MINISTRY OF HEALTH OF UKRAINE
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Faculty _____medical_____

(name of the faculty)

Department _____pediatric surgery_____

(name of department)

APPROVED

Acting vice-rector for scientific and educational work

_____ Svitlana KOTYUZHYNSKA

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METHODOLOGICAL PROGRAM FOR PRACTICAL/SEMINAR CLASSES

ON THE DISCIPLINE

Faculty, year _____medical_____

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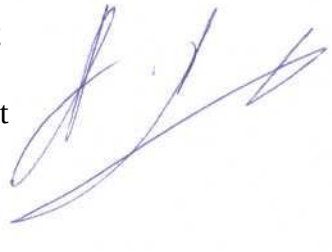
Head of department  Oleksandr LOSEV

Project group:

Head of the Department, Doctor of Medical Sciences, Professor Losev O.O.
Head of the educational part of the department, candidate of medical sciences, associate professor Aplevych V.M.
Doctor of Medical Sciences, Professor Melnychenko M.H.
Candidate of medical sciences, associate professor Eliy L.B.
Candidate of medical sciences, assistant PavlenkoK.B.
Candidate of medical sciences, assistant Kvashnina A.A.
Assistant Karavasili O.M.

Topic 1. Gastrointestinal bleedings in children

Topic: Syndrome of gastro-intestinal bleeding in children

a) From the upper part of gastro-intestinal tract (GIT)
b) From the lower departments.

I. Topic actuality, a professional orientation.

One of the major problems having social - medical value, the increase last years frequencies of ulcer processes in GIT (1 case on 630 children) is, (And. M. Dams it is new with other writers) with an opportunity of development of gastroenteric bleeding (GIT) in 5-6 % of cases. Alongside with ulcer bleedings, frequency not ulceral bleedings is increased (portal hypertension, Meckel′s diverticulum, disease of Mellore-Veise, Peits-Egerce, vascular ectasia etc.).

Despite of expressed enough clinical picture of bleedings and a wide spectrum of modern methods of researches, diagnostics of these tractologies leaves to wish the best. Even children with repeated bleedings are observed by doctors for a long time, but not always receive the appropriate treatment. Adjacent experts are not involved in diagnostics (endoscopists, radiologists, haematologists, genetics etc.).

Sharply arisen bleeding from GIT irrespective of a degree of expressiveness demands emergency diagnostics and rendering of the urgent reason of a bleeding specialised a help - duly establishment by basic image changes not only tactics of conducting the patient, but also the forecast of disease appreciably improves.

II. The educational purposes of the study.

To study the causes of bleeding in children of various age groups, localisation of the centres of defeat GIT, and also diseases which background bleeding based in.

a) ***As a result of the study the*** higher education applicant ***should know theoretically:***

1) Somatic and infectious diseases which can be complicated with bleeding from GIT.
2) A number of boundary (somatic-surgical) diseases at which treatment is appointed by pediatrists, but by virtue of its inefficiency pediatric surgeons continue.
3) Surgical diseases at which conservative methods are used as preparation for operation.
4) Features of bleedings from GIT depending on a level of the tractological process (a gullet, a stomach, various departments of intestine).
5) Frequency and localisation of bleeding depending on age of the child.
6) Classification by intensity of the bleeding.
7) Definition of a degree bleeding on BP Hb, Ht, to calculation of the erythrocytes, VCB, CVP, and also on fibrinogen′s, thrombocyte′s concentration, fibrinolitic activity, a shock index.
8) To know methods of investigations of the patients with the bleeding:
а) endoscopic (esofagogastroduodenoscopy, colonoscopy),
б) Radiological: investigations of a esophagus with revealing varicose the expanded veins of a esophagus and a stomach; tumours; ulcer processes in a stomach, a duodenal gut; Meckel′s diverticulum; polyps of a direct gut and polyposis.
9) Special methods of research: ULTRASONIC - dopplerography of the bodies, angyography, radioisotope methods of research.
10) As to carry out the control of a proceeding bleeding (a constant gastric
probe, definition VCB, CVP, analyses of blood in dynamics)
11) To know the methods of the surgical treatment of various kinds and localisation of the bleedings.

b) Higher education applicant ***must know to do practically***

1) To determine a source of a bleeding on two basic symptoms:

а) To characterise the blood by vomiting;
б) To characterise the blood by a stool.
2) To use the general principles of the treatment of the bleeding:
a) Struggle with hypovolemy and an anaemia (a method of the haemodilution′s control with transfusion of the erytrocytic mass, the frozen plasma, albumin etc.),
6} realising of haemostatic therapy, prescribing of the treatment accelerating thrombogenesis or lowering of the fibrinolisis).
3) To use the local methods of a stop of the bleeding.
4) To watch for the removing of the blood from intestinum (cleansing enema 3-4 times per day) for decrease of an intoxication and improvement of function of a liver,
5) To put the indications to sclerotherapy.
6) To take the anamnesis.
7) To carry out clinical researches.
8) To determine the height of the bleeding
9) Volume of the bleeding on data CVP, VCB and the BP, fibrinogen′s, thrombocyte′s concentration, fibrinolitic activity, a shock index.
10) To determine group of blood and the Rh factor.
11) To determine individual compatibility of group of blood of the donor with the recipient.
12) To issue the case record.
13) To enter a gastric tube into a stomach, to make washing of the stomach.
14) To estimate height and amount of the bleeding on colour of the blood selected of intestines.
15) On the basis of the data of the anamnesis, clinical and laboratory methods of investigations to put the presumable diagnosis.
16) To appoint general and local haemostatic therapy.

3. Questions for training.

1. Child with complains of vomiting with blood admixture came in the surgical department. He was ill with influence and took aspirin 3 times in a day. Your tentative diagnoses. Methods of investigation.
2. In neonatal period child took the infusive therapy through the umbilical catheter. What complications may develop background of this?
3. 4year old child cared out to the surgical department with losing of conciseness, low pulse rate, hypotension, and dark abundant blood discharges from the anus. Your diagnosis. Your tactic.

4. The plan and organisation of lessons.

Work type	Time	Activity		Equipment
		Higher education applicants	Teacher	
Organisation of the practical class	5	Listen	Presence control	Journal
Aim setting	5	Listen	Explain	
Checking of the knowledge of higher education applicants	15	Make	Control	Tests
Seminar	60	Answer	Control	X-ray-grams

Clinical discussion	30	Listen	Discuss patients	Patients
Personal work of higher education applicants	60	Make	Control	Patients
Results of the lesson	10	Listen	Appreciation of each activities	
Task for the next lesson	5	Listen	Explain the task. Indicates the literature.	

5. Substance of the lesson.

Bleeding from a digestive tract.

The bleeding from a digestive tract is a symptom of many diseases or developmental anomalies.
There are distinguishing:
1. Sharp, prophuse bleedings
2. Subacute one.
3. Chronic one.

The first results to development of the haemoragic shock, expressed hypovolemya and collapse, the second and the third one are accompanied by clinically increasing anaemia. Their diagnostics and treatment is based on complex clinical, and additional methods of investigations.

Amount of gastroenteric bleedings (GIB).

Definition of the bleeding amount is represented rather important by way of rendering the emergency help, and a choice of optimal tactics of treatment.

The light (1) degree is observed at:
Er ~ not less $3x10^{12}$ G/L
- Ht - not less than 0,3
- Moderate pallor of integuments
- Frequency of intimate reductions (heart rate) on 20-30 % exceeds age norm
- A BP - N

Average (II) degree:
- Er - before $2,5xl0^{12}$ G/L
- Ht- 0,29-0,25
- Anxiety, the cold sweat, the expressed pallor
- heartbeat rate on 20-30 % exceeds age norm
- BP is reduced on 10-15 %

Heavy (III) degree:
- Er-below $2,5\ x10^{12}$ G/L
- Ht - less than 0,25
- The expressed pallor, cold sticky sweat, block, quite often a stupor
- heartbeat rate on 50 % exceeds age norms
- BP is reduced on 30 % and more.

Usage of these simple parameters allows determining a complex of emergency actions. The last provide liquidation of infringement of the haemodinamic, metabolism, maintenance of normal functioning of the vital functions and systems.

Except it, a shock index is used for definition of the bleeding. It represents correlation of the heart rate to systolic arterial pressure.

Frequency of intimate reductions the Shock index (SI) = systolic arterial pressure (N^O.5-0,6).

If SI =0,8-1,2 prospective decrease VCB makes 15-20 %, that corresponds to an easy degree of the bleeding.

SI =1,3-2 - average the bleeding, prospective decrease VCB - 20-30 %. The heavy degree bleeding is characterised by a shock index from above 2, at decrease VCB more than on 35 %,

Depending on bleeding changes coagulograme of the patient. At an easy degree concentration of the fibrinogen, amount of the thrombocytes, thrombin time, and fibrinolitic activity are increased or are within the limits of norm. At an average degree the level of the fibrinogen, amount of the thrombocytes is reduced, other parameters remain same, as well as at an easy degree. The heavy degree is shown by significant decrease of concentration of the fibrinogen, thrombocytopenia, reduction of the thrombin time at simultaneous increase fibrinolitic activity. Thus coagulotractia consumption it is replaced by fibrinolisis.

Table I Clinical signs of a degree of the bleeding.

Parameter	Easy degree of the bleeding	Average degree of the bleeding	Heavy degree of the bleeding
Erythrocytes	3	2,5	Less than 2,5
Haemotocritic	0,3	0,29-0,25	Less than 0,25
Pulse	It is increased by 15-20 %	It is increased by 20-30 %	It is increased by 50 %
BP	Normal	It is reduced by 10-15 %	It is reduced by 30 %
Shock index	0,8-1,2	1.3-2	More than 2
VCB	It is reduced by 15-20 %	It is reduced by 25-30 %	It is reduced by 35 %
Pallor of a skin	Moderate	Expressed	Significant
Level of the fibrinogen	It is increased or in norm	It is reduced	It is reduced
Amount of the thrombocytes	It is increased or in norm	It is reduced	It is reduced
Thrombin time	It is lengthened or in norm	It is accelerated	It is short
Fibrinolitic activity	It is increased or in norm	It is increased or in norm	It is increased

At massive bleeding quickly there is changes of the haemostasis, a redistribution of the blood which has stayed at the human body. Reaction of the human body on sharp bleeding in many respects depends on the initial condition shown by "readiness" for a shock as chronic infringements in connection with various diseases of heart, a liver, kidneys. The leading part in tractogenesis of a shock belongs to infringement of acid-alkaline balance. At children quickly develops acidosis, the contents of standard hydrocarbonates is reduced, there is an oxygen starvation of fabrics. Metabolic acidosis it can be replaced alkalosis and on the contrary.

Haemoragic shock at children easily develops at ulcer bleedings of a stomach and duodenum, portal hypertension. Especially after repeated of the bleedings.

Conducting mechanism of adaptation of an organism to sharp of the bleeding is reaction of a vascular channel as a spasm and centralisation of blood circulation. At under acute and chronic bleedings there is a gradual adaptation and indemnification of bodies and fabrics to an anaemia

On character of disease or developmental anomaly allocate three groups of tractologies:

1). Children with somatic or infectious diseases at which the stop of a bleeding is made by conservative methods.

2). Patients who require consultation or supervision of the surgeon in connection with that spent conservative methods did not give desirable results. It is a so-called boundary tractology.

3). Children with surgical diseases to the Majority of them operation or special treatment will be carried out

Table II. The major reasons of bleedings from a digestive tract.

Somatic or infectious diseases	Boundary Diseases	Surgical Diseases
Dysentery	Haemoragic disease of the new-born	Invagination intestines
Haemoragic diathesis	Stomach ulcer of a stomach and duodenum	Portal hypertension
Sharp leucemoid and aleucemoid processes	Disease of the Sheinlein-Genocha	Hernia of the esophagal tract
Intestinal parasites	Non-specific ulcer colitis	Haemoragic gastritis
Lymphogranulomatosis intestines	Typho-paratyphosis infection	Meckel′s diverticulum
Sharp and under acute hepatites	Haemophilia	Polyps of a thick intestinum, disease of the Peitse-Agerse, disease of the Mellory-Veise
Exchange of the reticulosis		Doubling of intestines, tumour

Among surgical diseases classification of the bleedings by localisation of the centre is expedient.

Table III. Classification by localisation of a bleeding.

Esophagus	Haemoragic disease of the new-born, hernias of the esophagus apertures, chalazia of the esophagus, a bleeding from the expanded veins of a esophagus and a stomach
Stomach, duodenum	Haemoragic gastritis, a stomach ulcer of a stomach and duodenum, disease of the Mellory-Veise, polyps of a stomach, a tumour of a stomach
Thin intestinum	Meckel′s diverticulum, polyposis of a thin gut (disease of the Peitse-Agerse), Invagination, doubling of a thin gut, lymphopholicular hyperplasia a terminal department of the iliac intestinum, tumours
Thick intestinum the Direct intestinum	Polyps of a thick intestinum, non-specific ulcer colitis, Invagination, tumours, haemangioms

In practical work classification by age is very convenient.

Table 4 Classification of bleedings from a digestive tube on age.

Newborn	1. A birth trauma 2. Hypoxia, asphyxia 3. Haemoragic disease of the newborn
Nurse-child	1. Invagination intestines 2. Polyps of a thick intestinum Less often: doubling of intestines, a hernia of the esophagus apertures
1-3 years	1. Meckel′s diverticulum 2. Doubling of intestines 3. Polyps of a direct intestinum 4. Less often – syndrome of the Shereshevskii-Turner (teleangioectasia) 5. Tumours
older than 3 years	1. Polyps of a direct intestinum 2. Less often - disease of the Peitse-Agerse
	3. Tumours 4. Invagination
older Than 7 years	1. Portal hypertension 2. Haemoragic gastritis 3. disease of the Mellory-Veise 4. A ulcer of a stomach and a duodenal 5. Non-specific ulcer colitis

BLEEDING in NEWBORNs

Normal haemostasis depends on interaction thrombocytes and soluble fibres in plasma which, entering in interaction, form of the fibrinose clot.

The bleeding at new-born can be investigation of qualitative or quantitative change erythrocytes or coagulating fibres. For new-born decrease on 30-70 % vitamin - K-deficiency of factors coagulating systems (II, VII, IX is typical and X), at prematurely born this condition is even more dangerous as introduction of vitamin K appears inefficient because of транзиторной immaturity of a liver.

At children weak, prematurely born, transferred an intra-uterine sepsis or hypoxia, real coagulation or the DIC-SYNDROME can take place absent-minded intravascular. Occurrence of a bleeding at mature maturitive children is caused thrombocytopenia owing to immunological the conflict and shown classical Haemoragic by disease of the new-born,

Haemoragic disease of the NEWBORN (HDN)

HDN it is observed at 0,25-0,5 % new-born. Clinically disease is characterised by occurrence of bloody vomiting, melena which can be both unitary, and repeating, plentiful. Sometimes before occurrence is higher than the named attributes it is possible to observe pallor of integuments, mucous, a tachycardia, from a digestive tube bleedings can precede Bleedings skin haemoragia, at removing of the navel, kephalo hematomas, epistaxis. Sometimes disease is shown the general isolated petechias on a skin and mucous. The reason melena or bloody vomiting are small ulcer on mucous a stomach and duodenum.

Bleedings from mucous, puncsion of the vein, navel etc. testify about generalise infringement of coagul-ability of blood owing to deficiency of vitamin K, the DIC-SYNDROME and illness of a liver.

Nurse-child

From children after 1 month of a bleeding from GIT demand surgical intervention and treatment. Most it is frequently observed Invagination (3-10 months,), polyps of a thick intestinum, less often - doubling of a gut, a hernia of the esophagal apertures of the diaphragm.

1-3 YEARS

The reason of a bleeding at this age can be congenital developmental anomalies: Meckel′s diverticulum (most frequently), doubling of guts, polyps of a direct intestinum, less often - syndrome of the Shereshevskii-Turner, tumours.

Meckel′s diverticulum. Meckel′s diverticulum arises owing to infringement of return development umbilical duct. In 80 % of cases it is observed geterotopia mucous (a stomach, a pancreas, a thin gut). Among the disease, caused diverticulum bleedings, less often - impassability are observed peptic ulcers.

The bleeding can be unitary or recurring. Blood has be dark - cherry colour, an impurity of clots. At a plentiful bleeding stool mass practically are absent. Bleedings arise among full health. In heavy cases there can be a collapse.

At inspection reveal pallor, a tachycardia, falling the BP, decrease of amount erythrocytes, haemoglobin, In a direct intestinum the congestion of blood is determined. Treatment the Resection diverticulum, is preferable in the cold period. Doubling of intestines. The bleeding from rectal apertures less plentiful, is observed at children of 2-4 years more often. Seldom have chronic character. Treatment surgical.

Hernias of the esophagal apertures of the diaphragm. Are characterised by a moderate bleeding from erosive damages of a mucous membrane of a esophagus, have chronic character and conduct to anemisation of the patient. For this tractology characteristic clinical symptoms are dysphagia, pains in area of the epigastrium, backlog in physical development.

The diagnosis finally establish on the data of the XR in position of the Trendlenburg, and also on the basis esophagoscopy.

Bleeding at tumours. It is observed at haemangioms, sarcomas of an intestinal wall. In the first case it plentiful, can recurring, in the second - repeating, but not plentiful.

CHILDREN older than 3 YEARS

Most frequently the reason of a bleeding at children from 3 till 7 years are polyps of a thick gut, less often - disease of the Peitse-Agerse, tumours, Invagination.

For individual polyps typically allocation of a small amount of the not changed blood observed at the end of the act of the defecation. At a separation of a polyp the bleeding can be plentiful. The diagnosis establish after manual research, survey by rectoscopy or at endoscopy. At a polypose - the anaemia, constant allocation of blood is marked at each act of the defecation.

Syndrome of the Peitse-Agerse - heredo-familial polyposes, is combined with pigmentation of lips and a mucous mouth, and with age pigmentation decreases, and in puberty age disappeared.
Bleedings from a digestive tract can be intensive be accompanied by an anaemia. Quite often they are combined with invagination.

CHILDREN older than 7 YEARS of the Reason of a bleeding at this age are rather various.
Lymphopholicular hyperplasia a terminal department of the iliac intestinum. Last years it is established, that at some patients with the reason of an intestinal bleeding can be Lymphopholicular hyperplasia a terminal department of the iliac intestinum (LPH). In norm LPH meets practically at all children, it is located during 7-10 sm from illiocecal valve. This department of intestines is characterised also by features of local immunity, synthesising immunoglobulines, blast forms of the lymphocytes. The contents of these cells in 2-3 times is more, than in a mucous membrane of overlying departments.

On endoscopic and to the morphological data it is allocated 3 degrees LPH. The specified group of children is referred to group of risk whenever possible development in it of inflammatory process. The last can be accompanied by presence of erosion and even ulcers on a background of formation of conglomerates large lymph nodes, In this stage of the inflammatory process the bleeding can arise.
Disease is accompanied by periodic allocation of blood from a rectum, poorly changed on character. Falling the BP, НЬ, Er thus is not observed.
The diagnosis specify on the basis colonoscopy with obligatory survey of a terminal department iliac intestinum at which reveal sharply with edema, with hyperemia, mucous with erosion and ulcers, imposing of fibrin, but not always it is possible to establish presence of a bleeding.
Treatment provides purposeful specific anti-inflammatory therapy which appoint at ileitis and non-specific ulcer colitis.

Gastroenteric bleeding obscure ethiology. There are gastroenteric bleedings which source does not manage to be revealed, including laparoscopy and surgically. Such patients should be referred in group of risk and be subject to careful clinical inspection, including endoscopy, all digestive tract.
Portal hypertension. A principal cause portal hypertension - restriction of outflow from portal system. The barrier can settle down above, is lower or inside the liver (above, inside and underliver than the form).

Last to a thicket name exraliver the form.
The second factor of increase of pressure in portal vein is the increase of inflow of blood in visceral channel connected with hyper dynamic of the cardial syndrome. The last is observed at a cirrhosis of a liver, Subject of surgical interest is exraliver that form portal hypertension.

Varicose veins and occurrence of a bleeding count the reason of break:
1. A hypertonic crisis in system of portal vein.
2. Change mucous a stomach and an esophagus owing to infringement of the blood circulation, shown by erosion, ulcers.
3. Infringement coagulative systems of blood.

Clinically 1-2 days prior to a bleeding the patient has weakness, an indisposition, a pain in epigastral areas, rise in temperature up to 39-40°Ñ, that is quite often regarded as ARD. Further there comes deterioration: weakness amplifies, there is a pallor of a skin and mucous, thirst, dryness in a mouth, the tachycardia, is reduced filling and a pressure of pulse, the BP falls, the clinic haemoragic a shock develops. Then there is a vomiting of colour of a coffee grounds, at profuse bleedings - stool as crimson jelly in 30-40 minutes. The increased spleen quickly decreases, however it remains accessible for palpation.

Modern ultrasonic has considerably changed opportunities of early diagnostics exraliver forms PH.

Earlier at the majority of children the bleeding from veins of a esophagus was the first display of disease. Now the diagnosis can be established before development of a bleeding.

Ultrasonic attributes IPH is the normal structure of a liver without its increase. Main attribute Bill on ultrasonic is absence of correctly generated trunk portal veins and it intraliver branching. On ultrasonic the conglomerate twist veins and a fibrous fabric (portal cavernome) is determined. The second symptom is the thickening of a small omentum till the sizes of a belly aorta,

Using up to orography it is possible to find out return blood in vessels of a small omentum, and also delay of a current of blood in 2-3 times in vessels and lien to a vein.

During ultrasonic it is necessary to establish not only the reason portal hypertension, but also variants of anatomy visceral veins with which it is possible to impose decompressive anastomosis.

On ultrasonic visualised upmesenteric vein, it is possible to look after spleen a vein, vein cava inferior and left renal vein, which defects at IPH meet in 10 % of cases that can complicate its allocation during operation,

The second method of research is distal angyography which allows to see veins of a belly cavity in the real image, Spleen pro columns èÿ now it is used extremely seldom.

The third method is esophagoscopy at which it is possible to see expanded changed by varicosis veins of a esophagus and cardial a department of a stomach. Attribute PH is gastrotracty, expansion, their pressure. Presence of cherry spots 12 on veins of an esophagus, hyperemia, fibrinose imposing, twistering are terrible predecessors of a bleeding.

Treatment IPH at the present stage is reduced to prevention of a bleeding or treatment of a bleeding from the expanded veins of a esophagus and a stomach with the purpose of their prevention in the future.

Treatment of bleedings in the sharp period begin with conservative therapy and continue within several hours. In a stomach enter a probe and delete contents, and also it leave for the control of intensity of a bleeding. Despite of a proceeding bleeding, volume infusion therapies reduce up to 50 % of volume of daily requirement. Exclude the preparations directed on improvement reology. To the patient pour erytrocytic mass and chilled plasma. The purpose of similar transfusion – decrease of system pressure and increase of viscosity of blood.

Very important component of treatment is deep sedative therapy for exception of anxiety of the patient, tormenting thirst and presence of a probe in a stomach.

If within several hours the bleeding proceeds, put indications for imposing decompressive anastomosis. Methods of restoration of the blood in portal vein yet it is not developed. In the different countries of Europe and America in overwhelming majority of cases use endoscopic sclerotherapy. Special rings, with which blocked bleeding vessels are developed also at performance of endoscopy . This method allows to avoid necrosis a wall of a esophagus which can take place at sclerotherapy. In 10-15 % of cases after sclerotherapy repeated bleedings besides preservation of the increased pressure in portal system limits physical activity of the child are observed, demands observance of the strict diet, special supervision over the patient.

In Japan with the purpose of the prevention of bleedings use offered by Sigiura and becoming popular in other countries at unsuccessfulness sclerotherapy a method including devasculrisation bodies and a bottom of a stomach, the bottom third of esophagus, splienectomy, piloroplastic and vagotomy. The esophagus or a stomach at cardial cross or dissect for crossing varicose veins.

In Russia Prof. A. F. Leont'evym are developed and widely used venous shunting. At height of a bleeding it is expedient to use imposing of the widest anastomosis, capable completely to stop blood in varicose veins of a esophagus and a stomach. Most frequently impose spleen-renal or spleen-suprarenal anastomosis "side – in - side". Apply mesenterico-coval anastomosis less often, use an insert from internal jugular veins even less often

At performance of scheduled operations the order remains same.

As specifies Prof. A.J. Razumovsky (clinic of academician J.F.Isakova), shunting operations are shown at safe function of a liver at patients with exraliver PH, and also congenital fibrosis to a liver. At intraliver to form PH it is shown sclerotherapy or devasculrisation by Sigiura.

Haemoragic gastritis. It is characterised by plural haemorrhages, sharp erosion or superficial ulcers on mucous a stomach. Distinguish erosive and haemoragic gastritis. At the moment of a bleeding to distinguish haemoragy from erosion it is difficult. Most frequently haemoragic the gastritis develops as a result of system diseases (endocrine, infectious, toxic, septic) or under influence of local factors (medical products, vascular or alimentary frustration), and also mechanical irritation (trauma). The basic in haemoragic gastritis - infringement of exchange processes, coagulation systems of blood, increase of permeability of walls of capillaries. Occurrence of erosion and ulcers is promoted by amplification of digesting force of gastric juice owing to stress, hypercopny, necrobiotic changes in a mucous membrane, caused hypoxy, toxaemia or an angiospasm, and local injuring by medicinal substances.

Clinic of the bleeding.

1.Vomitting is a first attribute, intensity of it happens from significant up to fatal Sometimes to a bleeding pains in a stomach precede.

The diagnosis establish on the basis endoscopic investigation spent at height of a bleeding. Thus determine a hypostasis mucous, it haemoragy, erosion, small spot haemorrhages. The bleeding from sites visually intact mucous is characteristic.

Treatment. Washing of a stomach cooled up to 5-8°Ñ water, 0,5 % a solution of nitrate of silver, epsilon-aminocaprony an acid, fibrinogen, local cooling in time of endoscopy by mezaton, noradrenaline, fibrinogen, put on erosive surface - glue ÌÊ-6,7, film-forming aerosols. At absence of effect it is shown selective proximal vagotomy.

Syndrome Mellory-Veise. Syndrome Mellory-Veise - one of the reasons of the not ulcer bleedings connected to spontaneous break of a mucous membrane or deeper layers of a wall of a stomach esophago-gastral of an internal department. Meets very seldom at teenage age. In tractogenesis conducting place remove to increase of pressure in cardial a department at insufficient disclosing crude at strong cough, an attack of a bronchial asthma, epileptic an attack. Such picture can be observed and at strong vomiting. To promoting factors it is necessary to relate esophagal hernia, inflammatory diseases of a esophagus and a stomach.

Clinic. Vomiting of colour of a coffee grounds, less often - scarlet blood.

Before vomiting without an impurity of blood, as a rule, is observed.

The diagnosis: it is specified on a basis endoscopic researches during which determine the sizes of damages, cracks from 1 up to 5 see are more often. The bottom of breaks is filled with clots of blood. Alongside with breaks mucous deeper damages andermucouse and muscular layers can be marked.

Character of treatment is determined by intensity of a bleeding. Start with conservative therapy: washing by ice water, purpose of vasoconstrictive means (adrocson, adrenaline, novocain), with the help of the injection make a local irrigation of 96 % spirit, chloretil an acid. These means help to suspend a bleeding so that then to make a final stop of a bleeding with the help diatermic, laser coagulation. At absence of effect make gastrotomy.

Bleeding at a stomach ulcer of a stomach and a duodenal gut. The bleeding can arise both on a background of the "ulcer" anamnesis, and without any harbingers. Punching and a bleeding from a stomach at children (it is especial if they have arisen on a background of heavy diseases: the greenstone, a sepsis, a hepatites, a uraemia, thermal burns) are especially dangerous. So-called stressful ulcers at these patients, as a rule, develop in a terminal phase, is especial at realisation of massive hormonal therapy.

CLINICAL PICTURE of BLEEDINGS FROM the DIGESTIVE TRACT.

Diseases are characterised:

1. The general weakness

2. Senior children complain of noise in ears
3. Dizziness, flashing in eyes
4. At survey the attention sticky sweat pays to itself
5. Pallor of integuments and mucous
6. A cold snap of finiteness
7. A point of features
8. Block, on occasion euphoria, changes to blackout of consciousness
9. At a proceeding bleeding the tachycardia, arrhythmia is marked
10. Intimate tones are muffled
11. At attempt rise the patient can to lose consciousness
12. the BP falls, is reduced CVP, VCB
13. Disease is accompanied by bloody vomiting, a chair on type " crimson jelly, the expiration of scarlet blood from rectum.

DIAGNOSTICS of BLEEDINGS FROM GIT

Diagnostics of bleedings develops of a digestive tract from:
1. Scopings bleeding (according to pulse, the BP, Er, Hb, Ht, VCB, CVP),
2. Revealing a source of a bleeding. The last is based on definition:
a) Character of blood in vomiting;
b) Character of vomiting in stool,
The bleeding from top departments GIT (mucous a esophagus, a stomach, duodenum) is characterised by a gut of " a coffee thick " as haemoglobin, treat to action of a hydrochloric acid, turns to muriatic hematite, getting brown colour, however, at massive bleeding such change of haemoglobin does not occur, therefore emetic weights have colour less changed blood.

At their bleedings Meckel′s diverticulum blood, mixing up with contents of intestines, gets dark colour, colour of crimson jelly. At polyps of a direct gut blood settles down on a surface кала, colour of it is not changed.
At small bleedings from a stomach, a esophagus, vomiting can and to not be, it is possible to judge a bleeding on colour of a stool or on the laboratory data (the latent blood).

As additional methods use radiological, endoscopic and special researches.
The radiological method was widely used earlier at bleedings from varicosis the expanded veins of a esophagus, a stomach, a stomach ulcer of a stomach, duodenum guts (defect of filling), a polypose intestines. Now it is made fibroesophagogastroduidenoscopy, colonoscopy more often, allowing to examine mucous to determine a source of a bleeding and simultaneously to make coagulation or sclerosing of the bleeding vessels.

At portal hypertension deciding not only diagnostic, but also medical value has dopplerography.

Among special methods of diagnostics the important role isotope methods, angyography play radio etc.

The GENERAL PRINCIPLES of TREATMENT of BLEEDINGS.

Treatment of bleedings from the digestive channel depends on character of disease, its intensity, localisation, the mechanism of infringement coagulation systems of blood.
At new-born during adaptation, decrease of a level thrombin (2-3 days.), deficiency vitamin - to dependent factors treatment is reduced to purpose vicasole, to blood transfusion, adrocson.
At children of chest age at invagination treatment is reduced to conservative, less often operative disinvagination; at polyps of a rectum - to polipectomy

Surgical methods eliminate a bleeding at hernias esophagal apertures, tumours, doubling of intestines.

Tactics of treatment portal hypertension are stated in the appropriate section.

At haemoragic gastritis, a stomach ulcer of a stomach and a duodenal gut treatment will be carried out on 3 directions:

1. Struggle with hypovolemia and an anaemia (infusing therapies reduce volume up to 50 % of daily need), exclude the preparations improving reologic properties of blood. To the patient pour erythrocytar mass and chilled plasma with the purpose of reduction of system pressure and increase of viscosity of blood.
2. Haemostatic therapy: purpose of the preparations accelerating thrombogenesis or lowering fibrinolisis (vicasole 0,1 ml for year of life, chloride calcium of 10 % of 1 ml for one year of life, but no more than 10 ml, haemoglobin 1,5 % of 1-2 ml on kg, fibrinogen on 1-4 ã dry substance, adrocson 0,025 % on 1 ml i/m).
3. A local stop of a bleeding: coagulation, sclerotherapy etc.

6.

Main literature:

1. Pediatric surgery: textbook / Losev O.O., Melnychenko M.H., Dilanyan I.R., Samofalov D.O.; edited by Loseva O.O. – Odesa : ONMedU, 2011. — 224 pages

2. Pediatric surgery (Textbook / Grif of the Ministry of Health of Ukraine / Odesa: ONMedU, 2019, 224 c.) Losev O.O., Melnychenko M.H., and others, 7 people altogether

3. Kryvchenya D. Y., Lysak S.V, Plotnikov O.M Surgical diseases in children. - Vinnytsya: New book, 2008. - 256 p.

4. Pediatric surgery. / Edited by Sushka V.I. and co-authors // K. Health. - 2002. - 718 p.

5. Pediatric surgery. Tutorial. Part 2. Edited by Tolstanova O.K., Rybalchenko V.F., Danilova O.A. and others. Zhytomyr "POLISSYA". - 2016. - p. 225, 322-331

Additional literature:

6. Emergency surgery of the abdominal cavity (standards of organization and professionally oriented algorithms for medical care) / Edited by Fomina P.D., Usenko O.Y., Bereznytsky Y.S. - Kyiv: ‘Health of Ukraine’ Library, 2018. - 354 p.

7. Age aspects of acute appendicitis in children and the key to its recognition. Tutorial. 2019. 260 p. Edited by Bodnar B.M., Ribalchenko V.F., Bodnar O.B., Melnichenko M.H., and others. Publishing. ISBN 978-966-697-828-1

8. Nedelska S.M. Diseases of the hepatobiliary system and pancreas in children. Textbook for 6th-year higher education applicants of a medical faculty, interns, pediatricians, family doctors / Nedelska S.M., Mazur V.I., Shumna T.E.. - Zaporizhzhia: [ZDMU], 2017. - 113 p.

9. Violation of defecation in children: constipation and encopresis: Textbook / Rybalchenko V.F., Berezhnyy V.V. ,Konoplitsky V.S., and others; Edited by professor Rybalchenko V.F., Berezhnyy V.V., Konoplitsky V.S., Rusak P.S. - Kyiv: VIT-A-POL LLC, 2018. - 548 p .: illustrations

10. Intestinal malrotation in children: from embryogenesis to consequences / Monograph. Edited by Pereyaslova A.A., Rybalchenko V.F., Loseva O.O - K .: PE "INPOL LTM" Printing House "000000000", 2019. - 226 p: illustrations

11. Intestinal obstruction in children: a textbook for higher education applicants of the 5th year of medical faculties (specialties: "Medical business", "Pediatrics"). Spahi O.V., Barukhovych V.Y., Kokorkin O.D., Lyaturynska O.V., Pakholchuk O.P., Zaporozhchenko A.H. - Zaporizhzhia. -2015.- 75 p.

12. Developmental defects in children: a textbook for independent work of higher education applicants of the VI course of the medical faculty (specialties "Medical business", "Pediatrics"). Solovyov A.E., Lyaturynska O.V., Barukhovych V.Y., Spahi O.V., Shchokin O.V., Makarova M.O., Anikin I.O. Zaporizhzhia, 2013. 165 p.

13. Bachurin V.I. Malformations of the genitourinary system as a cause of urological pathologies: teaching methodology for independent work of fourth-year higher education applicants in the specialty: 7.12010001 "Medical business", 7.12010002 "Pediatrics", 6.120102 "Laboratory diagnostics" / Bachurin V.I.. - Zaporizhzhia: ZSMU, 2017. - 86 p.

14. Makarov A.V. Examination of the respiratory organs in children. Training manual. Makarov A.V., Danilov O.A., Sokur P.P., Rybalchenko V.F., Yurchenko M.I. - C .: Business entity Kolyada O.P., 2005. - 160 p.

Tests for control.

1. Most typical sing of presence of intestine polyps is:

A. Petition on abdominal pain
B. Signs anaemia
+ C. Intestinal bleeding

D. Unstable stool
E. Pain during defecation
2. There are periodic massive bleedings by a not changed blood from the lower evacuated ments intestine in 3-years old child. What is the most interquartile reason of such bleeding?
A. Hemorrhoids
B. Polyp of a rectum
C. Anal fissure
D. Ulcerative colitis
+ E. Ulcer Meckel's diverticulum
3. 7 monthly children in is delivered to the clinic in 18 hours after the beginning of disease with the complaints of paroxysmal abdominal pains, twice time vomiting, presence of "blackberry" jelly like stool. The diagnosis is invagination. What tactics is necessary to carry out?
A. Laparoscopy
B. Operating treatment

+C. Closed desinvagination

D. Observation
E. Siphon enema
4. Paroxysmal pains in abdomen, vomiting, a dark blood in a stool has appeared in 6-months old child after giving of vegetable mashes. The invagination is suspected at admitted department. What signs are characteristic for this disease?
A. "Vanka-vstanka" sign
B. Sign of "scissors"
+C. Dance's Sign
D. Filatov's Sign
E. Kocher's Sign
5. A child, 7 months old, admitted at the department in 8 hours after the beginning of disease with the complaints of paroxysmal anxiety, once vomiting. There is palpated tumorous formation in the right half of abdomen; dark blood was in rectal inspection. What disease is it talking about?
A. Doubling of intestine
+B. Invagination
C. Tumour of abdominal cavity
D. Ascaridas invasion
E. Enterocystoma
6. You are the doctor of first aid, who was called to the 8-months child in 8 hours from a beginning of disease. The mother has told, that among a full health attacks of anxiety have arisen, knocks by legs, the vomiting has appeared. The stool was on the eve. Discharges from a rectum like crimson jelly have appeared in rectal inspection. Your suggested diagnosis is:
A. Acute appendicitis
B. Capillary toxicosis
C. Meckel's Diverticulum
+D. Invagination
E. Dysentery
7. Point the reasons of bleedings from a digestive tube in children from 1 year till 3 years
A. Doubling an intestine
+B. Meckel's diverticulum

C. Hernia oesophageal opening of diaphragm
D. Halasya of oesophagus
E. All numbered reasons

8. A child, 12 years old, suddenly had collapse, the profuse vomiting by a not changed dark blood has appeared. At examination: there are paleness dermal covers, B/P - 80/50 mm Hg. Abdomen is painful. There is omphalitis in an anamnesis. Your suggested diagnosis is:
A. Halasya of oesophagus
+B. A portal hypertension
C. Peptic ulcer of abdomen
D. Capillary toxicosis
E. Bleeding from Meckel's diverticulum

9. A 3-year-old boy complains of pain on defecation with hard consistency of feces. Stool are with admixture of blood . Retention of stool is observed to. Make a diagnosis.
A. Intussusception .
B. Hemorrhoids .
+C. Anal fissure.
D. Dysentery.
E. Rectal polyp .

