**Subgect #4.**

**Non-specific pelvic inflammatory diseases. Urgent gynaecological diseases (pyosalpinx, tubo-ovarian, pelvic abscess).**

**INFLAMMATORY DISEASES OF FEMALE GENITALS**

The rate of inflammatory diseases is over 60% of all gynecologic diseases and about 30% patients of female hospitals have the inflammatory processes of genital organs. Especially the quantity of the inflammatory diseases has enlarged because of the increased sexual activity at the young age, permissive sexual attitude, prostitution. Those at the highest risk are young unmarried women with multiple sex partners. Primarily inflammatory diseases affect human fertility because of infections of the female upper genital tract and their sequel. Women with persistent virus infection are at particular risk for cervical dysplasia and intrauterine fetal death.

Normal flora has a significant role in defense against infection by genital pathogens. The female genital tract, especially the vaginal secretions, contain from 108 to 109 bacteria per gram of fluid examined. Lactobacilli produce lactic acid from glucose keeping the vagina at an acidic pH (3,8-4,2). Glycogen is metabolized by vaginal epithelial cells to glucose, which then serves as a substrate for Lactobacillus.

**Normal vaginal microflora contains:** Lactobacillus (70-90%), Staphylococcus epidermalis (30-60%), diphteroids (30-60%), Hemolytic Streptococci (10-20%), nonhaemolytic streptococci (5-30%), Escherichia coli (20-25%), Bacte-roides (5-15%), Peptococcus (10-60%), Peptostreptococcus (10-40%), Clostridium (5-15%).

Presence of pathogenic flora without inflammation isn't a sign of pathologic processes.

It is considered that normal vaginal flora is represented by Lactobacillus. But not only Lactobacillus acidophilus provide the self-cleaning of the vagina. The normal vaginal ecosystem of reproductive age women contains 7 kinds of Lactobacillus: L acidophilus (42,8%), L. Paracasei, L. Fermentum, L. Plantarum (10-18,6%), L.cateforme, L.corineformis, L. Brevis (2,5-5,7%), H202 producing Lactobacillus may play an important role in acting as a natural microbicide within the vaginal ecosystem.

Variation in vaginal colonization by Lactobacillus and other organisms could relate to estrogen level metabolism products of vaginal microflora, vaginal pH, and the type of Lactobacillus initially colonizing the vagina. Many endogenic and exogenic factors may change the balance of the vaginal ecosystem. Some vaginal microorganisms may cause the inflammation in certain conditions. Both vaginal and cervical epithelial cells have the capacity to convert glycogen to glucose, which is further metabolized to lactic acid. Vaginal acidity depends on adequate levels of estrogens as well as the presence of lactic acid-producing bacteria such as Lactobacilli. Concentrations of lactobacilli are probably important determinants of vaginal pH as well. The increased concentration of lactic acid producing bacteria in the vaginal fluid may result in a lower pH which determines decreased susceptibility to infection. Estrogens have a direct effect on the number of organisms and composition of the bacterial flora. The mucosal surface provides protection from invading pathogens. Mucous may act to eliminate a variety of pathogens or antigens. Mucous also serves for attachment of immunoglobulin A, lysozyme, lactoferrin and other biologically active substances. Mucous in the female genital tract is under hormonal control. Any abnormalities with low estrogen secretion and decreasing of estrogen level with age may damage defense mechanisms of the female genital tract. Using of contraceptives, shower can effect into vaginal ecosystem by changing vaginal pH, altering the vaginal fluid by direct dilution.

**Bartholinitis**

Bartholinitis is an inflammation of Bartholin's gland (large gland of vaginal vestibule). It may be caused by Staphylococcus, E.coli and N. gonorrhea. Any type of the pathogen initiates ductal inflammation and obstruction that can lead to Bartholin's abscess. There can be serous, serous-purulent, or purulent inflammation.

