed at night and remain hidden during the day in crevices or burrows or under wood, loose bark, or rocks on the ground. They seek cool spots under buildings and often enter houses, where they get into shoes, clothing, or bedding or enter bathtubs and sinks in search of water. Scorpions sting human beings only when disturbed [73].

Scorpions of the U.S. Of the 40 or so scorpion species in the U.S., only the bark scorpion *(Centruroides sculpturatus* or *Centruroides exilicauda)* produces venom that

can be lethal. Stings of the other species, such as the common striped scorpion *Centruroides vittatus*and the large *Hadrurus arizonensis,* cause immediate sharp local pain followed by edema, ecchymosis, and a burning sensation. Symptoms typically resolve within a few hours, and skin does not slough. Allergic reactions to the venom sometimes develop.

The deadly *C. sculpturatus* of the southwestern U.S. and northern Mexico measures about 7 cm in length and is yellow-brown in color. Its venom contains neurotoxins that cause sodium channels to remain open and neurons to fire repetitively. In contrast to the stings of nonlethal species, *C. sculpturatus* envenomations are usually associated with little swelling, but prominent pain, paresthesia, and hyperesthesia can be accentuated by tapping on the affected area (the tap test). These symptoms soon spread to other locations; dysfunction of cranial nerves and hyperexcitability of skeletal muscles develop within hours. Patients present with restlessness, blurred vision, abnormal eye movements, profuse salivation, lacrimation, rhinorrhea, slurred speech, difficulty in handling secretions, diaphoresis, nausea, and vomiting. Muscle twitching, jerking, and shaking may be mistaken for a seizure. Complications include tachycardia, arrhythmias, hypertension, hyperthermia, rhabdomyolysis, and acidosis. Symptoms progress to maximal severity in about 5 h and subside within a day or two, although pain and paresthesia can last for weeks. Fatal respiratory arrest is most common among young children and the elderly [74].

Other Dangerous Scorpions. Envenomations by ***Leiurusquinque striatus*** in the Middle East and North Africa, by ***Mesobuthus famulus*** in India, by ***Androctonus*** species along the Mediterranean littoral and in North Africa and the Middle East, and by ***Tityusserrulatus*** in Brazil cause massive release of endogenous catecholamine with hypertensive crises, arrhythmias, pulmonary edema, and myocardial damage. Acute pancreatitis occurs with stings of *Tityustrinitatis* in Trinidad, and central nervous toxicity complicates stings of *Parabuthus and Buthotus* scorpions of South Africa. Tissue necrosis and hemolysis may follow stings of the Iranian *Hemiscorpius lepturus*

Treatment. Identification of the offending scorpion aids in planning therapy. Stings of nonlethal species require at most ice packs, analgesics or antihistamines. Because most victims of dangerous envenomation (such as those produced by *C. SCULPTURATUS*) experience only local discomfort, they can be managed at home with instructions to return to the emergency department if signs of cranial-nerve or neuromuscular dysfunction develop. Aggressive supportive care and judicious use of antivenin can reduce or eliminate mortality from more severe envenomations. Keeping the patient calm and applying pressure dressings and cold packs to the sting site decrease the absorption of venom. Although narcotics and sedatives can control restlessness and hypertension, these agents interfere with protective airway reflexes and should not be given to patients with neuromuscular symptoms unless endotracheal

intubation is planned. Hypertension and pulmonary edema respond to nifedipine, nitroprusside, hydralazine, or prazosin, and bradyarrhythmias can be controlled with atropine [76].

Commercially prepared antivenins are available in several countries for some of the most dangerous species. A caprine *C.* ***sculpturatus*** antiveninis available as an investigational drug only in Arizona. Because of the risk of anaphylaxis or serum sickness following administration of goat serum, use of the antivenin is controversial. Intravenous administration of antivenin rapidly reverses cranial-nerve dysfunction and muscular symptoms but does not affect pain and paresthesia.

Prevention. In scorpion-infested areas, shoes, clothing, bedding, and towels should be shaken and inspected before being used. Removal of wood, stones, and debris from yards and campsites eliminates hiding places for scorpions, and household spraying of insecticides can deplete their source of food [72].

VENOMOUS SNAKEBITE

Epidemiology: The venomous snakes of the world are classified in Table 1.