10. A 3 years-old child suffered from tussis for a week. Suddenly appear pains in abdomen and vomiting with blood and melena. Doctor suspect Mallory-Weys's syndrome. What method of investigation should be undertaken?
A. Ultrasound investigation
B. Plain X-Ray
+C. Fibrooesophagoscopya
D. Blood count
E. Urinalysis

11. The child has a portal hypertension. Where bleeding in children prevail from?
A. Nose
B. Haemorrhoidal veins
C. Small intestine
+D. Cardial part of oesophagus and stomach
E. Pulmonary bleeding

12. The surgeon made a diagnosis of polyps of rectum. What tactic is indicated in this case?
A. Open operation
+B. Electrocoagulation of polyp
C. Conservative treatment
D. Cryodestruction of polyp
E. Sclerotising method of treatment

Topic 2. Acute surgical diseases of abdominal cavity organs

1. The topic: the Syndrome of a pain in a stomach (developmental anomalies, differential diagnostics, treatment; inflammatory diseases and a trauma of organs of a abdominal cavity, Differential diagnostics, treatment).

2. Actuality of The topic:

It is known more than 100 surgical diseases at children, accompanying with pains in a stomach. At many of them delay with realization of operative treatment conducts to progressing deterioration of a condition of the child, occurrence of a situation when disease threatens not only to health, but also life of the child. A pain in a stomach the otitis, a pneumonia, chicken pox, rubella, a pyelonephritis, a gastritis, intestinal infections can accompany also such diseases frequently meeting at children, as ARVI. Therefore with painful abdominal a syndrome at children it is necessary to meet in the practice not only to children's surgeons, but also podiatrists, doctors of a polyclinic, first aid, surgeons of the general structure.

At adult patients does not represent special difficulties to reveal character, intensity and localization of pains, that it is impossible to tell about children, it is especial children of the first years of life at whom in a abdominal cavity frequently it is possible to judge development of tractological process only on equivalents of a pain: to anxiety, crying, infringement of dream, refusal of meal,

Sometimes children try to hide presence of pains in a stomach, being afraid of hospitalization or operations.

Knowledge both surgical, and the somatic diseases proceeding with painful abdominal by a syndrome, a correct estimation of complaints and conditions of the child are a key to statement of the correct diagnosis and realization of necessary treatment.

3. The educational purposes of the study: to acquaint higher education applicants with a syndrome of a pain in a stomach at children in view of age of the child, to study a clinical picture most frequently the meeting diseases accompanying painful abdominal with a syndrome, to study methods of inspection and principles of realization of differential diagnostics at these diseases-

As a result of the study, higher education applicants should know:

1. Classification and clinical picture of internal hernias.
2. The reasons of development, a clinical picture, diagnostics and treatment chronic duodenal impassability.
3. Classification, clinical picture, diagnostics and treatment of hernias esophageal apertures of the diaphragm,
4. Clinic and diagnostics of the parasternal hernias.
5. Classification doubling a gastroenteric tract. A clinical picture of complications doubling a gastroenteric tract, methods of diagnostics and treatment.

6. Developmental anomalies of genitals at the girls, accompanying painful abdominal a syndrome.

7. Clinical picture and methods of diagnostics of developmental anomalies illiocecal angle. Classification and a clinical picture of the diseases caused by presence diverticulum of Mekkel.

8. ethiology, pathogenesis, a clinical picture sharp of appendicitis at typical and atypical forms.

9. Additional methods of research at children at suspicion on sharp appendicitis. Classification of complications sharp of appendicitis. Surgical tactics at complications.

10. Postoperative complications at sharp appendicitis. Methods of treatment. Features of a clinical picture and diagnostics sharp of appendicitis at children of the first 3 years of life,

11. The reasons of development and feature of a clinical picture mesanterial lymphadenitis.

12. Clinical picture of gynecologic diseases at the girls accompanying with pains in a stomach. Differential diagnostics with sharp appendicitis.

13. Features of a clinical picture, diagnostics and treatment at various forms of an initial peritonitis.

14. Diseases of the bile secretion systems at children, proceeding with pains in a stomach.

15. Diagnostics and treatment of a sharp cholecystitis at children.

16. Classification of a sharp pancreatitis. Features of a clinical picture depending on a stage of disease.

17. Conservative treatment of a sharp pancreatitis. Indications to operative treatment. A clinical picture, diagnostics and treatment of disease the Crone at children. Indications to operative treatment.

18. Features of a clinical picture nonspecific ulcer colitis. Methods of diagnostics. Conservative treatment, indications to operative treatment. Classification of traumas of organs of a abdominal cavity at children.

19. Classification of damages of a spleen and liver at children. Features of a clinical picture at various damages. Additional methods of diagnostics. Methods of treatment of damages of a spleen and a liver.

20. Classification of damages visceral organs of a abdominal cavity. Features of a clinical picture depending on localization and a kind of damage.

21. The reasons of damage of a pancreas at children. A clinical picture, diagnostics and treatment.

As a result of the study higher education applicants should be able:

1. To collect the anamnesis of disease at complaints to pains in a stomach.

2. To carry out objective inspection of children of various age at painful abdominal syndrome.

3. To appoint the plan of inspection at chronic duodenal impassability at children. To estimate results

radiological and endoscopic inspections at chronic duodenal impassability, hernias esophageal apertures of the diaphragm.

4. To appoint the plan of inspection at suspicion on sharp appendicitis. To carry out differential diagnostics sharp of appendicitis with others most frequently the meeting diseases causing pains in a stomach

5. To appoint the plan of inspection at a trauma of organs of abdominal cavity at children. To estimate results of laboratory, ultrasonic and radiological research at a trauma of various organs of abdominal cavity.

6. To determine tactics of treatment at various damages of organs of a abdominal cavity at children.

4. The plan and organisation of lessons.

Work type	Time	Activity		Equipment
		Higher education applicants	Teacher	
Organisation of the practical class	5	Listen	Presence control	Journal
Aim setting	5	Listen	Explain	
Checking of the knowledge of higher education applicants	15	Make	Control	Tests
Seminar	60	Answer	Control	X-ray-grams
Clinical discussion	30	Listen	Discuss patients	Patients
Personal work of higher education applicants	60	Make	Control	Patients
Results of the lesson	10	Listen	Appreciation of each activities	
Task for the next lesson	5	Listen	Explain the task. Indicates the literature.	

5. Substance of the lesson.

7.

4. Basic knowledge are necessary for realization of the purposes of the study for the higher education applicant:

Embriogenesis and anatomy of organs of abdominal cavity at children.

Inspection of organs abdominal cavity at children; survey, palpation, percussion, auscultation.

Radiological methods of inspection of a gastroenteric tract.

endoscopic methods of research of organs of abdominal cavity.

Children's infectious diseases: a chicken pox, a scarlet fever.

The infectious diseases amazing a gastroenteric tract: a dysentery, a salmonellosis, a virus hepatitis.

Helminthes invasion at children.

Diseases of respiratory ways at children: ARVI, a sharp pneumonia and a pleurisy,

Disease Shonlein.

Rheumatism at children.

Diseases of the urinosecretion systems at children: a pyelonephritis, glomerulonephritis, a cystitis, urolithic disease.

Diseases of a gastroenteric tract at children: a gastritis, duodenitis, a stomach ulcer of a stomach and a duodenal, a dyskinesia of the bile secretion ways.

4 5. Tasks with standards of answers:

1-Histologic classification sharp of appendicitis: cataral, phlegmonal, gangrenous, gangrenouse-perforative

II. Atypical forms of an arrangement of appendics: retrocecal in a small basin underliver left-hand

III. A diagnostic key sharp of appendicitis at children on With, J. Doletsky: Pressure of muscles of a stomach a bathing pain.

IV. The child of 5 years has arrived in clinic with complaints to the weakness, the reduced appetite, rise in temperature up to 38 degrees, cough, a headache, a pain in a stomach. Integument pure pale, in the act of breath the auxiliary muscles participate. Above easy it is marked shortening of the percussion sound under a angle of the scapula on the right, rigid breath, fine bubbling rale and crepitate rales here is listened. Frequency of respiratory movements - 36 a minutes, pulse - 130 in one minutes. The stomach of the correct form, participates in the act of breath, soft, painful in epigastrium and the right departments. At radiological

research amplification pulmonal figure, infiltrative a shadow borrowing the bottom share right easy is revealed.

The general analysis of blood: НЬ - 120 г/л, Ег - 3, 1 Т/л, ESR - 18 mm / h, L - 18 g/l, э-2 п-15

1. Diagnose.

2. Than you can will explain a pain in a stomach at a pneumonia? Right-hand infralobular pneumonia.

Pains in a stomach at a pneumonia and a pleurisy speak irritation of the bottom intercostal nerves, which innervated diaphragm, abdominal muscles and a mucous of a stomach.

V. The child of 9 years shows complaints to weakness, a fever, a headache, pains in muscles and a waist, rise in temperature up to 39 degrees, repeated vomiting, a diarrhea. Is sick 7 hours.

The child languid, integument pure pale, tongue dry. Breath rigid, is not present rattles. Intimate activity rhythmical, tones are muffled. Pulse - 110 in one minutes. The stomach is moderately inflated, soft, hums at palpation, morbidity in epigastrium, paraumbilical and right iliac areas is marked. A stool liquid, fetid, with an impurity of slime, greenish color.

Put the presumable diagnosis.

What from symptoms marked at the child concern to negative at sharp appendicitis?

Sharp gastroenteritis, presumably salmonellosis ethiology. - a headache

Muscular pains

hypertermy

Rumbling of a stomach

Fetid, liquid stool

6. The information for fastening initial knowledge can be found in the following manuals:

1. Andrievich M. A. Diagnostic, therapy and prevention of the inflectional diseases, - Lviv; 1996.-

352p.

2. Belozertsova V.N., Belakova A.V. Pediatrics.-S-P.:. 1999. 374 p..

3. Nikolaeva N.G. Pediatric surgery, Odessa, 1999. 212 p.,

4. Pokrovsky V-I. Infectious diseases.-M: Medicine, 1996. - 452 p.

5. Shabalov N.P. Children's of disease. - S-P.., 2000. 865 p,

7. The contents of a study material:

I. A syndrome of a pain at developmental anomalies of a gastroenteric tract:

Internal hernias.

Chronic duodenal impassability

Parasternal hernias.

Hernias esophageal apertures of the diaphragm.

Doubling of a digestive tract.

Developmental anomalies of genitals at girls.

diverticulum Of Mekkel.

Diseases illiocecal angle.

II. A syndrome of a pain at inflammatory diseases of organs of abdominal cavity.

Sharp appendicitis.

mesadenitis.

Gynecologic diseases,

Initial peritonitis.

Sharp cholecystitis.

Sharp pancreatitis.

Disease the Crone.

Nonspecific ulcer colitis.

III. A syndrome of pain at a traumatic organs of abdominal cavity.

Damages of a spleen.

Damages of a liver.

Trauma of a pancreas.

Damages of a stomach and intestines.

Internal hernias represent moving loops of intestines to defects of the mesentery, an omentum or pockets of the peritoneum. Such hernias meet seldom enough and come to light basically during operations

concerning sharp intestinal impassability Displacement of the intestinal loops covered lairs of the peritoneum which form a hernial bag, carry to true internal hernias. If internal organs move through apertures in peritoneum or other congenital defects they are not covered parietal peritoneum and form false internal hernias. Internal hernias can be located in any department of a abdominal cavity, but most frequently meet paraduodenal hernias (hernia Treitsa) which occurrence is connected to presence clefting defect in a back wall of a abdominal cavity in area duodenal a bend, paracecal the hernias representing displacement of intestinal loops in tractological pockets, located in area illiocecal a angle and moving of intestinal loops through apertures in peritoneum, most frequently in the field of a terminal department iliac and ascending colon,

Patients with internal hernias complain on periodic spasmodic pains in a stomach which decrease or disappear in position on a stomach. Sometimes internal hernias proceed without symptoms and for the first time come to light in connection with development of sharp intestinal impassability when realization of urgent operation which essence consists in extraction of intestinal loops from a hernial bag or through defect of the mesentery is shown, to an estimation of viability of intestines (at long infringement the resection of impractical loops of intestines can be demanded) and rhaphing a hernial gate. It is necessary to remember that at paraduodenal hernias the section of a hernial gate is inadmissible in connection with passage on edges of a gate of an ascending branch bottom peritoneal veins and left colon arteries.

Complaints to pains in epigastral areas, a nausea, vomiting, an eructation, a swelling in the top department of a stomach, it is especial after meal can be connected with chronic duodenal the impassability caused by crushing of a duodenal like a ring a pancreas or the vascular plug formed by an aorta and departing from it top peritoneal by the artery. On the survey roentgenogram of organs of a abdominal cavity it is possible to find out two levels of a liquid in a stomach and a duodenal . At endoscopic research of a duodenal narrowing its gleam in a place of the crushing with expansion above the located departments is determined, inflammatory process frequently comes to light on the part of a mucous membrane. The angle of the outcoming peritoneal arteries from an aorta can be revealed at aortography (in norm it makes 45 - 60 degrees) - Presence of a proof painful syndrome, the phenomena dysphagia and radiological attributes of impassability is the indication to operative treatment. At like a ring to a pancreas it is imposed at arteriomesenterial impassability-duadenojejunoanastomosis-

At parasternal hernias (hernias of a forward department of the diaphragm) there is a displacement of organs of abdominal cavity in a chest cavity through thin a forward department of the diaphragm in the field of crack Larrea (left-hand parasternal hernia) or apertures Morgany (right-hand parasternal a hernia). At parasternal hernias children complain of periodically arising pains and unpleasant sensations in epigastrium, sometimes there is a nausea and vomiting. In the time of percussion and auscultation in the field of the bottom department of a breast is determined timpanist and easing of intimate tones, on roentgenograms comes to light a shadow halfoval forms in area retrosternal. Usually in a hernial bag there are loops of intestines which are visible as macrocellular shadows on a background easy and hearts. Operative treatment parasternal hernias consists in bringing down of loops of intestines in a abdominal cavity and closing of the message between a abdominal and chest cavity by make a rhaphing of the diaphragm to soft fabrics of a forward chest wall.

Hernias esophageal apertures of the diaphragm are subdivided on esophageal and paraesophageal. At esophageal hernias there is a moving a stomach upwards, in mediastinum owing to what a gullet the but - gastric message settles down above a level of the diaphragm, and the gullet bends and deformed. For

paraesophageal hernias typically displacement of organs of abdominal cavity (more often a stomach) upwards near to a gullet. The clinical picture of hernias esophageal apertures of the diaphragm is caused by development gastro-esophageal reflux which at children of younger age is shown by anxiety, then vomiting which has constant character, for children of younger age anemia . More often these complaints arise after meal, in a prone position that is connected with leaking gastric contents in a gullet. Hernias esophageal apertures of the diaphragm can be restrained, that is accompanied strong spastic pains in hypogastrium areas and vomiting by "fountain". The diagnosis of hernias esophageal apertures of the diaphragm is specified at endoscopic and radiological research. Endoscopic attributes esophageal hernias are displacement of a line of esophageal-gastric transition above esophageal rings of the diaphragm, attributes gastro-esophageal reflux - mucous a gullet with hyperemia, is covered with erosion, easily bleeds, on border of an average and bottom third of gullet come to light strictures. Inflammatory changes on the part of mucous a gullet are characteristic and for paraesophageal hernias. On roentgenograms at paraesophageal hernias the bottom of the stomach displaced in a chest cavity is determined as cystic formations with the level of a liquid located on the right or to the left of a median line. The gas bubble of a stomach in abdominal cavity is reduced or is absent. At hernias esophageal apertures of the diaphragm research with contrast substance is obligatory. Characteristic for esophageal hernias is twisting course of a gullet, a high confluence of a gullet of a stomach, a stupid angle of a confluence of a gullet in a stomach, expansion of a gullet in the bottom third. After hard fillings of a stomach by contrast substance the child is stacked in position Trendlenburg′s and the moderate compression epigastrium is made. gastro-esophageal reflux it is determined at leaking contrast in a gullet - At папaesophagal hernias in position Trendlenburg′s the contrast substance becomes numb in area of a bottom of the stomach located above of the diaphragm. The stomach thus takes the form " of a sand-glass?). In treatment of hernias esophageal apertures of the diaphragm are applied both conservative, and operative methods. Conservative treatment (fractional feeding by small portions, application of rich mixes, vertical position of the child after feeding, use antacid, antispastic and sedative preparations) is shown at the small hernias which have been not complicated esophagitis or narrowing of a gullet - the Hernias proceeding with an expressed clinical picture, absence of effect from conservative treatment are indications to operative treatment.

Doubling of a digestive tract can meet in its any department, but most frequently they come to light on a thin. Distinguish cystic, diverticulum and tubular forms of doubling. diverticulum and tubular doubling are informed with a gleam of a digestive tract, cystics forms of doubling, settling down near to an intestinal tube, are connected with it the general muscular layer and having vessels. Less often cystics doubling have own peritoneum. All doubling are visceral the formations covered from within with a mucous membrane which structure not always corresponds to a that department of a digestive tract at which level there is a doubling .More often all doubling of a digestive tract are shown by attributes recurrent intestinal impassability. The child complains on periodic like attack pains in a stomach which are accompanied by a nausea, vomiting. The delay of gases and a stool . At the survey determinessome asymmetry of a stomach seen peristalsis of intestines. At palpation it is possible reveal tumor like formation in a abdominal cavity Attacks can to proceed from several minutes till one o'clock at radiological research with contrast substance it is possible to reveal a long delay of contrast in diverticulum. However in the sharp period this research is contra-indicated. Doubling of a thick can be revealed at irrigography or colonoscopy, High diagnostic value has laparoscopy To differentiate doubling follows from any mobile formations of a abdominal cavity mesenterial vasculars, vasculars an omentum - Frequently doubling gastro-intestinal a tract proceed without symptoms and for the first time are shown by complications: punching with development of a peritonitis, the impassability caused volvulus or node formation of a loop of bearing doubling, invagination or a bleeding. Small cystes or short

and wide diverticulums can cause invagination, that is shown spastic pains in a stomach, the Stool is detained by repeated vomiting, gases do not depart. At palpation in a abdominal cavity it is possible to reveal like sausage "tumor", mobile, slightly painful. At the rectal research it is possible to reveal dark blood with slime without stool mass.

The intestinal bleeding arises in connection with presence in doubles a mucous membrane of gastric type which secret causes formation peptic ulcers and erosion of blood vessels. The intestinal bleeding can be small and long time to remain unnoticed - At damage of a large vessel in stool a plenty of dark blood or brighter clots is found out at a low arrangement of a source of a bleeding. The anemia quickly develops.

Punching is caused by infringement of blood supply of a wall of doubling or occurs on a place peptic ulcers. Disease begins with strong pains in a stomach, vomiting, rises in temperature. Developing of a clinical picture of a peritonitis: a pressure of muscles of a forward abdominal wall, sharp morbidity at palpation, positive symptoms of irritation of the peritoneum. At radiological research free gas in a abdominal cavity comes to light. Complications doubling a digestive tract are subject to surgical treatment. The resection of doubling together most is frequently made with the basic intestinal tube with the subsequent restoration of a gleam of intestines anastomosis " the end in the end ".

In the prepubertal period at girls can arise painful abdominal syndrome caused by developmental anomalies of genitals. Most frequently meets haemocolpos - a congestion menstrual blood in a cavity of a vagina because of congenital absence of an aperture in virgin membrane. Disease is shown cyclic spastic by pains in the bottom of a stomach which repeat monthly. Pains are accompanied by weakness, a headache, is frequent rise in temperature, a nausea and vomiting. Despite of sufficient development of secondary sexual attributes, menstruations are absent - At survey comes to light full shutting hymen which is dome-shaped going out outside. Through it dark blood is appeared through and fluctuation is determined. At bimanual rectoabdominal research in the bottom of a stomach it is determined tumor like formation. By a similar clinical picture it can be shown full or partial aplasia of the vaginas. In diagnostics ultrasonic research during which it is possible to find out increased uteri, filled with a homogeneous liquid helps. At doubling a body of the uteri and vaginas from it malformation or aplasia are marked complaints on painful menstruations, and pains accrue with everyone menstruations, and in the bottom of a stomach is determined located asymmetrically above a bosom tumor like formation.

Diverticulum Of Mekkel located on the opposite side of the mesenterium in 20-100 sm. from illiocecal angle. More often diverticulum it is located freely in abdominal cavity, in some cases the top diverticulum is connected fibrous band with parietal peritoneum in the navel area. From within the gleam diverticulum is covered by a mucous membrane intestinal type, but it is sometimes marked heterotopy mucous of a stomach. Of Mekkel grows out incomplete return development of an intestinal part umbilical duct. Presence diverticulum Of Mekkel can result in development of the following complications: intestinal impassability, a bleeding, an inflammation. Strangulation loops of a around of fixed diverticulum result impassability in occurrence like attack pains in a stomach. Intensity of pains accrues with each attack, intervals between which decrease. There is a repeated vomiting. The condition of the child is progressively worsened. The stomach is asymmetric for the account increased loops of a thin intestinum, painful in the navel area, clearly comes to light peristalsis. A stool is not present, gases do not depart, On roentgenograms plural wide levels of a liquid in a abdominal cavity - invagination are visible, arisen on ground diverticulum Of Mekkel is characterized by the attributes specific to this kind of intestinal impassability. Among full health the child has attacks of pains in a stomach which intensity accrues, and the interval between attacks decreases. During

an attack of pains the child becomes restless, shouts, is sufficed by hands for a stomach spastic movements of the legs, there is a vomiting .At time between attacks the child calms down and behaves quite usually. The stool in the beginning normal, and in 4-6 hours after the beginning of disease is allocated dark blood with slime without stool mass. At palpation a stomach it is possible to find out smooth, mobile, slightly painful a tumor. At the rectal research the reduced tone sphincter, an empty ampoule of a direct , presence in a direct of dark blood with slime is determined. Sometimes diverticulum Of Mekkel it can be restrained in the inguenal hernia.

Presence in diverticulum a mucous membrane of gastric type can result in occurrence peptic ulcers and to a bleeding. The bleeding arises among full health and is characterized by allocation from a direct of a plenty of blood of be dark - cherry color . In time of significant bleeding pallor of integuments, a tachycardia, decrease the BP are determined. A stomach of the correct form, soft, without attributes of irritation of the peritoneum. To differentiate a bleeding from diverticulum Of Mekkel it is necessary with invagination, polyps of the intestines, abdominal form of disease Shonlein. Specification of the diagnosis probably at laparoscopic research. Detection diverticulum Of Mekkel is the indication to its resection.

Sharp diverticulum begins with strong pains in a stomach which are soon located in area navel or right iliac areas, vomiting, rise in temperature, a tachycardia Are marked. The child refuses meal, becomes languid. At palpation a stomach morbidity in area navel or right iliac areas, a pressure of muscles of a forward abdominal wall, positive symptom Shetkin-Blumberg is determined. Disease quickly progresses, in 10-15 hours after the beginning there is a punching diverticulum and the peritonitis develops. Differential diagnostics diverticulum and sharp of appendicitis is extremely complex, but it has no practical value. Both diseases require urgent operative treatment Used at sharp appendicitis access on Volkovich-Diakonov is convenient and for a resection diverticulum Of Mekkel.

The reason painful abdominal a syndrome can be the following congenital diseases illiocecal angle: insufficiency Bauhin′s valve, a mobile blind, membrane Of Jecson, sheaf of Lein. Bauhin′s valve interferes remove of the intestinal contents from a blind in thin. In some cases, in connection with incompleteness of formation of elements illiocecal angle. At revealing the given developmental anomaly it will be carried out hynoplastic on Vitebsk. Tractological mobility of a blind is caused by presence at a blind own of the mesentery and shown by nagging pains in right iliac areas which sometimes carry spastic character presence of a mobile blind can cause difficulties in diagnostics sharp of appendicitis. Sheaf of Lein, membrane of Jecson and others additional embrional soldering in area illiocecal a angle cause its deformation that can be shown by the painful syndrome amplifying at physical loading, At suspicion on a congenital tractology illiocecal a angle realization of radiological research with contrast substance or laparoscopy is shown.

The most widespread surgical disease at children's age - sharp appendicitis, With it is necessary to meet not only to children's surgeons, but also pediatrists, surgeons of the general structure, ambulance surgeons, and doctors of any other specialty. Sharp appendicitis at children - a source of often diagnostic mistakes, In one cases disease is diagnosed only after punching in a abdominal cavity, in others - for sharp appendicitis disease, apparently, not so similar to it is accepted. Diagnostic difficulties at sharp appendicitis at children are connected to features of reactance of a children's organism, relative immaturity CNS, often athipic an arrangement appendics, features of its) structure,

At histology distinguish the following forms sharp of appendicitis: cataral, phlegmonal and gangrenous at which there can be a punching . In typical cases sharp begins gradually. In the beginning there is a pain which within the first hours of disease is located in hypogastrium areas or near navel, and then moves in right iliac area. More often the pain has constant, whining character. Children become languid, refuse meal, restlessly sleep, frequently wake up. Sometimes the pain happens so intensive, that forces the child to accept the compelled position, the Often symptom sharp of appendicitis is vomiting at

the first o'clock of disease which has reflex character. For a typical picture sharp of appendicitis the temperature, increase of pulse and a delay of a stool are characteristic like subfebrile. At survey the form of a stomach is not changed, in initial stages of disease it participates in the act of breath. Superficial palpation a stomach begins with left iliac areas and it is made in a direction counter-clockwise. At transition to the right half of stomach it is necessary to watch reaction of the child, a look and to distract its attention conversation. At palpation a stomach the pressure of muscles of a forward abdominal wall in right iliac areas (the passive muscular pressure caused by inflammatory process in a abdominal cavity) comes to light. For its more precise revealing simultaneously it is necessary to carry out palpation a stomach from both sides. Thus at children it is very rare to be determined like a wall a stomach, the constant rigidity is marked only rather moderate, but. The second objective symptom of appendicitis - a local pain in right iliac areas. At children of advanced age the certain diagnostic value has symptom Shetkin-Blumberg. Value of other painful symptoms, characteristic for sharp of appendicitis at adults is rather doubtful at children. The data of laboratory researches at sharp appendicitis are not specific and can specify only presence of inflammatory process.

At the same time sharp appendicitis can proceed with a moderate painful syndrome or pains in the right hypogastrium, without vomiting and with normal temperature, with an often liquid stool and disuric the phenomena, without a muscular pressure. An athipic current sharp of appendicitis depends first of all on an arrangement appendics in abdominal cavity. At a retro cecal introperitoneal appendicitis the muscular pressure and a pain at palpation will be much less, than at a typical arrangement. At retroperitoneal an arrangement of the stomach will be soft on all extent, painless, symptom Shetkin-Blumberg will be negative. At the same time morbidity and a muscular pressure will be marked at palpation the right lumbar area, pains can irradiate in genitals or on a course of the ureter, that will cause disurical phenomena. In some cases the retro cecal proceeds from the very beginning with a heavy intoxication at feebly marked local symptoms. At pelvic an arrangement it is accompanied by pains in the bottom of a stomach, above pubis, the muscular pressure here is absent or feebly marked . Often pains irradiating in genitals, are marked disurical the phenomena, an often liquid stool with an impurity of blood and slime. Owing to transition of an inflammation to a bladder in analyses of urine leukocytes, erythrocytes, flat epithelium come to light. At underliver an arrangement appendics a of its inflammation begins with pains in the right hypogastrium, the muscular pressure and morbidity here is determined at palpation, morbidity is marked at hitting on the right costal arch. At a return arrangement of internal organs, a mobile blind or long appendics all clinical displays of appendicitis will be located in the left half of stomach. At gangrenous appendicitis the originality of a clinical picture is caused by defeat of the nervous device appendics, That is shown by painless in a stomach. The stomach participates in the act of breath, soft on all extent, insignificant morbidity is marked at deep palpation. Relative well-being is marked before development of a picture of a peritonitis. At such children it is possible to reveal a tachycardia which is not appropriate to a degree hypertermia, leukocytosis with shift leukocytes formulas to the left-

The clinical picture sharp of appendicitis at children till 3 years is characterized by the rough beginning. The child becomes restless, refuses meal, repeated vomiting and rise of temperature up to 38-40 degrees is marked, Frequently there is a liquid stool, urine often and painful. At palpation right iliac to area the child resists to survey, pushes away a hand of the surgeon, tightens the right leg, shout of the child considerably amplifies. It is desirable to examine children of younger age with suspicion on sharp during medicaments dream when the active muscular pressure disappears, and the passive pressure caused by an inflammation, it is kept.

At children with suspicion on sharp appendicitis make manual research of a direct . Thus it is

possible to reveal sharply painful edema of the walls of a direct, at late receipt of the child comes to light infiltrate. To state an objective estimation to a muscular pressure allows electromyography.

Research of a forward abdominal wall, laparoscopic research in doubtful cases practically correctly allows to confirm or reject destructive process in appendics. At absence sharp of appendicitis laparoscopy allows to reveal in 1/3 cases the true reason painful abdominal a syndrome.

At late diagnostics sharp of appendicitis it is necessary to meet its complications: a peritonitis and appendicular infiltrate. Punching appendics is shown by amplification of pains in a stomach and deterioration of a condition of the patient. There is a repeated vomiting, the temperature up to 39 - 40 degrees raises. Integuments become pale and dry, features are pointed. For a peritonitis the symptom of a divergence of a pulse rate and temperatures is characteristic. The stomach does not participate in the act of breath, at palpation sharp morbidity and a muscular pressure in all departments of a stomach is determined, it is more in right iliac than area. Symptom Shetkin-Blumberg also is positive in all departments. In process of increase of an intoxication the child becomes adynamic, languid, sleepy, intensity of pains in a stomach decreases. Laboratory researches confirm presence of heavy inflammatory process.

Appendicular infiltrate can be formed at children of advanced age for 3-5 day from the beginning of disease. At children of younger age of an opportunity of restriction of inflammatory process in a abdominal cavity are insufficient because of weak plastic properties of the peritoneum and malformation an omentum. At formation appendicular intensity of pains decreases, but the phenomena of an intoxication are kept, the high temperature, In the right half of stomach is determined dense, sharply painful, tumor like formation without precise borders. At atypical an arrangement of an appendix can be revealed in a small basin, the left half of stomach, the right lumbar area, are frequently marked disurical the phenomena and a liquid stool. In blood come to light leukocytosis, shift leucocytar formulas to the left, increased ESR.

Main principle of treatment sharp of appendicitis there is an early operative intervention, Children with the expressed phenomena of an intoxication, metabolic infringements require short-term and intensive preoperative preparation. The unique indication to conservative treatment is presence dense motionless infiltration. Treatment consists in purpose of antibiotics of a wide spectrum of action, physiotherapeutic procedures with anti-inflammatory action. At active treatment return development of the infiltrate - the Child who has transferred appendicular infiltrate is marked, in 1 month after end of treatment it is necessary to hospitalize for performance appendectomy, At a suppuration appendicular infiltrate it is necessary to make its opening, In the postoperative period therapy will be carried out active antibacterial, anti-inflammatory and desintoxicative therapy. In 2-3 months in the scheduled order is carried out appendectomy.

Diseases with which most frequently it is necessary to differentiate sharp appendicitis at children:

Intestinal acute pain, coprostasis. ARVI, a sharp bronchitis, a pneumonia, a pleurisy.

Intestinal infection (a salmonellosis, a dysentery), helminthes invasion.

Diseases of the bile secretion systems - a dyskinesia of the bile secretion ways, a sharp cholecystitis.

Diseases of the urinosecretion systems - a pyelonephritis, glomerulonephritis, a cystitis, urolithic disease.

Diseases of sexual sphere at girls.

Inflammation diverticulum of Mekkel,

Children's infections.

Rheumatism, colagenosis.

Abdominal the form of disease Shonlein

Diseases illiocecal angle - membrane of Jecson, soldering of Lein, a mobile blind .

Psychogenic pains in a stomach.

Mesadenitis - the inflammation of lymph nodes of the mesentery intestines - is caused by an infection more often, therefore frequency mesadenitis is increased during distribution of a sharp respiratory virus infection, Disease is shown by sharp pains in a stomach, a nausea, vomiting, rise in temperature up to 38-39 degrees. Are marked a moderate pressure of muscles of a forward abdominal wall and large morbidity. For mesadenitis symptom Shternberg (morbidity is characteristic at deep palpation on a course of an attachment of a root of the mesentery intestines). Sometimes it is possible to probe packages of the increased lymph nodes. At the expressed painful syndrome realization laparoscopy which allows not only to inspect a abdominal cavity is shown but also to make puncsion biopsy of the lymph node. If it is found out during operation also it is necessary to make biopsy a lymph node of the mesentery. Treatment mesadenitis consists in purpose of antibiotics of a wide spectrum of action, multi-vitamins.

From gynecological diseases painful abdominal the syndrome can be marked at inflammatory diseases of appendages uterine, apoplexy of the ovarian. At acute salpingitis or ooforitis morbidity in the bottom part of a stomach is marked, the temperature is more on the right, sometimes vague attributes of irritation of the peritoneum, like subfebrile, leukocytosis. At the rectal research morbidity of appendages comes to light. Disease basically meets at girls of 10-14 years, more often on the right. Infringement of integrity ovarian with a hemorrhage in a abdominal cavity is marked in the period ovulation when owing to increase of a level of hormones ovaries it is observed hyperemia, vasodilatation and increase of permeability of a wall of vessels ovarian more often, Disease begins sharply, among full well-being with strong pains in the bottom of a stomach which are accompanied by dizziness, a nausea, vomiting. Usually in abdominal cavity it is given vent 20-100 ml of blood that is not accompanied haemodinamic by infringements. At more significant of the bleeding increase of pulse, decrease the BP, falling of hemoglobin is marked, At the rectal research comes to light painful , a pain at displacement uterine operative treatment is shown at the big bleeding and consists in rhaphing break. If the bleeding small also has stopped independently conservative therapy will be carried out: a confinement to bed, a cold on a bottom of a stomach.

The initial peritonitis, develops at girls in the age of 5-10 years more often and is connected to

penetration of an infection into a abdominal cavity through a vagina. Clinically distinguish two forms of disease - simple and toxic, the Toxic form begins sharply with strong pains in the bottom of a stomach, repeated vomiting, rise in temperature up to 39 degrees are frequently marked mucopurulent allocation from a vagina, high leukocytosis, In some hours after the beginning of disease the general condition of the child is estimated as heavy. At inspection of a stomach the muscular pressure and morbidity in all departments comes to light, but it is more on the right below, positive symptoms of irritation of the peritoneum. To thicket there is a simple form of an initial peritonitis at which the intoxication is not expressed, temperature like subfebrile, moderate pains in the bottom of a stomach are marked or only in right iliac areas Application laparoscopy in urgency surgeries of children's age allows with high to reveal accuracy the reason painful abdominal a syndrome. At sharp salpingitis uterine tubes with edema, vessels also have dim color, it is sometimes marked hyperemia vessels parietal of the peritoneum a small basin. Patients with inflammatory changes of appendages uterine should receive a rate antibacterial therapy. Laparoscopic attributes of the pelvioperitonitis are detection of a plenty transparent or muddy exudate in a cavity of a small basin, sticky, therefore lasts behind the manipulator, it is marked hyperemia and puffiness uterine and its appendages. The greatest inflammatory changes are found out in ampular of parts tubes of the uteri . At the toxic form of the pelvioperitonitis also gets purulent character. Treatment of the pelvioperitonitis consists in aspiration exudate from a abdominal cavity, introduction of a solution antiseptics. In the postoperative period therapy will be carried out antibacterial and desintoxicative.

Increased synthesis of the prostaglandin’s before the menstruation causes spastic reductions uterine. For 1-2 days prior to the beginning menstruations the girl complains on spasmodic pains in the bottom of a stomach which can be accompanied by a nausea, vomiting, dizziness. Similar complaints sometimes force to think about sharp appendicitis. But correctly collected anamnesis, absence an attribute of irritation of the peritoneum allow to put the true diagnosis and to appoint necessary treatment: antispastic means and preparations, inhibited synthesis of the prostaglandin’s.

Courvoisier′s gallbladder at children is the big rarity. Usually sharp cholecystitis begins strong pains in the right hypogastrium or epigastrium. Pains frequently carry spastic character and last from several minutes till several o'clock. At presence of an obstacle to outflow of the belie, and it more often a mucopurulent clot, pains become very intensive and force the child to rush in bed or to accept the compelled position in which the child remains for a long time. Attacks are usually accompanied by the repeated vomiting which is not bringing simplification, increase of breath and pulse, rise in temperature up to 38-39 degrees, a swelling of a stomach, a delay of a stool. Integuments pale, are covered sticky then, approximately in half of cases there is a jaundice. At palpation a stomach sharp morbidity and a muscular pressure in the right hypogastrium, positive symptoms Ortner′s, Merfy comes to light. The liver is increased and painful. Treatment of a sharp cholecystitis begins with conservative actions: a confinement to bed, antibacterial and desintoxicative therapy, are appointed analgetic, spasmolitic and antifermental preparations. Absence of effect from conservative treatment is the indication to emergency operation. If the phlegmon or a gangrene of a bilious bubble it is shown cholecistectomy is revealed. If to make removal of a bubble it is impossible because of a heavy condition of the patient or presence expressed infiltration it is necessary to execute cholecistectomy.