Obstruction of the opening of the main duct into the vestibule leads to abscess formation. Infection of Bartholin's glands can lead to secondary infections, abscess or cyst formation. When the gland becomes full and painful, incision and drainage is appropriate. Patients with abscess usually require abscess incision with insertion of the catheter in abscess cavity. Recurrent infection from vaginal flora and mucous cyst formation are common sequelae of bartholinitis. If the infection of gland is caused by N.gonorrhea specific antibacterial treatment is prescribed.

**Vulvitis**

Vulvitis is a vulvar inflammation. It may be primary and secondary. Primary vulvitis is caused by local irritants (including feminine hygiene sprays, deodorants, tight-fitting synthetic undergarments in women with obesity or diabetes mellitus. Secondary vulvitis are caused by accompanying discharge from vagina.

Reduced estrogens levels in reproductive age women, and more frequent in girls and menopause women may lead to vulvitis.

**Clinic.** Erythema, edema of vulva and skm ulcers are all indices of the infection.

Patient's complains are itching or burning Excoriation caused by the patient's scratching of the skin of vulva are often seen m vulvar irritation

To relieve inflammation and itching the mam suspected cause must be removed The therapy includes local application of boric acid solution or KMn04 solution Candidasis is treated with Gyno-pavenl 150mg in suppositories — 3 days, or Orungal lOOmg twice a day during 6-7 days orally, and then one capsule per day every first day of menstrual cycle during 3-6 cycles. Treatment with local antibiotics and steroids is successful

**Vaginitis (colpitis)**

Vaginitis (colpitis) is an inflammation of vagina It is the most frequent cause of visits to gynecologists It may be caused by Staphylococcus, Streptococcus, E coh and other.

Excessive vaginal discharge is associated with an identifiable microbiologic cause m 80% to 90%of cases. Hormonal or chemical causes account for most of the remaining cases Vaginitis may be acute, subacute and chronic There are two forms of vulvitis purulent and granulosa-diffusional.

The main symptom is the increased, gray-white or yellow discharge generally serous or purulent with rancid odour. The patients complain of dysuria, vulvar itching, burning and dyspareunia. Examination may reveal edema or erythema of vulva and vagina, petechia or patches in the upper vagina or on the cervix. In case of chronic vaginitis all these signs are not so expressed.The cultures from vagina, cervix,urethra, ductus of Bartholin's gland should be microscopically examined.

**Treatment**of nonspecific vaginitis is comlex:

* using of antiinflammatory medicines
* treatment of neuroendocrinologic and immunodificiency conditions
* treating of male sexual partner; patients should avoid sexual contacts while therapy

*Local treatment* includes using of syringing with antiseptic fluid (KMn04, furacilin, chlorhexidin) no more than 3-4 days. In case of acute or chronic vaginitis laser therapy may be used.

Metronidazol (vaginal suppositories), chlorhinaldin, terginan, betadin, gyno-paveril may be prescribed. For normalization of vaginal ecosystem solkotry-chovac, vagilak, Lactobacterin and Bifidumbacterin are used.

**Bacterial Vaginosis**

10-25% of all gynecologic patients have this disease. Among sexually transmitted diseases, bacterial vaginosis is diagnosed in 60-65%) of women. Bacterial vaginosis is a result of an overgrowth of both anaerobic bacteria and the aerobic bacteria Gardnerella vaginalis. Anaerobes and G. vaginalis are normal inhabitants of vagina, but these bacteria overgrowth dominant of the normal Lactobacillus flora results in the appearance of a thin, fishy odor, gray vaginal discharge that adheres to the vaginal walls.

A small amount of vaginal discharge may be normal (2ml) particularly at the midcycle. Bacterial vaginosis causes an increased vaginal discharge (15-20ml), vulvar irritation, pruritus, dysuria and foul odour.

**The diagnosis** of bacterial vaginosis is based on the presence of the following characteristics of the discharge:

* pH is higher than 4,5
* a homogeneous thin appearance
* a fishy amine odour produced by anaerobes when 10% KOH is added
* presence of clue cells (vaginal epithelial cells to which organisms are attached)

Cultures aren't helpful because anaerobes and Gardnerella vaginalis can be recovered from normal flora of healthy women, but the concentration of both bacteria is higher in patients with bacterial vaginosis. Factors that lead to overgrowth of G.vaginalis and anaerobes have not been identified.