A number of snakes in the family Colubridae can be dangerous to humans because of toxic salivary secretions. The incidence of venomous snakebites is low in most developed countries, but in regions of the world where people are engaged in manual agriculture, often with exposed lower extremities, attack rates are higher. About 30000 to 40000 persons die each year from venomous snakebite; incomplete reporting in disadvantaged regions probably makes this range an underestimate.

Table 1

Venomous Snakes of the World

Family	Subfamily	RepresentativeSpecies	Remarks
Viperidae	Crotalinae	Rattlesnakes (Crotalus and Sistrurus species), water moccasins and copperheads (Agkistrodon species), lancehead vipers (Bothrops species)	New World and Asian pit vipers
	Viperinae	Russell's viper (Vipera russelli), saw-scaled viper (Echis carinatus), puff adder (Bitis arietans)	European, Asian, African vipers
Elapidae		Cobras (Naja species), mambas (Dendroaspis species), taipan (Oxyuranus scutellatus)	Temperate and tropical New and Old World; all venomous terrestrial snakes of Australia
Hydrophiidae		Pelagic sea snake (Pelamis pla-	Pacific and Indian

		turus)	oceans
Atractaspididae		Burrowing asps ***(Atractaspis*** species)	Africa, Middle East
Colubridae		Boomslang (Dispholidus typus),twig snake **(**Thelotornis kirtlandii)	Rear-fanged snakes with toxic salivary secretions

Anatomy. The typical snake-venom apparatus consists of paired venom glands - one on each side of the head, below and behind the eye - connected by ducts to hollow, anterior maxillary teeth. In viperids, these teeth are large, mobile fangs that retract against the roof of the mouth when the animal is at rest. In elapids and sea snakes, the fangs are only slightly enlarged and are fixed in an erect position. For reasons that are unclear but may be related to the venom apparatus itself, venomous snakes can bite without injecting any venom. Approximately 20% of pit viper bites and an even higher percentage of bites inflicted by some other snake families (up to 75% for sea snakes) are «dry»[66].

Differentiation of venomous from nonvenomous snake species can be difficult unless one is familiar with local fauna. Viperids are characterized by somewhat triangular heads (a feature shared with many harmless snakes); elliptical pupils (also seen in the nonvenomous boas and pythons as well as some colubrids); enlarged maxillary fangs; subcaudalscalation that involves a single scale running the full width of the ventral surface of the tail for several rows just distal to the anal plate (as opposed to the two scales in each subcaudal row in most nonvenomous snakes); and, in the case of the pit vipers, the heat-sensing pits (foveal organs) for which they are named, located slightly inferior and anterior to the eyes on each side. Color pattern is notoriously unhelpful in identifying most venomous snakes except for the coral snakes, whose other body characteristics are similar to those of harmless colubrids. The American coral snakes can be identified by red, yellow (or white), and black bands completely encircling the body; a few species have red and black bands only. North of Mexico City, the immediate contiguity of red and yellow bands is fairly reliable for distinguishing a coral snake front) one of its harmless colubrid mimics. Further south, differentiation by color pattern is less reliable.

Immunodiagnostic techniques have been developed for species identification of the snakes involved in bites. An enzyme-linked immunoassay (ELISA) can be used to identify a specific type of snake venom in a victim's blood, wound aspirate, or urine, and this method is finding clinical application around the world. However, no commercial ELISA kit is currently available in the U.S.

Venoms and clinical manifestations. Snake venoms are complex mixtures of enzymes, low-molecular-weight polypeptides, glycoproteins, and metal ions. The

enzymes and polypeptides affect the human body in a multisystem fashion. Among the deleterious components are hemorrhagins that render the vasculature leaky and thus cause both local and systemic bleeding; various proteolytic enzymes that cause local tissue necrosis, affect the coagulation pathway at various steps, or impair organ function; myocardial depressant factors that reduce cardiac output; and neurotoxins that act either pre- or postsynaptically to inhibit peripheral nerve impulses. Most snake venoms can adversely affect multiple organs[66-69].

Treatment. Field Management First-aid or «field» measures to be used in the management of venomous snakebite should focus on delivery of the victim to definitive medical care as quickly as possible; the victim should be as inactive as is feasible to limit systemic spread of the venom. Beyond this, any measure employed should at least do no further harm.