Much more often than an inflammation, there are a dyskinesia of bilious ways, diets connected to infringement, neurosis’s, helminthes invasion, disbacteriosis, a gastritis, duodenitis .abnormality of the development of bilious ways,

Sharp pancreatitis disease. Described by a hypostasis, an inflammation and necrosis a pancreas, meets at children sufficient seldom. The reasons of it can be duodeno-pancreatic reflux, the dyskinesia of a duodenal, a trauma including operational, stressful situations, infectious diseases, proceeding with a allergic component, In a basis pathogenesis a pancreatitis lays autolisis fabrics of a pancreas as a result of emission in blood of a plenty tripsin, elastase and others proteolitic enzymes. The first stage of a sharp pancreatitis - a hypostasis of a pancreas, begins with the general nonspecific symptoms: weakness, refusal of meal, uncertain complaints in a stomach. In some hours of a pain are located in hypogastrium areas, have surrounding character, irradiating in accompanied by repeated vomiting. The child can borrow the compelled position, is more often on left to a side. The stomach is not inflated, participates in the act of breath, the muscular pressure is not present. Similar discrepancy between the expressed complaints to painful sensations in a stomach and absence of objective attributes of inflammatory process in a abdominal cavity is typical of a hypostasis of a pancreas. The most informative laboratory parameter is definition of the raised level amylase in blood and urine. To diagnostics ultrasonic research is useful. At progressing disease develop fatty and haemoragic pancreonecrosis. Children of younger age in the beginning are restless, shout from a pain, borrow the compelled position, then the impellent anxiety that caused. Children specify strong pains in the top departments of a stomach which frequently have surrounding character. Despite of the thirst caused by repeated vomiting, the child refuses drink as each drink of water causes repeated vomiting. Integuments pale, with cyanotic shade. Language dry, is impose. Pulse become more frequent, the BP it is reduced. The temperature usually like subfebrile, its rise up to 38 ~ 39 degrees testifies to development fatty necrosis .The state of the child is progressively worsened, quickly develop exicosis, a toxicosis. At survey the inflated stomach, the expressed pressure of muscles of a forward abdominal wall, positive symptoms of irritation of the peritoneum comes to light. In blood it is determined high leukocytosis, infringements. The level amylase in blood and urine is raised. Its sharp falling at deterioration of a condition of the patient is an attribute total necrosis of the glandular, the most informative method of diagnostics of a sharp pancreatitis is laparoscopic research during which find out the centers fatty necrosis as spots of wight-yellow color on parietal and visceral peritoneum, in an omentum, haemoragic exudate, haemoragic imbybition an omentum and retroperetoneum spaces, In exudate a abdominal cavity the raised level amylase is determined, Difficulties at differential diagnostics first of all are connected by that the sharp pancreatitis is rare disease at children, and the doctor first of all thinks of diseases more typical for children's age . Against sharp of appendicitis will speak acute pain in the top departments of a stomach, repeated vomiting and fast deterioration of a condition of the child, Are absent the most typical for sharp of appendicitis symptoms local morbidity and a pressure of muscles of a forward abdominal wall in right iliac areas. Laboratory researches allow to specify the diagnosis. Principles of conservative treatment of a sharp pancreatitis are: maintenance of rest of a pancreas for what for 3-4 days it is cancelled enteral a feed; struggle against a painful syndrome, the most effective method is prolonged epydural anesthesia; antifermental therapy, antibacterial therapy; liquidation of infringements; desintocsisation therapy. To surgical treatment resort at absence of effect from conservative therapy, at progressing disease. In the postoperative period active medicamentous therapy continues to be carried out.

Disease the Crone is nonspecific granulomatosis inflammatory process which can amaze any department of a digestive tract. Most frequently, approximately in three quarters of cases, disease amazes a terminal department iliac. Exact ethiology diseases the Crone is unknown, though now the majority of authors carry this disease to colagenosis. Chronic granulomatosis the inflammation amazes all layers of an intestinal wall with formation of narrow deep longitudinal ulcers on a mucous membrane, appear granulome, are increased mesenterial lymph nodes. Clinically disease is characterized spastic by pains in a stomach, repeated vomiting, rise in temperature up to 38 degrees. Sometimes the painful syndrome is so expressed, that children operate, suspecting sharp appendicitis. During operation find out hyperemia,

infiltration and a thickening of a wall of a terminal department iliac s are sometimes amazed blind and an initial department colon. Local lymph nodes are considerably increased in the sizes. Sometimes disease accepts character and is shown by periodic complaints to pains in a stomach, the weakness, the reduced appetite, dyspeptic frustration, loosing of the weight the child. At children complications of disease the Crone seldom develop: punching, stenoses of intestines, a bleeding, formation of fistulas. In diagnostics help radiological and endoscopic researches of thick intestines. Treatment of disease the Crone at children conservative also consists in purpose of a diet with the low contents of fats and the high contents of fibers and carbohydrates, vitamins, antihistamine and sedative preparations shown antibacterial therapy are applied. Surgical treatment is shown at development of complications.

Nonspecific ulcer colitis represents inflammatory defeat of all or a segment of a thick in which basis lay autoimunal infringements. On a mucous membrane of thick intestines erosion and ulcers on which place in a consequence expands a fabric are formed and there is a scarring. Disease usually begins with moderate pains in the left half of stomach, occurrence of blood in stool mass. The stool depending on weight of disease departs from 3 up to 10 times per day, rise of temperature is marked, quickly develop an anemia, disproteinemia, disbacteriosis. At endoscopic research of thick intestines come to light hyperemia, puffiness and bleeding a mucous membrane on which surface erosion and ulcers are formed. As against disease the Crone of a ulcer have a cross direction, are not distributed more deeply a mucous layer, the direct, Treatment basically conservative is frequently amazed: dietotherapy (a plenty high-grade), preparations of group salasopirine (course of treatment of 4-8 months), in heavy cases - desintaxicative and steroid therapy. Surgical treatment is shown at development of complications - punching of ulcers, profuse a bleeding, toxic dilatation a thick , lightning current of disease.

Traumatic damages of organs of a abdominal cavity meet more often at boys in the age of 7-14 years and are subdivided on closed and open which in turn can be penetrating and not penetrating. At children damages of a spleen most are frequently observed. Distinguish the following kinds of damages of a spleen:

Superficial anguishes of a capsule. Subcapsular hematomas. Breaks of a capsule and parenchyma lease spleens, leisure of spleens from a vascular leg, the Basic and constant attribute in one time break of a spleen is the constant whining pain in left hypogastrium and hypogastrium areas that coincides with a place of a bruise. Children of younger age precisely can not locate a pain and more often complain on pains like the lake, Children of advanced age start to complain on pains a bit later after a trauma that is connected to distribution of blood on a abdominal cavity. However the greatest intensity of pains is kept in a projection of a spleen. The pain, as a rule, amplifies at a deep breath and irradiate in left scapula. Crises of edges at damages of a spleen at children practically do not meet. Much typical display of an internal bleeding at children pallor of a skin and mucous membranes are. Thus pulse and the BP can keep quite satisfactory properties within several hours. Sometimes children borrow the compelled position.

Attempt to change results position in amplification of a pain that forces the child to return to a former pose. In time of the survey of a stomach is marked backlog of its left half in the act of breath, grazes in a projection of a spleen are sometimes visible. Morbidity and a muscular pressure are located in left

hypogastrium, less often distributed on all stomach. In some cases discrepancy of sharp morbidity in a stomach is observed and insignificant pressure of muscles - symptom Culencampha. In a abdominal cavity already in the near future after a trauma it is possible to determine a free liquid. At the rectal research it is possible to determine a congestion of blood in a small basin - In connection with that in a abdominal cavity basically blood which is deposited in a spleen is given vent, at the first o'clock after a trauma parameters of red blood change a little bit - growth of number of leukocytes is more characteristic for damages of a spleen, is especial at the first 6 o'clock after a trauma. In diagnostics of damages of a spleen help radiological, ultrasonic research, laparocentesis or microlaporotomy with application of a technique fumbling, laparoscopy. Thus the condition of the child is worsened and develops a picture of a bleeding in a abdominal cavity.

Conservative treatment of damages of a spleen probably only at exception introabdominal bleedings and revealing of precise regress of symptoms of damage. Conservative treatment consists in a strict confinement to bed within 8-12 days, application of a cold on area of a trauma, purpose haestatic therapies. At a bleeding in a abdominal cavity operative intervention which technique is defined by a damage rate of a spleen, but, whenever possible, is shown is necessary to aspire to carry out operation.

Damages of a liver are consequence of a trauma though "spontaneous" breaks can be observed at tumors more often and cystic the liver, the lesion liver can divide into three groups:

Inracapsular hematomas, Breaks of a liver with damage of a capsule.

The central breaks of a liver at which inside of the parenchyma the cavity filled with blood and bile is formed.

In most cases the condition of the child after a trauma is regarded as heavy because of development of a shock. The basic symptom of damage of a liver is the constant pain, is more often in the right hypogastrium or the right half of stomach. Localization of damage influences distribution of pains. At damage of a dome of a liver the pain is focused in the bottom departments of the right half of chest cell; at damage of a back surface of a liver or its separation from a coronal sheaf the pain is distributed in lumbar area; at damage of the bottom surface of a liver the pain is defined closer to navel. The pain can irradiate in right to the scapula, at distribution of blood on the right lateral channel, morbidity will be defined in right iliac areas, above a bosom. Frequently after a trauma there is a reflex vomiting. Vomiting of color of a coffee grounds - consequence (break of the central hematomas in bilious courses) . At the majority of victims the pressure(voltage) of muscles of a forward abdominal wall, positive symptom Shetkin-Blumberg, a free liquid in a abdominal cavity is defined, a swelling of intestines . Symptom "navel" - sharp morbidity at pressing on navel, arising owing to a tension of a round sheaf of a liver later. The anemia rather quickly develops, appears leukocytosis. In doubtful cases the same methods are applied to diagnostics, as at a trauma of a spleen. The diagnosis of break of a liver with a bleeding in a abdominal cavity serves as the indication for urgent operative. Interventions.

Damages of a pancreas at children meet seldom enough. more often they are marked at combination to a trauma of organs of a abdominal cavity and retroperitoneal spaces. Damages of a pancreas result from the forced impact in epigastral area (impact is most typical for children about a rudder of a bicycle at falling) or as a result of long of the crushing the top half of stomach. Right after traumas at children appear an acute pains in epigastral areas, the collapse and a shock develop. Pains can irradiate in lumbar area, in left costo-vertebral angle, at significant damages of a pain have surrounding character. There is a vomiting which at

development of a traumatic pancreatitis becomes unrestrained. Pulse often. Weak filling, the BP in most cases does not change. At survey of a stomach it is possible to find out traces of a trauma in epigastral areas, the stomach lags behind in the act of breath, is marked morbidity above navel and at the left, a moderate pressure of muscles of the stomach, especially expressed in epigastrium. Symptom Shetkin-Blumberg poorly positive. At dynamic supervision amplification of attributes of irritation of the peritoneum is marked. Then suddenly a condition of the child there are strongest pains in a stomach, unrestrained vomiting, develops clinic of a shock. In diagnostics of damages of a pancreas repeated definitions of a level amylase in blood and urine have the big value. At the first o'clock after a trauma there can be a decrease of a level amylase, that is connected to an angiospasm of a gland. Then than more time passes from the moment of a trauma, increase of activity of enzyme especially is expressed. Activity of thelipase, also raises only for 2-3 day after a trauma. At suspicion on a bruise of a pancreas the child is translated on parental feed, the strict confinement to bed and a rate of the therapy directed on the prevention of development of a pancreatitis are appointed, each 4-6 hours the level amylase in blood and urine is defined. At absence of effect from conservative therapy within day realization of operative treatment is shown

The clinical picture of penetrating wound of a stomach is characterized by a heavy shock and fast development of a peritonitis. There are the sharp pains especially expressed in epigastral of area, often desires on vomiting, emetic weights thus poor, can contain an impurity of blood. The stomach does not participate in the act of breath, the muscular pressure and positive symptom Shetkin-Blumberg are defined, at percussion disappearance of borders of hepatic dullness. XR in a abdominal cavity free gas is quite often defined comes to light. Bruises of a wall of a stomach can be accompanied by formation subserosis or submucouse hematomas, anguishes of a mucous or serous-muscular environment of a stomach. At these damages the condition of the child after a trauma can be heavy, the shock can be developed, however antishock therapy results a condition of the patient in improvement. At damage of a seros-muscular layer attributes introperitoneal bleedings can be developed. At penetrating breaks of a stomach it is shown urgent laparotomy.

Damages of intestines are subdivided into the bruises accompanying with formation of hematomas in a wall of a or anguishes of a wall; full breaks of a wall of a and from of the mesentery. At nestling close breaks of a wall of a the picture of a peritonitis. Child develops complains of pains in a stomach which are almost always accompanied by vomiting. The general condition of the child heavy. The child is pale, adynamic, features are pointed, pulse often, weak filling, the body temperature raises. At palpation a stomach sharp morbidity above a place of damage is defined, with development of a peritonitis the pain is distributed on all stomach. Come to light разлитое a muscular pressure and positive symptom Shetkin - Blumberg - XR free gas in a abdominal cavity is determined. However absence of it an attribute does not allow to exclude damage visceral organ. In some cases punching of intestines can be covered or the small sizes that conducts to slower development of a peritonitis. Bruises of a wall of a can proceed without symptoms, however presence of hematomas in a wall of a can result to necrosis of the walls, punching and development of a peritonitis.

At a separation from of the mesentery the picture introperitoneal bleedings develops.

Features of clinical current are marked at damage retroperitoneal parts of a duodenal. The first hours after a trauma by the only thing of display of damage there can be only complaints to moderate pains in epigastral areas. However in due course intensity of pains accrues, they are located a little bit above and more to the right navel. Deterioration of a condition of the child, occurrence of vomiting, rise in temperature up to 38 degrees, increase of the phenomena of an intoxication is connected to formation extraperitoneal phlegmon .B epigastral areas the muscular pressure and sharp morbidity is determined at palpation - Pains can be determined and in lumbar area on the right.

At damages of intestines at the first o'clock it is marked moderate leukocytosis which with development of a peritonitis accrues. The big value has radiological research. At a not clear picture of a trauma of a stomach the technique " fumbling cateter " and laparoscopy is applied. At suspicion on a trauma retroperitoneal realization RX-scopy with water-soluble contrast substance is shown a part of a duodenal - at penetrating breaks the contrast substance will fall outside the limits contours of a duodenal.

The established diagnosis of damage of intestines is the indication to urgent operative treatment after realization of short-term antishock therapy.

8. Independent work of higher education applicants:

Themes for self study:

Doubling of a stomach

Strangulation a stomach

Diseases of an omentum - cyster, Strangulation, omentitis

Trauma of organs of abdominal cavity at newborn

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7. Age aspects of acute appendicitis in children and the key to its recognition. Tutorial. 2019. 260 p. Edited by Bodnar B.M., Ribalchenko V.F., Bodnar O.B., Melnichenko M.H., and others. Publishing. ISBN 978-966-697-828-1

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13. Bachurin V.I. Malformations of the genitourinary system as a cause of urological pathologies: teaching methodology for independent work of fourth-year higher education applicants in the specialty: 7.12010001 "Medical business", 7.12010002 "Pediatrics", 6.120102 "Laboratory diagnostics" / Bachurin V.I.. - Zaporizhzhia: ZSMU, 2017. - 86 p.

Topic 3a. Peculiarities of traumatology in children.

Topic: Peculiarities of traumatology in children.

I. Topic actuality, a professional orientation.

Children are very active part of citizen and take different trauma comparatively often. Wrong treatment of fractures may lead to the different complications, especially in small children and bring to development of acquired malformations. Peculiarities of fractures in children depend on the anatomo-physiological features of the bones. Still rate of mortality in cases of polytrauma keeps very high. Also trauma may result in social disadaptation.

II. The educational purposes of the study.

a) ***As a result of the study higher education applicants should know theoretically:***

1. Anatomo-physiological features of the bones in children.

2. Peculiarities of fractures in children.

3. Structure of children's traumatism.

4. Correlation of causes of injures with the age of the child.

5. Correlation of character of injures with morpho-functional peculiarities.

6. Correlation of manifestation and course of injures with morpho-functional peculiarities of children.

7. Clinical manifestation of fractures.

8. General principles of treatment of bone fractures in children.

9. Methods of treatment of bone fractures.

10. Peculiarities of fractures in children depend on the anatomo-physiological features of the bones.

11. Means of acceptable and unacceptable dislocation.

b) Higher education applicants must know to do practically

1. To issue the case record.
2. To carry out clinical researches.
3. To take the anamnesis.
4. To interpret the X-ray film.
5. To make immobilization of extremity.

6. To make a plan of treatment.

3. Questions for training.

1. Newborn of 13 days of life is carried out to the surgical department with suspicion on fracture of clavicle. What method of treatment is indicated?

2. The 13-year-old boy has an oblique fracture of both bones of right calf. What method of treatment is indicated in this case?

3. The 6-years boy has an osteoepiphyseolisis of the distal epiphysis of radius with dislocations by width, by length and angular. What complication is possible in such fracture?

4. The plan and organisation of lessons.

		Activity		
		Higher education applicants	Teacher	
Organisation of the practical class	5	Listen	Presence control	Journal
Aim setting	5	Listen	Explain	
Checking of the knowledge of higher education applicants	15	Make	Control	Tests
Seminar	60	Answer	Control	X-ray-grams
Clinical discussion	30	Listen	Discuss patients	Patients
Personal work of higher education applicants	60	Make	Control	Patients
Results of the lesson	10	Listen	Appreciation of each activities	
Task for the next lesson	5	Listen	Explain the task. Indicates the literature.	

5. Substance of the lesson.

1. Structure of children's traumatism: school (identical with industrial traumatism in adults), sport, street, domestic, traumatism of birth.

Traumatism of birth happens during deliveries. In structure of children's traumatism of birth the first place by frequency is taking by craniocerebral trauma; the second place is taking by fractures of

extremities, 505 of which relate to fracture of clavicle; at the third place there are injures of the peripheral nervous system (paresis, paralysis); than the spinal trauma and the injures of internal organs.

2. Correlation of causes of injures with the age of the child.

In newborns such injures may happen that never happen in older children and adults (cefalohematoma, swelling of birth). In children of age under 3, domestic trauma prevails burns are the most frequent. In preschoolers domestic trauma also prevails, but it is mostly due to fallings. In children of school age domestic and street traumas have an equal frequency.

3. Correlation of character of injures with morpho-functional peculiarities.

There are present fractures at growth zone (epiphyseolisis, apophyseolisis, osteoepiphyseolisis). Fractures in forearm shafts are typical and they can be compared with a cracked pussy-willow branch (angulation of fragments with partial integrity of bone in a zone of compression).

4. Correlation of manifestation and course of injures with morpho-functional peculiarities of children.

Due to abundant vascularization of the bones, injures are always accompanied with significant edema and haematoma; due to immaturity of the central nerves system, receptory apparatus in children traumatic shock is often observed.

5. Priority of sparing methods in diagnosis and treatment.

Such symptoms as crepitation and pathologic mobility are never revealed intentionally in children. Conservative methods of treatment are always preferred in other equal conditions; in case of operation, less traumatic methods are used.

6. Appearance of acquired defects of development due to injury of immature structures. Only in children affection of growth zone can lead to infringement in their function, appearance of deformations and shortening of extremities and invalidisation.

Bone fractures.

Peculiarities of fractures in children depend on the anatomo-physiological features of the bones. In children there is high specific gravity of organic component of bones. The younger the child, the less mineral substances is contained in his bone tissue, the more manifestative the elastic features of the skeleton. In a baby epiphysis is mostly cartilage tissue that performs amortization function. Periosteum of a child is thick elastic its circulation is abundant. That conditions appearance of subperiostal fractures and their fast healing. Good vascularisation of the skeleton leads to significant edema and haematoma and at the same time to fast consolidation. Due to elasticity of ligaments in children dislocations and traumas occur rarely, presence of growth zones explains frequency of fractures that cross through physal plates.

The following peculiarities of fractures in children can be underlined:

1. Fractures occur rare if to compare with frequency of traumatic factors action and injuries frequency.

2. Traumatic dislocations occur rarely;

3. Fragmental fractures occur rarely;
4. Often fractures cross through growth zones: epiphyseolisis (fragmentation of epiphysis from metaphysis), apophyseolisis (fragmentation of apophysis), osteoepiphyseolisis (fragmentation of epiphysis with a part of metaphysis);
5. Subperiostal fractures are often found;
6. Not rarely fractures resemble a broken branch of palm;
7. Fractures are accompanied with significant edema and haematoma.

Clinical manifestation of fractures.

Signs of fractures are divided into possible and reliable.

Deformation, local pain and infringement of function refer to *possible clinical symptoms*. Degree of deformation depends upon presence or absence of dislocation of fragments, thickness of sheath of soft tissues around affected segment, sizes of accompanying edema and haematoma. The leading symptoms are pain and dysfunction. Pain may be independent and provoked. Percussion, palpation, reveals provoked pain as well as using subsidiary means.

The reliable signs are pathologic mobility and crepitation of fragments, but in children they are not determined intentionally because it can additionally injure the child and make dislocation worse. Above-mentioned signs nay be determined spontaneously during transport immobilization, patient's transfer so on.

The main mean of subsidiary diagnosis is X-ray investigation that allows not only to reveal fracture, but also to determine its character and type of dislocations. X-ray picture must be made in 2 positions, including 2 opposite joints. When it is difficult to interpret the data there can be performed X-ray investigation of counter-lateral extremity that helps to diagnose the disease by the way of comparison.

The process of **reparation of fracture** pass through the following stages:

I stage – *fibrous callus* – is characterized by organization of blood that have been stored in the fractured zone. On 4-5th day of the trauma, blood turns into the blood clot, fibroblasts appear there and during first two weeks in the fractured zone fibroreticular tissue that surrounds fragments forms. Fibroblasts are osteogenous cell-predecessors. Their source is endosteum, periosteum, and endothelium of vessels. Fibrous callus is deprived of mineral components and doesn't have mechanic density and is not contrast in X-rays.

II stage – *soft bone callus* - develops passing the cartilage callus in case of not affected osteogenesis in adaptation of fragments and reliable fixation. Fibroblasts that relate to melanocytes proliferate while producing collagen, activity of alkaline phosphatase increases in them and deposition of calcium occurs, i.e. parallel – fibrous tissue appears, as well as mineralization. Primary bone structures are formed. Soft bone callus is contrast in X-rays, it has mechanic solidity that is enough for keeping fragments at their place, but it can be easily deformed due to load.

III stage – *maturation of bone structures* – last 2,4-4 month and is characterized by formation of plate bone tissue, reconstruction of regenerate and structural adaptation.

General principles of **treatment** of bone fractures in children are following:

1 Sparing treatment;

2 Individual approach;

3 Timely reposition of fragments;

4 Reliable fixation;

5 Early functional treatment.

Methods of treatment of bone fractures can be conservative and operative

According to means of reposition and fixation they are divided into immobilizational (full immobility of the injured segment during the entire period of fixation); functional (uniting of principles of rest and movement – immobility of the injured segment while saving the joints function) and combined (uniting of functional and immobilizational methods).

Choice of treatment mode depends on the age of the patient, type of fracture and dislocation of fragments, localization of injury, time since trauma and state of the child.

In children the conservative method of treatment is prior when it supposes single-moment closed reposition (when necessary) and fixation of injured segment with plaster bandage. Reposition of fragments is not conducted when there is fracture without dislocation, when dislocation is insignificant (subperiostal fracture) and when dislocation is acceptable.

An acceptable dislocation supposes such dislocation of fragments that can be neglected because in the process of growth bones become longer and thicker and conditions for spontaneous correction appear. The younger the child the more possibilities are for leveling the deformation.

Due to that acceptable dislocation of fragments in fractures of diaphysis in newborns is complete dislocation according to width; dislocation not over 1-2 cm or less according to length; angular dislocation at an angle of 30^0 or less.

In children of first year of life complete dislocation according to width and 1 cm or less according to length is acceptable. Angular dislocation is unacceptable.

In children of the older age group in fractures of metaphysis and diaphysis when angular and rotational dislocations are absent, almost complete dislocation according to width is acceptable, and state of the fragments is estimated as following: excellent – dislocation according to width by 1/3; good - dislocation according to width by S; satisfactory - dislocation according to width by 2/3.

In all noted cases reposition is not conducted, only fixation and rehabilitation therapy is performed.

Reposition is indicated in unacceptable dislocations of fractures. These are angular and rotational

dislocations, fractures along the growth zone even with small dislocations in children of all age groups because they may lead to furder deformation and shortening.

Single-moment closed reposition is performed in cases when it can be followed by effective immobilization. It is used mainly in epiphyseolisis, transverse meta- and diaphysary fractures of long bones, in para- and intraarticular fractures and injures of small bones. Reposition is performed mainly by hand with the use of narcosis. In single-moment closed hand reposition in case of affection of large segment with significant dislocation according to length sometimes traction apparatuses are used. These are mainly in children of older age.

During closed reposition three Beller's rules are used:

1. Confrontation of the distal fragment to the central one;
2. During traction there should be counter-traction;
3. Exertion should be directed along the fragment's axis.

Plaster splints and bandages are used as major immobilisational means. They provide reliable fixation only when the injured joint is immobilized together with two approximate joints. It should be kept in mind that in children due to morpho-functional peculiarities there is significant edema of soft tissues and due to that plaster immobilization should be only in type of splints that occupy no more that 2/3 of diameter of extremity or in type of plaster bandages with incision along its entire length, that prevents compressing of soft tissues.

In newborns in birth traumas plaster immobilization is not used because the skin is easily damaged, its intertrigo and maceration appear, inflammation can accompany it. In such children extremities are fixed with soft bandages or cardboard splints are used.

The ***functional*** method of treatment includes functional reposition of fragments ("reposition in time") and fixation of injured segment by means of constant traction. Traction can be skeletal, soft (adhesive plaster, cleol), traction by "English swing" (Glisson's loop in fractures of vertebral column) or by plaster "high-boot".

The soft traction is conducted with the soft tissues, the skeletal traction – directly through the bone; the force of the first one is no more than 3 kilos, the second one can bare higher load. That's why soft traction is used in children of 2-3 years old, and skeletal one is used in children after 3 years old in oblique fracture of the hip, shoulder, bones of cnemis, transverse fractures of the hip and shoulder. During functional treatment following rules should be kept:

1. The extremity should be placed into average physiologic state;
2. It is necessary to provide rest of the extremity;
3. Fragments should be confronted;
4. The load should be gradual;
5. It is necessary to provide counter-traction (more often it's a body of the patient himself).

Combined method is indicated in fractures of the hip, shoulder, bones of cnemis and consist of the traction at first and immobilization after formation of bone callus. This method of treatment we use because some negative aspects of skeletal traction: bed regimen, long time of staying in the hospital and disorder of the blood circulation. In case of combined treatment the patient by the end of the total term of fixation is discharged from the hospital. Total term of fixation depends on the age of the patient and localization of injury.

Rehabilitation measures are started right after ending immobilization (MPE, massage, physiotherapy, sanatorium treatment).

Operative treatment is rarely conducted (3-4% of cases). It is performed when conservative methods are ineffective (unsatisfactory location of fragments), and in case of old and incorrectly consolidated fractures.

Open reposition is conducted according to morpho-functional peculiarities of infant age with the use of sparing methods: Kirshner's wire is the most often used as means of fixation, Bogdanov's rods are used in fracture of the hip; bone osteosynthesis with metal plates is not used. In case of combined injures prevalence is given to external fixation with compression-distraction wire apparatuses according to Illizarov that allow not only reposition and reliable fixation of the injured segment, but also provide optimal conditions of treatment of wounds in condition of patient mobility.

Tests for control.

1. Oedema and pain was revealed at a palpation of the right clavicle labour in newborn after delivery. The upper right extremity is immobile. What pathology can be suspected?

+A. Fracture of clavicle

B. Metaepiphyseal osteomyelitis

C. Paralysis Duchenne-Erb

D. Right-side torticolis

E. Children's cerebral paralysis

2. A newborn has got a fracture of the left femur in a mean third during labour. Objectively: there are an oedema of soft tissues, deformation and linear shortness of a femur. What medical tactics would you choose?

A. Plaster extension on Shede

B. Plaster bandage

C. Skeletal traction

+D. Bandage on Krede

E. Opened reposition

3. A child, 3 years old, was transported to traumatology with the suggested diagnosis "Subluxation of the head of radius". What from following signs is most typical for this trauma?

A. The child holds an ill arm by a healthy arm

+B. The arm is pronationated, is prolated along a trunk, is slightly bent in elbow

C. There are motions in elbow

D. Deformation of elbow

E. There is tumescence in the field of elbow

4. A child, 8 years old, was delivered in traumatology department with the diagnosis: closed oblique fracture of bones of the right calf in middle part without displacement. What tactics of treatment of the given fracture should you prescribe?

A. Opened reposition

B. Plaster bandage

C. Ilizarov's apparatus

D. Plaster extension

+E. Skeletal traction

5. A boy, 10 years old, after a dip has felt a sever pain in the region of a hip, where the tumescence, deformation are determined, and he can't stand on a leg. What from following signs concerns to authentic signs of fracture?

A. Tumescence

B. Deformation

+C. Crepitation parts of fracture

D. Pain in palpation

E. Disorders of function

6. A boy, 9-years old admitted to traumapunkt with the complaints of a pain, absence of movements in left shoulder joint. When he was rolling, he felt on his left hand. Objectively: there is tumescence of upper third of the left shoulder, painfulness, and deformation. On the X-ray is epiphysiolyisis of the head of the left shoulder bone with displacement. What kind of treatment is administered at this pathology?

A. Closed reposition, bandage Dezo

B. Opened reposition

+C. Closed reposition, bandage by Gromov

D. Skeletal traction

E. Plaster extension

7. In children's traumatology there is a lot of fractures caused by anatomical-physiological features of a skeleton. You should point, after what type of fractures there can appear a shortness of extremity, as complication?

A. Subperiostal fracture

B. Fracture by the type of " green tree "

+C. Epiphysiolyisis, osteoepiphysiolyisis

D. Fracture with many parts

E. Apophysiolyisis

8. The pain, tumescence, deformation in low third of the left forearm has appeared in a child, 10 years old, after dip on an arm. Fracture of a radial bone in low third with displacement lengthways, width and bevel way was revealed in X-ray examination. What type of displacement of breaking off is it possible to consider permissible in this case?

A. Displacement lengthways up to 1 sм

B. Displacement bevel way up to 10°

C. Displacement bevel way more than 10°

+D. Displacement by width (on 1/3 diameters)

E. Displacement by width in all diameter

9. A child was in the accident with a motor vehicle. He complains of pains in the down part of abdomen, vast hematoma in the region of a symphysis. Fracture horizontal branches of a pubic bone was revealed in X-ray examination. What clinical sign is specific for this type of fracture?

A. Limitation of flexion in p/f joints

B. Verneil's Sign

C. Outside rotation of limb

+D. Sign of "adhered heel"

E. Limitation of driving back in hip joint

10. A child is on treatment in traumatology department with diagnosis: a gap of a symphysis. You should determine the medical tactics at a gap of a symphysis with a divergence:

A. Imposing of a skeletal traction

+B. Fixing on a board in a position of bellows

C. Juncture of a pubic articulation

D. Fixing by a plaster bandage

E. Fixation in bed

11. A child, 10 years old, admitted to traumatology punkt with the diagnosis: opened fracture of bones of the right calf in middle third with displacement, syndrome of a prelum of soft tissues. A trauma was received on building, where concrete design has pressed down a leg. Objectively: there is an oedema of soft tissues in middle and low third of the right calf, lacerated wound on anterior surface, paleness of dermal covers of calf, toes are warm, coloured normally. What is the plan of local treatment of this patient?

A. Closed reposition, plaster bandage, FSI

B. Skeletal traction, primary surgical treatment of a wound

C. Opened reposition of parts metalostoesynthesis

D. Closed reposition, FSI, Plaster extension

+E. FSI, processing of a skin on Krasovitov's, imposing of the Ilizarov's apparatus

12. Parents of 12-yearsold child, who had fracture of femur 2 months ago, have applied for consulting aid. The boy was treated at regional hospital with a skeletal traction. Objective: there is deformation in middle third of femur, limb is shortened. Osteal callosities, angular displacement about 30°, deformation by width on half of diameter are seen in X-ray examination. What types of displacement parts of fracture are corrected during growth of a child?

+A. Displacement by width is corrected completely (up to cortical layer)

B. Displacement lengthways up to 1 sм

C. Displacement bevel way 15°

D. All types of displacement are corrected during growth

E. Residual displacement isn't corrected

13. A child, 10 years old, complains of pain in the region of the left clavicle after dip from a tree. In examination: there is tumescence, crepitating in palpation. Fracture of a clavicle in middle third with lengthways displacement and by width on all diameters is seen on X-ray examination. The reposition of breakings off and imposing immobilising bandage is ordered. What the type of bandage, using in this

displacement?

A. Bandage Deso

B. Imposing of rings

C. Trunk Alberts

D. Thoracic-brachial bandage

E. Bandage such as the trunk Kuzminskogo

14. A boy, 13 years old, has fallen from an altitude on the arm, bent in elbow. In inspection: there is signed tumescence in the field of elbow, Marks' sign is disturbed. The passive motions are possible. The suggested diagnosis is:

A. Bruise of elbow

B. Dislocation of bones of forearm

+C. Transepicondylar fracture

D. Fracture of radial neck

E. Intermuscular hematoma

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Topic 3b. Blunt trauma of thorax, abdominal cavity, and retroperitoneal space

Theme: "Acute abdomen" at a trauma of the parenchymatous and hollow organs of abdominal cavity

1. Actuality of theme: A trauma of the abdominal organs represents a serious problem for modern medicine. Ruptures of the organs demand an urgent operative intervention. Its in-time diagnostics is not always possible because of blind clinical picture, presence of the multiple traumas, unconsciousness of victims. Developing intra-abdominal bleeding or peritonitis represent a serious problem for the health and life of patients with a trauma.

2. Educational aims:

As a result of the independent working on this theme higher education applicants must:

- know:

1. Classification of traumas of abdominal organs in children.
2. Classification of damages of the spleen and liver in children. Features of the clinical picture at various damages. Additional methods of diagnostics.
3. Methods of treatment of damages of the spleen and liver.
4. Classification of damages of hollow organs. Features of the clinical picture depending on organ localization and type of damage.
5. Reasons of a damage of the pancreas in children. Clinical picture, diagnostics and treatment.

- be able:

1. To appoint the inspection plan in children with the trauma of abdominal organs.
2. To estimate results of laboratory, ultrasonic and radiological research in children with the trauma of abdominal organs.
3. To define a treatment tactics at various damages of abdominal organs in children.

3. Materials for preliminary preparation of higher education applicants:

3.1. Basic base knowledges, abilities, skills necessary for independent study, which are based on the interdisciplinary communications:

№	Discipline	to know	to be able
1	Anatomy	Structure of hollow and parenhymatous organs of the abdominal cavity	
2	Pathological anatomy	Anatomical changes at a trauma of hollow and parenhymatous organs of the abdominal cavity	
3	Pathological physiology	The changes caused by the acute intra-abdominal bleeding and peritonitis	
4	Surgery	Clinical symptoms and inspection methods in patients with intra-abdominal bleeding and peritonitis	To collect the anamnesis, to perform a palpation, percussion of the abdominal organs, recatal examination, to estiimate the results of laboratory, ultrasonic and radiological inspection

3.2. The contents of theme is expounded in the recommended literature.

3.3. Recommended literature:

1. Ashkraft T., Kholder T. Pediatric surgery, II vol.. - SPb. - 1997. - 406 p.

2. Isacov Yu.F. Surgical disease in children. - M.- 1988.- 415 p.

3. Nikolayeva N. G. Pediatric surgery. - Odessa. - 2004. - 180 p.

3.4. Reference card for independent preparation of higher education applicants with the use of literature on the theme.

Basic tasks	Instructions	Answers
1	2	3
Classification of traumas of spleen	Name types of traumas depending on spleen damage rate	
Clinical picture of rupture of spleen	Complaints of the child, data of objective survey, data of additional methods of research	
Treatment of traumas of spleen	Indications for conservative and operative treatment, their technique	
Classification and clinical picture of traumas of liver	Name the basic clinical symptoms, make the inspection and treatment plan	
Classification of ruptures of stomach and intestines.	Give the characteristic to different variants of the full and incomplete rupture	
Clinical picture of rupture of hollow abdominal organs	Name the basic clinical symptoms, the additional methods of examination. Define the indications for operation.	
Diagnostics and treatment of traumas of a pancreas.	Name the basic symptoms, resuts of the ultrasonic and laboratory research, the indication for conservative and operative treatment	

3.5. Materials for self-control.

3.5.1. *Questions for self-control.*

1. Name four types of the spleen trauma.
2. How long is «the light period» in patient with double-moment rupture of the spleen?
3. Classification of damages of liver.
4. What is a danger of incomplete ruptures of intestine?
5. What radiological sign confirms a rupture of hollow abdominal organs?
6. Appoint the plan of inspection of a child with a trauma of parenhymatous abdominal organ.
7. Estimate the results of ultrasonic inspection in various damages of spleen.
8. Appoint a plan of the treatment in a child with subcapsule hematoma of the spleen.
9. Appoint the plan of inspection of the child with a pancreas trauma.
10. Appoint the plan of treatment of the child with a pancreas bruise.

3.5.2. Test tasks for self-control.

3.6. The subject for scientific work on this theme:

1. Frequency of the various traumas of spleen.
2. Definition of the immune status in children after spleenectomy.

THORACIC TRAUMA

HP horacic trauma is an important cause of morbidity ^ and mortality in children. Although it accounts for a small minority of pediatric trauma injuries (4%-25%), it is associated with a 20-fold increase in mortality when compared with pediatric trauma patients without thoracic trauma.[1-10] Moreover, the mortality rate of head and abdominal trauma in association with thoracic trauma in children increases to 25%. Children with neurosurgical trauma, thoracic trauma, and abdominal trauma may have a mortality rate that approaches 40%. Isolated thoracic trauma in a child is associated with a mortality rate of approximately 5%, which is largely due to penetrating trauma.[1]

Epidemiologic studies have reported a twofold to threefold higher incidence of thoracic trauma in boys as compared with girls.[9-15] Most injuries (80%-95%) are the result of blunt trauma, typically resulting from a traffic accident in which the child involved is a passenger or pedestrian.[210] Not surprisingly, many children will have involvement of other organ systems with a high Injury Severity Score (ISS). When penetrating trauma does occur, older children and adolescents are more likely to be the victims. This is associated with a higher mortality rate.[5]

Contusion or laceration of the pulmonary parenchyma is the most common injury and may be associated with rib fractures and pneumothorax or hemothorax. Injuries to other organs such as the tracheobronchial tree (<1%), esophagus (<1%), aorta (<1%), diaphragm (4%), and heart (6%) are uncommon but not insignificant.[12]

ANATOMY AND PHYSIOLOGY

Children have unique anatomic and physiologic properties that are salient to the diagnosis and management of thoracic trauma. As in any trauma patient, sequential management of the airway, breathing, and circulation is of primary importance. The pediatric airway may be complicated by numerous factors. The head of an infant is proportionally much larger than that of an adult, thus predisposing to neck flexion and occlusion of the airway in the supine position. The larger tongue

and soft palate, as well as the more anterior glottis, can make the airway difficult to visualize. The child's trachea is shorter relative to body size, narrower, and more easily compressed compared with the adult. The subglottic region is the narrowest part of the airway in children. Because of its small cross-sectional diameter, the pediatric airway is more susceptible to plugging with mucus and to small amounts of airway edema.