Treatment includes elimination of anaerobic agent of microflora, inducement of local and general immunity and then the normal microflora should be renewed.

Oral using of metronidazol (Flagyl) 500mg twice a day for 7 days or by intravaginal Metrogel 0,75% cream twice a day for 5 days, 2% Clindamycin cream (Cleocin) once daily for 7 days.

For normalization of vaginal microflora the local bifidumbacterin insertion or 2-3% solution of Lactic acid is used. The treatment of the male parthner with Metronidazol can be advocated only when bacterial vaginosis recurs, but effectiveness is not proven.

**Endocervicitis**

Endocervicitis is the inflammation of mucosa layer of the endocervix. Bacteria cause infection of the columnar epithelium. Chlamidia trachomatis, Mycoplasma, Trichomonada vaginalis, N. Gonorrhoeae, viruses, Candida, E.coli, Staphylococci cause endocervicitis.

Cervix is constantly exposed to trauma during childbirth, abortion.The abundant mucus secretion of the endocervical glands both with the bacterial ascend from the vagina creates a situation that is advantaging to infection.

The inflammatory process is chiefly confined to the endocervical glands. The squamous epithelium of the exocervix may be involved into the process called acute exocervicitis. The extent of endocervical involvement as compared with exocervical one appears to have some relation to the infecting agent.

Chronic cervicitis manifestation is cervical erosion. Erosion indicates the presence around the cervical os a zone of infected tissue that has a granular appearance. It implies the loss of superficial layers of the stratified squamous epithelium of the cervix and overgrowth of infected endocervical tissues.

The inflammatory process stimulates a reparative attempt in the form of anupward growth of squamous epithelium, causing some of the ducts of the endocervical glands to be obstructed. Retention of mucus and other fluid within these glands results in the formation of Nabothian cycts. These cysts are endocervical glands filled with infected secretion. Their ducts become secondarily included into the inflammation and reparative processes.

The most important in the diagnosis of chronic cervitis is the exclusion of the malignant process. Before the begining of treatment, examination with colposcope should be carried out. The cervicitis may appear as a reddish granulation raised above the surrounding surface, giving the impression of being papillary.

A Papanicolaou smear should be obtained and suspicious areas should undergo biopsy.

**Treatment.**Acute cervicitis is treated with appropriate antibiotics (it depends on bacterial agent). Local treatment of acute phase is a real danger of dissemination of infection. Laser therapy is used in treatment of acute and chronic cervicitis.

Electocautherization is the traditional treatment of chronic cervicitis, especially with erosion, cervical ulcers or ectropion. Nowadays cryosurgery or laser surgery has replaced electrocautherization.

**Acute endometritis**

Acute endometritis is an inflammation of endometrium (mucus layer of uterine). It may occur in such cases as: endometritis after uterine curettage or suction and puerperal endometritis. Endometritis is caused by bacterias, viruses, mycoplasmas. The most frequent the associations of 3-4 anaerobic bacteria and 1-2 aerobic are the main reason of endometritis.

Anaerobic bacteria compose a part of the normal cervicogenital flora. There are two known mechanisms which cause anaerobic infection: antibiotic selection that preferentially inhibits aerobic bacteria and tissual trauma that occurs after surgery which reduces the redox potencial. Anaerobes produce odorous metabolic products.

Uterus has endometrium factors of local immunity. There are T-lymphocytes and other factors of cellular imunity in endometrial stroma. Lymphocytes and neutrophiels normally appear in the endometrium in the second half of menstrual cycle; their presence does not necessarily constitute endometritis. The appearing of plasma cells represents immune response, usually to foreign bacterial antigen. The organism should be cultured before applying of antimicrobal therapy. As anaerobes compose a part of normal flora, deep tissual cultures not contaminated by surface bacteria are required. Forty eight or more hours are required for anaerobe recovery, and treatment usually is based on clinical signs. There are nonspecific and specific endometritis. Specific endometritis is caused by M. Tuberculosis, N. Gonorrhea, Chlamidia trachomatis, Actinomyces.