After viperid bites, local mechanical suction may be beneficial if applied to the puncture wounds within 3 to 5 min. A useful device is the Extractor, which delivers one atmosphere of negative pressure to the wound. Suction should be continued for at least 30 min. Mouth suction should be avoided as it inoculates the wound with oral flora and theoretically can also result in the absorption of venom by the rescuer through lesions of the upper digestive tract. A proximal lymphatic-occlusive constriction band may limit the spread of venom if applied within 30 min. To avoid compounding of tissue necrosis, however, the band should not be allowed to interrupt arterial flow. A bitten extremity should be splinted if possible and kept at approximately heart level. Incisions into the bite site should never be made, and no form of cooling or electric shock is advantageous [67,68].

For elapid or sea snake bites, the Australian pressure-immobilization technique, in which the entire bitten extremity is wrapped with an elastic or crepe bandage and then splinted, is highly beneficial. The bandage is applied as tightly as it would be to treat a sprained ankle. This technique greatly restricts the absorption and circulation of venom from the bite site. However, an assessment of the potential utility of this method in viperid poisoning requires further research, as it may compound local tissue damage following these bites.

Hospital Management. The victim should be closely monitored (vital signs, cardiac rhythm, and oxygen saturation) while a history is quickly obtained and a brief but thorough physical examination is performed. The level of erythema/swelling in a bitten extremity should be marked and the circumferences measured in several locations every 15 min until swelling has stabilized.

Large-bore intravenous access in unaffected extremities should be obtained in the event that hypotension develops. Early hypotension is due to pooling of blood in the pulmonary and splanchnic vascular beds; hours later, hemolysis and loss of intravascular volume into soft tissues may play important roles. Fluid resuscitation with

normal saline or Ringer's lactate should be initiated for clinical shock. If the blood pressure response is inadequate after the administration of 20 to 40 mL/kg body weight, then a trial of 5% albumin (10 to 20 mL/kg) is in order. If volume resuscitation fails to improve tissue perfusion, vasopressors (e.g., dopamine) should be administered. Invasive hemodynamic monitoring (central venous and/or pulmonary arterial pressures) can be helpful in such cases. Central access must be obtained with extra caution if coagulopathy is evident [77].

Blood should be drawn for laboratory evaluation (including determination of blood type and cross-matching) as soon as possible, before the effects of circulating venom interfere with typing. Also important are a complete blood count to evaluate the degree of hemorrhage or hemolysis, studies of renal and hepatic function, coagulation studies to identify signs of consumptive coagulopathy, and testing of urine for blood or myoglobin. In severe cases or in the face of significant comorbidity, arterial blood gas studies, ECG, and chest radiography may be necessary.

Attempts to locate a source of appropriate antivenin should begin early in all cases of known venomous snake bite, regardless of symptoms. If signs or symptoms develop, they may progress rapidly, making any delay in the administration of antivenin dangerous for the victim. Antivenins rarely offer cross-protection against snake species other than those used in their production unless the species are closely related. An example of good cross-protection is in the use of Australian tiger snake (*Notechisscutatus)* antivenin for sea snake bites. The package insert accompanying a particular antivenin should be consulted for information regarding the spectrum of coverage. In the U.S., assistance in finding antivenin can be obtained 24 hours a day from the University of Arizona Poison and Drug Information Center [74].

Rapidly progressive and severe local findings (soft tissue swelling, ecchymosis, petechiae, etc.) or manifestations of systemic toxicity (signs and symptoms or laboratory abnormalities) are indications for the administration of intravenous antivenin. The package insert outlines techniques for reconstitution of antivenin (when necessary), skin-testing procedures (for potential allergy), and appropriate starting doses. Most antivenins are of equine origin and carry a risk of anaphylactic, anaphylactoid, and delayed-hypersensitivity reactions. Skin testing does not always reliably predict which patients will have an allergic reaction to equine antivenin; a skin test can be either false negative or false positive. Before antivenin infusion, the patient should receive appropriate loading doses of intravenous antihistamines (e.g., diphenhydramine, 1 mg/kg to a maximum of 100 mg; and cimetidine, 5 to 10 mg/kg to a maximum of 300 mg) in an effort to limit acute reactions. Expanding the patient's intravascular volume with crystalloids may also be beneficial in this regard (unless contraindicated by the patient's cardiac status). Epinephrine should be immediately available, and the antivenin dose to be administered should be diluted (e.g., in 1000 mL of normal saline, Ringer's