With regard to breathing, the chest wall is more compliant in children, with less muscle mass for soft tissue protection. This allows a greater transmission of energy to underlying organs when injury occurs. The thinner chest wall also allows for easier transmission of breath sounds, which may obscure the diagnosis of a hemothorax or pneumothorax. Children are also at an increased risk for hypoxia owing to their higher oxygen consumption per unit body mass and their lower functional residual capacity to total lung volume ratio.

When assessing circulation, it is important to note that the mediastinum is more mobile than in older patients, and this is particularly true in young children. Unilateral changes in thoracic pressure, such as with a pneumo- thorax, can lead to a tension pneumothorax. This can shift the mediastinum to the extent that venous return is markedly reduced. The pathophysiologic effect is similar to hypovolemic shock. This response is more pronounced than is typically seen in an adult.

Children compensate for a decrease in cardiac output by increasing their heart rate. In the infant, improvement in stroke volume provides little in the way of compensation in the hypotensive child. Pediatric patients also have a higher body surface area to weight ratio than the adult, which predisposes them to hypothermia. This, in turn, may complicate the assessment of perfusion.

SPECIFIC INJURIES AND MANAGEMENT

Thoracic injuries in children can be categorized by

location:

I. Chest Wall

a. Flail chest

b. Open pneumothorax

c. Rib fracture

d. Traumatic asphyxia

II. Pleural Cavity/Pulmonary Parenchyma

a.Tension pneumothorax

b. Hemothorax

c. Simple pneumothorax

d. Pulmonary contusion/laceration

e. Diaphragmatic injury

III. Mediastinum

a.Pericardial tamponade

b. Aortic/great vessel injury

c. Tracheobronchial injury

d. Cardiac contusion

e. Esophageal injury

Chest Wall

Rib Fractures

Young children have a compliant thorax and begin to resemble adults around 8 to 10 years of age. As a consequence, rib fractures are relatively uncommon in young children and occur more frequently in adolescents. Rib fractures are often suspected with physical examination and are identified on a chest radiograph (CXR) during the initial assessment. By themselves, rib fractures are infrequently a cause of major morbidity or mortality, but they are indicators of significant energy transfer. If a rib fracture is found in a child younger than 3 years of age, child abuse should be considered. Bone scans and bone surveys are useful in diagnosing remote fractures of the bony thorax in abused children, and follow-up studies improve identification of these injuries. In older children, rib fractures should draw attention to the risk of an associated underlying injury. Fractures and dislocations of the bony thorax and joints may cause significant long-term pain. In addition to pneumothorax and hemothorax, children with first rib fractures may have fractures of the clavicle, central nervous system injury, facial fractures, pelvic fractures, extremity injuries, and major vascular trauma. When children present with multiple rib fractures, mortality has been reported to be as high as 42%. A careful survey of the child must be performed to look for significant injuries in other regions of the body.

The management of rib fractures is typically supportive. Good pain relief will prevent atelectasis and pneumonia. Because rib fractures can be associated with a hemothorax or pneumothorax, immediate drainage of fluid and blood collections or air with a tube thoracostomy is appropriate.

Flail Chest

Because of the increased pliability of the chest wall, multiple rib fractures in series (flail chest) are not commonly seen in younger children. However, when flail chest does occur, respiratory effort can be depressed with a paradoxical motion of the flail segment. The large force required to produce this injury invariably results in injury to the underlying lung, which contributes to the respiratory compromise. Treatment of the pediatric flail chest includes good pain relief, adjusted to avoid respiratory depression. Nonsteroidal anti-inflammatory medications are useful to treat the pain from rib fractures after the acute phase. Occasionally, positive-pressure ventilation may be required in children with a flail chest and respiratory insufficiency. In the case of a severe flail chest there is some evidence to suggest that operative fracture stabilization of a flail chest will decrease morbidity and improve outcome in selected cases.

Open Pneumothorax

Open pneumothorax (sucking chest wound) occurs when there is a gaping defect in the chest wall typically caused by blast injury, severe avulsion injuries, or impalement. This is not commonly seen in children but may be acutely life threatening when present. The negative pressure in the pleural cavity sucks air into the thorax. Air trapping results in collapse of the ipsilateral lung and mediastinal shift as with a tension pneumothorax. Treatment requires placement of an occlusive dressing to prevent further air from entering the chest cavity as well as chest tube insertion to drain any hemo/pneumothorax that may have accumulated.

Traumatic Asphyxia

Traumatic asphyxia is typically caused by a large compressive force on the chest combined with deep inspiration against a closed glottis (Valsalva maneuver). The increased thoracic pressure compresses the right atrium, precluding blood return from the superior vena cava, and resulting in rupture of venules and capillaries of the face and head. Patients will exhibit conjunctival hemorrhages, facial swelling, and petechial hemorrhages on the face and upper chest. Although severe cases may result in loss of vision or

other permanent neurologic sequelae, the morbidity and mortality associated with traumatic asphyxia is generally related to associated injuries. The majority of children who survive exhibit good outcomes.

Pleural Cavity and Pulmonary Parenchyma

Pneumothorax—Pulmonary Lacerations

Pneumothorax may occur with penetrating injury to the chest wall or air leak into the pleural space from a pulmonary laceration or disruption of the airway more proximally. It is a relatively common finding in children with blunt and penetrating thoracic trauma. An air leak from the injury may dissect under the pleura to cause pneumomediastinum and subcutaneous emphysema. A simple pneumothorax is often asymptomatic because the lack of increased intrathoracic pressure limits the recognition of symptoms. For this reason, a screening CXR is an important component of the evaluation of pediatric thoracic injury. Air within the pleural cavity can layer anteriorly, posteriorly, or in the subpulmonic space, and a simple pneumothorax can

be easily missed on chest film but can be identified on a subsequent computed tomographic (CT) scan. However, a recent study analyzing the utility of CT scan as a screening modality to replace initial CXR concluded that although a CT scan is highly sensitive, it should not be used as a primary imaging tool given its cost and the acceptable sensitivity of routine CXR. Ultrasonography is another diagnostic modality that has been shown to be nearly as sensitive as CT in determining the presence of an occult pneumothorax and has gained wide acceptance as a screening tool for traumatic pneumothorax.

The need for intervention in the presence of a simple pneumothorax will depend on the degree of pneumo- thorax and the patient's clinical condition. Some authors have suggested that if the volume of the pneumothorax is greater than 20% of the pleural space, then drainage is indicated. Although insertion of a chest tube can be considered appropriate in almost every circumstance of traumatic pneumothorax, there are alternatives to conventional chest tubes, such as pigtail catheters. Additionally, there may be a benefit in treating with supplemental oxygen alone. The rationale for this therapy is that atmospheric gas (78% nitrogen) comprises the majority of the entrapped air collection. If the nitrogen level in the blood is "washed out" by increased inspired oxygen, a nitrogen gradient will be created that will cause accelerated absorption of the air collection. Oxygen can be delivered by way of nasal cannula, a hood, or a mask. Treatment with supplemental oxygen is usually only required for 24 to 48 hours.

In contrast, tension pneumothorax is a life- threatening condition that requires expeditious decompression of the involved hemithorax. A tension pneumothorax likely causes symptoms due initially to hypoxemia and later to increased intrapleural pressure with subsequent decreased venous return and cardiovascular collapse. If the clinician suspects a tension pneumothorax in a patient with appropriate signs and symptoms, it is reasonable to proceed with needle decompression of the pneumothorax without waiting for a CXR to confirm the clinical suspicion. If rapid drainage of intrapleural air cannot be accomplished with a needle, insertion of a pigtail catheter or a chest tube should be performed. A tension pneumothorax treated initially with needle decompression will usually require placement of a chest tube due to the continuing collection of air under pressure in the involved hemithorax. If one or both lungs have been under compression due to a prolonged tension pneumotho- rax, re-expansion pulmonary edema can occur.

Systemic air embolism can occur with any pulmonary parenchymal injury and increased intrabronchial pressure, creating a bronchopulmonary venous fis- tula. This is most often seen when positive-pressure ventilation is required to support the injured patient. Sudden neurologic findings or cardiovascular decom- pensation may be the initial sign that air has embolized to the coronary or cerebral vessels. If this complication is recognized, steps should be taken to prevent further air embolism. If possible, the removal of the intravascu- lar air should be considered. Treatment options include tube thoracostomy, but more often an emergency tho- racotomy will provide immediate reversal of the physiology promoting air embolism. The hilum of the lung should be occluded to prevent further escape of air into the venous system, and operative control of the bronchial-venous interface should be obtained. The mortality associated with this complication is high.

Hemothorax

Hemothorax can result from blunt or penetrating injury to any of the intrathoracic vessels, the chest wall ves-

sels, the pleura, or the pulmonary parenchyma. Occasionally, a rib fracture may lacerate intercostal blood vessels or the lung. Rarely, the aorta or vena cava may be injured by pressure or shearing. Blood in the thorax may be asymptomatic unless the volume of blood is large. Smaller volumes may be more easily detected on CT scan, which also allows for measurement of Hounsfield density to aid in the diagnosis. Each hemithorax can hold approximately 40% of a child's blood volume. It is difficult to estimate the amount of blood loss on a CXR. Prompt chest tube placement allows for evacuation of blood from the pleural space and re-expansion of the lung. It also allows the surgeon to assess the volume of blood lost and whether the blood loss is ongoing.

There are instances in which an operation may be needed to stop ongoing intrathoracic blood loss. The immediate blood loss after tube thoracostomy of 15 mL/kg or ongoing losses of 2 to 3 mL/kg/hr for 3 or more hours are indicators for thoracic exploration in children to control bleeding. If undrained, the hemothorax can become organized with the clot eventually replaced by a fibrothorax that can cause a restrictive lung defect. This predisposes to atelectasis, ventilation-perfusion mismatching, and pneumonia.

Residual blood is an excellent culture medium. Empyema and sepsis can result from infection of an undrained hemothorax. Tube thoracostomy may not adequately evacuate an organizing post-traumatic hemothorax in up to 12% of patients. In this situation, thoracoscopy is indicated to evacuate the residual clot. Patients who undergo earlier thora- coscopy may experience less morbidity according to some authors. However, there are also data to suggest that thrombolytic therapy with streptokinase or urokinase is equally effective at treating a retained hemothorax.

Chylothorax

Chylothorax caused by injury to thoracic lymphatic channels is an uncommon complication of thoracic trauma. Chylothorax usually becomes evident 3 to 7 days after injury. The diagnosis is made by obtaining a sample of the pleural fluid and identifying the lymphatic and lipid content. Treatment includes tube thoracostomy and either enteral feedings with medium-chain triglycerides or parenteral nutrition.

Pulmonary Contusion

One of the most common thoracic injuries in children is a pulmonary contusion, which can occur with blunt or penetrating trauma. The flexible chest wall of the child allows contusion of the lung without rib fracture, resulting in areas of lung consolidation and chest wall contusion. Microscopically, pulmonary contusions show alveolar hemorrhage, consolidation, and edema. The presence of a pulmonary contusion contributes to decreased pulmonary compliance, hypoxia, hypoventilation, and a ventilation-perfusion mismatch. A CXR taken during the initial assessment may demonstrate a pulmonary contusion. However, because this is invariably a supine film, it is sometimes difficult to differentiate fluid/blood in the pleural space from a lung contusion. To this end, a chest CT scan can show areas of pulmonary contusion not appreciated on the radiograph and can differentiate a parenchymal process (contusion) from free fluid. When a contusion is seen on CXR, however, these children typically have a larger volume of lung that has been injured with a higher degree of impaired oxygenation. Also, a significant percentage will require ventilatory support. When a pulmonary contusion is seen only on CT, the morbidity of the injured child does not appear to be affected

when compared with children with normal CT findings. The overall injury severity, associated injuries, and outcomes in these patients are similar to those seen in adults. Treatment includes appropriate fluid resuscitation, supplemental oxygen, pain management, and strategies to prevent atelectasis and pneumonia.

A significant percentage of patients may develop pneumonia or the acute respiratory distress syndrome (ARDS) after pulmonary contusion. In an occasional patient, the pulmonary contusion may cause life-threatening hypoxia that cannot be supported with conventional ventilation, including high-frequency oscillation. Extracorporeal life support has been used in extreme circumstances to support patients with severe pulmonary contusions or ARDS. Children with pulmonary contusions may have prolonged changes in respiratory function and radiographic abnormalities. These changes may persist for an extended period of time after resolution of the symptoms.However, these children do not appear to suffer from any significant long-term sequelae.

Mediastinum

Airway Injury

Injuries to the tracheobronchial tree are infrequent in children. Airway disruption may occur with penetrating injury or with blunt injury such as high-energy acceleration or deceleration. Up to three fourths of these injuries are noted within 2 cm of the carina and almost half occur within the first 2 cm of the right main-stem bronchus. Most patients with tracheal injuries have mediastinal air on CXR, although more distal injuries may rupture into the pleural space and present as a tension pneumothorax. Other findings associated with a major airway injury include a persistent large air leak from a chest tube, mediastinal air, cervical subcu-taneous emphysema without pneumothorax, or florid respiratory compromise. Rarely, complete transection of a distal main-stem bronchus will appear on CXR with total lung collapse and mediastinal displacement. Persistent pneumomediastinum and pneumothoraces on CXR after adequate tube thoracostomy should alert the clinician to consider an injury to the tracheobron- chial tree.

Once recognized, these injuries require prompt diagnosis and treatment. Pleural air or fluid collections should be drained until an accurate diagnosis of the airway injury is made. Mechanical ventilation may be necessary because of respiratory failure in this setting. Fiberoptic bronchoscopy allows for evaluation of the airway and may improve the probability of successful intubation. Many airway injuries are diagnosed by rigid or flexible bronchoscopy. Chest CT with a multiple-array scanner may have a role in visualizing tracheal or bronchial injuries especially if three- dimensional reconstructions of the airway are used (virtual bronchoscopy) .

Esophagus

Pediatric esophageal injuries are uncommon, occurring in less than 1% of children sustaining either blunt or penetrating thoracic trauma. The esophagus is a relatively elastic, mediastinal structure that is largely protected by the bony thorax. This elasticity allows the esophagus mobility so that when blunt force is applied it can move or decompress, which limits the likelihood of rupture. Penetrating injuries are more

likely to cause esophageal trauma. Esophageal disruption may manifest as dyspnea, dysphagia, cyanosis, mediastinal air, subcutaneous emphysema, pleural effusion, chest or epigastric pain, fever, or sepsis. Initial symptoms, however, may be vague and nonspecific. Esophagography with a water-soluble contrast agent and esophagoscopy are typically the studies that will identify an esophageal injury.

If the esophagus is ruptured or perforated, conventional treatment is operative repair. This is performed for the purpose of drainage and/or repair of the injured esophagus. Treatment is initiated with fluid resuscitation and parenteral antibiotics. Operative repair consists of direct suture closure of the injury. If possible, pleural flap coverage and tube thoracostomy are performed. If an operation is undertaken early after the injury, this treatment, along with bowel rest and total parenteral nutrition has good success. If the perforation is not identified early, treatment becomes more difficult. If the perforation is more than 24 hours old, operative closure becomes technically much more difficult owing to the degree of inflammation and the amount of contamination. Techniques used in this circumstance include attempted repair, esophageal isolation, multiple drain placement, gastrostomy, and total parenteral nutrition.

In selected cases of esophageal perforation, nonop- erative management may be successful. This might be the case with certain types of blunt trauma and with iatrogenic injury such as a perforation at the time of endoscopy. Nonoperative management is based on the clinical status of the patient and the injury seen on imaging studies. For example, a patient might have a small leak identified, manifested by mediastinal air or pneumothorax. If there is no fever, no effusion, and the patient looks well, nonoperative treatment with total parenteral nutrition and intravenous antibiotics, along with serial examinations, may be reasonable.

ABDOMINAL AND RENAL TRAUMA

T he management of children with major abdominal injuries has changed significantly in the past 2 decades. An increased awareness of the anatomic patterns and physiologic responses characteristic of trauma in children has resulted in the successful non- operative treatment of most abdominal solid organ injuries. Our colleagues in adult trauma care have acknowledged this success and have applied many of the principles learned in pediatric trauma to their patients. A recent review of the National Pediatric Trauma Registry (NPTR) indicates that 8% to 12% of children with blunt trauma have an abdominal injury. Fortunately, more than 90% survive. Only 22% of the deaths in the NPTR were related to the abdominal injury. Although abdominal injuries are 30% more common than thoracic injuries, they are 40% less likely to be fatal.

Historically, adult trauma surgeons unfamiliar with the nonoperative management of solid organ injuries raised doubts about the wisdom of this approach. Their concerns included the potential for increased transfu- sion requirements, increased length of hospitalization, and missed associated injuries. Some even questioned the need for involvement of pediatric surgeons in pedi- atric trauma care. The clinical experience accumulated over the past 20 to 30 years, which has settled these concerns, is reviewed.

Few surgeons have extensive experience with massive abdominal solid organ injury requiring immediate surgery. It is imperative that surgeons familiarize themselves with current treatment algorithms for life-threatening abdominal trauma. Important contributions have been made in the diagnosis and treatment of children with abdominal injury by radiologists and endoscopists. The resolution and speed of computed

tomography (CT), screening capabilities of focused assessment with sonography for trauma (FAST), and the percutaneous, angiographic, and endoscopic interventions of nonsurgeon members of the pediatric trauma team have all enhanced patient care and improved outcomes. Each section of this chapter focuses on the more common blunt injuries and unique aspects of their care in children.

DIAGNOSTIC MODALITIES

The initial evaluation of the acutely injured child is similar to that of the adult. Plain radiographs of the cervical spine, chest, and pelvis are obtained after the primary survey and evaluation of the airway, breathing, and circulation. Plain abdominal radiographs offer little in the urgent evaluation of the pediatric trauma patient. The rapid availability and quality of imaging modalities has improved dramatically in recent years, both in the urban and in the rural settings. Prompt identification of potentially life-threatening intra-abdominal injuries with rapid resuscitation and therapeutic intervention is now possible in the overwhelming majority of children.

Computed Tomography

CT has become the standard of care in the evaluation of the pediatric trauma patient. Newer-generation CT scanners are now readily accessible in most health care facilities. CT is noninvasive, rapid, and highly accurate in identifying and qualifying the extent of abdominal injury. This has significantly reduced the incidence of nontherapeutic exploratory laparotomy. A head CT, when indicated, should be performed without use of a contrast agent before abdominal CT to avoid having the contrast conceal a hemorrhagic brain injury. Intravenous contrast is then administered, and the vascular and parenchymal resolution is enhanced utilizing a "dynamic" scanning mode. The finding of a contrast "blush" on CT in children with blunt liver injury has been associated with larger transfusion requirements and a higher mortality rate. A CT blush after significant hepatic or splenic injury should prompt the surgeon caring for the child to consider surgical or interventional radiologic control of the bleeding based on the hemodynamic status and stability of the child. Enteral contrast for enhancement of the gastrointestinal tract is generally not required in the acute trauma setting and can actually be detrimental secondary to aspiration of the contrast agent.

Not all children with potential abdominal injuries are candidates for acute CT evaluation. Obvious penetrating injury necessitates immediate operative intervention. Diagnostic laparoscopy may be very beneficial in determining if peritoneal penetration has occurred in the setting of an abdominal or flank stab wound. The hemodynamically unstable child should never be taken from an appropriate resuscitation arena for a CT scan. These children may benefit from an alternative diagnostic study, such as diagnostic peritoneal lavage (DPL), FAST, or urgent operative intervention. Hemodynamically unstable patients with clinical evidence of abdominal trauma who are hypotensive and not responsive to resuscitative efforts should go immediately to the operating room without imaging.

Modern generation CT scanners are highly sensitive in the evaluation of possible solid organ and retroperitoneal injuries. The greatest limitation of abdominal CT in trauma is the lack of ability to reliably identify acute intestinal rupture. Findings suggestive but not diagnostic of intestinal perforation are pneumoperitoneum, bowel wall thickening, free intraperitoneal fluid, bowel wall enhancement, and dilated

bowel. There must be a high index of suspicion for a hollow viscus injury in the child with free intraperitoneal fluid and no identifiable solid organ injury on CT

Recent concerns about the risk of the radiation involved in CT have arisen, particularly in the pedia- tric population. Epidemiologic studies of populations exposed to radiation have shown that children are considerably more sensitive to radiation than adults. Because children have more rapidly dividing cells and have a longer life expectancy, the odds that children may develop cancers from x-ray radiation may be significantly higher than adults. The actual lifetime risk from CT is not clearly known. Efforts to reduce the numbers of CT scans, unnecessary scans, and efforts to reduce the CT-related radiation dose in individual patients are important measures that should be kept in mind when caring for and evaluating the injured child.

Focused Abdominal Sonography for Trauma

Clinician-performed sonography for the early evaluation of the injured child has been shown to be useful in many situations but does have limitations. Examination of Morrison's pouch, the pouch of Douglas, the left flank to include the perisplenic region, and a subxi- phoid view to visualize the pericardium is the standard four-view FAST examination. This bedside study may be useful as a rapid screening study, particularly in the patient too unstable to undergo an abdominal CT scan. Free intraperitoneal fluid seen on FAST examination in the unstable child not responding to resuscitative measures may support a decision to operate immediately. This modality is the study of choice for the evaluation of possible hemopericardium with tamponade.

A negative FAST exam (absence of hemoperito- neum) does not exclude a significant solid organ or hollow viscus injury. There is at least a 15% false-negative rate for detecting hemoperitoneum with sonography. FAST may miss up to 25% of liver and spleen injuries, most acute renal injuries, retroperitoneal bleeding, and virtually all pancreatic, mes- enteric, and bowel and bladder injuries. Compared with CT, FAST is only about 63% sensitive for detecting moderate amounts of free intraperitoneal fluid in the trauma setting. Also, the documentation of free fluid in the abdomen does not necessarily indicate the need for surgical intervention. FAST may be very useful in decreasing the number of CT scans performed for "low-likelihood" injuries. The study may be repeated on a serial basis and results correlated with the clinical scenario.

Diagnostic Peritoneal Lavage

Since its description in 1965, the use of DPL has diminished significantly, especially in the pediatric population. The ready availability of high-resolution CT scanners and the nonoperative management of many pediatric injury patterns have increased, thus minimizing the utility of DPL. Although very accurate for identifying hemoperitoneum, retroperitoneal injuries may be missed with DPL. Incisional pain after a negative DPL may interfere with serial examinations in a child being managed nonoperatively. Also, a positive DPL may lead to a nontherapeutic laparotomy if based on hemoperi- toneum alone. Because the majority of solid organ injuries do not require surgical intervention, intraperitoneal blood documented by DPL has little clinical significance. The need for operative management is determined by clinical instability, associated injuries, and the requirement for ongoing blood replacement.

There are clinical situations in which a DPL may prove to be very beneficial. The hemodynamically unstable child may have a rapid DPL to exclude the abdomen as a source of significant hemorrhage. Children with a lap belt injury pose a particular diagnostic challenge, particularly if concomitant neurologic injury is present. The initial abdominal CT is frequently normal in those with lap belt injuries, including acute hollow viscus perforations, but a DPL may document the presence of an occult visceral injury with the return of bile, bacteria, feculent matter, or an increased leukocyte count. Infusion of 10 mL/kg of normal saline into the peritoneal cavity is followed by allowing the infusate to drain.

Diagnostic and Therapeutic Laparoscopy

Diagnostic laparoscopy in a stable patient after a stab wound to the upper abdomen revealed this diaphragmatic injury. Repair was performed by using minimal access techniques. the hemodynamically stable patient. The sensitivity is comparable to that of DPL, but the specificity is higher, as would be expected by actually visualizing the injury. A decrease in the number of nonthera- peutic laparotomies has been demonstrated in adult series. Studies also have shown that not only may the traumatic injury be identified with laparoscopy but the definitive repair also can be frequently performed

SOLID ORGAN INJURY Spleen and Liver

The spleen and liver are the organs most commonly injured in blunt abdominal trauma, with each accounting for one third of the injuries. Nonoperative treatment of isolated splenic and hepatic injuries in stable children is now standard practice. Although nonoperative treatment of children with isolated, blunt spleen or liver injury has been universally successful, great variation is seen in the management algorithms used by individual pediatric surgeons. Review of the NPTR and recent surveys of the American Pediatric Surgical Association (APSA) membership confirm the wide disparity in practice. Controversy also exists regarding the utility of CT grading as a prediction of outcome in liver and spleen injury. In 1999, the APSA Trauma Committee defined consensus guidelines for resource utilization in hemodynamically stable children with isolated liver or spleen injury. These guidelines are based on CT grading and were developed by analyzing a contemporary, multi-institution database of 832 children treated nonoperatively at 32 centers in North America from 1995 to 1997. Consensus guidelines on the length of intensive care unit (ICU) stay, length of total hospitalization, use of follow-up imaging, and physical activity restriction for clinically stable children with isolated spleen or liver injuries (grades I to IV) were defined by analysis of this database.

The guidelines were then applied prospectively to 312 children with liver or spleen injuries treated nonoperatively at 16 centers from 1998 to 2000. (It is imperative to emphasize that these proposed guidelines assume hemodynamic stability.) Patients with other minor injuries such as nondisplaced, noncom- minuted fractures or soft tissue injuries were included as long as the associated injuries did not influence the variables in this study. The patients were grouped by severity of injury and defined by CT grade. Compliance with the proposed guidelines was analyzed for age, organ injured, and injury grade. All patients were followed for 4 months after injury. The extremely low rates of transfusion and need for operation document the stability of the study patients.

Specific guideline compliance was 81% for criteria for ICU hospitalization, 82% for length of

hospitalization, 87% for follow-up imaging, and 78% for activity restriction. A significant improvement in compliance was noted from year 1 to year 2 for ICU stay (77% vs. 88%; $P < .02$) and activity restriction (73% vs. 87%; $P < .01$). No differences in compliance were found by age, gender, or organ injured. Deviation from guidelines was the surgeon's choice in 90% and patient related in 10%. Six (1.9%) patients were readmitted, although none required operation. Compared with the previously studied 832 patients, the latter 312 patients managed prospectively under the proposed guidelines had a significant reduction in ICU stay ($P < .0001$), total hospitalization ($P < .0006$), follow-up imaging ($P < .0001$), and interval of physical activity restriction ($P < .04$) within each grade of injury.

From these data, it was concluded that prospective application of specific treatment guidelines based on injury severity resulted in conformity in patient management, improved utilization of resources, and validation of guideline safety. Significant reduction of ICU care, hospital stay, follow-up imaging, and activity restriction was achieved without adverse sequelae when compared with the retrospective database. Recent single-institution studies have suggested that further reduction in resource utilization may be safe. A large retrospective series showed that once hemodynamic stability was achieved and there was no evidence of ongoing bleeding, patients are unlikely to begin bleeding again. This evidence challenges the assumption that bed rest is the key variable in treatment. In reality, bed rest is simply a period of observation, for which the current recommendations may be longer than necessary. Another institution has employed a management strategy based on the patient's hemodynamic status instead of injury grade and recorded a dramatic decrease in hospital stay compared with the current guidelines without an adverse event. A prospective, multicenter trial is underway to delineate the safety of an abbreviated protocol.

The surgeon's decision to operate for spleen or liver injury is best based on evidence of continued blood loss such as hypotension, tachycardia, decreased urine output, and decreasing hematocrit unresponsive to crystalloid and blood transfusion. The rates of successful nonoperative treatment of isolated blunt splenic and hepatic injury now exceed 90% in most pediatric trauma centers or adult trauma centers with a strong pediatric commitment. A recent study of more than 100 patients from the NPTR indicated that non-operative treatment of spleen or liver injury is indicated even in the presence of associated head injury, if the patient is hemodynamically stable. In this study, the rates of operative intervention for blunt spleen or liver injury were similar with or without an associated closed-head injury.

Surgeons unfamiliar with current treatment algorithms for blunt splenic injuries in children occasionally question the nonoperative approach. This is important because the majority of seriously injured children are treated outside dedicated pediatric trauma centers. Although several adult trauma services have reported excellent survival rates for pediatric trauma patients, analysis of treatment for spleen and liver injuries reveals an alarmingly high rate of operative treat- ment. It is possible that adult trauma surgeons, influenced by their past experience with adult patients, are more likely to favor operative treatment than are their pediatric surgical colleagues. Adult trauma surgeons caring for injured children must consider the anatomic, immunologic, and physiologic differences between pediatric and adult trauma patients and incorporate these differences into their treatment protocols. The major concerns regarding nonoperative management are related to the potential risks of increased transfusion requirements, missed associated injuries, and increased length of hospital stay. Each of these concerns has been shown to be without merit.

Outcomes in the Treatment of Blunt Spleen Injury

Many early attempts at comparing care in pediat- ric trauma focused on the treatment of blunt spleen injury. Hospital and physician expertise were used as the basis for comparison in the treatment of children with blunt splenic injury in seven studies between 1985 and 1998

Failure of nonoperative management can have serious consequences. Therefore, patient selection is important. Two recent multi-institutional reviews sought to evaluate the timeline and the characteristics of patients who fail nonoperative management. One hundred twenty of 1813 (6.6%) children with solid organ injury underwent laparotomy in a median time of 2.4 hours, with 90% of the patients having surgery within 24 hours. Pediatric patients who sustained pancreatic injuries were more likely to fail nonoperative management (odds ratio [OR] 7.49; 95% CI, 3.7415.01) compared with those who suffered other injuries. The patients who failed nonoperative management had a higher Injury Severity Score (28 ± 17) than those who were managed successfully nonoperatively (14 ± 10, $P < .001$). Severely head-injured patients (Glasgow Coma Scale score [GCS] = 8) had a higher failure rate for nonoperative management (OR 5.09; 95% CI, 3.04-8.52). Factors associated with an increased failure rate include a bicycle-related injury mechanism, isolated pancreatic injury, more than one solid organ injury, and an isolated grade 5 solid organ injury. The time to failure of nonoperative management peaked at 4 hours and then declined over 36 hours from admission. Thus, continued surgical evaluation and assessment during the entire hospitalization is required to limit morbidity and mortality.

Missed Associated Abdominal Injuries

Advocates of surgical intervention for splenic trauma cite their concern about missing associated abdominal injuries if an operation is not performed. One study reported successful nonoperative treatment in 110 (91%) of 120 children with blunt splenic trauma. In that report, 22 (18%) had associated abdominal injuries. Only 3 (2.5%) of these 120 patients had gastrointestinal injuries, and each was found at early celiotomy performed for a specific indication. No morbidity occurred from missed injuries or delayed surgical intervention. Similarly, a review of the NPTR from 1988 through 1998 revealed 2977 patients with solid abdominal visceral injury. Only 96 (3.2%) had an associated hollow viscus injury. Higher rates of hollow viscus injury were observed in assaulted patients and those with multiple solid visceral injury or pancreatic injury. Differences in mechanism of injury may account for the much lower incidence of associated abdominal injuries in children with splenic trauma. No justification exists for an exploratory celiotomy solely to avoid missing potential associated injuries in children.

Complications of Nonoperative Treatment

Nonoperative treatment protocols have been the standard for most children with blunt liver and spleen injury during the past 2 decades. The cumulative experience allows an evaluation of both the benefits and risks of the nonoperative approach. Fundamental to the success of the nonoperative strategy is the early, spontaneous cessation of hemorrhage. Transfusion rates for children with isolated spleen or liver injury have decreased to less than 10%, confirming the lack of continued blood loss in the majority of patients. Despite these many favorable observations, obvious ongoing hemorrhage and hemo- dynamic instability require the presence of skilled surgeons, operating room staff, and blood bank capabilities. The role of angioembolization in adult trauma patients with splenic injury is expanding with variable success. To date, most pediatric trauma

centers have not utilized angioembolization in acutely bleeding patients with splenic injury.

Isolated reports of significant delayed hemorrhage with adverse outcome continue to appear. Two children with delayed hemorrhage 10 days after blunt liver injury have been reported. Both children had persistent right upper quadrant (RUQ) and right shoulder pain despite having normal vital signs and stable hematocrits. The authors recommended continued in- house observation for injured patients until symptoms resolve. Recent reports described patients with significant bleeding 38 days after a grade II spleen injury and 24 days after a grade IV liver injury. These rare occurrences create anxiety in identifying the minimal safe interval before resuming unrestricted activities.

Routine follow-up imaging studies have identified pseudocysts and pseudoaneurysms after splenic injury. Splenic pseudoaneurysms are often asymptomatic and appear to resolve with time. The true incidence of self-limited, post-traumatic splenic pseudoaneurysms is unknown as routine follow-up imaging after successful nonoperative management has been largely abandoned. Once it is identified, the actual risk of splenic pseudoaneurysm rupture also is unclear. Angiographic embolization techniques can be used to treat these lesions successfully, obviating the need for operation and loss of splenic parenchyma.

Splenic pseudocysts may reach enormous size, leading to pain and gastrointestinal disturbance). Simple percutaneous aspiration leads to a high recurrence rate. Laparoscopic excision and marsupialization is highly effective.

The immunocompetence of a shattered spleen that heals without surgery is debated, and vaccination practices in these children vary widely. Recent evidence using differential interference contrast microscopy in adult trauma patients indicates immunocompetence in patients with healed grade IV injuries.

Sequelae of Damage Control Strategies

Even the most severe solid organ injuries can be treated nonoperatively if a prompt response to resuscitation occurs. In patients who are hemodynamically unstable, despite fluid and packed red blood cell transfusion, emergency laparotomy is indicated. Most spleen and liver injuries requiring operation are amenable to simple methods of hemostasis, using a combination of manual compression, direct suture, and an increasing array of topical hemostatic agents. In young children with significant hepatic injury, the sternum can be divided rapidly to expose the suprahepatic or intrapericardial inferior vena cava. Children will tolerate clamping of the inferior vena cava above the liver as long as their blood volume is replenished. With this exposure, the liver and major perihepatic veins can be isolated and the bleeding controlled to permit direct suture repair or liga- tion of the injured vessel.

The early morbidity and mortality of severe hepatic injuries are related to the effects of massive blood loss and replacement with large volumes of cold blood products. The consequences of prolonged operations with massive blood-product replacement include hypothermia, coagulopathy, and acidosis. Although the surgical team may keep pace with blood loss, serious physiologic and metabolic consequences are inevitable, and many of these critically ill patients are unlikely to survive. A multi-institutional review iden-

tified exsanguination as the cause of death in 82% of 537 intraoperative deaths at eight academic trauma centers. The mean serum pH was 7.18, and the mean core temperature was 32°C before death. Survival in only 5 (40%) of 12 consecutive operative cases of ret- rohepatic vascular or severe parenchymal liver injury in children has been reported.

Maintenance of physiologic stability during the struggle for surgical control of severe bleeding is a formidable challenge even for the most experienced operative team, particularly when hypothermia, coagulopathy, and acidosis occur. This triad creates a vicious cycle in which each derangement exacerbates the others. The physiologic and metabolic consequences of this triad often preclude completion of the procedure. Lethal coagulopathy from dilution, hypothermia, and acidosis can rapidly occur.

Abbreviated laparotomy with packing for hemosta- sis allowing resuscitation before planned reoperation is an alternative in unstable patients in whom further blood loss would be untenable. This "damage control" philosophy is a systematic, phased approach to the management of the exsanguinating trauma patient. Packing of the retroperitoneum while avoiding entry into the abdomen in unstable patients with severe pelvic fractures has also gained popularity. The three phases of damage control are detailed in Table 16-8. Although controversial, several resuscitative endpoints have been proposed beyond the conventional vital signs and urine output, including serum lactate, base deficit, mixed venous oxygen saturation, and gastric mucosal pH. Studies from recent military experience indicate that the optimal ratio of transfused packed red blood cells and fresh frozen plasma should approach 1:1 in massive transfusion scenarios.

There has been increasing experimental and clinical evidence that recombinant activated factor VII (rFVIIa) may be a useful adjunctive therapy in injured patients with ongoing hemorrhage. In the laboratory, reduced clot formation due to hypothermia was reversed with rFVIIa. Military experience indicates a survival advantage in injured soldiers requiring massive transfusion with early use of rFVIIa. The use of rFVIIA has been described in eight children with blunt injuries to the spleen, liver, or kidney and signs of ongoing hemorrhage. Bleeding was successfully controlled after a single dose of rFVIIa. Only three patients required transfusion, and there were no thromboembolic events. The indications, optimal dosages and intervals, and risk profile still need to be developed before rFVIIa can be a mainstay in pediatric trauma care.

Once patients become normothermic, coagulation factors replaced, and oxygen delivery optimized, a second procedure is performed for pack removal and definitive repair of the injuries. A review of nearly 700 adult patients from several institutions who were treated with abdominal packing demonstrated hemo- stasis in 80%, survival of 32% to 73%, and abdominal abscess rates of 10% to 40%.

Although abdominal packing with planned reopera- tion has been used with increasing frequency in adults during the past 2 decades, little published experience has been reported in children. Nevertheless, this technique has a place in the management of children with massive intra-abdominal bleeding, especially after blunt trauma. A 3-year-old child required abdominal packing for a severe liver injury, making closure of the abdomen impossible. A polymeric silicone "silo" was constructed to accommodate the bowel until the packing could be removed. The patient made a complete recovery.

It is essential to emphasize that the success of the abbreviated laparotomy and planned reoperation

depends on a decision to use this strategy before irreversible shock. Abdominal packing, when used as a desperate, "last-ditch" effort after prolonged, failed attempts at hemostasis, has been uniformly unsuccessful. Indications for abdominal packing based on physiologic and anatomic criteria have been identified. Most have focused on intraoperative parameters including pH (~7.2), core temperature (<35°C), and coagulation values (prothrombin time >16 seconds) in the patient with profuse hemorrhage requiring large volumes of blood-product transfusion.

The optimal time for re-exploration is controversial because neither the physiologic endpoints of resuscitation nor the increased risk of infection with prolonged packing is well defined. The obvious benefits of hemostasis provided by packing also are balanced against the potential deleterious effects of increased intra-abdominal pressure on ventilation, cardiac output, renal function, mesenteric circulation, and intra- cranial pressure. Timely alleviation of the secondary "abdominal compartment syndrome" may be a critical salvage maneuver for patients. We recommend temporary abdominal wall expansion in all patients requiring packing until the hemostasis is obtained and visceral edema subsides.