**Clinic.** Fever is the characteristic feature in the diagnosis of endometritis, and it may be accompanied by uterine tenderness. If the infection has spread to the parametrium and adnexa, tenderness may be present there as well. Temperature elevation is probably proportionate to the extention of the infection and when confined to the decidua, the cases are mild and there is minimal fever. Chills may accompany fever. Women usually complain of abdominal pain. There is tenderness on one or both sides of the abdomen and parametrial tenderness is elicited upon bimanual examination. The uterus is lightly enlarged.

A leukocytosis and increased erythrocyte's sedimentation rate is revealed in patient' blood test. In some cases acute endometritis may become a chronic one.

**Treatment.** Various choices of initial antibiotic therapy are used. Most of them are successful. Single-agent therapy has the benefit of easy administration; Cephalosporins such as Cefotetan and Cefoxitin are commonly used. A combination of AmpiciUin and Aminoglycoside is also popular. The combination of Clindamycin with Gentamicin or Metronidasol with Unasyn (AmpiciUin with Sulbuctam) and Gentamicin is applied. It is desirable to provide additional antibiotic coverage if there has been no responce within 48 to 72 hours. Intravenous antibiotic therapy is continued until the patient is asymptomatic and afebrile period lasts for at least 24 hours.

Local uterine douching with antiseptic solution of chlorhexidin or furacilin has a good effect. In some cases uterine curettage is performed after temperature normalization.

**Chronic endometritis**

Chronic endometritis is a sequale of untreated acute endometritis or nona-dequate treatment of postabortion or purperal endometritis. The chronic endometritis sometimes is associated with the use of intrauterine device (IUD). In some cases it may occur without acute stage.

**Clinic.** The chronic endometritis results from organisms that are normally in lower genital tract (Protei, E. Coli, Staphylococcus, Mycoplasma). Bacteria that can be recovered are usually of low pathogenicity, but more virulent intrauterine bacteria occasionally cause the serous purulent' discharge, abnormal uterine bleeding and moderate uterine tenderness. Diagnosis is based on anamnesis and clinical manifestation. It could not be diagnosed unless plasma cells are found in the endometrium. Ultrasonography can identify gas vesicules in uterine cavity, hyperechogenic places (local fibrosis, sclerosis) in basal layer of endometrium.

**Treatment.** A complex treatment is used. It includes a medicines for curing of accompaning deseases, desensibilisative medicines and additional general health measures, vitamines.

Physiotherapy has an important role. It improves pelvic hemodynamics. Diathermy on lower abdomen, electrophoresis with copper, zinc, ultrasound, inductothermy, laser radiation are used. If during physiotherapy the process becomes strained antibiotic therapy is recomended. While remission antibiotic using is not proved.

Physiotherapy promotes to activation of hormonal ovarian function. If effect is not enouph than a hormonal therapy'is used (taking into account the patient's age, term of deseases, degree of ovarian hypofunction). Health resort treatment is effective (balneologic therapy, mudcure resort).

**Salpingoophoritis**

Salpingoophoritis is the inflammation of the uterine tubes and the ovaries. Salpingoophoritis is the most frequent among all pelvic inflammatory deseases. Most cases of oophoritis are secondary to salpingitis. The ovaries become infected by the purulent material that escapes from fallopian tube. If the tubal fimbriae are adherent to the ovary, the tube and ovary together may form a large retort-shaped tubo-ovarian abscess.

Most patients with salpingoophoritis have lower abdominal, adnexal tenderness (unilateral or bilateral) purulent cervical exudate or purulent vaginal discharge (fig. 88).