lactate, or 5% dextrose in water for adults or in 20 ml/kg for children). This volume can be decreased if necessary for the treatment of patients with compromised cardiovascular reserve. The antivenin infusion should be started slowly, with the physician at the bedside to intervene in the event of an acute reaction. The rate of infusion can be increased gradually in the absence of allergic phenomena until the total starting dose has been administered (over a period of 1 to 4 h). Further antivenin may be necessary if clinical abnormalities worsen. Laboratory values should be rechecked hourly, particularly if abnormal, until stability is ensured [77].

The management of a life-threatening envenomation in a victim with an apparent allergy to antivenin requires significant expertise. Consultation with a poison specialist, an intensive care specialist, or an allergist is recommended. Often, antivenin can still be administered in these situations under closely controlled conditions and with intensive premedication (e.g., with epinephrine, antihistamines, and steroids).

Care of the bite wound should include application of a dry sterile dressing and splinting of the extremity with padding between the digits. Because of the risk of central spread of venom, an extremity should be elevated only when antivenin is available. Tetanus immunization should be updated as appropriate. The use of prophylactic antibiotics is controversial, as the incidence of secondary infection following venomous snakebite appears to be low. Many authorities, however, prescribe a broad-spectrum antibiotic (such as ampicillin or a cephalosporin) for the first few days [75].

If swelling in the bitten extremity raises concern that subfascial muscle edema may be impeding tissue perfusion (muscle-compartment syndrome), intracompartmental pressures should be checked by any minimally invasive technique (e.g., the wick catheter). If pressures are elevated, prompt surgical consultation for possible fasciotomy should be obtained while antivenin administration continues. Compartment syndromes are quite rare after snakebites.

Whether or not antivenin is given, any patient with signs of venom poisoning should be admitted to the hospital for observation for at least 24 h. A patient with an apparently «dry» bite should be watched for at least 6 to 8 h before discharge. An occasional viperid «dry» bite progresses to significant toxicity after a delay of several hours, and the onset of systemic symptoms is commonly delayed for a number of hours after bites by several of the elapids (especially the coral snakes) and sea snakes. Patients bitten by these reptiles should be observed in the hospital for 24 h.

Morbidity and mortality. The overall mortality rates for venomous snakebite are low in areas of the world with rapid access to medical care and appropriate antivenin. In the U.S., for example, the mortality rate is <1% for victims who receive antivenin. Eastern and western diamondback rattlesnakes (*Crotalus adamariteus* and *Crotalus atrox,* respectively) are responsible for most snakebite deaths in the U.S. Snakes responsible for large numbers of deaths in other regions of the world include the cobras

(Naja species) of Asia and Africa, the carpet and saw-scaled vipers of the Middle East and Africa *(Echis* species), Russell's viper (*Viperarusselli)* of the Middle East and Asia, the large African vipers *(Bitis* species), and the lancehead pit vipers of Central and South America *(Bothrops* species) [71,73].

The incidence of morbidity in terms of permanent functional loss in a bitten extremity is difficult to estimate but is probably substantial. Such loss may be due to muscle, nerve, or vascular injury or to scar contracture.

VII. Tasks for final control

1. The Man of 30 years old with a total body overheating and heatstroke after work under conditions of high temperatures is concerned with uncontrollable vomiting. What solution for intravenous infusion should be prescribed for reduction of uncontrollable vomiting?

A. Hypotonic glucose solution
B. Solution of atropine sulphate
C. Hypertonic solution of sodium chloride
D. Solution of cerukal 4.0 ml
E. Polarizing mixture

2. Patients admitted to hospital immediately after snake bite. condition is of medium severity. In the place of bite the victim feels pain and burning. HR - 100/minute, BP - 100/60 mm Hg, consciousness is not impaired. What immediate treatment should be?

A. Glucocorticosteroids
B. Aqueous salt solutions
C. Cardiotonic agent
D. Specific serum against snake
E. Narcotic painkiller

3. After a bite of wasps, itchy skin hoarse voice, barking cough, anxiety are emerged. In review: swelling of the lips, eyelids, and cyanosis. Which drug should be the first?