A staged operative strategy for unstable trauma patients represents advanced surgical care and requires sound judgment. Intra-abdominal packing for control of exsanguinating hemorrhage is a lifesaving maneuver in highly selected patients in whom coagulopa- thy, hypothermia, and acidosis render further surgical procedures unduly hazardous. Early identification of patients likely to benefit from abbreviated laparotomy techniques is crucial for its success.

Abdominal Compartment Syndrome

Abdominal compartment syndrome is a term used to describe the deleterious effects of increased intra-abdominal pressure. The "syndrome" includes respiratory insufficiency from worsening ventilation/ perfusion mismatch, hemodynamic compromise from preload reduction due to inferior vena cava compression, impaired renal function from renal vein compression as well as decreased cardiac output, intracranial hypertension from increased ventilator pressures, splanchnic hypoperfusion, and abdominal wall overdistention. The causes of intra-abdominal hypertension in trauma patients include hemoperitoneum, retroperitoneal and/or bowel edema, use of abdominal/pelvic packing, and secondary to massive resuscitation of non-abdominal trauma. The combination of tissue injury and hemodynamic shock creates a cascade of events including capillary leak, ischemia-reperfusion, and release of vasoactive mediators and free radicals that combine to increase extracellular volume and tissue edema. Experimental evidence indicates significant alterations in cytokine levels in the presence of sustained intra-abdominal pressure elevation. Once the combined effects of tissue edema and intra- abdominal fluid exceed a certain level, abdominal decompression must be considered.

The adverse effects of abdominal compartment syndrome have been acknowledged for decades. However, abdominal compartment syndrome has only recently been recognized as a life-threatening, yet potentially treatable entity. The measurement of intra- abdominal pressure can be useful in determining the contribution of abdominal compartment syndrome to altered physiologic and metabolic parameters. Intra-abdominal pressure can be determined by measuring bladder pressure. This involves instilling 1 mL/ kg of saline into the urinary catheter and connecting it to a pressure transducer or manometer via a three- way

stopcock. The symphysis pubis is used as the zero reference point, and the pressure is measured in centimeters of H_2O or millimeters of mercury. Intra- abdominal pressures in the range of 20 to 35 cm H_2O or 15 to 25 mm Hg have been identified as an indication to decompress the abdomen. Many surgeons prefer to intervene according to alterations in other physiologic and metabolic parameters rather than a specific pressure measurement. One series reported 11 adult trauma patients with abdominal compartment syndrome, measured by pulmonary artery catheters and gastric tonometry, had improved preload, pulmonary function, and visceral perfusion after abdominal decompression.

Experience with abdominal decompression for abdominal compartment syndrome in children is limited. Nonspecific abdominal CT findings in children with abdominal compartment syndrome include narrowing of the inferior vena cava, direct renal compression or displacement, bowel wall thickening with enhancement, and a rounded appearance of the abdomen. One study reported the use of patch abdominoplasty in 23 infants and children, of whom only 3 were trauma patients. These authors found that patch abdominoplasty for abdominal compartment syndrome effectively decreased airway pressures and oxygen requirements. Failure to respond with a decrease in airway pressures or Fio_2 was an ominous sign in their series. Several authors found that abdominal decompression resulted in decreased airway pressures, increased Po_2, and increased urine output in children with abdominal compartment syn- drome. Many materials have been suggested for use in temporary patch abdominoplasty, including Silastic sheeting, polytetrafluoroethylene (PTFE) sheeting, intravenous bags, cystoscopy bags, ostomy appliances, and various mesh materials The vacuum pack technique, used increasingly in adults, is applicable in pediatric patients and seems promising

Bile Duct Injury

Nonoperative management of pediatric blunt liver injury is highly successful but is complicated by a 4% risk of persistent bile leakage. The majority of patients with bile leaks sustained high grade (III-V) injuries. Radionuclide scanning is recommended when biliary injury is suspected. Delayed views may show a bile leak even if early views are normal. Endoscopic retrograde cholangiopancreatog- raphy (ERCP) can be performed with placement of transampullary biliary stents for biliary duct injury after blunt hepatic trauma. Although ERCP is invasive and requires conscious sedation, it can pinpoint the site of injury and allow treatment of the injured ducts without laparotomy. Endoscopic transam- pullary biliary decompression is a recent addition to treatment for patients with persistent bile leakage. The addition of sphincterotomy during ERCP for persistent bile leakage after blunt liver injury has been advocated to decrease intrabiliary pressure and promote internal decompression. Also, it is important to note that endoscopic biliary stents may migrate or clog.

Injury to the Pancreas

Blunt traumatic injury to the pancreas occurs infrequently in children and can be very difficult to diagnose. It is estimated that injuries to the pancreas compose 3% to 12% of intra-abdominal injuries in children sustaining blunt trauma. The lack of surrounding fat planes and the small size of the retroperi- toneal gland make it challenging to document even a major ductal injury by routine CT. A dynamic CT pancreatogram, with multiple thin slices while infusing a contrast medium, gives much more detail than routine abdominal

CT. Magnetic resonance cholangiopancreatography also is a useful diagnostic modality but is not appropriate in the acute resuscitative phase of the child with multiple injuries. Elevations in serum amylase and lipase levels are very common in abdominal trauma but are not indicative of the extent of injury or need for surgical intervention. Hyperamylasemia also may occur with salivary gland injury, bowel perforation or obstruction, intracranial hemorrhage, or other nonspecific mesenteric injuries. In contrast, ERCP is a very accurate technique in evaluating potential injury to the pancreatic ductal system. It is not widely used in the early evaluation of the acutely injured child because of the highly specialized skills required, the need for general anesthesia, and the likelihood of significant need for treatment of associated head and thoracic injuries.

Nonoperative management of blunt injuries to the liver, spleen, and kidney in children is accepted as the standard of care in the majority of cases. Controversy exists when discussing management of the child with a significant pancreatic injury. Those with a pancreatic contusion without major ductal disruption will heal spontaneously. Successful nonoperative treatment of 28 children with pancreatic injuries ranging from contusions to complete transections by CT scan has been described. Pseudocysts developed in 36% of patients and were treated with observation or percutaneous aspiration.

Direct visualization of the pancreas is very important when exploring a child's abdomen for a traumatic injury. The lesser sac is entered above the transverse colon, and the body and tail of the gland are carefully inspected. A Kocher maneuver is then used to inspect the duodenum and head of the pancreas. The posterior surface of the gland may be accessed by mobilization of the spleen along with the tail of the pancreas from a lateral to medial direction.

In the child with a major pancreatic ductal injury, early operative intervention has been reported to shorten hospitalization and lessen dependence on total parenteral nutrition compared with those children who were initially managed nonopera- tively. Pseudocyst formation occurs in 45% to 100% of ductal injuries managed nonoperatively. Of these, a significant number, up to 60%, may resolve with time. Percutaneous or cyst-enteric drainage procedures may be needed if resolution does not occur spontaneously. These children have an increased length of hospitalization as well as an increased dependence on total parenteral nutrition compared with those undergoing early distal pancreatectomy.

ERCP as a diagnostic and therapeutic option has recently gained some favor in selected centers with pediatric ERCP expertise. Documentation of a ductal injury, sphincterotomy, and possible stenting of the injury are all maneuvers useful for healing.

HOLLOW VISCUS INJURY

Gastrointestinal tract injuries in children may occur by either blunt or penetrating mechanisms. Penetrating injuries to the abdomen account for less than 10% of cases of abdominal trauma in most series. They require minimal diagnostic evaluation before operative management if penetration of the peritoneal cavity is evident, if the child has a clinical examination consistent with peritoneal irritation, or in the unstable child. Blunt intestinal injury may be seen with obvious indications for surgical intervention or may have a more subtle, insidious presentation. The mechanism may be from a compressive force (crush injury or child abuse), a

deceleration shearing force (fall from a height), or a combination of the two (lap belt injury).

CT diagnosis of a bowel injury is often quite difficult. CT findings have been described in an effort to improve its diagnostic accuracy in victims of blunt abdominal trauma. Bowel wall thickening and enhancement, mesenteric stranding, and free intra- peritoneal fluid in the absence of solid organ injury should alert the caregiver to a possible hollow vis- cus injury. Pneumoperitoneum often is not present, even with a full-thickness bowel wall disruption. Virtually all neurologically intact patients have findings or symptoms suggestive of peritoneal contamination from a perforated viscus, such as pain, tachycardia, rebound tenderness, or guarding. Laparoscopy can be useful for diagnostic purposes in some patients. The unconscious patient with multiple injuries presents a significant diagnostic challenge, and a high index of suspicion is needed. These neuro- logically injured patients may benefit from DPL if the abdominal CT shows suspicious findings but laparot- omy poses substantial risks due to associated injuries.

Children who have a visible contusion of the anterior abdominal wall from a seat-belt lap restraint have been documented to have an increased incidence of abdominal injuries. These include both solid and hollow abdominal organs as well as associated fractures of the lower thoracic and lumbar spine. These children must have frequent in-hospital assessments, serial physical examination by the same surgeon, and diagnostic imaging to assess for abdominal and spinal injuries. Several series have documented 80% to 100% of children with major blunt intestinal injuries who had evidence of abdominal wall ecchymosis. The presence of a visible contusion does not, in itself, mandate exploration but should prompt careful observation.

Injury to the Stomach

Injuries to the stomach due to penetrating mechanisms are variable in the amount of tissue destruction and are dependent primarily on the velocity of the offending missile or penetrating object. Blunt injury to the stomach, which is relatively infrequent, occurs when a compressive force causes a burst injury in a patient with a full stomach following a meal.

The diagnosis of a gastric injury from blunt force is often problematic. Free air may not be seen on the initial radiograph or CT scan. A nasogastric tube lying outside the stomach contour on a radiograph or CT scan is diagnostic. The child with abdominal wall ecchymosis, tenderness, and/or free fluid in the peritoneal cavity should prompt the suspicion of hollow viscus injury.

Surgical exploration for a suspected gastric injury is initiated with an upper midline laparotomy incision. The stomach and duodenum should be adequately mobilized, and hemorrhage should be controlled. Par- ticular attention should be paid to the lesser curve and to the posterior stomach near the gastroesophageal junction, which are sites of possible missed injuries. The lesser sac should always be opened to explore the posterior wall of the stomach adequately. Owing to the excellent blood supply of the stomach, repairs usually heal well.

Injury to the Duodenum

Duodenal injuries may be extremely difficult to manage because of the intimate association with the pancreas, extrahepatic biliary system, and intra-abdominal vascular systems. Thorough mobilization is key to identifying injuries, both blunt and penetrating. The technique used to repair a duodenal injury is dependent on the overall status of the patient, the location of the injury, and the amount of tissue destruction encountered. Extravasation of air or enteral CT contrast material into the paraduodenal, pararenal, or retroperitoneal space is the key finding in those patients with a duodenal perforation. This is not seen in patients sustaining a duodenal hematoma.

An intramural duodenal hematoma due to blunt force applied to the epigastrium (kick, punch, handlebar) may be managed nonoperatively if the child has no evidence of full-thickness injury or peritonitis. The CT scans or upper gastrointestinal contrast studies typically reveal duodenal narrowing, spiraling, or partial obstruction of the duodenum. No contrast or air is seen outside the bowel lumen. When managed nonoperatively, nasogastric decompression and total parenteral nutrition may be required for an average of 1 to 3 weeks. If a duodenal hematoma is encountered during exploration for additional abdominal trauma, the serosa may be incised and the clot carefully evacuated, taking care not to enter the duodenal lumen.

Full-thickness duodenal injuries may be closed primarily if the amount of tissue destruction is not excessive and if the closure will not compromise the duodenal lumen or drainage of the biliary and pancreatic ductal system. Adequate surgical drainage of the paraduodenal area is important if a pancreatic injury or major duodenal tissue devitalization has occurred. Duodenal closure may be obtained with the aid of a Roux limb of jejunum sewn to the viable debrided duodenum. A duodenal drainage tube for decompression is left in place. Temporary pyloric exclusion with an absorbable suture via a gastrotomy also may be valuable in the management of a complex duodenal repair. This allows healing of the duodenum before spontaneous recanalization of the pyloric channel once the pyloric sutures resorb. A gastrostomy tube for decompression may also be utilized instead of a nasogastric tube. Placement of a feeding jejunostomy will be helpful for enteral nutritional support. Surgical drains placed near the injured tissue at the time of initial repair will control a fistula resulting from any enteric leak. Somatostatin analogs may be helpful in decreasing pancreatic and intestinal secretions and are useful in management of a fistula should one occur. Rarely is a pancreaticoduodenectomy required for blunt trauma in children.

Small Bowel Injury

Penetrating injury to the abdomen frequently injures the small intestine. The small bowel must be carefully and systematically visualized for mesenteric and bowel wall injuries. Extensive injuries should be managed by resection and anastomosis. Isolated perforations and lacerations may be repaired with debridement and closure, with care to avoid luminal narrowing.

Hematomas in the mesentery should be explored, and control of bleeding vessels should be meticulously performed. If a segment of bowel is in jeopardy because of a mesenteric injury, bowel resection and primary anastomosis should be performed to avoid stricture formation. Injuries to the superior mesenteric artery or vein should be repaired with a vein patch as needed to avoid stenosis of the affected vessel. Delayed diagnosis of injuries may also be seen with a delayed bowel infarction due to rapid deceleration injury with avulsion of the bowel from its mesentery. A late sequela of blunt intestinal injury is intestinal stricture.

Crush injury to the bowel wall results in ischemic injury and resulting scarring and fibrosis. Intolerance of feeding, bilious emesis, and evidence of a bowel obstruction 1 to 6 weeks after blunt abdominal trauma is the most common presenting scenario.

Injury to the Colon

Injuries to the colon in children may occur by the same mechanisms as small bowel injuries, but the consequences may be more significant. Delayed diagnosis with peritoneal contamination may result in severe and life-threatening septic complications. If isolated colonic injuries are identified and repaired early in a hemodynamically stable child, a primary bowel anastomosis with appropriate perioperative antibiotic coverage is safe. This approach also avoids the potential complications of stomas and the need for reoperation for stoma closure. Once a colonic injury has been identified and controlled, multiple factors influence surgical management. In the absence of shock or delayed diagnosis with severe fecal contamination, many colon injuries may be safely repaired primarily. A diverting stoma may be necessary if extensive abdominal wall or perineal tissue devitalization is present, if the child is in shock, or if severe peritoneal soilage has occurred. Care must be taken in selecting the site for placement of the proximal diverting stoma to avoid ongoing wound contamination.

Rectal Injuries

The etiology of the majority of perineal and rectal injuries in children is either accidental falls in a straddle fashion onto sharp or blunt objects or sexual abuse. Typically, rectal perforations require proximal diversion with an end colostomy and drainage of the distal injured perirectal space. Identification of these injuries may be difficult. The ability to ascertain the extent of the injury will frequently require a formal examination under general anesthesia. In the female, care is taken to carefully inspect the vaginal vault for evidence of a penetrating injury either through or into the vagina. Proctosigmoidoscopy in a nonpre- pared colon may not clearly localize an obvious perforation, but the presence of endoluminal blood must be assumed to be evidence of injury. After proximal colostomy and presacral drainage has been performed, the distal rectum should be cleared of any remaining feces. Meticulous repair of the injured anal sphincter musculature must be performed. Any nonviable tissue should be debrided at this setting. Closure of the rectal injury or perforation may be accomplished transanally if the injury is low enough to permit this approach.

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Topic 4. Purulent surgical infections in children

Topic: Peculiarities of the purulent infection in newborns (phlegmon, mastitis, omphalitis, paraproctitis). Hematogenous osteomyelitis in children. Bacterial destructive pneumonia and its complications (pyothorax, pneumothorax). Etyology, pathogenesis, classification, diagnostics and treatment

I. Topic actuality, a professional orientation.

Among pyo-inflammatory diseases of childhood osteomyelitis occupies one of the top-level positions by prevalence and severity of the clinical course.

Anatomic and physiological features of newborns, uncontrollable use of the antibacterial therapy, a problem of an intrahospital infection promote that a frequency of purulent diseases in newborns remains on high level. A danger of the purulent infection in newborns is caused by imperfection of the protective mechanisms of the newborn organism, possibility of damage of the growing structures and organs that can lead to early complications as well, as to late ones.

Children most often have hematogenous osteomyelitis and generaly among all the patients with this disease the children take 75%. Acute hematogenic osteomyelitis may lead to the different complications, especially in small children and bring to development of acquired complications.

Diseases of the lungs occupy one of the leading places among all causes of the pediatric morbidity and mortality. The most frequent pulmonary disease is a pneumonia. The majority of the patients with pneumonia are treated by pediatrics, but sometimes the course of pneumonia are followed with complications, required the surgical interventions. This is a bacterial destructive pneumonia.

II. The educational purposes of the study.

a) ***As a result of the study higher education applicants should know theoretically:***

2. Pathogenesis of acute hematogenous osteomyelitis in cyildren.
3. Clinical forms of acute hematogenous osteomyelitis.
4. Clinical picture and peculiarities of different clinical forms.
5. Early methods of diagnostic of acute hematogenous osteomyelitis.
6. X-ray symptoms of acute hematogenous osteomyelitis.
7. Principles of treatment of acute hematogenous osteomyelitis.
8. Peculiarities of course of acute hematogenous osteomyelitis in children under 3-years old.
9. What means methaepyphiseal osteomyelitis?
10. What methods of diagnostic of acute hematogenous osteomyelitis in children under 3-years old?
11. What complications may develop after acute hematogenous osteomyelitis in children under 3-years old and

in older children?

12. What means primary and secondary chronic osteomyelitis?
13. What X-ray signs of secondary chronic osteomyelitis?
14. Atypical forms of chronic osteomyelitis.
15. Treatment of primary and secondary chronic osteomyelitis.
16. Higher education applicants of V year must know etiology and pathogenesis of bacterial destructive pneumonia, its clinical symptoms, diagnostic methods and methods of treatment in children with different forms of the bacterial destructive pneumonia, make a differential diagnostics with other diseases.
17. Classification of the purulent diseases in newborns.
18. Features of the structure of skin in newborns.
19. The plan of the examination of newborn.
20. Clinical symptoms of the necrotic phlegmon of newborns.
21. Treatment tactics in newborn with necrotic phlegmon of newborns.
22. Clinical picture, differential diagnostics and the plan of treatment of mastitis in newborns depending on its stage.
23. Classification of omphalitis, clinical picture of the different clinical forms and treatment depending on the stage of disease.
24. The reasons for development of paraproctitis and its classification.
25. Clinical picture of the acute and chronic form of paraproctitis.
26. Treatment of the acute and chronic form of paraproctitis.

b) **Higher education applicants** ***must know to do practically***

1. To issue the case record.
2. To carry out clinical researches.
3. To take the anamnesis.
4. To interpret the X-ray film.
5. To make immobilization of extremity.
6. To make a plan of treatment.

7. As result of the lecture, a philosophic rationale to management of children with surgical diseases must be formed, that is based on the main diagnostic and treatment principles of the national school of pediatric surgery.

8. To collect the anamnesis of disease in the presence of signs of the purulent infection in newborn.

9. To exam a newborn .

10. To make surgical intervention in newborn with necrotic phlegmon of newborns.

11. To make a differential diagnostics of mastitis and physiological edema of mammary glands in newborn.

12. To estimate a stage of mastitis and appoint the necessary therapy.

13. To appoint the treatment plan of omphalitis depending on its stage.

14. To estimate a stage of paraproctitis, define its form and make the treatment plan.

3. Questions for training.

1. Newborn of 13 days of life is carried out to the intensive care department with increasing of temperature, arthritis of right elbow joint and pseudoparalisis of the right arm. What is your diagnosis?

2. The 13-year old boy has an osteomyelitis of the left fibula. His condition becomes worth, temperature increases, appear signs of intoxication. What complication may develop in acute hematogenous osteomyelitis?

3. The 6-years boy has been suffering from acute hematogenous osteomyelitis for 14 days. What X-ray signs should appear in this period?

4. In X-ray film of femur in the lower methaphysis is visualised oval cavity with clear borders. What is your diagnosis?

4. The plan and organisation of lessons.

		Activity		
		Higher education applicants	Teacher	
Organisation of the practical class	5	Listen	Presence control	Journal
Aim setting	5	Listen	Explain	
Checking of the knowledge of higher education	15	Make	Control	Tests

applicants				
Seminar	60	Answer	Control	X-ray-grams
Clinical discussion	30	Listen	Discuss patients	Patients
Personal work of higher education applicants	60	Make	Control	Patients
Results of the lesson	10	Listen	Appreciation of each activities	
Task for the next lesson	5	Listen	Explain the task. Indicates the literature.	

Bacterial destructive pneumonia and its complications (pyothorax, pneumothorax). Etyology, pathogenesis, classification, diagnostics and treatment"

№	Main parts of the lecture	Aims	Lecture type, lecture contents	Time
	Preparative part			5 %
1	Educational aims presentation			
2	Formation of positive motivation			
	Main part		Multimedia presentation	85 %
3	Bacterial destructive pneumonia, its classification.	II		
4	Etiology and pathogenesis of the bacterial destructive pneumonia	III		
	Pulmonary complications.			
5	Pleural complications.	III		
6	Treatment of the bacterial destructive pneumonia	III		
7		III		
	Final part			10 %
8	Conclusions of the lecture			

9	Higher education applicants' questions			
10	Tasks for independent work of higher education applicants			

Diseases of the lungs occupy one of the leading places among all causes of the pediatric morbidity and mortality. The most frequent pulmonary disease is a pneumonia. The majority of the patients with pneumonia are treated by pediatrics, but sometimes the course of pneumonia are followed with complications, required the surgical interventions. This is a bacterial destructive pneumonia.

Classification of the BDP:

Route of infection:

1. Primary – by an aerogenous route through the bronchi,

2. Secondary - by hematogenous route from other purulent focus.

Infecting agent:

1. Gram-negative.

2. Gram-positive.

3. Mixed flora.

Complications:

1. Pulmonary:

a) abscess,

b) blebs (residual cavities).

2. Pleural:

a) exudative pleuratis,

b) pyothorax – local and total,

c) pneumothorax – tension and non-tension,

d) pyopneumothorax – tension and non-tension.

3. Emphysema of mediastinum.

3. Materials for preliminary preparation of higher education applicants:

3.1. Basic base knowledges, abilities, skills necessary for independent study, which are based on the interdisciplinary communications:

№	Discipline	to know	to be able
1	Anatomy	Structure of the skin and other soft tissues of newborn, structure of the rectum and surrounding adipose tissue.	
2	Pathological physiology	The local and general changes caused by purulent infection in newborns	
3	Surgery		Surgical technique in adults with phlegmons, abscesses, mastitis and paraproctitis
4	Propaedeutics of the pediatric diseases	Features of the immunity in newborns.	Survey and inspection of the newborn
5	Pediatrics	Physiological conditions of the newborn period. Diseases of the skin in newborns.	
6	Infectious diseases	Clinical picture, diagnostics and treatment of erysipelas.	

3.2. The contents of theme is expounded in the recommended literature.

3.3. Recommended literature:

1. Ashkraft T., Kholder T. Pediatric surgery, II vol.. - SPb. - 1997. - 406 p.

2. Isacov Yu.F. Surgical disease in children. - M.- 1988.- 415 p.

3. Nikolayeva N. G. Pediatric surgery. - Odessa. - 2004. - 180 p.

3.4. Reference card for independent preparation of higher education applicants with the use of literature on the theme.

Basic tasks	Instructions	Answers
Features of the structure of the skin in newborns	Protective properties of the skin, features of the blood supply	
Clinical picture of necrotic phlegmon of newborns	Clinical symptoms (simple and septic forms), survey data, differential diagnostics with erysipelas, sclerema,	

	hypodermic adiponecrosis.	
Treatment of necrotic phlegmons of newborns	1. Local treatment (features of incisions). 2. General treatment.	
Classification and clinical picture of omphalitis	Symptoms of «wet umbilicus», catarrhal, phlegmonous, and gangrenous omphalitis	
Treatment of omphalitis	Describe the local and general treatment depending on the stage of disease	
Mastitis of newborns	Classification of clinical forms, differential diagnostics with physiological edema and treatment depending on its stage.	
Classification of paraproctitis	Definition of the acute sharp and chronic paraproctitis. Anatomic forms of the acute paraproctitis. Types of fistulas.	
Treatment of paraproctitis.	1. Treatment of the acute form. 2. Treatment of the chronic form.	

3.5. Materials for self-control.

3.5.1. Questions for self-control.

1. Why is the necrotic phlegmon diagnosed in newborns only?
2. What are features of incisions at necrotic phlegmon of newborns?
3. What complications can develop in newborns with necrotic phlegmon?
4. What diseases are followed with discharge from the umbilicus?
5. Name clinical forms of mastitis.
6. Describe the typical symptoms of the physiological edema of mammary glands.
7. Name the features of incisions, performed over a mammary gland.
8. What anatomic localization of acute paraproctitis are found most often?
9. What is a treatment of acute paraproctitis?

10. What symptom is the main sign of chronic paraproctitis?

11. What is a treatment of chronic paraproctitis?

3.5.2. Test tasks for self-control.

3.6. The subject for scientific work on this theme:

1. Estimation of the efficiency of conservative treatment of newborns with omphalitis.

2. Estimation of the local and general immunity in patients with different clinical forms of omphilitis.

3. Efficiency of the surgical treatment of chronic paraproctitis in children.

Substance of the lesson.

Among pyo-inflammatory diseases of childhood osteomyelitis occupies one of the top-level positions by prevalence and severity of the clinical course.

Children most often have hematogenous osteomyelitis and in general among all the patients with this disease the children make up 75%. In 70% of cases the pathological process affects the long tubular bones, primarily those with the intensive growth (femur, tibial bone, shoulder) and, as a rule, starts with the metaphysis.

Hematogenous osteomyelitis disease producing germ mostly is staphylococcus. At first the process develops as a monoinfection, but with the course of time in most cases flora becomes mixed, the microbial associations predominate.

The pathogenesis.

The entrance of the infection may be pyo-inflammatory diseases of the skin, mucous membranes, tonsils, ears and so forth. With the blood flow the microbes are carried into the organs with an abundant blood supply and hampered by the cells of reticuloendothelial system. A sedimentation of microorganisms exactly in metaphyses of long tubular bones is promoted by a great number of macrophages which are red marrow is rich in and by physiological slowing down of blood flow in the bloody lakes – sinusoids where a stasis is observed up to 30 – 40 sec.

Under a prolonged infections persisting, additional infecting, action of provoking factors (trauma, supercooling) against a background of immunological insufficiency, the microflora of metaphyseal cells realizes its pathogenic properties. First of all it causes damage of intraosseal vascular wall under the action of microbial toxins and enzymes. The alteration of vascular wall leads to the exit of microbial toxins outer vascular space, where the exudative sings manifest. The exudates accumulation in a closed rigid osteal cavity results in an increase of intraosseal pressure in 2-3 times, that in its turn stipulates an occlusion of intraosseal vessels from the outside, first of all in haversian canals, i.e. the bone's own blood supply system suffers affliction first of all. So the 1-st phase of hematogenous osteomyelitis is the ***marrow edema*** and develops 1-2 days.

In the 2-nd phase (***medullar phlegmon***) exudates transforms to pus. Intraosseal pressure increases up to 300-500mm H_2O (normal rate 60-80 mm H_2O). These make worth trophy disorders, intraosseal necrosis

and distribution of the pathological process from metaphysis into diaphysis.

At the 4-5th day the 3rd phase - ***subperiostal phlegmon -*** of hematogenous osteomyelitis develops. Pyoexudate along Volkmann's canal breaks an osteal cortical layer, flows off under a periosteum, causes its inflammation (periostitis), and shifts a periosteum on the stretch that it is turn disturbs subperiosteal blood supply, aggravates local trophic violations and becomes an additional factor of necrotic process.

Phlegmon of soft tissues is the 4th phase. It characterized by a periosteum necrosis, output of pus via periosteum in paraossal tissues (into intramuscular or hypodermic layer) on the 6-7th day of the disease.

According to the clinical course hematogenous osteomyelitis divided into acute, subacute and chronic. Depending on the clinical signs intensity T.P. Krasnobayev distinguishes toxic, septicopyemic and local forms of acute hematogenous osteomyelitis.

Toxic (superacute, adynamic, lightning) form is characterized by the symptoms of toxicosis and microcirculation disturbances caused by endotoxic shock. Hyperthermia, excitation, hallucinations suddenly develop in the child, he faints consciousness, sometimes cramps and vomiting occur. At first the skin is acyanotic, acrocianosis is observed, then petechias develop. The breathing becomes surface, frequent. Tachicardia, progressive decrease of arterial pressure, impossibility of pulse definition on peripheral arteries, olyguria, anuria develop. As a result "shock lung", "shock kidney", brain edema develop. The syndrome of disseminated intravascular coagulation appears. The local clinical signs cannot develop. In 70% of cases toxic form of hematogenous osteomyelitis causes fatality during first 2-3 days of the disease.

The ***septicopyemic*** form of hematogenous osteomyelitis is also accompanied by expressed toxicosis, but homodynamic damages are not as fatal as in the lightning one. The onset of the disease is acute, hypertermia, collapse-like attacks, pathological changes on the part of the central cerebral system (disorientation, excitation, delirium), pneumonia, and instability of hemodynamics take place, soon the liver and spleen enlarge, and the icterus can join. General clinical signs of toxicosis are expressed, but they are relatively stable, without rapid progression. The local clinical signs of osteomyelitis are rather precisely determined since the 1st day of the disease. There are megalgia in the aria of impact zone, disturbance of joint function, pain contraction, increase of local temperature and pastosity of paraosseal tissues. Multiple pyemic foci can emerge at once, simultaneously with the onset of disease (some damaged bones, bilateral pneumonia, carditis) or reappear after a while (secondary metastasing of infection).

The ***local*** form is characterized by a prevalence of local signs above total ones. But therewith the child is fevering and his general condition can be severe. The onset of disease is characterized full-blown pain syndrome of a hypertensive genesis gradually passing to classic sings of a pyo-inflammatory process though after a spontaneous decompression of a medullar phlegmon in paraosseal tissue the pain becomes a bit weaker.

Diagnosis of acute hematogenous osteomyelitis includes early methods and late. Puncture of an inflamed bone is the early and crucial important method. On its basis one can define medullar pressure (presence of a medullar hypertension); evaluate macroscopic investigation of a punctate; conduct a microscopic investigation (express bacterioscopy, cytologic investigation); establish etiological diagnosis (bacteriological investigation); evaluate pH. The laboratory investigations (haemogram, proteinogram, immunigram, biochemical indices…) testify to general inflammatory process, its intensity, intoxication manifestation and character of immunological disturbances.

As to X-ray examination within first two weeks of the disease, the roentgen negative phase of hematogenous osteomyelitis takes place, during this time one cannot observe any osteal changes. On the 10-14th day from the onset of the disease X-ray signs appear (roentgen positive phase). They include a spotted osteoporosis, and linear periostitis.

In contrast to pathological process in children of a school age in babies it develops in a little bit different way. Starting traditionally with metaphyses further inflammation is distributed into epiphysis through a zone of growth. A zone of growth in babies is immature, the chondroblasts are arranged chaotically, they regulation takes place parallel to epiphysis ossifications and according to it the zone of growth gains step-by-step barrier properties. That is why under the lack of ossification nucleus due to morpho-functional immaturity of the growth zone the inflammatory process penetrates its central part, if the ossification nucleus appears, penetration takes place in a peripheral part of the growth zone. That is why in the little kids we speak about metaepiphysial one with different sings and complications.

The damage of epiphysis entails a destruction of the latter, involving a joint into a pathological process and an origin of arthritis, which in its turn to pathologic dislocation and paraarticular phlegmon. A discredited zone of growth is partially or completely perished and later on it stimulates the development of acquired malformations. The destruction of epiphysis leads to the defects of joint ends, formation of destructive dislocations, instability in joints.

Clinical picture of acute hematogenous osteomyelitis in babies is different with it in older children. In baby's disturbance of general condition, increase of temperature, flexibility, paleness, and sometimes icterus, increase of liver and spleen, dyspepsia testify to a toxicosis. A pain equivalent is anxiety, which intensifies at motions. The damaged limb takes an enforced position by a **"pseudoparesis"**, active mobility is reduced, the passive motions cause the kids anxiety and cry. At the 2-3rd day from the onset of the disease they determine pastosity of paraarticular tissues, later on their edema, hypertermia, which is conditioned by metaepiphiseal localization of the pathological process with an involvement of a joint into inflammation and development of the main sign of acute hematogenous osteomyelitis in babies – **arthritis**.

In little kids X-ray negative phase is shorter than in older children and takes 7-10 days. Infiltrative changes of paraosseal tissues, widening of the slit of the affected joint and dislocation in it point to the inflammatory process. Later on in a X-ray positive phase the segment of subphisar destruction of metaphysis with penetration through a zone of growth into epiphysis, signs of periostitis are determined. For early diagnostic of osteomyelitis in babies we should use the puncture of joints with bacteriological, macro- and microscopic investigations.

Treatment of acute hematogenous osteomyelitis is executed according to the directions formulated by T.P.Krasnobayev:

1 Effect on the macroorganism

2 Effect on the microorganism

3 Effect on the focus of disease.

Effect on the macroorganim provides disintoxication, support and stimulation of immunological forces of the organism, syndromatic therapy, desensibilisation and vitamin therapy.

The effect on the microorganism is carried out by rational antibacterial therapy.

The local treatment includes:

3 timely and adequate local decompression;

4 local sanitation (antibacterial therapy);

5 immobilization of affected extremity.

Decompression of intraosseal focus is advisable to conduct by injection into the damaged segment of constant needles designed by K.P.Aleksyuk. The needles allow not only to drain a bone softly, but to inject the antibiotics, antiseptics directly into the focus of inflammation.

In subperiostal phlegmon or phlegmon of soft tissues there is indicated applying of sparing cuts (up to 2 cm.) and drainage. If arthritis develops in babies they should be punctured.

The purpose of fixation is not only maintenance of the damaged with the osteomyelitis segment, but also the prophylaxis of such complications as pathological fracture, dislocations, immobility of joints. Immobilization also decreases painful feelings in affected extremity.

To the **complications** of acute hematogenous osteomyelitis the false joint (it forms after a pathological fracture), destructive dislocation and instability in joints (due to complete or partial destruction of epiphysis), growth disorders and deformation of bones (on the basis of complete or partial destruction of growth zone) are referred.

Chronic osteomyelitis.

Osteonecrosis in the terms from 1 to 4 months is separated by granular tissues from the healthy bone, transforming in sequester. Depending on the volume of blood supply disturbance sequesters may be: total, central and surface laminar. Chronic stage of hematogenous osteomyelitis takes next changes: cambial layer of the saved periosteum proliferates, gets thicker, harder, foci of sclerosis appear. Around central osteonecroses plastic process also take place. It results in derivation of intraosseal cavities, “sequestral coffins”, “osteal sarcophagus” in which pus and granular tissue “eat away” the holes, through which pus and the small sequesters evacuate out, creating fistulas.

Chronic osteomyelitis is divided on **primary and secondary** one. If the pathologic process lasts more 4-6 months, we talk about transition to the chronic stage – the secondary chronic osteomyelitis.

Secondary chronic osteomyelitis is characterized by alternation of remissions and exacerbations. At the phase of remission the child feels well, he has no complains, the temperature of the body is normal. Among the local data one can determine only moderate increase of the volume of damaged segment. In the phase of exacerbation the patient’s condition is worsening, the temperature of the body increases, the signs of intoxication aggravate, the pain appears in locus morbid and other signs of inflammation occur. Soon paraosteal phlegmon or the fistula opens, from which the pus and sometimes small sequesters discharge. Such an alternation of phases of remission and exacerbation may last for years and initiate distrophic damages of kidneys, liver, heart as well as convert into chroniosepsis.

The X-ray examination of the secondary chronic hematogenous osteomyelitis reveals both destructive

and proliferative changes of a bone, such as sclerosis, eburnation, cavitary intraosteal formations with sequesters. The fistulography helps to determine the fistula channel and its connection with intraosteal focus of inflammation and sequester.

Unlike the secondary chronic process, which is preceded by the acute hematogenous osteomyelitis, at primary-chronicle course of the disease there is no acute phase, from the very beginning the disease is characterized by "cold" course and "blurred" clinical symptoms. Discomfort, slight pains for a long time remain non-localized, the symptom of "sparing" of a damaged limb, an increase of the temperature to mild pyrexia may take place.

Among primary chronic hematogenous osteomyelitis (its another name – *atypical osteomyelitis*) there are distinguished the following: Brodie's abscess, Garre's osteomyelitis, Ollier's (albuminous) osteomyelitis, Popkorov's (antibiotic) osteomyelitis, tumor-like osteomyelitis, Probst's (plyfocal) osteomyelitis.

All kids of chronic osteomyelitis both primary- and secondary- are subjected to surgical treatment. Surgical intervention includes radical necrectomy, intraoperative sanitation, multiple puncturing of the remained osteal walls. If necessary an operated-on segment is stabilized with a spire distraction-compressive apparatus.

Bacterial destructive pneumonia

Microbes, reaching the pulmonary tissue, begin to produce the different toxins (one of them – necrotoxin) and proteolytic ferments, which cause the tissue necrosis and formations of the purulent cavities. These cavities join and form the pulmonary abscess. The clinical course of the abscess has two stages. The first one (formation of abscess or undrained abscess) is followed with severe clinical symptoms of the respiratory insufficiency and intoxication: shortness of breath, tachypnea, cyanosis, tachycardia, high temperature, raised white blood cells (WBC) level and erythrocyte sedimentation rate (ESR).The X-ray shows the round shadow, that occupies a few segments or entire pulmonary lobe. If conservative treatment (antibiotherapy, disintoxication) is ineffective, the puncture of the undrained abscess is indicated.

The second stage of the abscess is a drained abscess. Usually the abscess drains into the bronchi, what is followed with the violent cough with pus, decreased temperature and improvement of the patient condition. The X-ray shows presence of air into the abscess cavity. If the bronchial fistula within the bronchus and abscess cavity is wide and the pus leaves the cavity rapidly the conservative treatment is used. This includes the antibiotherapy, bronchiolitic inhalation, expectorants, postural drainage.

After the successful treatment of the pulmonary abscesses the residual air-filled cavities (blebs) are present into the lung. These cavities need no special treatment and usually disappear in 3 – 4 months.