**Clinic.** There are four stages of salpingoophoritis. The first— salpingitis without irritation (inflammation), of the peritoneum, the second— with signs of peritonitis, the third with occlusion of uterine tubes and tuboovarian abscess and the fourth is the rupture of tuboovarian abscess. During bimanual examination adnexal inflammatory mass is revealed.

**The diagnosis** of salpingoophoritis is based on the history, physical examination and laboratory tests. Besides that additional ultrasonography and laparoscopy can be used.

Laparoscopy provides the most accurate way to diagnose the inflammatory process and its stage. It should be used in cases when the diagnosis is unclear, especially in patients with severe peritonitis, to exclude a ruptured abscess and appendicitis. Besides diagnostic laparoscopy is used to provide treatment procedures.

Ultrasound can be used to distinguish the presence of an abscess from an inflammatory mass within the adnexal mass. It may also be helpful in defining mass in the obuse patient or if the bimanual examination is unsatisfactory because of the excessive tenderness.

**Treatment.**All patients with acute salpingoophoritis should be hospitalized. Adequate therapy of salpingitis includes the assessment of severity, antibiotic treatment, additional general health measures.

Before the culture test performing the antibiotic therapy is provide with broad spectrum antibiotics. The most effective is the combination of Clindamicin with Chloramphenicol, Gentamicin and Lincomicin, Doxycyclin, Clacid, Cefobid, Cyfran, Claforan, Dalacin C and Unasyn.

When anaerobic agents are suspected metronidazol should be used, in severecases intravenously. After temperature normalization and cessation of peritonitissigns antibacterial therapy is continued for 5 days. Detoxycation is indicated andis provided by using of 5% glucose solution, polyglucin, reopolyglucin, solutionsof proteins, correction of pH balance by using of 4% solution of Sodium bicarbo-nates. Among physical methods of treatment cold on the lower part of the abdomenis used. Appropriate antibacterial treatment is combined with laparoscopy activedrainage. . The tuboovarial abscess is drained of pus by puncture and rinsed with bacteriostatic solution and local application of antibiotics. In subacute stage aloe, ultraviolet radiation, authohaemotherapy is used. They prevent the chronic processes.

**Chronic salpingoophoritis**. In most cases chronic salpingoophoritis is the sequale of non treated acute process. Chronic stage of the process is characterized by tubal occlusion with periovarial adhesions, tubal dysfunction.

**Clinic**. The main complains of the patient are: mild tenderness in lower part of abdomen that becomes severe during menstruation. Pelvic nerves have more painful sensitivity (pelvic plexitis, ganglionevritis due to chronic inflammation). In some cases menstrual dysfunctions such as oligomenorrhea, polymenorrhagia, algodismenorrhagia occur. Changing in uterine tubes and hypofunction of ovaries lead to infertility or miscarriage. Secretory dysfunction like vaginal discharge or cervical exudate may be observed as a clinical finding of colpitis or endocervitis. Some patients complain of low libido, painful coitus, dysfunction of urinary bladder, liver tenderness.

Menstrual dysfunction (menorrhagia or metrorrhagia) is the most frequent symptom of chronic salpingoophoritis as a sequel of disorders of neurohomoral regulation of menstrual function. Metrorrhagia often occurs after cessation of menstruation and then the differential diagnosis should be made in case of ectopic pregnancy.

**Diagnosis.** Correct history taking (reveal of inflammation after abortion, delivery or dilatation and curettage) makes it possible to suspect the chronic inflammatory process. Primary chronic salpingoophoritis is found in more than 60% of cases. Some information gives physical examination and laboratory tests. Bimanual examination gives nonspecific information. Enlargement, consistency and degree of adnexa mobility should be examined. Sometimes because of peritubal and periovarian adhesions the sizes and mobility of adnexa are changed.

Additionally, ultrasound and laparoscopy, hysteroscopy should be held. Tomography or endoscopy may be used. Laparoscopy is the most informative diagnostic method to differentiate salpingoophoritis, external endometriosis, uterine myoma with inflammatory changes, cysts. Disorders of adjacent organs (bladder, intestine) while serous inflammation is present without structural changes. But women with disorders of urinary tract, gastro-intestinal tract must be additionally examined (urography, irrigoscopy).