A. Diklofenak sodium
B. Seduksen
C. Adrenaline
D. Laziks
E. Prednisolone

4. 30 years old received thermal burns. He has been examined by the family doctor, who determined the presence of epidermal burns of patient’s head and neck, and

deep dermal burn of all over his hand. The manifestation of what burn shock by the degree should expect?

A. Hard

B. Medium

C. Burn shock will be absent

D. Extremly hard

E. Light

5. The boy was bitten by snake. In 20 minutes after biting he was examined by family doctor. On the place of bite there is a swelling of tissue; HR - 84/min., BP - 104/60 mm Hg. Antitoxic serum was injected. What is the prognosis of the victim's condition?

A. Full recovery

B. Delayed anaphylactic shock will develop

C. Partial recovery

D. Doubtful

E. Renal failure will develop

6. The man who was on the beach under heavy influence of solar radiation (from 11 to 14 hours) lost consciousness. What was the reason of such a reaction of the body?

A. Skin burns

B. Photodermatosis of skin

C. Photochemical effect of solar radiation

D. Sunstroke

E. Features of autonomic nervous system

7. Influence of electric current on the body primarily manifested injury:

A. Of central Nervous System

B. Of the cardiovascular system - shock

C. Of Heart - the development of myocardial dystrophy

D. Of Heart - development of ventricular fibrillation

E. Of peripheral nervous system

8. From the place of disaster, where oil was fired, the group of burnt arrived to the hospital. What of the main criteria should be followed by the physician of receiving department for early diagnosis of burn shock?

A. Dyspnea, tachycardia

B. Cyanosis, shortness of breath

C. The depth and lesion area

D. Thirst, muscle tremors

E. The feeling of anxiety, paleness of skin out of burns

9. Following changes are typical for drowning in sea water:
A. Hypovolemia, hyponatremia, hyperkalemia
B. Hypervolemia, hyperkalemia, hypernatremia
Hypervolemia, hypoproteinemia, hyperchloremia
D. Hypovolemia, hypokalemia, hyponatremia
E. Hypovolemia, hypernatremia, hypercalcemia

10. A man of 24 years old was admitted to the hospital. He walked home in winter during 12 hours. Physical examination: dopey, "gooseflesh, slight cyanosis, hypertonus muscles of limbs and body. What is the situation of the victim?
A. Acute respiratory infection
B. Undercooling
C. Hypothermia
D. Pneumonia
E. Tiredness

Answers:

1	2	3	4	5	6	7	8	9	10
C	D	E	B	A	D	D	C	E	B

VIII. Practical skills

Task 1. Drowned man of 23-years-old was brought at the shore of the sea. Physical examination: without consciousness, pale cyanotic skin, foam from the mouth; auscultation - heterogeneous crackles above lungs. RR 32/min., BP 100/70 mm HgHR 104/min,central venous pressure 160 mm Hg. Which is the reason of this patient status?
A. Cervical spine fracture
B. Acute heart failure
C. Cardiogenic shock
D. Genuine drowning in sea water
E. Pulmonary embolism

Task 2. The bee bites the female of 19-years-old by finger on right hand. 30 minutes later the victim was admitted to the hospital. She complains on weakness, feeling of heat throughout the body, compression of the chest, ringing in ears. Bee sting was removed at once. The doctor diagnosed anaphylactic shock of moderate severity. Which drug should be injected primary?
A. Calcium Chloride
B. Adrenalin

C. Prednisolone
D. Tavehil
E. Kordiamin

Task 3. Family doctor was appealed to the 46-years-old man. After the alcohol abuse during 2 days, he complains on stinging, itching skin, paresthesia in the feet. Physical examination: cyanotic toes of feet, humid skin, tense vesicle with clear yellow liquid. Which is your diagnosis?

A. III degree frostbitten
B. crush syndrome
C. I degree frostbitten
D. IV degree frostbitten
E. II degree frostbitten

Task 4. The 54-years old man was admitted to the hospital ward. He rested on the snow at temperature -10^0C. Physical examination: pungent smell of alcohol, mental confusion, lie motionless, pale cyanotic skin, cold by touch. HR 50/min, BP 80/40 mmHg, body temperature 32^0C. Which is your diagnosis?