The pneumonia almost always is followed with a serous exudate accumulation in pleural cavity. In case of the destructive pneumonia the suppuration of the exudate happens and it becomes purulent. This is a pyothorax (the pus accumulation in the pleural cavity). The pyothorax is most frequent complication of the bacterial destructive pneumonia. If auscultating a child with severe pneumonia (respiratory insufficiency, fever, intoxication) you found the weak or absent breath sounds and don't hear the moist rales, this usually means a presence of the pus into pleural cavity. At the roentrenograms the lung field shadow (local or total) is visible. This shadow closes the pleural sinus and has an oblique upper line. In case of the total pyothorax the upper line reaches the pleural top. To confirm the diagnosis the pleural puncture at the 6^{th} or 7^{th} intercostal space by linia axillaris media or posterior should be done. Presence of the pus in the pleural

cavity confirms the diagnosis of pyothorax, what indicates a necessity for the pleural tube insertion (drainage of the pleural cavity).

Sometimes the abscess cavity empties into the pleural cavity with formation of the bronchial fistula between the bronchus and pleural cavity. This situation leads to pus and air accumulation into the pleural condition. This complication is known as pyopneumothorax. This is complication is more severe than the pyothorax. In this case the severe condition of the patient with pneumonia deteriorates significantly and may be life-threatening. The dyspnea increases, cyanosis and apprehension appear, the accessory muscle help to breathe. The auscultation reveal the absence of breath sounds, although a few hours before the moist rales and coarse breath sounds were heard. During the percussion the tympanic sound, which indicates presence of the air, is found. The tension pyopneumothrax is followed with progressive air accumulation in the pleural cavity, what causes the mediastinum and heart shift to the opposite side. The tension pyopneumothorax is life-threatening condition, causing the acute cardiac and respiratory insufficiency. The X-ray shows the the air and pus presence into the pleural cavity with a clear horizontal line between them. The lung is compressed. In the case of the tension pyopneumothorax the shift of the mediastinum and heart to the healthy side is visible. The treatment in this case is emergency and includes the pleural tube insertion. The system of the passive aspiration should be applied.

In case of the pneumothorax the air accumulates it the pleural cavity. Like the pyopneumothorax it may be tension and non-tension and requires the puncture of the pleural cavity to remove the air. The puncture is done at the 2^{nd} or 3^{rd} intercostal space by linia subclavia media. Sometimes a few puncture should be done.

The emphysema of mediastinum is a rare complication of the bacterial destructive pneumonia. The presence of the air in the mediastinum is always followed with its spread to neck, where the subcutaneous emphysema is visible. This symptom and X-ray, which shows the presence of air in the mediastinum, allow to make a correct diagnosis. The local treatment of the emphysema is a suprajugular mediastinotomy and drainage of the mediastinum.

All these complication of the pneumonia require a general treatment as well, as mentioned above local treatment. The general treatment includes the antibiotherapy, infusion therapy, symptomatic therapy. The antibiotherapy is begun with wide-spread antibiotics, then this therapy is adjusted due to results of the microbial sensitivity. The intravenous route for antibiotherapy is preferable. Quite often the children with bacterial destructive pneumonia need the oxygen. The nasal cannulas or oxygen tent are used for this purpose. The severe cases may require the ventilator support.

The secondary pneumonia develops as a complication of other purulent diseases. The most common among these diseases is an osteomyelitis. Usually the bacterias reach the lungs through the hematogenous route. Such pneumonia have a double-side localization and may be followed with any above-mentioned complication (pyothorax, pyopneumothorax and the rest).

5. Material to promote higher education applicants' attention on the lecture.

11. What are the clinical symptoms of respiratory insufficiency?

12. What classification of pneumonia do you know?

13. What other diseases may cause respiratory insufficiency in children?

14. What is X-ray picture of pneumonia?

15. What points a pleural puncture must be done at?

6. Material for independent work of higher education applicants.

1. Classification of the bacterial destructive pneumonia.

2. What microorganism cause the bacterial destructive pneumonia usually?

3. What are clinical symptoms of pulmonary abscess?

4. What are the differences of X-ray presentation of pulmonary abscess, pulmonary cyst and tuberculosis?

5. What is a treatment of the pulmonary abscess?

6. What are the main clinical symptoms of pyothorax, pneumothorax and pyopneumothorax?

7. What forms of pyothorax do you know?

8. What forms of pneumothorax do you know?

9. What are the principles of treatment at purulent diseases?

10. What are the blebs?

11. What are the symptoms of mediastinum emphysema?

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Topic 5. Peculiarities of oncology in children. Benign tumors

Theme: "**Peculiarities of oncology in children. Benign tumors.**

1. Actuality of theme: **Peculiarities of oncology in children. Benign tumors** represents a serious problem for modern medicine. This confusion among patients and physicians led the International Society for the Study of Vascular Anomalies to adopt a general, biologic classification scheme for vascular anomalies based on physical findings, natural history, and cellular kinetics. In this system, vascular anomalies are described as either malformations or tumors. Vascular tumors exhibit abnormal endothelial cell proliferation while malformations are products of abnormal embryonic vessel development.This schema presents a useful framework for discussing the diagnosis and treatment of vascular anomalies.

2. Educational aims:

Concrete aims:
1. To master the diseases which cause a tumors of soft tissues.
2. To recognize the basic clinical symptoms of tumors of soft tissues.
3. To differentiate a tumors depending on a kind.
4. To interpret principles of medical treatment of tumors of soft tissue and their complications.
5. To recognize the basic clinical symptoms of tumors of bones.
6. To identify the features of course of separate diseases on the basis of clinical and roentgenologic signs.
7. To differentiate the benign tumors of bones on the basis of clinical and auxiliary methods of examination (roentgenologic, ultrasound, computer tomography, angiography, pucture and open biopsy of tumor).
8. To work out a plan of inspection of patient and algorithm of actions on the exposure and treatment of complications of tumors of bones (pathological fracture, false joints, metastasis).
9. To interpret principles of medical treatment depending on the type of tumor of bone.

4. The plan and organisation of lessons.

Work type	Time	Activity		Equipment
		Higher education applicants	Teacher	
Organisation of the practical class	5	Listen	Presence control	Journal
Aim setting	5	Listen	Explain	
Checking of the knowledge of higher education applicants	15	Make	Control	Tests

Seminar	60	Answer	Control	X-ray-grams
Clinical discussion	30	Listen	Discuss patients	Patients
Personal work of higher education applicants	60	Make	Control	Patients
Results of the lesson	10	Listen	Appreciation of each activities	
Task for the next lesson	5	Listen	Explain the task. Indicates the literature.	

5. Substance of the lesson.

Benign tomors of soft tissues.

Determination of reasons of tumor growth, main clinical symptoms, localization of hemangiomas, lymphangiomas, pigmental tumors, dermoid cysts, ateromas, teratomas. Use of auxiliary methods of inspection. Medical tactic depending on the type of formations, treatment in case of complications apperance (puncture, control of bleeding, dissection of inflammation)

Methods of conservative medical treatment of hemangiomas (injection, cryotherapy, electro-coagulation). Surgical method of medical treatment, features of deleting of lympangiomas of neck, teratomas of sacro-coccygeal area.

Benign tumors and tumorous masses of bones.

Concept about the benign tumors and tumurous masses of bones: osteoma, oeteoid, osteochondroma, osteoblasoclastoma, fibrous dysplasia, cysts of bones. Features of clinical course depending on the type of formations, complications (pathological fracture, false joints, deformations). Differential diagnostics of diseases. Principles of surgical medical treatment, types of resection of the bones, the plastic operations. Conservative medical treatment of cysts of bones.

Bone and Soft Tissue Tumors in Children

Osteosarcoma. Common (red) and less common (orange) locations are shown. The age of onset in decades is shown in blue.

We do not know exactly what causes children to develop these tumors. Only about two in every 10 children who get malignant bone tumors have a family history of the condition. Benign bone tumors are not inherited, except for the tumor called exostosis.

Children between the ages of 6 and 12 are the most likely to develop benign bone tumors, although the tumors sometimes show up in children as young as age 2.

Exostosis tumors are slightly more common in boys than girls.

Ewing sarcoma. Common (red) and less common (orange) locations are shown. The age of onset in decades is shown in blue.

The bone cancers osteosarcoma and Ewing sarcoma usually begin when children are young teenagers. This is a time when their bones are growing very rapidly and they often are taking part in sports and other physical activities. The most common age at which children are diagnosed with these cancers is 14.

Boys are slightly more likely to get a malignant bone tumor than girls.

Malignant soft tissue tumors are more common in teenagers. Children who are younger than age 6 are most likely to get benign soft tissue tumors.

Bone and Soft Tissue Tumors at Seattle Children’s

To ensure that your child’s diagnosis and treatment is the best it can be, it is important to choose a medical center and doctors who specialize in pediatric bone and soft tissue tumors.

Seattle Children’s has the first clinic in the United States where doctors who specialize in bones, joints and muscles (orthopedists) and specialists in cancer (oncologists) work together in the same program. They are called a multidisciplinary team, and these teams are an important part of Children’s care.

For 20 years, our multidisciplinary team of doctors, nurses and staff in the Bone Tumor and Sarcoma Clinic has specialized in diagnosing tumors and providing exceptional care for children.

Each year we see about 350 new patients at our Bone Tumor and Sarcoma Clinic. About 90% of them have benign bone tumors.

Between 30 and 40 of the children we treat every year have new malignant bone tumors, and each year we diagnose and care for about 30 children with malignant soft tissue tumors.

Doctors from throughout North America refer children to us. We see your child within one week of the call for an appointment. We develop a plan for treatment at your child's first visit.

While our clinic is specifically for children and teens, we work closely with adult sarcoma programs. We share expertise and access to highly sophisticated equipment with UW Medicine.

Infantile Hemangioma

Infantile hemangiomas are benign vascular neoplasms that have a characteristic clinical course marked by early proliferation and followed by spontaneous involution. During the proliferative phase in the neonatal period or early infancy, a rapidly dividing endothelial cell proliferation is responsible for the enlargement of infantile hemangiomas. Finally, an involutional phase occurs, whereby most infantile hemangiomas are clinically resolved by age 9 years.

Hemangiomas are the most common tumors of infancy, and most infantile hemangiomas are medically insignificant. Occasionally infantile hemangiomas may impinge on vital structures, ulcerate, bleed, cause high-output cardiac failure or significant structural abnormalities or disfigurement. Rarely, a cutaneous infantile hemangioma may be associated with one or more underlying congenital anomalies.

Pathophysiology

Infantile hemangiomas are composed of proliferating, plump endothelial cells. Early in proliferation, the cells are in disarray, but, with time, they form vascular spaces and channels replete with blood cells (see image below).

Histopathology of a proliferating infantile hemangioma with plump endothelial cells in the dermis.

These benign-appearing endothelial cells produce limited basement membrane structures. Hemangiomas assume a lobular architecture as proliferation slows and ends. Mast cells appear to affect this process and are implicated in the promotion of feeding arterioles and veins that supply each lobule. They also have been found in high concentrations during involution.

Takahashi hypothesized that during the third trimester of fetal development, immature endothelial cells coexist with immature pericytes, which maintain their proliferative capacity for a limited period during postnatal life.1 Angiogenic peptides, such as beta-fibroblast growth factor, vascular endothelial growth factor (VEGF), and proliferating cell nuclear antigen, induce proliferation of these immature cells, resulting in the development of the hemangioma. As the endothelial cells differentiate, an influx of mast cells, various myeloid cells, and tissue inhibitors of metalloproteinases (TIMPs) occurs.2 TIMPs, along with interferon and transforming growth factor produced by the mast cells, terminate the endothelial cell proliferation and passively induce involution by senescence of endothelial cells.

Infantile hemangiomas occur in 10-12% of white non-Hispanic infants, 1.4% of black infants, and 0.8% of Asian infants. The incidence of infantile hemangiomas is approximately 22-30% of preterm infants with birthweight less than 1 kg; for preterm infants with birthweight greater than 1.5 kg,3 the incidence is the same as for term infants. An increased incidence is recognized in infants from multiple gestations. The

incidence is increased with older maternal age, maternal placenta previa, and preeclampsia.4 Some, but not all, surveys have demonstrated increased incidence in infants born to mothers who have undergone prenatal chorionic villus sampling.

Most infantile hemangiomas are benign and do not cause any morbidity or mortality. Occasionally, they may impinge on vital structures and interfere with breathing, vision, eating, or hearing. Ulceration of certain areas (eg, diaper area, neck, mucosal surfaces) is not uncommon. Excessive bleeding is infrequent and rarely, if ever, life threatening. In the past, infantile hemangiomas were confused with other vascular neoplasms, particularly kaposiform hemangioendothelioma and tufted angiomas, which can incite a consumptive coagulopathy that may be life threatening. This is referred to as Kasabach-Merritt phenomenon (KMP). It is now generally accepted that infantile hemangiomas are rarely, if ever, responsible for KMP.5,6

Large cutaneous or visceral hemangiomas (particularly liver) can result in high-output cardiac failure resulting from increased vascular flow. Permanent significant structural abnormalities may result, particularly when facial structures are involved. The highest risk appears to be with involvement of the nasal tip, lips, and ears.7 Segmental hemangiomas, which cover a particular section or area of skin, may be markers for underlying malformations or developmental anomalies of the heart, blood vessels, or nervous system (PHACES and PELVIS syndromes and lumbosacral hemangiomas) and, depending on the severity of the associated anomaly, can result in increased morbidity or mortality.

*PHACES syndrome (see image below) - Posterior fossa abnormalities (Arnold-Chiari and Dandy-Walker malformations), hemangiomas (cervicofacial and/or laryngeal), arterial anomalies (carotid, cerebral, and vertebral), cardiac anomalies (especially coarctation of the aorta), eye abnormalities, and sternal or abdominal clefting)

Segmental infantile hemangioma in a female infant with PHACES syndrome involving the posterior neck and right forehead associated with an absent right vertebral artery and a laryngeal hemangioma.

*PELVIS syndrome (see image below) - Perineal hemangioma with any of the following: external genital malformations, lipomyelomeningocele, vesicorenal abnormalities, imperforate anus, or skin tag)

Abortive (telangiectatic) segmental infantile hemangioma of the bilateral buttocks and posterior thigh in this male infant with PELVIS syndrome (complicated by cutaneous ulceration, hypospadias, anal stenosis, intraspinal lipoma with tethered cord). The white material is a barrier diaper cream.

Females are affected more often than males by a ratio of 3:1. This disparity is higher (9:1) in those infants with large cervicofacial segmental hemangiomas associated with PHACES syndrome.

Infantile hemangiomas exhibit a characteristic evolution, with early rapid growth (proliferation) followed by slow involution.10 The earliest sign of an infantile hemangioma is blanching of the involved skin. This may be followed by fine telangiectasias and then a red or crimson macule. Rarely, a shallow ulceration may be the first sign of an incipient infantile hemangioma.

Rapid growth during the neonatal period (birth to 4 wk) is the historical hallmark of infantile hemangiomas. This rate is characteristically beyond the growth rate of the infant, thereby differentiating this neoplasm from vascular malformations that grow commensurate with the infant. As proliferation ensues, the infantile hemangioma becomes elevated and may be dome shaped, lobulated, plaquelike, tumoral, or any combination of these morphologies.11,12 The proliferation phase occurs during the first year, with the most growth occurring during the first 4-6 months of life. Proliferation slows considerably between the middle and end of the first year of life; however, most hemangiomas have completed this phase by age 4 months. During this time, the hemangioma may remain quiescent or may begin to involute.13

The involutional phase of an infantile hemangioma may be rapid or prolonged. No specific characteristics appear to influence the rate or completeness of involution of infantile hemangiomas. The exception is a separate type of hemangioma referred to as a rapidly involuting congenital hemangioma (RICH), which proliferates in utero and is fully developed at birth.14 RICHs tend to completely involute during the second year of life. Congenital hemangiomas are not considered to be a variant of the infantile hemangioma.

Fifty percent of infantile hemangiomas complete involution by age 5 years and 70% by age 7 years; the remainder may take an additional 3-5 years to complete the process.15 Of lesions that have involuted by age 6 years, 38% have residual evidence with scar formation, telangiectasia, or redundant or anetodermic skin. Infantile hemangiomas that take longer to involute have a higher incidence of permanent cutaneous residua. Eighty percent of infantile hemangiomas that complete involution after age 6 years may exhibit cutaneous residua.16

Eighty percent of infantile hemangiomas are focal and solitary. Sixty percent of cutaneous hemangiomas occur on the head and neck, 25% on the trunk, and 15% on the extremities (see image below). Hemangiomas also can occur in extracutaneous sites, including the liver, gastrointestinal tract, larynx, CNS, pancreas, gall bladder, thymus, spleen, lymph nodes, lung, urinary bladder, and adrenal glands.

This proliferating superficial infantile hemangioma on the trunk required no therapy.

Features of early proliferating infantile hemangiomas (birth to age 6 wk) include blanching of the involved skin, followed by fine telangiectasias, and then a red or crimson macule or papule that often is surrounded by a faint halo of vascular blanching. Occasionally, an infantile hemangioma is heralded by a shallow ulceration, especially lip and buttock lesions (see image below). Exquisitely painful ulcerated mixed hemangioma (superficial and deep) of the left deltoid in a 6-month-old female infant. This lesion was treated successfully with pulsed dye laser. As infantile hemangiomas proliferate (birth to age 12 mo), depending on their size and depth, their morphology and texture may be dome shaped, bosselated, plaquelike, tumoral, or any combination of these morphologies.

If the infantile hemangioma is located in the subcutaneous tissue, the overlying skin may be completely normal. Color varies with the depth from the surface and can be bright red or crimson (superficial dermis), purple, blue, or flesh colored with predominant involvement of the deeper tissues. Telangiectases and large superficial veins radiating from the infantile hemangioma often are associated. The consistency is firm, rubbery, and tense and expands with increased intravascular pressure (eg, with crying when on the head and neck). Tenderness to palpation is a variable generally uncommon feature of infantile hemangiomas.

Most infantile hemangiomas reach a maximum size of 0.5-5 cm, but they can range from the size of a pinhead to greater than 20 cm in diameter. Most infantile hemangiomas remain well circumscribed and focal. A minority may be segmental in nature, covering a larger portion of the cutaneous surface. This variant has more superficial than deep involvement, as is seen with extensive facial lesions (see image below).

This superficial and deep infantile hemangioma resulted in astigmatism of the left eye, requiring spectacles to correct the refractive error and to prevent amblyopia. Further growth of this hemangioma necessitated a course of oral prednisolone. The hemangioma shrunk rapidly, and the patient's astigmatism decreased such that the spectacles were unnecessary 1 month after beginning steroids.

Another variant is an abortive or reticular infantile hemangioma, previously referred to as a telangiectatic hemangioma. This variant of infantile hemangioma is almost completely flat and may simply present with an erythematous blush of the affected skin. As its prior name suggests, an abortive or reticular infantile hemangioma often has telangiectasia coursing through it. This variant may be confused with a capillary malformation; however, the rapid growth characteristics and presence of visible telangiectases assist in differentiation. This variant has been seen in association with underlying vascular and other congenital anomalies (PHACES and PELVIS syndromes and with underlying vascular anomalies on an extremity).18

During involution, which may begin as early as a few months from birth or as late as 2-3 years, the infantile hemangioma shrinks centrifugally from the center of the lesion. This is less notable with deeper lesions. The superficial lesions become less red, taking on a duskier maroon-to-purple color, and finally regaining normal flesh tones (often referred to as "graying"). With involution, the infantile hemangiomas become softer and more compressible with decreased tenderness, and they exhibit less expansion during increased intravascular pressure (eg, crying).

During the late involution phase (quiescent residual lesions), the skin may return to normal with no evidence of a previous pathologic process. Approximately 50-60% of all hemangiomas resolve incompletely, leaving permanent changes in the skin. These changes include telangiectases, superficial dilated veins, stippled scarring, anetoderma or epidermal atrophy (particularly with superficial lesions), hypopigmentation, and/or redundant skin with fibrofatty residua (especially with subcutaneous lesions).16

It is believed that infantile hemangiomas may be derived from endothelial progenitor cells (EPCs). EPCs are bone marrow-derived CD133 (or AC133) – positive, CD34-positive, and KDR (VEGFR-2)-positive pluripotent cells that demonstrate the ability to develop into Glut-1–positive endothelial cells. A hemangioma model using these cells exhibits growth characteristics typical of an infantile hemangioma, with both proliferating and involuting phases and the development of fibrofatty residua during involution. The endothelial cells in an infantile hemangioma are thought to be primitive endothelial clonal cells that exhibit abnormal behavior.

Two possibly interrelated theories exist regarding the pathogenesis of hemangiomas with regard to the EPCs, intrinsic and extrinsic. The intrinsic theory proposes that EPCs are monoclonal and behave differently from normal endothelial progenitor cells. The extrinsic theory postulates that the EPCs are polyclonal and behave normally and proliferate in response to the surrounding tissue angiogenic and angiostatic factors.

Mesenchymal stem cells may also play a role in the formation of infantile hemangiomas. These cells have been identified in hemangioma tissue. Mesenchymal stem cells retain the capacity to differentiate into numerous mesodermal cells, including adipocytes, suggesting that these cells may be the source of the resultant adipose tissue found in involuted hemangiomas.

Evidence to support a hereditary/genetic component in the development of most infantile hemangiomas is minimal; most appear to be sporadic. However, at least one report described a kindred in which infantile hemangiomas may be the result of an autosomal dominant trait. These infantile hemangiomas were seen in association with an increased incidence of vascular malformations (mostly capillary malformations) in various members of the same family.

Laboratory Studies

No laboratory studies have been universally accepted for the diagnosis and treatment of infantile hemangiomas; MRI with and without intravenous gadolinium is the imaging modality of choice to delineate the location and extent of both cutaneous and extracutaneous hemangiomas. MRI also helps in differentiating other high-flow vascular lesions (eg, arteriovenous malformations vs proliferating hemangiomas). Involuting hemangiomas have features that resemble low-flow lesions (eg, venous malformations).

Ultrasonography is useful in differentiating hemangiomas from other deep dermal or subcutaneous structures, such as cysts or lymph nodes. Ultrasonography is generally limited by its inability to fully evaluate the magnitude and extent of the hemangioma. Dubois et al found that an evaluation exhibiting high vessel density (>5 vessels/cm2) and high peak arterial Doppler shift (exceeding 2 kHz) was both sensitive and specific for infantile hemangiomas compared with other soft tissue masses.

Plain radiography is fairly limited but may be useful for evaluating hemangiomas that impinge on the airway.

Procedures

Specimens may be evaluated for tissue-specific immunohistochemical markers such as GLUT-1, merosin, Fc-gamma-RII, and Lewis Y antigens. These markers may aid in differentiating infantile hemangiomas (positive staining for all) from other vascular neoplasms or malformations, such as the congenital hemangiomas (eg, rapidly involuting congenital hemangioma, noninvoluting congenital hemangioma), kaposiform hemangioendothelioma, tufted angioma, or pyogenic granuloma, none of which stains positively for these antigens. These markers are coexpressed by infantile hemangiomas, erythrocyte cell membranes,

and placental microvessels.The vast majority of infantile hemangiomas do not require any medical or surgical intervention. Medical care of clinically significant hemangiomas has been limited to a few medications, including glucocorticosteroids (topical, intralesional, and oral), interferon alfa, and, rarely, vincristine and topical imiquimod. Beta-blockers, most specifically propranolol, have serendipitously been shown to induce involution of infantile hemangiomas. The individual therapies are discussed in detail under Medication. Also see Further Reading for a partial listing of clinical trials that are currently recruiting.

Surgical Care

Pulsed-dye laser surgery is effective for treating ulcerated hemangiomas and thin superficial hemangiomas, especially those on areas likely to result in significant functional or psychological impact (eg, fingers, eyes, lips, nasal tip, ears, face). Many ulcerated hemangiomas respond with decreased pain (sometimes as early as a few days after the initial treatment), rapid reepithelialization, and hastened involution.

Treatments generally are performed every 2-4 weeks until complete healing results. Occasionally, particularly with deep or combined superficial and deep lesions, ulceration may worsen with pulsed-dye laser treatment.

The risk of scarring or residual skin changes associated with pulsed-dye laser surgery of hemangiomas may be greater than without early laser treatment or with the treatment of capillary malformations (port wine stains), but the benefits of early involution should be weighed against the risks of a passive approach or alternative therapies.

Other lasers that appear to be efficacious in treating hemangiomas include the pulsed Nd:YAG, frequency-doubled Nd:YAG, and KTP lasers. Carbon dioxide lasers are occasionally used for airway hemangiomas.46 Each of these lasers has specific benefits and limitations regarding depth of penetration, absorption of skin chromophores, and caliber of the vessel treated. Complications also vary depending on the laser, settings, and site treated.

The use of nonablative fractional photothermolysis (nFP) for the treatment of the anetodermic fibrofatty residua in involuted infantile hemangiomas may be an alternative to surgical excision.

See Laser Treatment of Acquired and Congenital Vascular Lesions for a detailed discussion.

Surgical excision of involuted hemangiomas is not uncommon because of the cutaneous defects resulting from them.49 Atrophic and hypertrophic scars, as well as anetodermic and tumoral fibrofatty skin, may result in significant cosmetic or functional impairment. The benefits of excision during late involution include a reduced risk of hemorrhage and a potentially smaller lesion because of the natural course. In addition, because involuted hemangiomas are composed primarily of fibrofatty tissue, complete removal of all tissue is unnecessary, while removing too much tissue could detract from proper contours.

Surgical excision of proliferating hemangiomas is potentially hazardous because of the risk of hemorrhage and damage to vital structures associated with them (ie, head, neck); therefore, only specially trained surgeons should perform this procedure. Certain benefits to early excision include saving a life or preserving vision and decreasing the negative psychosocial effects associated with a cosmetically disfiguring lesion during early childhood. Other benefits of early excision include the use of naturally expanded skin to aid in primary closure and the ability to use a relatively avascular tissue plane surrounding actively growing hemangiomas. New advancements in surgical instruments that cauterize while cutting lessen the risk of hemorrhage.

An ophthalmologist or a pediatric ophthalmologist should evaluate children with periorbital hemangiomas, particularly with involvement of the upper eyelid. Refraction with retinoscopy is performed to evaluate for visual disturbances, particularly astigmatism, and to prevent visual deprivation amblyopia. Also see Hemangioma, Capillary in the eMedicine Ophthalmology section.

Infants with rapidly growing hemangiomas that are impinging on vital structures of the head and neck, particularly the airway or auditory canals, should be referred to an otolaryngologist or a pediatric otolaryngologist for evaluation and treatment. Infants with large V3 dermatomal hemangiomas (beard area

hemangiomas) have a higher incidence of upper airway hemangiomas, and early consultation for mild signs or symptoms (noisy breathing or stridor) may prevent possible future complications.

Consultation with a plastic surgeon is indicated for symptomatic involuting or proliferating lesions that are unresponsive to medical therapy and for which surgical excision is being contemplated.

The presence of an infantile hemangioma over the midline lumbar back may be a cutaneous sign of an underlying occult spinal dysraphism, such as a tethered cord. MRI or ultrasonography if the infant is younger than 5 months is indicated for midline hemangiomas, especially if any other signs of spinal dysraphism (eg, deviated gluteal cleft, atypical sacral dimple, tuft of hair, tail) are present. MRI is the more sensitive study, even in infancy, and should be considered when clinical suspicion is high. Consultation with a pediatric neurosurgeon should be sought for any questionable or worrisome lesions.

The goals of pharmacotherapy for infantile hemangiomas are to reduce morbidity and mortality and to prevent complications. Note that none of the treatments is approved for treatment of infantile hemangiomas by the US Food and Drug Administration, and all therapies should be considered off-label usage.

Oral and intralesional corticosteroids are effective at slowing the growth and decreasing the size of proliferating infantile hemangiomas. The mechanism of action has not been elucidated completely; however, corticosteroids have been shown to inhibit VEGF-A expression and subsequent proliferation in hemangioma stem cells in a murine hemangioma model.50 Evidence indicates that corticosteroids block estradiol receptors in hemangiomas in vitro. Response vary widely, from less than 40% to greater than 90%, depending on dose, duration of treatment, and age at which corticosteroid therapy is initiated.51 Corticosteroid therapy should be administered during the proliferative phase because it has a negligible effect on involuting and otherwise stable infantile hemangiomas. The oral route generally is preferred over intralesional therapy; however, the location, size, patient age, and physician experience factor into the decision-making process.

Teratomas and Other Germ Cell Tumors

Teratomas (from Greek terato meaning "a monster" and onkoma meaning "swelling or mass") and other germ cell tumors are relatively common solid neoplasms in children. They may occur in both gonadal and extragonadal locations. Locations and specific tumor types depend on the age of the child. The tumors are grouped together because they all appear to arise from postmeiotic germ cells. Most of the malignant tumors produce markers that can be serologically assessed.

Several theories about the origin of these tumors are recognized. The best evidence suggests that most are due to abnormal differentiation of fetal germ cells that arise from the fetal yolk sac. Normal migration of these germ cells may cause gonadal tumors, whereas abnormal migration produces extragonadal tumors. Teratomas are typically found in the midline or gonads. Frequencies of the most common sites are as follows:

Sacrococcygeal - 40%.Ovary - 25%, Testicle - 12%, Brain - 5%,Other (including the neck and mediastinum) - 18% By definition, teratomas include components derived from all 3 embryonic layers: ectoderm, endoderm, and mesoderm. These tissues are foreign to the location in which they are found. Teratomas may be classified as mature or immature on the basis of the presence of immature neuroectodermal elements within the tumor. Mature tumors (grade 0) have no immature elements. In grade 1 tumors, immature elements are limited to one low-power field per slide; in grade 2 tumors, less than 4 fields are present per slide; and in grade 3 tumors, more than 4 fields are present per slide.

In the past, survival was linked to the degree of immaturity in the teratoma. Close histologic evaluation of immature teratomas reveals a good correlation between the degree of immaturity and the presence of microscopic foci of frankly malignant elements. These malignant elements are typically yolk sac tumors but may also represent primitive neuroectodermal tumor (PNET). Charoenkwan et al (2002) found overexpression of p53 in the more aggressive immature teratomas at all sites.

The risk of recurrence also appears to be related to the degree of immaturity. Recurrence in a completely resected mature teratoma is less than 10%; in an immature teratoma, recurrence may be as high as 33%. The likelihood of recurrence depends on the site of the tumor as well as the completeness of resection. The German MAKEI trials suggest that the recurrence rate for immature teratomas can be decreased to 9.5% with chemotherapy.2 Sacrococcygeal teratomas are more likely to recur than those in the ovary or other sites. Molecular biologic and cytogenetic studies are providing a firmer scientific basis to these observations.

Mutter describes genetic imprinting as a major factor in the development of some of these tumors.4 The developmentally expressed genes insulinlike growth factor 2 (IGF II) and its receptor RNA (H-19); small nuclear riboprotein (SNRPN); mas proto-oncogene; and the tumor suppressor genes WT1 and MASH2 are imprinted, depending on their maternal or paternal origin. Mutter suggests that these genes or the cells have only the maternal imprint because many teratomas arise from a parthenogenetically activated egg. Therefore, maternally active genes are present in higher-than-usual concentrations, and maternally inactive products are present at lesser concentrations if at all. These abnormalities may account for the lack of organization of the 3 germ cell layers.

Oosterhuis et al suggest that tumors may be grouped on the basis of their chromosomal abnormalities as follows: Group 1 includes immature teratomas and yolk sac tumors. The immature teratomas are usually diploid, whereas yolk sac tumors may be diploid, tetraploid, or aneuploid. The chromosomal aberrations include overrepresentation of chromosomes X, 1, 3, 8, 12, and 14 and underrepresentation of Y and X. Deletions in 1p and rearrangements of 3q and 6q may be present. Isochromosome 12p (i12p) has been found. An abnormal number of centromeres is frequent in both diploid and aneuploid tumors. Group 2 includes most nonseminomatous malignant germ cell tumors and typically includes numeric abnormalities in X, 7, 8, 12, and 21 as excess and deletions of Y, 11, 13, or 18. Once again, isochromosomes 12p with other aberrations of 12p and 1p are present.

Group 3 includes mature teratomas or mature cystic teratomas. Numeric abnormalities, including extra X, 7, 12, and 15, have been found. No chromosomal structural anomalies have been found. Group 4 includes spermatocytic seminoma, a type usually confined to older men. The cytogenetics of this group have not been characterized. As with abnormalities and imprinting patterns, these chromosomal rearrangements can lead to overproduction of certain gene products and underproduction of others; these lead to the abnormal growth characteristics of the tumor.

Hara et al suggest that the MAGE gene family of tumor rejection antigens may also be involved in the pathogenesis of these tumors.6 These genes appear to be more active in pure seminoma or mixed type of seminomatous elements than in other germ cell tumors. In their limited study of 22 patients, MAGE expression was not correlated with disease progression. It is likely to be only an indicator of maturity or differentiation of the tissues.

The concept of teratoma with malignant transformation indicates the development of non–germ cell malignancies within a teratoma. Among 641 patients in the MAKEI protocols 83/86/89/96, 9 patients were identified with this finding.7 Five patients presented with a carcinoma, 2 patients presented with glial tumors, and 2 patients presented with embryonal tumors. Resection and chemotherapy were typically used. Because these tumors are quite rare, response to treatment is difficult to generalize.

When platinum-based chemotherapy–resistant tumors are evaluated, between one third and one half of tumors exhibit microsatellite instability.

Sacrococcygeal teratoma occurs in 1 in 30,000-70,000 live births. The female-to-male predominance is 4:1. Ovarian teratomas are almost as common, whereas testicular teratomas are about one third less frequent. The overall incidence of malignant germ cell tumors is approximately 3% of all childhood malignancies, or approximately 3 cases per million population per year. The frequency of all germ cell tumors has increased over the last several decades.

The mortality rate for congenital teratomas depends on gestational age and the size and location of the tumors. Survival of preterm infants younger than 30 weeks' gestation with sacrococcygeal teratoma is only 7%, whereas the survival for infants older than 30 weeks' gestation is 75%.

Rapid early growth is associated with the yolk-sac phenotype and carries a poorer prognosis.8 Early tumors are frequently large relative to the size of the infant and may induce congestive heart failure. Cervical teratomas may frequently lead to airway problems and death when they are large.

Prior to recent chemotherapeutic successes, the 10-year survival rate for malignant germ cell tumors ranged from 25% for embryonal carcinoma to 75% for dysgerminoma. Today, overall survival rates are greater than 90%.

Sacrococcygeal teratoma has a 4:1 female-to-male predominance. In other germ cell tumors, the female-to-male ratio is roughly 2:1 in children.

Sacrococcygeal teratomas are congenital.

Those with a significant external component are identified at birth. Tumors without an external component (Altman type 4) are discovered later.

When the tumors are resected before the patient is aged 2 months, 7-10% are malignant. After that age, the risk of malignancy greatly increases to more than 50% by age one year.

The incidence of ovarian germ cell tumors increases with age and peaks around age 15-19 years.

When girls younger than 15 years were examined, fewer than 10% of tumors occurred in girls younger than 5 years, 20% of tumors were found in girls aged 5-9 years, and more than 70% of tumors were found in girls aged 10-14 years.

Benign ovarian tumors, largely teratomas, predominate.

Roughly 70% of malignant ovarian tumors in childhood are germ cell tumors, one quarter are epithelial, and the remainder are stromal tumors. The ratio of germ cell tumors to epithelial malignancies decreases with increasing patient age.

Chromosomal abnormalities also appear to be related to age at presentation for teratomas. In girls less than 5 years old, no chromosomal abnormalities were found, whereas older girls often have gains of 12p and chromosomes 7 and 8.

Testicular tumors

Testicular germ cell tumors in childhood are split between teratomas and yolk sac tumors. They are more common from birth to age 5 years. From age 6 years until puberty, testicular tumors are exceedingly uncommon. Thereafter, the incidence increases, with a more adultlike tumor pattern with seminomas gradually becoming the predominant histology.

Both teratomas and yolk sac tumors may be associated with contralateral in situ dysgenesis in 9% of patients compared with 0.5% of otherwise healthy males. Contralateral tumors are often found. These are occasionally synchronous but are more often metachronous. Ongoing surveillance of the contralateral testis is therefore needed.

The clinical presentation of these tumors depends on the location of the tumor.

Sacrococcygeal teratomas may be prenatally diagnosed as an incidental ultrasonographic finding; they may occur in an infant who is large for age, in premature infants, or in infants with fetal hydrops. Fetal hydrops is an ominous sign. It is typically due to high flow through the tumor with high-output cardiac failure and placentomegaly. A teratoma larger than 5 cm is likely to cause dystocia and possible rupture; elective cesarean delivery should be performed. Sacrococcygeal teratomas that are not prenatally diagnosed may be noted at delivery, within the first few weeks after birth, or discovered late. Ovarian masses typically cause abdominal pain, mass, distention, or emesis. Two thirds of affected girls present with pain as their primary symptom. Acute and chronic pain occur with equal frequency. In situations of acute pain, the diagnosis is often related to torsion of the ovary with consequent compromise of the blood supply. Palpable masses are less frequent and appear later in the clinical course. Testicular tumors typically occur as a scrotal mass with

or without pain. The differential diagnosis may include hydrocele because some cystic teratomas may transilluminate. In some situations, the tumor may cause symptomatic metastasis; this is more common in older patients. The distribution of the patients' age at presentation for testicular tumors is bimodal. In the youngest children (aged 0-4 y), teratomatous lesions and yolk sac tumors are predominant. In children older than 10 years, teratomas are increasingly rare. Yolk sac tumors are still predominant, but other malignant germ cell types start to become clinically relevant.

The epidemiology of the tumors suggests that they are increasing in frequency. With sacrococcygeal teratomas, no causative agents are known. With respect to ovarian germ cell tumors, a familial predilection may be present. Cases in 7 families have been reported in which female first-degree relatives had germ cell tumors. In an additional 7 families, males had germ cell tumors. This observation suggests that certain genes may be present in these families, predisposing them to germ cell malignancy.

One study that examined the effect of diet on the development of ovarian tumors revealed that diets high in polyunsaturated fat were associated with the development of teratomas.10 Likely, plant estrogens, and not the polyunsaturated fat, are associated with an increased tumor risk.

The risk factors and epidemiologic features of testicular cancer suggest that cryptorchidism increases the risk of germ cell tumor by a factor of 10. Tumors may appear in the ipsilateral or contralateral testicle. Hernia is similarly associated with germ cell tumors. One study also revealed that a history of pyloric stenosis leads to a 4-fold risk of germ cell malignancy.11 Boys whose father or brother has had a teratoma have a 5-15% increased risk for developing a teratoma. Whether this is due to genetic causes or is a consequence of shared environment is unclear.

Intersex anomalies have also been associated with development of germ cell tumors. Gonadoblastoma is observed in roughly one third of patients with intersex anomalies. Although gonadoblastoma is a carcinoma in situ, it frequently evolves into dysgerminoma; yolk sac tumors, immature teratomas, and choriocarcinomas are possible as well. Turner syndrome is similarly a risk factor for gonadoblastoma. Klinefelter syndrome has been linked with an increased risk of extragonadal malignant germ cell tumors. The highest risk seems to be among patients who carry some Y-chromosome genes in ectopic locations where they may not be normally regulated.

Children with intersex anomalies are typically male pseudohermaphrodites with antigen insensitivity or 5-alpha reductase deficiency. These patients with testicular feminization are sometimes discovered serendipitously during a hernia repair. Debate surrounds the optimal timing for gonadal resection in these situations. Gonadal estrogen production may benefit the patient in terms of growth and development. However, gonadoblastoma has been observed in patients as young as 2 months, and frank tumors have been observed in those younger than 2 years. The decision to leave or remove the gonads early should be made with the family after thorough discussion of these risks and potential benefits.

Surgical Care

In general, gross total resection of tumor is the goal. The tumor and involved adjacent structures should be resected en bloc, if this is possible and does not lead to disfigurement.

Sacrococcygeal tumor

Typically, the surgeon approaches this tumor through a posterior trans-sacral route. The coccyx must be resected en bloc with the tumor to minimize the risk of recurrence. Control and division of the middle sacral artery early in the procedure is advisable. If the sacrum or rectum is invaded by the tumor, complete resection may not be advisable at the initial operation. Treating these tumors with chemotherapy is reasonable, with resection after the maximum response is obtained.

When the tumor extends high into the pelvis and abdomen, laparotomy or laparoscopy is required in addition to the posterior incision. Ascitic fluid should be collected or peritoneal washings obtained. The tumor may then be mobilized for removal from below or above, depending on the anatomy. Samples should be obtained

in lymph nodes from the retroperitoneum. In tumors with a moderate pelvic component, laparoscopy may allow clip placement in the middle sacral artery and mobilization of the pelvic portion of the tumor.

Cowles et al (2006) reported preoperative embolization of the major vessels that supply a large teratoma followed by radiofrequency ablation of the zone between normal tissue and tumor.13 Damage to the nerves supplying the leg has been reported with prenatal radiofrequency ablation.

Ovarian tumor

Open resection is the preferred approach to these tumors. Typically, laparoscopy requires morcellation of the tumor in a bag. The consequent destruction of the tumor capsule prevents pathological staging; thus, patients must be treated as stage II. Ascites or peritoneal washings should be undergo cytologic analysis. The entire peritoneal cavity should be inspected. Any suspicious implants should be sampled or resected. Gliomatosis peritonei does not worsen the stage of a tumor, but all implants must have mature glial tissue. Immature tissue suggests metastatic disease and requires more intensive therapy. The omentum must be inspected. If disease is possible (eg, adherence, nodules, implants), the affected area should be resected at this time.

Ipsilateral oophorectomy or salpingo-oophorectomy should be performed. Uninvolved fallopian tubes should be preserved if possible. In cases of mature teratoma, the contralateral ovary should be inspected. If it appears normal, it should be left alone. Bilateral malignant tumors require bilateral oophorectomy, but hysterectomy is unnecessary for germ cell tumors. Some authors advocate ovary-sparing resection of mature teratomas. This is not always possible.

Samples of suspicious and involved lymph nodes should be obtained. Random bilateral sampling is no longer required because it did not have an impact on survival in the last Intergroup study.

Testicular tumor

Testicular teratomas may be treated with local resection in prepubertal patients. The tumor should be removed with a small rim of normal testicle. If the testicular tissue shows signs of pubertal change, radical inguinal orchiectomy should be performed.

In all malignant cases, radical inguinal orchiectomy should be performed with high ligation of the spermatic cord. For very large tumors, the incision may be enlarged by extending the medial portion of the incision downward into the upper scrotum. Transscrotal resection with intact capsule is now treated as a stage I tumor, provided the cord structures are completely removed and are uninvolved. If trans-scrotal biopsy was performed prior to resection, the stage is at least stage II. Because most of these preadolescent tumors are responsive to chemotherapy, hemiscrotectomy is rarely necessary.

If images do not reveal lymph node enlargement, sampling of ipsilateral retroperitoneal lymph nodes is not required. When images show positive findings of nodal enlargement of 2-4 cm, perform a biopsy of the enlarged nodes. Nodes larger than 4 cm diameter are treated as stage III metastatic disease and do not require biopsy. Tumor debulking is no longer recommended.

Mediastinal tumor

The approach to the resection may be via median sternotomy or lateral thoracotomy. Small lesions have been resected by using video-assisted thoracic surgery (VATS). Large lesions may cause airway compromise and require intubation and care in the intensive care unit. Many of these large tumors are best managed with initial biopsy, neoadjuvant chemotherapy, and delayed complete resection.

Adherent nonvital structures such as the pericardium and thymus should be removed en bloc with the tumor. Lymph nodes should be sampled

Neck tumor

These lesions present special surgical challenges. In large congenital lesions, the airway may be compromised, and intubation may be difficult. The ex utero intrapartum treatment (EXIT) procedure, in which a cesarean delivery is performed and the neonate remains attached to the placenta, may allow enough time for bronchoscopic airway placement.

Resection should be total but not at the expense of vital structures. A staged procedure is acceptable in this circumstance. Complete resection may then be possible after chemotherapy.

Recurrent disease

Recurrent disease must be surgically staged. The extent of disease is an important prognostic factor. Surgically resectable recurrent disease has a far more favorable prognosis than unresectable disease. The best prognosis exists when complete surgical resection is accompanied by high intensity chemotherapy with autologous stem cell rescue.

Additionally, recurrent disease may have a different tissue type than that of the original tumor. PNET, for example, is a frequent component of germ cell tumors that may not respond to bleomycin, etoposide, and cisplatin (BEP)–type therapy.

Metastatic disease: When these tumors are metastatic, initial chemotherapy may lead to resolution of metastatic disease. When it does not, residual disease may be necrotic tumor, mature teratoma, persistent malignant disease, or combinations of the above. No current radiologic test reliably distinguishes between these possibilities. Surgical biopsy may help guide therapy. Resection is recommended when possible.

Psychological support is important for both the patient and the family after any diagnosis of cancer. For older patients, fertility issues, as well as issues of sexual identity, may also be important.

Maintaining adequate nutrition is often difficult during chemotherapy. Additionally, intestinal obstruction may be a consequence of an abdominal tumor. Nutritional supplements or parenteral nutrition may be necessary. In cases other than those involving frank obstruction, enteral tube feeding has proven useful.

Medication

Since the introduction of platinum-based therapy for this disease, the survival rate has improved considerably. First-line therapy includes the use of cisplatin, etoposide, and bleomycin. Survival with carboplatin containing regimens has not been as favorable. For low risk tumors (testicular stage II, and ovarian stage I and II) 4 cycles of BEP has a survival rate of 94-100%. For high risk tumors (stage III and IV testicular and ovarian tumors and stage I-IV extragonadal tumors) high-dose BEP has better overall survival at the cost of some increase in toxicity.

Salvage therapy typically consists of a combination of paclitaxel, ifosfamide, carboplatin, etoposide, and vinblastine or vincristine plus peripheral blood stem cell (PBSC) transplantation. Gemcitabine has been used as salvage therapy in a phase 2 protocol.

Kollmannsberger et al (2003) reported improved 2-year survival with a second course of high-dose chemotherapy with stem cell rescue and complete surgical resection in high-risk patients who failed initial ablative chemotherapy and autologous stem cell transplant.15

Antineoplastic agents

Cancer chemotherapy is based on an understanding of tumor cell growth and how drugs affect this growth. After cells divide, they enter a period of growth (ie, phase G1), followed by DNA synthesis (ie, phase S). The next phase is a premitotic phase (ie, G2). Finally, a mitotic cell division occurs (ie, phase M). Cell division rates vary for different tumors.

Antineoplastic agents interfere with cell reproduction. Some agents are cell cycle specific, while others (eg, alkylating agents, anthracyclines, cisplatin) are not phase specific. Cellular apoptosis (ie, programmed cell death) is also a potential mechanism of many antineoplastic agents.Current protocols use these agents in combinations that exploit differences in growth and recovery between tumors and normal tissues.

Bone Tumors (Benign)

Most bone tumors are benign, and unlikely to spread. They can occur in any bone, but they usually are found in the biggest ones. These include the thighbone (femur), shinbone (tibia), upper arm bone (humerus) and pelvis. Some types are more common in specific places such as the spine or near the growth plates of the largest bones.

There are many specific types of tumors within the category of benign bone tumors. The most common ones are endochondromas, osteochondromas, nonossifying fibromas, chondroblastomas, osteoid osteomas, osteoblastomas, periosteal chondromas, giant cell tumors and chondromyxoid fibromas. Some conditions such as aneurysmal bone cyst, unicameral bone cyst and fibrous dysplasia are grouped with benign bone tumors. They often are treated in a similar way, although they aren't truly tumors.

Symptoms of Benign Bone Tumors

A lump or swelling can be the first sign of a benign tumor. Another is ongoing or increasing aching or pain in the region of the tumor. Sometimes tumors are found only after a fracture occurs where the bone has been weakened by the growing tumor.

Causes and Risk Factors of Benign Bone Tumors

Benign bone tumors occur most often in children whose skeletons are still growing and people up to age 30. These tumors are often strongly affected by the hormones that cause growth. Many benign tumors stop growing once a child's bones do. This usually is between the ages 14 to 16 in girls and 16 to 19 in boys.

Treatment of Benign Bone Tumors

The type of tumor, its size, its location and how old the individual is all affect treatment decisions. Some tumors will heal after a fracture. Others may stop growing if the patient is near maturity when the tumor is discovered. Still other tumors are only discovered when an X-ray is taken for another reason. These may only need to be watched to make sure they aren't growing or becoming aggressive.

Usually, however, surgery is needed. Surgery removes the tumor and rebuilds new, healthy bone where the tumor was removed. At the Cedars-Sinai Orthopedic Center, specialized, minimally invasive techniques are used to protect the surrounding healthy tissue. This gives young patients the greatest chance of returning to full and unlimited activities.

Benign bone tumors include:

Osteoblastomas, which affect children and adolescents. These tumors can be large, aggressive and painful. They are best treated by a multidisciplinary team of oncologists, orthopedic surgeons and pain management specialists. They sometimes cause spinal deformity and paralysis. Treatment of depends on the tumor's size and location.

Osteoid osteoma, which is a small bone tumor (smaller than two centimeters). It usually affects adolescents, causing pain at night. It may also result in spinal deformity. Treatment depends the tumor's size and location. Surgery may be necessary if spine stability is compromised.

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Topic 6. Blunt trauma of thorax, abdominal cavity, and retroperitoneal space

Theme: "**Peculiarities of oncology in children. Malignant tumors.**

1. Actuality of theme: **Peculiarities of oncology in children. Malignant tumors** represents a serious problem for modern medicine. This confusion among patients and physicians led the International Society for the Study of Vascular Anomalies to adopt a general, biologic classification scheme for vascular anomalies based on physical findings, natural history, and cellular kinetics. In this system, vascular anomalies are described as either malformations or tumors. Vascular tumors exhibit abnormal endothelial cell proliferation while malformations are products of abnormal embryonic vessel development.This schema presents a useful framework for discussing the diagnosis and treatment of vascular anomalies.

2. Educational aims:

Concrete aims:

1. To master the malignant tumors of soft tissues.
2. To recognize the basic clinical symptoms of malignant tumors of soft tissues.
3. To differentiate a tumors depending on their kind.
4. To expose the signs of malignant regeneration of tumors and complications (bleeding, inflammations, squeezing of nervous-vascular structures).
5. To interpret principles of medical treatment of malignant tumors of soft tissue and its complications.
6. To recognize the basic clinical symptoms of tumors of bones, distinguish the signs of malignant course of diseases.
7. To identify the features of course of separate diseases on the basis of clinical and roentgenologic signs.
8. To differentiate the malignant tumors of bones on the basis of clinical and auxiliary methods of examination (roentgenologic, ultrasound, computer tomography, angiography, puncture and open biopsy of tumor).
9. To work out a plan of inspection of patient and algorithm of actions on the exposure and treatment of complications of tumors of bones (pathological fracture, false joints, metastasis).
10. To interpret principles of surgical medical treatment, chemotherapy, radial therapy depending on the type of tumor of bone.

4. The plan and organisation of lessons.

Work type	Time	Activity		Equipment
		Higher education applicants	Teacher	
Organisation of the practical class	5	Listen	Presence control	Journal
Aim setting	5	Listen	Explain	
Checking of the knowledge of higher education applicants	15	Make	Control	Tests
Seminar	60	Answer	Control	X-ray-grams
Clinical discussion	30	Listen	Discuss patients	Patients
Personal work of higher education applicants	60	Make	Control	Patients
Results of the lesson	10	Listen	Appreciation of each activities	
Task for the next lesson	5	Listen	Explain the task. Indicates the	

			literature.	

5. Substance of the lesson.

Malignant tumors of soft tissues.

Determination of clinical symptoms of melanoma, differential diagnostics. Surgical treatment of melanoma, chemotherapy, radial therapy. Prognosis.

Clinical symptoms of rabdomiosarcoma. Role of auxiliary methods of inspection in differential diagnostics (sciagraphy, angiography, cytoogical and histological diagnostics). Surgical treatment of rabdomiosarcoma, radial and chemotherapy depending on the stage of disease. Prognosis.

Osteogenous sarcoma and Yuing's sarcoma, characteristic clinical and roentgenologic symptoms. Differential diagnostics with chondrosarcoma, malignant form of osteoblastoclastoma, eosinophil granuloma, aneurismal cyst of bone on the basis of symptoms, X-ray, ultrasound, CT, angiography, biopsy of tumor. Principles of combined medical treatment of malignant tumors of bones, including surgical, chemotherapy, radial therapy. Prognosis.

Nephroblastoma and neuroblastoma

Concrete aims:

1. To master the characteristic signs of "palpated tumor of abdomen" in children with nephroblastoma and neuroblastoma.
2. To distinguish nephroblastoma and neuroblastoma depending on the clinical symptoms and stage of diseases.
3. To interpret auxiliary information of methods of examination (ultrasound, sciagraphy, urography, computer tomography, puncture biopsy).
4. To conduct differential diagnostics of nephroblastoma, neuroblastoma, hydronephrosis, polycystosis, renal anomalies, tumors of liver, tumors of adrenal gland.
5. To explain the principles of complex medical treatment of nephroblastoma and neuroblastoma depending on the stage of course of disease.

Nephroblastoma in children.

Nephroblastoma (Willms tumour) is malignant tumor of kydney. Clinical symptom sand diagnostics of nephroblastoma. Variants of course and clinical stages of nephroblastoma.

Differential diagnostics. Complex medical treatment of nephroblastoma. Surgical (nephrectomy), radial, chemotherapy before and after the surgery. Prognosis.

Neuroblastoma in children.

Neuroblastoma is a malignant tumor of neurogenous origin. Clinical symptoms of neuroblastoma, features of localization of tumor, and its stages of development. Diagnostics of neuroblastoma. Complex medical treatment of neuroblastoma: chemotherapy and radial therapy, surgical treatment. Prognosis.

Tumors of mediastinum.

1. Actuality of problem.

A frequency of tumors of mediastinum is 0,5-3 % among all tumors.

B. Diagnostics of tumors of mediastinum is responsible and difficult diagnostic problem which needs urgency in determination of pathological process.

2. Recognition of basic clinical symptoms of tumors of mediastinum.

The clinical symptoms of disease consist of symptoms of squeezing and destruction of tissue and organs of thoracic cavity and symptoms of intoxication, general symptoms of the tumors: cyanosis, asphyxia, stridor breathing. Every tumor has its characteristic clinical signs.

Neurogenous tumors:

- in the case of benign forms a clinical picture arises up as the growth of tumor,

- in case of malignant forms the symptoms appear in children of the first year of life and they are determined by catecholamines intoxication, because the tumor produces the precursors of adrenalinim.

Vascular tumors:

- arise up in the area of large vessels and trachea,
- the main symptoms is a syndrome of squeezing of vena cava superior,
- squeezing of trachea shows up as a cyanosas, pain, paresis of vocal cords.

Brochogenous tumors more frequent have asymptomatic course, but may appear with such symptoms: coughing, stenotic breathing, symptoms of squeezing of esophagus.

Teratomas and dermoids have long asymptomatic course. In case of quick growth these tumors cause the typical violations of hemodynamics and deformation of thorax.

Lypoma, fibroma, ?chondroma are the tumors of small sizes, on initial stages the clinical symptoms are absent, latter the pain, unpleasant feeling of mass presence appear.

Thymoma grows slowly, reaching the large sizes, the tumor squeezes veins, that causes difficulty of outflow of blood from the vein system.

Cysts of pericardium meet rarely. In 30% of cases a clinical picture is absent, but the next symptoms may appear: pain, heartache, coughing, stuffiness, general weakness.

The malignant regeneration of tumors of thymus is observed in children . Immature nerigenous tumors attribute to the group of potentially malignant tumors. The general percent of malignant tumors is 2%.

Medical treatment.

Medical treatment of tumors and cysts of mediastinum is mainly operative. In case of occurring of hemodynamic and respiratory violations, which arise up suddenly and quickly progress, immediate operative medical treatment is conducted.

Medical treatment of malignant tumors of mediastinum is perphormed after the international protocols of medical treatment, which, are based on type of tumor and its stage.

Present achievements in the field of soft tissue tumors are the result of advances in molecular biology, oncogenetics, imaging techniques, immunochemistry, diagnosis by fine-needle aspiration, surgical reconstruction, radiation therapy, and tissue banking. Benign soft tissue tumors are fairly common and are treated with surgery alone. Prior to the 1970s, surgery was the primary therapy for malignant soft tissue tumors, and most patients with high-grade tumors had a poor prognosis and a significant mortality rate. Since the mid-1970s, radiation therapy, chemotherapy, and advanced surgical techniques have helped increase long-term survival and decrease the need for ablative surgery.1 Future advances in molecular oncology may further improve diagnostic, prognostic, and treatment protocols for patients with soft tissue sarcomas.

Soft tissue is defined as the supportive tissue of various organs and the nonepithelial, extraskeletal structures exclusive of lymphohematopoietic tissues. It includes fibrous connective tissue, adipose tissue, skeletal muscle, blood/lymph vessels, and the peripheral nervous system. Embryologically, most of it is derived from mesoderm, with a neuroectodermal contribution in the case of peripheral nerves.

A computed-tomography (CT)–guided needle biopsy of a high-grade soft tissue sarcoma arising in the left hemipelvis. The CT artifact from the needle can be seen in the upper right corner of the image as the needle enters the lesion just anterior and medial to the dome of the left hip joint. Courtesy of Howard A. Chansky, MD

Magnetic resonance imaging (MRI) is used to demonstrate involvement of critical structures by tumor. This recurrent, high-grade soft tissue sarcoma in the posterior calf abuts the tibial nerve and posterior tibial vessels. An extensive reactive zone surrounds the structures. This patient was treated with below-knee amputation. Courtesy of Howard A. Chansky, MD

Soft tissue tumors are a large and heterogeneous group of neoplasms. Traditionally, tumors have been classified according to histogenetic features. (Fibrosarcoma, for example, is categorized as a tumor arising

from fibroblasts.) However, histomorphologic, immunohistochemical, and experimental data suggest that most, if not all, sarcomas arise from primitive, multipotential mesenchymal cells, which in the course of neoplastic transformation differentiate along one or more lines. A liposarcoma appears to arise from a lipoblast but may actually develop through lipoblastic differentiation of a precursor multipotent mesenchymal cell. At the clinical level, soft tissue tumors are classified according to various parameters, including location, growth pattern, likelihood of recurrence, presence and distribution of metastases, patient age, and prognosis.

Although most soft tissue tumors of various histogenetic types are classified as either benign or malignant, many are of an intermediate nature, which typically implies aggressive local behavior with a low to moderate propensity to metastasize.

In general, benign soft tissue tumors occur at least 10 times more frequently than malignant ones, although the true incidence of soft tissue tumors is not well documented.

However, some insight regarding the incidence of soft tissue sarcomas can be derived from the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program, which, between 1973 and 1983, accumulated data on 6883 such tumors.

Overall, age-adjusted annual incidence of soft tissue sarcomas ranges from 15-35 per 1 million population. The rate increases steadily with age and is slightly higher in men than in women.

Malignant soft tissue tumors occur twice as often as primary bone sarcomas.

Approximately 45% of sarcomas occur in the lower extremities, 15% in the upper extremities, 10% in the head-and-neck region, 15% in the retroperitoneum, and the remaining 15% in the abdominal and chest wall. Visceral sarcomas, arising from the connective tissue stroma in parenchymal organs, are not common.

The different types of soft tissue tumors have distinct age distributions.

Rhabdomyosarcoma is seen more frequently in children and young adults.

Synovial sarcoma arises in young adults.

Malignant fibrous histiocytoma and liposarcoma generally occur in older adults.

Benign deep masses in adults usually are due to intramuscular lipoma.

In general, the prognosis in older patients with a diagnosis of high-grade sarcoma is poor.

The incidence of soft tissue tumors is slightly higher in males than in females.

Etiology

Good evidence exists suggesting that certain genetic disorders and gene mutations are predisposing factors for some benign and malignant soft tissue tumors. The NF1 gene in neurofibromatosis is a classic example, predisposing patients to multiple neurofibromas with a proclivity for malignant transformation. Many tumor suppressor genes, oncogenes, and cytogenetic defects are now associated with various soft tissue sarcomas. Other clinical risk factors account for a small proportion of soft tissue malignancies.

A partial list of reported cytogenetic abnormalities is shown in Table 1. They have a significant role in diagnosis, and, in the future, some of these abnormalities may become therapeutically significant. Specific translocations involving selected genes have been observed. One of these, the t(X;18) translocation in synovial sarcoma, results in fusion of the SYT gene from chromosome 18 to either of 2 highly homologous genes at Xp11, SSX1 or SSX2. SYT-SSX fusion transcript may be detected by reverse transcriptase-polymerase chain reaction assay, using a cytologic specimen from fine-needle aspiration biopsy (FNAB), histologic material from paraffin block, or frozen material.

Similar to postirradiation bone tumors, postirradiation fibrosarcomas have been described. The pathogenetic mechanism is the emergence of radiation-induced genetic mutations that encourage neoplastic transformation.

Chronic lymphedema

As observed in patients with late-stage breast carcinoma, chronic lymphedema may predispose individuals to the development of lymphangiosarcoma.

Environmental carcinogens

An association between exposure to various carcinogens and an increased incidence of soft tissue tumors has been reported. The occurrence of hepatic angiosarcoma, for example, has been linked to arsenic, thorium dioxide, and vinyl chloride exposure.

A classic example of an infection-induced soft tissue tumor is Kaposi sarcoma resulting from human herpesvirus type 8 in patients with human immunodeficiency virus (HIV). Infection with Epstein-Barr virus in an immunocompromised host also increases the likelihood of soft tissue tumor development.

The relationship between trauma and soft tissue tumors appears to be coincidental. Trauma probably draws medical attention to a pre-existing lesion.

Pathophysiology

Generally, soft tissue tumors grow centripetally, although some benign tumors, such as fibrous lesions, may grow longitudinally along tissue planes. Most soft tissue tumors respect fascial boundaries, remaining confined to the compartment of origin until the later stages of development. Once the tumor reaches the anatomic limits of the compartment, the tumor is more likely to breach compartmental boundaries. Major neurovascular structures usually are displaced as opposed to being enveloped or invaded by tumor. Tumors arising in extracompartmental locations, such as the popliteal fossa, may expand more quickly because of a lack of fascial boundaries; they are also more likely to involve neurovascular structures.

The peripheral portion of the tumor compresses surrounding, normal soft tissue because of centripetal expansile growth. This results in the formation of a relatively well-defined zone of compressed fibrous tissue potentially containing scattered tumor cells. This zone may also consist of inflammatory cells and demonstrate neovascularity. A thin layer of tissue called the reactive zone surrounds the compression zone, especially in higher-grade tumors. Together, the compression and reactive zones form a pseudocapsule that encloses the tumor and is useful in defining the extent of surgical resection.

Some extremely aggressive lesions with infiltrative growth patterns, such as childhood rhabdomyosarcoma, may not respect anatomic compartmental boundaries and frequently will invade fascial planes.

Soft tissue sarcomas have the propensity to recur locally. Because recurrences are more difficult to treat than the primary lesion is, complete resection and appropriate use of radiation therapy are critical during the initial treatment. The pseudocapsule provides surgeons with a more or less obvious plane of dissection; however, such an excision can leave behind microscopic or occasionally gross tumor. This may lead to local recurrences in up to 80% of patients.4 The addition of postoperative radiation therapy decreases the risk of recurrence associated with a marginal resection. Technical ease of resectability (and, thus, the likelihood of local control) may be affected by the location of a soft tissue sarcoma. For example, lesions of the head and neck are more likely to involve or abut vital structures; consequently, they often are more difficult to resect than are lesions of the extremities. Even in an extremity, the tumor site may have prognostic implications. For proximal tumors, local control is more difficult to achieve than in tumors located more distally. Retroperitoneal sarcomas, which typically have a poor prognosis, have a higher proclivity for local recurrence and for intra-abdominal dissemination.

The pattern of recurrence generally is predictable, and most tumors destined to recur do so within the first 2-3 years. Adjuvant radiation therapy clearly minimizes local recurrence, but its ability to increase overall chances of survival, although likely, is not certain. Adjuvant chemotherapy may decrease the risk of local recurrence of high-grade tumors, presumably because of a reduction in the size of the tumor and an increase in the reactive zone, but this notion is very controversial.

Regional lymph node involvement is rare in soft tissue sarcomas; fewer than 4% of cases have nodal metastases at presentation. Lymph node involvement is more frequent in epithelioid sarcoma, rhabdomyosarcoma, synovial sarcoma, and clear cell sarcoma. Carcinoma and melanoma should be included in the differential diagnosis for any mass presenting with lymph node metastases.Many patients with high-grade soft tissue sarcomas, as well as a few with the low-grade type, progress to metastatic disease, even

following adequate local control of the primary tumor. The lung is by far the most common site of metastasis, which occurs in up to 52% of patients with high-grade lesions.5 Although, at the time of presentation, most patients do not have clinically evident metastases, they may have occult micrometastases that eventually manifest clinically. This would appear to be an impetus for the development of chemotherapeutic methods of systemic disease control. At present, however, this is a controversial area of investigation, and it is uncertain whether systemic chemotherapy can improve long-term survival rates for patients with high-grade sarcomas.

A mass is the most common sign of a soft tissue tumor. It usually is painless and does not cause limb dysfunction. However, depending on the anatomic location of the tumor, it may cause pain or neurologic symptoms by compressing or stretching nerves, by irritating overlying bursae, or by expanding sensitive structures. A rapid rate of increase in the size of a mass should arouse suspicion that the lesion is malignant.

Physical examination can be used to determine the location and size of a mass and to exclude other, more common causes of pain. Whether the mass is deep or subcutaneous, transilluminates (cysts), and adheres to underlying structures also can be gleaned from physical examination. Regional lymph nodes should be examined as well. Neurovascular examination is useful for the detection of either primary or secondary tumor involvement.

Extremity masses larger than 5-7 cm and deeper than subcutaneous tissue favor a diagnosis of a malignant soft tissue tumor. However, up to 30% of soft tissue sarcomas occur in subcutaneous tissue and exhibit relatively less aggressive behavior.

Medical Therapy

High-grade soft tissue sarcomas often are treated with ifosfamide- and doxorubicin-based chemotherapy. This is controversial, as no definitive studies exist proving that adjuvant chemotherapy contributes to prolonged overall survival.20,21

Surgical Therapy

Complete local excision is adequate treatment for benign soft tissue tumors. However, a variety of treatment options, including surgery alone or combined with radiation therapy or chemotherapy, may be considered for treatment of localized primary and recurrent sarcomas.

Extremity sarcoma

Extremity sarcomas may be treated surgically, with or without radiation therapy and adjuvant chemotherapy. Surgery is the most important component of any treatment plan for a clinically localized primary or recurrent soft tissue sarcoma. On the basis of the achievable margin, 4 types of excisions may be performed.

Intracapsular excisions and amputation - The excision or amputation passes within the tumor itself. The tumor inside the pseudocapsule is removed (often piecemeal). Incidence of local recurrence with these types of excisions is virtually 100%; these procedures are performed only in unusual circumstances.

Marginal excisions and amputation - The excision is performed through the pseudocapsule surrounding the tumor. Shelling-out procedures and most excisional biopsies belong to this category. The chance of local recurrence is 20-75%, depending on the nature of the tumor and whether or not radiotherapy is used.

Wide excisions and amputation - The tumor is excised with a wide margin of surrounding normal tissue but within the muscular compartment. Without adjuvant therapy, the incidence of local recurrence following wide excision varies but may reach 30%; the rate of recurrence depends on the selection criteria used and the adequacy of the histologically assessed surgical margin. A wide amputation is performed through the normal tissue proximal to the reactive zone around the tumor but remains within the involved compartment. Limb-sparing procedures belong to this category.

Radical excisions and amputation - These are en bloc excisions of the tumor along with the entire muscle compartment. Amputation with disarticulation of the joint proximal to the involved compartment is called radical amputation. The risk of local recurrence is lowest with this procedure.

Small, superficial, or low-grade tumors treated with only a wide, local excision have a very low risk of local recurrence.12 For better local control, many patients undergoing surgical excision receive radiation therapy. In patients who refuse or cannot tolerate surgery, radiation alone can be an effective treatment for certain extremity sarcomas.

Postoperative radiation therapy - Following wide surgical excision, radiation therapy enhances local control for primary extremity sarcomas. The concept of limb-sparing surgery with postoperative radiation has been validated by randomized trials of amputation versus wide local excision.22 Usually, a total dose of about 60 grays (Gy) is adequate.

Brachytherapy - Postoperative radiation can also be delivered to the tumor bed by means of brachytherapy (in which radioactive sources are implanted in the patient). The advantage of this approach is that it requires a much shorter time for initiation and completion of therapy than does external radiation. External beam radiation is used for 6 weeks beginning a month or more following surgery; brachytherapy usually is started within a week of surgery and completed in 4 or 5 days. Because of its technical complexity, brachytherapy requires an experienced radiation oncologist during the operating procedure. Brachytherapy and external beam radiation appear to be equally effective when properly administered.

Preoperative radiation therapy - The employment of preoperative radiation therapy may allow less radical forms of surgery to be used, specifically on large tumors that otherwise may compromise limb-sparing procedures. Radiation-induced tumor shrinkage decreases the magnitude of resection needed and reduces the risk of seeding by viable tumor cells. Local fibrosis may make the resection more challenging.

Even after achieving local control in patients with intermediate- and high-grade soft tissue sarcomas, the risk of metastatic disease following multimodality treatments without amputation is as high as 50%. The risk is even greater if stage IIIB tumors are included. Thus, effective systemic, adjuvant chemotherapy is desirable following definitive treatment of local disease. However, conclusive evidence that adjuvant chemotherapy for extremity sarcomas increases overall survival rates is lacking. Randomized trials have not demonstrated that higher overall survival rates occur with surgery and adjuvant doxorubicin therapy than with surgery alone.

In randomized clinical trials, multiagent chemotherapy with doxorubicin, cyclophosphamide, and methotrexate following surgery improved disease-free survival rates for patients with high-grade extremity sarcomas (except when the lesions were associated with the trunk or retroperitoneum).23 However, the toxicity associated with this regimen was substantial.24

Preoperative chemotherapy, also called neoadjuvant chemotherapy, is an option for most patients with osteosarcomas of the extremity. However, it has not been established that this treatment is superior to conventional chemotherapy for soft tissue tumors. Preoperative chemotherapy may be used alone or with preoperative or postoperative radiation therapy.

A significant hypothetical advantage of neoadjuvant chemotherapy is that it allows treatment effectiveness to be monitored through evaluation of the degree of necrosis in the resected primary tumor. However, no evidence exists that this results in improved clinical prognosis.

Nonextremity sarcoma

As with sarcomas of extremities, options for therapeutic management of nonextremity sarcomas include surgery, radiation, and chemotherapy. Sarcomas arising in the head and neck, thoracic or abdominal wall, mediastinum, or retroperitoneum are difficult to treat. Most of these tumors develop in areas where surrounding normal tissue limits the maximum dosage of radiation that can safely be delivered to the tumor bed. In general, the risk of local recurrence is high. For retroperitoneal tumors, the patient usually succumbs as a result of local complications, before metastases are evident.

Compressive bandages and suction drains should be used to minimize seroma formation that can delay administration of chemotherapy or radiation therapy. Physical therapy and rehabilitation support may be required.

Follow-up

General follow-up care includes surveillance studies to evaluate local recurrence and distant metastasis of malignant and intermediate tumors. The precise interval between and the duration of various follow-up studies are not well defined. In general, vigorous surveillance continues for 3-5 years after treatment. Benign tumors generally do not require such surveillance.

Complications

Complications can be divided into those that occur before therapy is completed and those that develop after its completion.

Before completion of therapy

Related to the tumor: Depending on histopathologic category and anatomic site, the tumor may cause complications such as skin ulceration, thrombocytopenia, hemorrhage, and fracture.

Related to operative procedures: Infection and wound dehiscence are possible.

After completion of therapy

Related to the tumor: Complications include local recurrence and distant metastasis.

Related to chemotherapy and radiation therapy: Infections may result from immunosuppression. Postirradiation sarcomas can occur, usually 10 years or longer after radiation therapy

Wilms Tumor

Wilms tumor (WT) is the fifth most common pediatric malignancy and the most common type of renal tumor in children. The treatment used to treat Wilms tumor is an example of success achieved through a multidisciplinary collaboration of the National Wilms' Tumor Study Group (NWTSG) and the Societe Internationale d'Oncologie Pediatrique (SIOP).

Fifty years ago, with surgery alone, the survival rate 2 years after nephrectomy was 20%. The introduction of adjuvant radiotherapy raised the survival rate to 50% overall. Owing to the cooperative efforts of oncologists, surgeons, and pathologists and with the introduction of chemotherapy with vincristine, dactinomycin (actinomycin D), and doxorubicin, the overall survival rate has risen to 90% in the last 30 years.

The incidence of Wilms tumor is approximately 0.8 cases per 100,000 persons. Approximately 500 new cases are diagnosed each year in the United States, with 6% involving both kidneys.

Etiology

Wilms tumor may arise in 3 clinical settings, the study of which resulted in the discovery of the genetic abnormalities that lead to the disease. Wilms tumors can arise sporadically, can develop in association with genetic syndromes, or can be familial. Although some of the molecular biology of Wilms tumor is coming to light, the exact cellular mechanisms involved in the etiology of the tumor are still being investigated.

Sporadic Wilms tumor

Most cases of Wilms tumor are not part of a genetic malformation syndrome and occur in the absence of a family history of the malignancy; however, familial Wilms tumor is very common in certain families. Genetic syndromes that predispose to and may include Wilms tumor include the following:

Beckwith-Wiedemann syndrome (macroglossia, gigantism, and umbilical hernia)

Hemihypertrophy

Congenital aniridia

Wilms tumor, aniridia, genitourinary malformations, and mental retardation (WAGR syndrome)

Denys-Drash syndrome (Wilms tumor, pseudohermaphroditism, and glomerulopathy)

Trisomy 18 mutation

These clinical observations have led to genetic and molecular studies that have enhanced discovery of the genetic mechanism that promotes Wilms tumor genesis. In addition, the molecular genetic characterization of Wilms tumor plays a major role in the understanding of the genetic aspects of carcinogenesis in general.

Based on the model developed originally for retinoblastoma, Knudson and Strong proposed that Wilms tumor results from 2 mutational events based on loss of function of tumor suppressor genes.

The first mutation, the inactivation of the first allele of the specific tumor suppressor gene, involves prezygotic and postzygotic aspects. Prezygotic (constitutional or germline) mutations are inherited or result from a de novo germline mutation. This mutation is present in all body cells and predisposes the patient to familial and/or multiple Wilms tumor. Postzygotic mutations occur only in specific cells, and they predispose to single tumors and sporadic cases of Wilms tumor.

The second mutation is inactivation of the second allele of the specific tumor suppressor gene.

Although the model of the retinoblastoma suppressor gene has been used to explain the genetics, clinical characteristics of Wilms tumor suggest that the molecular genetic mechanism in the second type of mutation depends on more than one tumor suppressor gene.

The WT1 gene (at chromosome 11p13) is a tissue-specific gene for renal blastemal cells and glomerular epithelium, with both renal precursor cells thought to harbor sites of origin of Wilms tumor. The expression of WT1 peaks around birth. As the kidney matures, the expression declines. It is also a dominant oncogene; hence, a certain mutation in only 1 of the 2 alleles is enough to promote changes that may lead to the formation of Wilms tumor. The WT2 gene (at chromosome 11p15) remains isolated.

In addition, several genetic factors have been identified as possible prognostic factors in individuals with Wilms tumor. One such factor is loss of heterozygosity at chromosomes 1p and 16q. Children with loss of heterozygosity at 16q appear to be at greater risk of relapse and mortality than children without this genetic change.2 According to the latest NWTS-5 study, tumor-specific loss of heterozygosity for both chromosomes 1p and 16q, identified in about 5% of patients with favorable-histology Wilms tumor, was shown to be associated with a significantly increased risk of relapse and death.

The pathophysiology of Wilms tumor is characterized by an abnormal proliferation of the metanephric blastema cells, which are believed to be primitive embryologic cells of the kidney.

Presentation

Wilms tumor is diagnosed at a mean age of 3.5 years. The most common feature at presentation is an abdominal mass. Abdominal pain occurs in 30%-40% of cases. Other signs and symptoms of Wilms tumor include hypertension, fever caused by tumor necrosis, hematuria, and anemia.