A. Severe alcohol intoxication
B. Acute pancreatitis
C. cerebral contusion
D. General hypothermia
E. alcohol poisoning

Task 5. During few minutes a worker was under the influence of electric current. Physical examination: without consciousness, tonoclonic convulsion, no breathing, determined carotid pulsation, mydriatic pupil, not light reaction. What will be your primary action?

A. adrenalin intracardiac introduction
B. anticonvulsive drugs introduction
C. Closed-heart massage and artificial ventilation
D. respiratory analeptic introduction
E. lung contusion

Task 6. The man was admitted to the hospital ward with general hypothermia. Physical examination: muscle tremors, body temperature 33^0C, BP 110/60 mm HgHR 96/min, normal breathing, II-III degree frostbite of hand. Which will be your following action?

A. external rewarming
B. 30% alcohol oral administration
C. warm fluids injection

D. Bandager on foot and hand
E. Drinking warm drinks

Task 7. Family doctor was appealed to the patient with frostbite limbs. Physical examination: distal cyanosis of feet, cool by touch, no pain sensation, swelling of affected skin, several medium-sized strained vesicles with hemorrhagic fluid. What is a tactic of doctor?

A. Expand vesicles
B. Transportation to burns department
C. ethanol disinfection of affected places
D. heat-insulated bandage
E. Call a surgeon

Task 8. The 17-years-old female worked in the garden on a hot day (air temperature 31^0C). She felt headache, dizziness, nausea, and tinnitus. Physical examination: edematous face, body temperature 38^0C, tachypnoe, HR 110/min, BP 110/70 mm Hg. Which is your diagnosis?

A. Vegetable hypersensitivity
B. Migraine attack
C. Heatstroke moderate severity
D. Heatstroke light severity
E. Acute respiratory disease

The task 9. In the cold season a man was admitted to the hospital. He was extracted from water. Contacts have respiratory water was not. He complains on pain, hands and feet numbness. Physical examination: agitated, pale skin, cold shivering, rectal temperature of $34,5^0$C, HR 110/min., BP 120/90 mm HgRR 22/min. What is the kind of victim‘s warming?

A. Warming compresses
B. Warm bath
C. Infusion solutions 37.0^0C
D. Hemodialysis with warm blood
E. Passive warming

Task number 10. The victim of electric shock was at home. After half an hour after hospitalization he came to consciousness. Physical examination: stable hemodynamics, adequate breathing, orientation is preserved. What is further management of the patient?

A. ECG without changes may be issued each 3 hours
B. ECG without changes may be issued after 3 day
C. ECG without changes may be issued each 12 hours

D. ECG without changes may be issued after 6 hours
E. ECG without changes may be issued after 9 hours

Task 11. In the hot season the 38-years-old male started to work in open-hearth furnaces. After some hours he felt heat around the body, uncontrolled sweating, frequent palpitations, headache and dizziness. Physical examination: hyperemia of skin, body temperature 38,2^{0}C, HR 110/min, BP 160/60 mmHg. What emergency help is needed?

A. blowing body by cool air, oxygen inhalation
B. antibiotics introduction
C. hypotensive and diuretic drugs introduction
D. ice pack, oxygen inhalation
E. ice pack, putting pain

Task 12. While diving from a height of depth a young boy bump his head against bottom. He was immediately brought to shore in a state of clinical death, with signs of cervical spine trauma. What will be emergency measures in this situation?

A. Do not throw back victim's head
B. Do not open the mouth of the victim, mouth-to-nose respiration
C. Hohera-Nielsen's artificial respiration
D. Do not move the victim's lower jaw up and forward
F. Sylvester's artificial respiration

Task 13. summercamp the 20-year-old girl was taken out from the river without consciousness. Physical examination: pale skin, no spontaneous respiration, carotid pulsation is determined, midriatic pupils. Which is primary resuscitation measure in this case?

A. Heart defibrillation
B. adrenaline hydrochloride injection
C. closed-chest cardiac massage
D. calcium chloride injection
E. respirogenesis

Answers:

1	2	3	4	5	6	7	8	9	10	11	12	13
D	B	E	D	C	A	B	C	E	B	D	A	E

IX. Basic questions after theme

Emergency in the case of bites.
Emergency in the case of electric injuries.

Emergency in the case of insolation.

Emergency in the case of low and high temperatures exposure.

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