Major congenital anomalies include genitourinary anomalies (WAGR and Denys-Drash syndromes, 5% of cases), ectopic solitary horseshoe kidney, hypospadias and cryptorchidism, hemihypertrophy and organomegaly (Beckwith-Wiedemann syndrome, 2% of cases); and aniridia (1% of cases). Children with such syndrome anomalies should undergo periodic testing for Wilms tumor. Ultrasonography of the kidneys (once or twice per year) is a good screening tool.

Indications for primary surgical excision of a Wilms tumor (WT) include tumors confined to the kidney, extending beyond the kidney but not crossing the midline, and with or without vascular extension. Postchemotherapy excision of the tumor is indicated in patients with bilateral tumors, tumors that extended beyond the midline and have shrunk, and tumors with vascular extension. Surgery alone is not recommended for Wilms tumor based on the results of the NWTS-5 study.

Wilms tumor (WT) arises from the primitive embryonal renal tissue. Grossly, Wilms tumor is typically an intrarenal solid or cystic mass, which may displace and, in rare cases, invade the renal collecting system. The tumor extends into the renal vein in 40% of cases. In very rare cases, it extends into the ureter and down to the bladder, where it may cause hematuria. Wilms tumor is bilateral in 6% of cases. Local invasion is rare and tumor spread is usually through lymphatic and vascular routes.

Contraindications to primary surgery for Wilms tumor (WT) include bilateral tumors and documented metastatic disease. Large tumors that extend beyond the midline, have vascular extension, or both are relative contraindications since some surgeons elect to obtain tissue via surgical excision, but this may expose patients to increased surgical risks.

Laboratory Studies

Complete blood count, Basic metabolic panel, including serum calcium levels, Coagulation abnormalities (to rule out acquired von Willebrand disease, which is coincident in up to 8% of individuals with Wilms tumor),Liver function tests,Renal function tests,Urinalysis and urine culture,Imaging Studies,Ultrasonography

Initial diagnosis of a renal or abdominal mass, possible renal vein or inferior vena cava (IVC) thrombus (Doppler flow study may be helpful in the setting of vascular invasion.)

Information regarding liver and other kidney: CT scanning of the chest and abdomen, Differential diagnosis of a kidney tumor versus adrenal tumor (neuroblastoma), Liver metastases, Status of opposite kidney, Lymph node assessment, Status of chest with respect to metastases, Chest radiography - As a baseline for pulmonary metastases, Bone scan - Necessary for children with clear cell sarcoma of the kidney , Magnetic resonance imaging

Typically, these tumors appear inhomogeneous on gadolinium-enhanced MRI, while the nephrogenic rests (which sometimes are precursors of Wilms tumor [WT]) appear as homogeneous masses.MRI is also useful for magnetic resonance venography to aid in the diagnosis of thrombus of the renal vein of the IVC. MRI scanning of the head is recommended in patients with suspected rhabdoid and clear cell carcinoma of the kidney.

Wilms tumor arises from the primitive embryonal renal tissue and contains epithelial, stromal, and blastemal elements.

Favorable histology (90% of cases) is characterized by all 3 histological elements, without any anaplastic features. The cure rate in these cases is close to 90%. Occasionally, foci of cartilaginous, adipose, or muscle tissue may appear (ie, teratoid Wilms tumor).

Unfavorable histology (10% of cases) is characterized by the presence of anaplasia. Clear cell carcinoma of the kidney (bone-metastasizing renal tumor of childhood) and rhabdoid tumor of the kidney are now considered distinct type tumors and should not be included.

Anaplasia is defined by nuclear enlargement, nuclear hyperchromasia, and abnormal mitoses. Focal anaplasia is defined as less than 10% anaplastic features in a specimen, whereas more than 10% is considered diffuse anaplasia. Nephrogenic rests are foci of abnormally present nephrogenic renal blastemal cells (metanephric blastema). These are considered precursors of Wilms tumor. Nephroblastomatosis is the diffuse presence of nephrogenic rests. It may be perilobar; intralobar (usually the more primitive elements are situated intralobarly), which has been associated more frequently with the development of Wilms tumor than the perilobar blastemal rests; or panlobular.

Grossly, Wilms tumor is typically an intrarenal solid or cystic mass that displaces the collecting system. It usually has a pseudocapsule and may contain hemorrhage and necrosis. The tumor extends into the renal vein in 40% of cases. It rarely extends into the ureter and bladder.

The partially differentiated cystic nephroblastoma (ie, multilocular cystic nephroma) with possible Wilms elements is generally considered a benign lesion.

Staging

NWTSG recommends surgical staging in every case. In addition, recent data have suggested that patients with Wilms tumor undergo an individual risk assessment based on biological determinants or markers.

Stage I: The tumor is limited to the kidney and is excised completely.

Stage II: The tumor extends beyond the kidney but is excised completely. Capsular penetration, renal vein involvement, and renal sinus involvement may also be found. A biopsy of the tumor is performed, and local spillage occurs.

Stage III: Residual intra-abdominal tumor (nonhematogenous) exists after the completion of surgery. Lymph node findings are positive, or peritoneal implants are found. The resected specimen has histologically positive margins, or the tumor has been spilled into the abdominal cavity.

Stage IV: Hematogenous or lymph node metastasis has occurred outside the abdomen or pelvis.
Stage V: Synchronous bilateral involvement has occurred. Each side is assigned a stage from I to III, and histology is based on biopsy findings.
Chemotherapy is essential in the treatment of Wilms tumor (WT). Refinements in the combination, length, and mode of administration of the various chemotherapeutic agents have resulted from the successive NWTS trials and have helped to optimize survival rates while minimizing acute and chronic toxicities.
Chemotherapy protocols vary from study to study; however, the main agents administered include vincristine, dactinomycin, and doxorubicin. Cyclophosphamide can be added in the presence of unfavorable histology and advanced stage.
In the SIOP trials, chemotherapy is administered up front to reduce tumor volume, thereby decreasing the risk of surgical spillage of tumor.
Radiation therapy is restricted to treatment of higher-stage (stage II with unfavorable histology and stages III and IV) disease.
According to the NWTSG protocol, the first step in the treatment of Wilms tumor is surgical staging followed by radical nephrectomy, if possible. Begin the abdominal exploration through a transverse incision. The kidney is explored by mobilizing the ipsilateral colon and opening the Gerota fascia. Exploration of the contralateral kidney is currently not recommended because of the improvement in imaging techniques (CT scan, MRI). If bilateral disease is diagnosed, nephrectomy is not performed, but biopsy specimens are obtained. New protocols in the management of bilateral Wilms tumor are being explored. If the disease is unilateral, radical nephrectomy and regional lymph node dissection or sampling are performed.
The role of partial nephrectomy remains controversial. Although end-stage renal disease after unilateral radical nephrectomy is uncommon (0.25% in NWTSG trial), preserving healthy renal tissue may prevent this complication, especially in patients with an underlying intrinsic renal disease (eg, WAGR syndrome, Denys-Drash syndrome). Partial nephrectomy may be feasible in only 10%-15% of patients, as most tumors are too large at initial diagnosis. The main concern regarding a nephron-sparing procedure is that of local recurrence. The NWTS-4 study showed an 8% local recurrence rate following partial nephrectomy for patients with bilateral disease.3 In the presence of bilateral Wilms tumors, solitary kidney, or renal insufficiency, partial nephrectomy is a reasonable consideration.
If the tumor is unresectable, biopsies are performed and the nephrectomy is deferred until after chemotherapy, which, in most cases, will shrink the tumor. Contiguous involvement of adjacent organs is frequently overdiagnosed. The overall surgical complication rate for Wilms tumor is approximately 20%. If IVC thrombus is present, preoperative chemotherapy will reduce the cavotomy rate by 50%.
With bilateral Wilms tumor (6% of cases), surgical exploration, biopsies from both sides, and accurate surgical staging (including lymph node biopsy of both sides) are performed. This is followed by 6 weeks of chemotherapy that is appropriate to the stage and histology of the tumor. Then, reassessment is performed using imaging studies, followed by definitive surgery with (1) unilateral radical nephrectomy and partial nephrectomy on the contralateral side; (2) bilateral partial nephrectomy; and (3) unilateral nephrectomy only, if the response was complete on the opposite side. This approach dramatically reduces the renal failure rate following bilateral Wilms tumor therapy.
The overall 2-year survival rate is higher than 80% with this approach, and the nephrectomy rate drops by 50% in patients with bilateral Wilms tumor. Bilateral partial nephrectomy is possible after chemotherapy or, if the tumor on one side responds completely to chemotherapy, with no subsequent need for nephrectomy.
Tumor biomarkers, histology, and stage are the most important prognostic factors in cases of unilateral disease. Bilateral high-stage tumors with unfavorable histology are associated with a poor prognosis in spite of the multimodal therapy.

Multimodal therapy (ie, surgery, radiation, chemotherapy) is the key to success when treating Wilms tumor. The NWTSG recommends preoperative chemotherapy (after initial exploratory laparotomy and biopsy) in the following situations:4

Intracaval tumor extension: This occurs in 5% of cases of Wilms tumor. It is associated with a 40% rate of surgical complications, even in experienced hands. Upfront chemotherapy after staging and biopsy reduces tumor and thrombus size, which account for 25% of surgical complications. Inoperable tumors: Large tumors that involve vital structures make resection difficult. The complication rate is high, and the incidence of tumor spill soilage is also high. Upfront chemotherapy reduces soilage by 50%.

Chemotherapy without proper surgical staging (eg, staging by means of imaging studies only) may alter the actual initial stage of the disease by the time of surgery and may subsequently alter decisions regarding the adjuvant chemotherapy and radiation therapy, which is based on the surgical staging.

Intraoperative Details

Through a transperitoneal approach, enter the Gerota (perinephric fascia) fascia to examine the kidney. In cases of unilateral tumor, perform a radical nephrectomy if the opposite side is normal. Evaluate the liver, lymph nodes, and peritoneum for metastases. The renal vein and IVC should be palpated to assess intravascular extension (present in 6% of the cases).

In cases of bilateral disease, excisional biopsy of visible tumor is indicated, followed by re-resection with nephron-preservation after chemotherapy. Identify the involved nodes with clips to facilitate postoperative radiation therapy.

Integrity of the surgical specimen is of paramount importance, as tumor spillage has a deleterious impact on prognosis (6-fold increase in local abdominal recurrence).

Postoperative chemotherapy and radiotherapy protocols are based on the surgical staging and follow the guidelines of the NWTSG.

Stage I favorable histology and unfavorable histology or stage II favorable histology

Neuroblastoma in children.

Neuroblastoma is the most common extracranial solid tumor in infancy. It is an embryonal malignancy of the sympathetic nervous system arising from neuroblasts (pluripotent sympathetic cells). In the developing embryo, these cells invaginate, migrate along the neuraxis, and populate the sympathetic ganglia, adrenal medulla, and other sites. The pattern of distribution of these cells correlates with the sites of primary disease presentation.

Age, stage, and biological features encountered in tumor cells are important prognostic factors and are used for risk stratification and treatment assignment. The differences in outcome for patients with neuroblastoma are striking. Patients with low-risk and intermediate-risk neuroblastoma have excellent prognosis and outcome. However, those with high-risk disease continue to have very poor outcomes despite intensive therapy. Unfortunately, approximately 70-80% of patients older than 18 months present with metastatic disease, usually in the lymph nodes, liver, bone, and bone marrow. Less than half of these patients are cured, even with the use of high-dose therapy followed by autologous bone marrow or stem cell rescue.

Histologic subtypes of neuroblastoma. Top right panel, neuroblastoma: A monotonous population of hyperchromatic cells with scant cytoplasm. Bottom left panel, ganglioneuroblastoma: Increased schwannian stroma. Bottom right panel, ganglioneuroma: Mature ganglion cell with schwannian stroma.

Over the last 2 decades, many chromosomal and molecular abnormalities have been identified in patients with neuroblastoma. These biologic markers have been evaluated to determine their value in assigning prognosis, and some of these have been incorporated into the strategies used for risk assignment.

The most important of these biologic markers is MYCN. MYCN is an oncogene that is overexpressed in approximately one quarter of cases of neuroblastoma via the amplification of the distal arm of chromosome 2. This gene is amplified in approximately 25% of de novo cases and is more common in patients with advanced-stage disease. Patients whose tumors have MYCN amplification tend to have rapid tumor

progression and a poor prognosis, even in the setting of other favorable factors such as low-stage disease or 4S disease.

In contrast to MYCN, expression of the H-ras oncogene correlates with lower stages of the disease. Cytogenetically, the presence of double-minute chromatin bodies and homogeneously staining regions correlates with MYCN gene amplification. Deletion of the short arm of chromosome 1 is the most common chromosomal abnormality present in neuroblastoma and confers a poor prognosis. The 1p chromosome region likely harbors tumor suppressor genes or genes that control neuroblast differentiation. Deletion of 1p is more common in near-diploid tumors and is associated with a more advanced stage of the disease. Most of the deletions of 1p are located in the 1p36 area of the chromosome.

A relationship between 1p loss of heterozygosity (LOH) and MYCN amplification has been described. Other allelic losses of chromosomes 11q, 14q, and 17q have been reported, suggesting that other tumor suppressor genes may be located in these chromosomes. Another characteristic of neuroblastoma is the frequent gain of chromosome 1.

DNA index is another useful test that correlates with response to therapy in infants. Look et al demonstrated that infants whose neuroblastoma have hyperdiploidy (ie, DNA index >1) have a good therapeutic response to cyclophosphamide and doxorubicin.1 In contrast, infants whose tumors have a DNA index of 1 are less responsive to the latter combination and require more aggressive therapy. DNA index does not have any prognostic significance in older children. In fact, hyperdiploidy in children more frequently occurs in the context of other chromosomal and molecular abnormalities that confer a poor prognosis.

Three neurotrophin receptor gene products, TrkA, TrkB, and TrkC, are tyrosine kinases that code for a receptor of members of the nerve growth factor (NGF) family. Their ligands include p75 neurotrophin receptor (p75NTR) NGF, and brain-derived neurotrophic factors (BDNFs). Interestingly, TrkA expression is inversely correlated with the amplification of the MYCN gene, and the expression of the TrkC gene is correlated with TrkA expression. In most patients younger than 1 year, a high expression of TrkA correlates with a good prognosis, especially in patients with stages 1, 2, and 4S. In contrast, TrkB is more commonly expressed in tumors with MYCN amplification. This association may represent an autocrine survival pathway.

Disruption of normal apoptotic pathways may also play a role in neuroblastoma pathology. Disruption of these normal pathways may play a role in therapy response as a result of epigenetic silencing of gene promoters in apoptotic pathways. Drugs that target DNA methylation, such as decitabine, are being explored in preliminary studies.

Other biologic markers associated with poor prognosis include increased levels of telomerase RNA and lack of expression of glycoprotein CD44 on the tumor cell surface. P-glycoprotein (P-gp) and multidrug resistance protein (MRP) are 2 proteins expressed in neuroblastoma. These proteins confer a multidrug-resistant (MDR) phenotype in some cancers. Their role in neuroblastoma is controversial. Reversal of MDR is one target for novel drug development.

Origin and migration pattern of neuroblasts during fetal development explains the multiple anatomic sites where these tumors occur; location of tumors varies with age. Tumors can develop in the abdominal cavity (40% adrenal, 25% paraspinal ganglia) or other sites (15% thoracic, 5% pelvic, 3% cervical tumors, 12% miscellaneous). Infants more commonly present with thoracic and cervical tumors, whereas older children more frequently have abdominal tumors.

Most patients present with signs and symptoms related to tumor growth, although small tumors have been detected due to the common use of prenatal ultrasonography. Large abdominal tumors often result in increased abdominal girth and other local symptoms (eg, pain). Paraspinal dumbbell tumors can extend into the spinal canal, impinge on the spinal cord, and cause neurologic dysfunction.

Stage of the tumor at the time of diagnosis and age of the patient are the most important prognostic factors. Although patients with localized tumors (regardless of age) have an excellent outcome (80-90% 3-year

event-free survival [EFS] rate), patients older than 18 months with metastatic disease fare poorly. Generally, more than 50% of patients present with metastatic disease at the time of diagnosis, 20-25% have localized disease, 15% have regional extension, and approximately 7% present during infancy with disseminated disease limited to the skin, liver, and bone marrow (stage 4S).

More than 90% of patients have elevated homovanillic acid (HVA) and/or vanillylmandelic acid (VMA) levels detectable in urine. Mass screening studies using urinary catecholamines in neonates and infants in Japan, Quebec, and Europe have demonstrated the ability to detect neuroblastoma before it is clinically apparent. However, most of the tumors identified occur in infants with a good prognosis. None of these studies show that mass screening decreases deaths due to high-risk neuroblastoma. Markers associated with a poor prognosisinclude (1) elevated ferritin levels, (2) elevated serum lactate dehydrogenase (LDH) levels, and (3) elevated serum neuron-specific enolase (NSE) levels. However, these markers have become less important due to the discovery of more relevant biomarkers (ie, chromosomal and molecular markers). In fact, ferritin was not included in the recent formulation of the International Neuroblastoma Risk Group Classification System because it was not found to be of prognostic difference in the high risk group.

Pluripotent sympathetic stem cells migrate and differentiate to form the different organs of the sympathetic nervous system. The normal adrenal gland consists of chromaffin cells, which produce and secrete catecholamines and neuropeptides. Other cells include sustentacular cells, which are similar to Schwann cells, and scattered ganglion cells. Histologically, neural crest tumors can be classified as neuroblastoma, ganglioneuroblastoma, and ganglioneuroma, depending on the degree of maturation and differentiation of the tumor.

The undifferentiated neuroblastomas histologically present as small, round, blue cell tumors with dense nests of cells in a fibrovascular matrix and Homer-Wright pseudorosettes. These pseudorosettes, which are observed in 15-50% of tumor samples, can be described as neuroblasts surrounding eosinophilic neuritic processes. The typical tumor shows small uniform cells with scant cytoplasm and hyperchromatic nuclei. A neuritic process, also called neuropil, is a pathognomonic feature of neuroblastoma cells. NSE, chromogranin, synaptophysin, and S-100 immunohistochemical stains are usually positive. Electron microscopy can be useful because ultrastructural features (eg, neurofilaments, neurotubules, synaptic vessels, dense core granules) are diagnostic for neuroblastoma.

In contrast, the completely benign ganglioneuroma is typically composed of mature ganglion cells, Schwann cells, and neuritic processes, whereas ganglioneuroblastomas include the whole spectrum of differentiation between pure ganglioneuromas and neuroblastomas. Because of the presence of different histologic components, the pathologist must thoroughly evaluate the tumor; the regions with different gross appearance may demonstrate a different histology.

Neuroblastic nodules are present in the fetal adrenal gland and peak at 17-18 weeks' gestation. Most of these nodules spontaneously regress and likely represent remnants of fetal development. Some of these may persist and lead to the development of neuroblastoma.

Shimada et al developed a histopathologic classification in patients with neuroblastoma.2 This classification system was retrospectively evaluated and correlated with outcome in 295 patients with neuroblastoma who were treated by the Children's Cancer Group (CCG). Important features of the classification include (1) the degree of neuroblast differentiation, (2) the presence or absence of Schwannian stromal development (stroma-rich, stroma-poor), (3) the index of cellular proliferation (known as mitosis-karyorrhexis index [MKI]), (4) nodular pattern, and (5) age. Using these components, patients can be classified into the following histology groups:

Favorable histology group

Patients of any age with stroma-rich tumors without a nodular pattern

Patients younger than 18 months with stroma-poor tumors, an MKI of less than 200/5000 (200 karyorrhectic cells per 5000 cells scanned), and differentiated or undifferentiated neuroblasts

Patients younger than 60 months with stroma-poor tumors, an MKI of less than 100/5000, and well-differentiated tumor cells

Neuroblastoma accounts for approximately 7.8% of childhood cancers in the United States. Approximately 650 new cases are diagnosed in the United States each year. According to the Surveillance, Epidemiology, and End Report (SEER), incidence is approximately 9.5 cases per million children.

Incidence in other industrialized nations appears to be similar to that observed in the United States. International reports have shown that the incidence rates of neuroblastoma are highest among high income countries in Europe and North America, and lower in low income countries in Africa, Asia, and Latin America. No published data are available on the incidence in the emerging high-income countries of Asia.5

According to the SEER data, the overall 5-year survival rate for children with neuroblastoma has improved from 24% in 1960-1963 to 55% in 1985-1994.4 In part, this increase in survival rate may be due to better detection of low-risk tumors in infants. The survival rate 5 years from diagnosis is approximately 83% for infants, 55% for children aged 1-5 years, and 40% for children older than 5 years. Improvements in diagnostic imaging modalities, medical and surgical management, and supportive care have contributed to the improved survival rates.6

Most patients with neuroblastoma present with disseminated disease, which confers a poor prognosis and is associated with a high mortality rate. Tumors in these patients usually have unfavorable pathologic and/or molecular features. The 3-year EFS for high-risk patients treated with conventional chemotherapy, radiation therapy, and surgery is less than 20%. Differentiating agents and dose intensification of active drugs, followed by autologous bone marrow transplant, have been reported to improve the outcome for these patients, contributing to an EFS of 38%. A recent single-arm study of tandem stem cell transplantation reported a 3-year EFS of 58%, but this has not been tested in a randomized fashion.7

Morbidity of high-dose chemotherapy approaches can be substantial, although the treatment-related mortality rates have decreased with improvements in supportive care and hematopoietic support with growth factors and stem cells instead of bone marrow.

Males have a slightly higher incidence of neuroblastoma than females, with a male-to-female ratio of 1.2:1.

Age distribution is as follows: 40% of patients are younger than 1 year when diagnosed, 35% are aged 1-2 years, and 25% are older than 2 years when diagnosed. According to SEER, incidence decreases every consecutive year up to age 10 years, after which the disease is rare.4

The following may be noted in patients with neuroblastoma:

Signs and symptoms of neuroblastoma vary with site of presentation. Generally, symptoms include abdominal pain, emesis, weight loss, anorexia, fatigue, and bone pain. Hypertension is an uncommon sign of the disease and is generally caused by renal artery compression, not catecholamine excess. Chronic diarrhea is a rare presenting symptom secondary to tumor secretion of vasoactive intestinal peptide secretion.

Because more than 50% of patients present with advanced stage disease, usually to the bone and bone marrow, the most common presentation includes bone pain and a limp. However, patients may also present with unexplained fever, weight loss, irritability, and periorbital ecchymosis secondary to metastatic disease to the orbits. The presence of bone metastases can lead to pathologic fractures.

Approximately two thirds of patients with neuroblastoma have abdominal primaries. In these circumstances, patients can present with an asymptomatic abdominal mass that usually is discovered by the parents or a caregiver. Symptoms produced by the presence of the mass depend on its proximity to vital structures and usually progress over time.

Tumors that arise from the paraspinal sympathetic ganglia can grow through the spinal foramina into the spinal canal and impinge on the spinal cord. This may result in the presence of neurologic symptoms, including weakness, limping, paralysis, and even bladder and bowel dysfunction. Thoracic neuroblastomas (posterior mediastinum) may be asymptomatic and are usually diagnosed by imaging studies obtained for other reasons. Presenting signs or symptoms may be insignificant and involve mild airway obstruction or

chronic cough, leading to chest radiography. Thoracic tumors extending to the neck can produce Horner syndrome. Primary cervical neuroblastoma is rare but should be considered in the differential diagnosis of masses of the neck, especially in infants younger than 1 year with feeding or respiratory difficulties.

In a small proportion of infants younger than 6 months, neuroblastoma presents with a small primary tumor and metastatic disease confined to the liver, skin, and bone marrow (stage 4S). If this type of tumor develops in neonates, skin lesions may be confused with congenital rubella, and, if the patient has severe skin involvement, the term "blueberry muffin baby" may be used.

Approximately 2% of patients present with opsoclonus and myoclonus a paraneoplastic syndrome characterized by the presence of myoclonic jerking and random eye movements. These patients often have localized disease and a good long-term prognosis. Unfortunately, the neurologic abnormalities can persist or progress and can be devastating. Finally, intractable diarrhea is a rare paraneoplastic symptom and is associated with more differentiated tumors and a good prognosis.

The following may be noted in patients with neuroblastoma:

Children are usually referred to a pediatric oncologist by primary care providers who have identified a persistent unexplained symptom or sign, either upon physical examination or based on screening test findings. In patients with suspected neuroblastoma, performing a thorough examination with careful attention to vital signs (eg, blood pressure), neck, chest, abdomen, skin, and nervous system is essential.- Metastatic lesions of the skin are common in infants younger than 6 months and may represent stage 4S disease.

Examination of the abdomen may reveal an abdominal mass, leading to the appropriate workup.

Neurologic examination may reveal Horner syndrome. In the case of dumbbell tumors, compression of the spinal cord may produce lower extremity weakness or paraplegia. Patients with neurologic involvement by tumor should be treated emergently, secondary to the risk of permanent neurologic sequelae.

The cause of neuroblastoma is unknown, and no specific environmental exposure or risk factors have been identified.

Because of young age of onset with this disease, investigators have focused on events before conception and during gestation.

According to SEER data, factors investigated for which evidence is limited or inconsistent include medications, hormones, birth characteristics, congenital anomalies, previous spontaneous abortion or fetal death, alcohol or tobacco use, and paternal occupational exposures.

The vast majority of neuroblastoma arises sporadically without family history of the disease. However, 1-2% of newly diagnosed cases do have a family history of neuroblastoma.

Patients with familial neuroblastoma often present at earlier age or with several distinct primary tumors.

Neuroblastoma has been known to occur in the setting of other disorders that are linked to abnormal development of neural crest tissues, such as Hirschsprung disease or central congenital hypoventilation syndrome.

Recent work using genome-wide analysis of neuroblastoma from these rare familial cases has identified a genetic defects involved in these cases.

Cases of neuroblastoma that accompany other congenital abnormalities of the neural crest have been associated with a germline mutation in PHOX2B. This gene is a homeobox gene that acts as a regulator of autonomic nervous system development.

In familial neuroblastoma cases that are not associated with other congenital disorders of neural crest development, ALK mutations have been identified in the germline.8 These mutations largely occur in the kinase domain causing activation of ALK signaling. Efforts are ongoing to investigate the incidence of ALK mutations across all subsets of neuroblastoma, but initial evidence indicates that somatic mutations of the ALK gene are also present in some cases of sporadic neuroblastoma.

Any child with a presumed diagnosis of neuroblastoma or any other childhood cancer should be referred to a pediatric cancer center for proper care and evaluation. Laboratory studies should include the following:
CBC count and differential (Anemia or other cytopenias suggest bone marrow involvement.)
Urine collection for catecholamines (VMA/HVA) and UA
A single sample or collected urine test for VMA/HVA is highly accurate in CLIA approved laboratories. Centers usually send samples to a specialty laboratory and/or perform a timed collection of urine.
A urinary catecholamine level is considered to be elevated if it is 3 standard deviations higher than the age-related reference range levels.
The following studies may be indicated in patients with neuroblastomas:
Obtain chest and abdominal radiographs to evaluate for the presence of a posterior mediastinal mass or calcifications.
A CT scan of the primary site is essential to determine tumor extent. The main body of the tumor is usually indistinguishable from nodal masses. See the images below.
CT scan of abdomen in a patient with a retroperitoneal mass arising from the upper pole of the left kidney and elevated urine catecholamines.
MRI of a left adrenal mass. The mass was revealed by fetal ultrasonography at 30 weeks' gestation. During infancy, the mass was found on the inferior pole of the left adrenal and was completely resected. Before surgery, the metastatic workup was negative. Surgical pathology service confirmed a diagnosis of neuroblastoma. After 3 years of follow-up care, no recurrence was observed.
I123/131 -methyliodobenzylguanadine (MIBG) accumulates in catecholaminergic cells and provides a specific way of identifying primary and metastatic disease if present. Increasing numbers of institutions have access to MIBG scanning.
A technetium-99 bone scan can also be used to evaluate bone metastases. This may be especially helpful in patients with negative MIBG study findings. Most current therapeutic protocols require both a bone scan and MIBG scan.
Skeletal surveys may also be useful, especially in patients with multiple metastatic lesions.
Positron emission tomography (PET) scan are under evaluation but are not currently recommended as part of the radiographic workup.
Biopsy or resection of the primary tumor (stage I or II disease) is performed to collect tissue samples for biologic studies used to assign the patient into the appropriate risk category. Most centers in the United States perform limited open biopsies when the primary tumor is unresectable upfront. Adequate tissue is needed to perform molecular studies that aid in risk assignment. Extensive resections should be avoided upfront if they may place patient at excessive risk from morbidity or mortality from surgery. Neuroblastoma is a chemo-sensitive tumor; thus, second-look surgery to resect a residual primary may be a safer procedure with biopsy only performed upfront.
Tissue samples from a primary or metastatic tumor may be undifferentiated and confused with other small, round, blue cell tumors of childhood; however, immunohistochemical stains can aid with tissue diagnosis.
Molecular techniques, such as fluorescent in situ hybridization (FISH), can detect MYCN amplification, an important prognostic marker. Polymerase chain reaction (PCR) can identify specific translocations, such as t(11;22), in Ewing sarcoma and t(2;13) in alveolar rhabdomyosarcoma, thus ruling out neuroblastoma.
Neuroblastoma in bone marrow can be difficult to distinguish from other small, round, blue cell tumors of childhood.
Biopsy findings are usually required to diagnose neuroblastoma. Depending on the extent of disease at presentation, consider complete surgical resection, especially in patients with low-stage disease. Even without a biopsy, the presence of elevated urinary catecholamines and a bone marrow aspirate or biopsy with unequivocal neuroblastoma cells is diagnostic.

Histologically, neural crest tumors can be classified as neuroblastoma, ganglioneuroblastoma, and ganglioneuroma, depending on the degree of maturation and differentiation of the tumor. Undifferentiated neuroblastomas histologically present as small, round, blue cell tumors with dense nests of cells in a fibrovascular matrix and Homer-Wright pseudorosettes. These pseudorosettes, observed in 15-50% of tumor samples can be described as neuroblasts surrounding eosinophilic neuritic processes. The typical tumor shows small uniform cells with scant cytoplasm and hyperchromatic nuclei. A neuritic process, also called neuropil, is a pathognomonic feature of neuroblastoma.

Neuron-specific enolase (NSE), chromogranin, synaptophysin, and S-100 immunohistochemical stain findings are usually positive. Electron microscopy can be useful because ultrastructural features (eg, neurofilaments, neurotubules, synaptic vessels, dense core granules) are diagnostic for neuroblastoma. In contrast, the completely benign ganglioneuroma is typically composed of mature ganglion cells, Schwann cells, and neuritic processes, whereas ganglioneuroblastomas include the whole spectrum of differentiation between pure ganglioneuromas and neuroblastomas.

The pathologist must thoroughly evaluate the tumor because regions with different gross appearance may exhibit a different histology.

The patient should undergo a staging workup along with surgical resection or biopsy, as appropriate. Using various molecular features in conjunction with pathology and staging is essential to appropriately stratify patients and determine the best therapy.

The International Neuroblastoma Staging System (INSS) is currently used in all cooperative group studies in the United States. Recently, the International Neuroblastoma Risk Group Staging System (INRGSS) and International Neuroblastoma Risk Group Consensus Pretreatment Classification were released.9 The current INSS system is based on degree of surgical resection and thus is not appropriate for use with the INRG Pretreatment Classification. This is especially important because not all groups use upfront surgical resection as part of their staging system. The INRG was formulated to be used in international settings and to facilitate comparison of treatment outcomes across studies to allow common definitions among all groups. Thus, development of the INRGSS was facilitated using pretreatment tumor imaging rather than extent of surgical resection.

The INRGSS is as follows:

L1 - Localized tumor not involving vital structures, as defined by the list of image-defined risk factors and confined to one body component

L2 - Locoregional tumor with presence of one or more image-defined risk factors

M - Distant metastatic disease

MS - Metastatic disease in children younger than 18 months with metastases confined to skin, liver, and/or bone marrow

The INSS is as follows:

Stage 1

Localized tumor with complete gross excision, microscopic residual disease, or both

Ipsilateral lymph nodes negative for tumor (Nodes attached to the primary tumor may be positive for tumor).

Stage 2A

Localized tumor with incomplete gross resection

Representative ipsilateral nonadherent lymph nodes microscopically negative for tumor

Stage 2B

Localized tumor, complete gross excision, or both with ipsilateral nonadherent lymph nodes positive for tumor

Enlarged contralateral lymph nodes, which are negative for tumor microscopically

Stage 3

Unresectable unilateral tumor infiltrating across the midline, regional lymph node involvement, or both

Alternatively, localized unilateral tumor with contralateral regional lymph node involvement
Stage 4 - Any primary tumor with dissemination to distant lymph nodes, bone, bone marrow, liver, skin, and/or other organs (except as defined for stage 4S)
Stage 4S
Localized primary tumor (as defined for stages 1, 2A, or 2B) with dissemination limited to skin, liver, and/or bone marrow (<10% involvement)
Limited to infants
Care of children with neuroblastoma is provided by a multidisciplinary team involving pediatric oncology, radiation oncologists, surgeons, and anesthesiologists, as well as nurse practitioners, nurses, pharmacists, psychologists, and physical and occupational therapists dedicated to the special needs of these children.
The table below outlines criteria for risk assignment based on the International Neuroblastoma Staging System (INSS), age, and biologic risk factors. This, in turn, determines the intensity of the therapy. These treatment strategies have been developed from more than 2 decades of experience with clinical trials in Children's Cancer Group (CCG) and Pediatric Oncology Group (POG), now known as the Children's Oncology Group (COG). Correlative biologic studies were pivotal in identifying biologic risk factors important for outcome. Currently, efforts are ongoing to develop an International Neuroblastoma Risk Group (INRG).
In addition, recently published results on correlative biologic studies and clinical outcome have lead to changes in an age cut-off of more than 365 days (365-547 d) for some patients with tumors in stages 3 and 4.10,11 These criteria are based on the analysis of several thousands of patients treated in cooperative group protocols in Australia, Canada, Europe, Japan, and the United States.Local control involves surgical resection of primary tumor site as well as radiation to primary tumor site. Primary tumors are often more amenable to surgical resection after receiving upfront induction chemotherapy. Neuroblastoma is a very radiosensitive tumor, and chemotherapy plays an important role in control of disease in the high-risk setting.
Myeloablative consolidation therapy has shown to improve EFS for patients with high-risk neuroblastoma. Current data from trials in the United States and Europe support improved outcomes for patients receiving myeloablative consolidation therapy with etoposide, carboplatin, and melphalan. Recently, a single-arm study of tandem stem cell transplantation reported an EFS of 58%. A randomized study of tandem stem cell transplant against a single transplant is currently ongoing in the Children’s Oncology Group.7 Because of significant improvements in time to recovery and a lower risk of tumor cell contamination, most centers now recommend the use of peripheral blood stem cell support over bone marrow for consolidation therapy.
Surgical resection plays an important role in the treatment of patients with neuroblastoma. For patients with localized disease, surgical resection is curative. For patients with regional or metastatic disease, surgery to establish a diagnosis and obtain adequate samples for biologic studies is essential. Typically, second-look surgery postchemotherapy is used to attempt a complete resection. The emphasis in the second-look procedure is as complete a debulking as possible without sacrificing major organ function. Patients with residual disease postchemotherapy and surgery may benefit from the use of radiotherapy.
Consultations
Neuroblastoma can be confused with other neoplastic or nonneoplastic diseases of childhood. The diagnosis can be challenging in the 10% of patients who present with normal urinary catecholamines.
Radiation oncologists may participate in the care of patients with neuroblastoma. Typically, they are consulted to evaluate patients whenever radiation therapy is a consideration. Usually, radiotherapy is localized to areas of residual microscopic disease, persistent disease, or both after chemotherapy and surgery.
In high-risk patients, the need for stem cell harvest and transplantation should be anticipated. These services should be included early in the planning phase of treatment.

Diet

Nutrition plays an important role in therapy. Children need adequate caloric intake to attain normal growth and development, and to recover from the adverse effects of therapy. Nutritionists typically help to provide adequate supportive care during therapy. Supplemental nutrition is often required during therapy. This should occur via the enteral route (nasogastric or gastric tube). The parenteral route should be used only after failure to supplement adequately using enteral feedings.

No specific restrictions are placed on activity. Patients who are thrombocytopenic should avoid strenuous activity and contact sports. Patients should avoid ill contacts, especially if neutropenic.

Medication

All chemotherapy orders are written by pediatric oncologists and countersigned, usually by another physician. With recurrent disease, various salvage protocols may be used; with refractory disease, a limited number of phase I/II studies are available through the Children's Oncology Group (COG) and New Approaches to Neuroblastoma Therapy (NANT) consortia.

Resources presented in this section should serve as a guide to indication, usual dosages, and adverse effects of specific agents. Antineoplastic drugs have a narrow therapeutic index and effective doses usually cause severe toxicities, some of which can be life threatening.

Individual chemotherapy drugs are discussed below. These agents are almost invariably given in combination. Commonly used combinations include the following: Vincristine, cyclophosphamide, and doxorubicin, Carboplatin and etoposide, Cisplatin and etoposide

Ifosfamide and etoposide, Cyclophosphamide and topotecan, Consolidation regimens used in neuroblastoma include the following: Carboplatin and etoposide with melphalan r cyclophosphamide,-Thiotepa and cyclophosphamide, Melphalan and total body irradiation In Europe, several studies have used busulfan with melphalan or cyclophosphamide. One commonly used salvage or relapse therapy regimen is the combination of topotecan and cyclophosphamide. The use or retinoids have been incorporated in maintenance regimens in the posttransplant setting. Irinotecan is also under investigation.

Antineoplastic Agents

Cancer chemotherapy is based on an understanding of tumor cell growth and how drugs affect this growth. After cells divide, they enter a period of growth (ie, phase G1), followed by DNA synthesis (ie, phase S). The next phase is a premitotic phase (ie, G2), which is followed by a mitotic cell division (ie, phase M).

Cell division rate varies for different tumors. Most common cancers increase very slowly in size compared with normal tissues, and the rate may decrease further in large tumors. This difference allows normal cells to recover more quickly from chemotherapy than malignant cells; it is the rationale behind current cyclic dosage schedules.

Antineoplastic agents interfere with cell reproduction. Some agents are cell cycle specific, whereas others (eg, alkylating agents, anthracyclines, cisplatin) are not phase specific. Cellular apoptosis (ie, programmed cell death) is also a potential mechanism of many antineoplastic agents.

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