**Treatment** of chronic salpingoophoritis is provided with minding of pathogenesis and clinic. Antibiotics are indicated in acute period, when there are signs of inflammation.

Nonsteroidal antiinflammatory drugs (Voltaren, Butadion) are prescribed. To stimulate immune system immunomodulators are used: (Decaris, T-activin). FIBS, aloe, autohaemotherapy are also used. Analgesia both by medicines and by reflextherapy is of great importance. Physiotherapy is conducted in hospital while in case of acute process and remission it can be used in ambulatory conditions. Ultrasound has analgetic and fibrinolityc influence and is prescribed in sinusoid and modulate of high frequency. Laserotherapy is also used. To escape chronic salpingoophoritis the acute salpingoophoritis must be treated in proper way and the quantity of abortion should be reduced.

**Parametritis**

Parametritis is an inflammation of parametrium. Inflammation of the whole pelvic cellular is called pelviocellulitis. According to international statistics these diseases are classified as acute parametritis or pelvic phlegmona.

Infection agents may be staphyloccocus, streptoccocus, E.coli, etc. It can be caused by one microbic agent or microbe association. It occurs after pathologic delivery, abortion, operation on genitals. The main way of infection spreading is lymphogenic. Morphologically parametritis is characterized by all signs of inflammation: dilation of blood and lymphatic vessels, peripheral edema, exudation. There are 3 stages in course of parametritis (infiltration, exudation, firming). Exudation may be serous, and very rarely it is purulent. Sometimes it undergo resorbtion and dissolves, sometimes a fibrose connective tissue grows and leads to uterine dislocation to the side of previous inflammatory process.

**Clinic.** Moderate tenderness in lower parts of abdomen, in back, high body temperature (38-39°C), tachicardia are found. Signs of peritoneal irritation and diminished or absent bowel sounds, especially associated with ileus, indicate more serious infection, including the possibility of abscess formation. Fever is a characteristic feature in the diagnosis of metritis and it is accompanied by uterine tenderness. Bimanually before or behind on left or right side of the uterus infiltration may be palpated. It is firm and immovable. Infiltration is classified into anterior, posterior and lateral.

**Treatment** begins from using antibiotic of broad coverage against a variety of common microorganisms and is usually prescribed without cultures.

Various choices of initial antibiotic therapy are used. Most of them are successful. Cephalosporins such as Cefotetan and Cefoxitin are commonly used. A combination of Ampicillin and Aminoglucoside and also the combination of Clindamycin with Gentamicin are used.

A bottle with ice on the lower part of abdomen is used in case of infiltrative stage of disease. Biostimulators should be prescribed. Management of a persistent pelvic abscess includes drainage by colpotomy, or laparotomy. Intraabdominal rupture of pelvic abscess is a surgical emergency. Sepsis may occur in association with pelvic infection, with or without frank abscess formation. Phisiotheraputic precedures are used for rehabilitation.

**Tuboovarian abscess**

Tuboovarian abscess (TOA) may occur as a complication of salpingoophoritis. It begins from acute purulent salpingitis when all layers of uterine tubes arc involved into the process. The tubes characteristically become swollen and redde i as the muscularis and serosa are inflamed. If exudate drips from the fimbriated ends of the tubes a pelvic peritonitis is produced then it can give rise to peritoneal adhesions. The swollen and congested fimbriaes may adhere to one another and produce tubal occlusion. The fimbriae may occlude tubes producing permanent tubal infertility. The swollen and congested fimbriae may adhere to ovary, trapping the exudate in the tube and giving rise to pyosalpinx or if the ovary becomes infected, a tuboovarian abscess. The mucosal folds may adhere to one another forming gland-like spaces that are filled with exudate. If the infection subsides after agglutination of the fimbria and closure of the peripheral end of the tube, secretion accumulates and distends the tube, forming pyosalpinx. Each recidive of chronic salpingoophoritis has more clinical manifestation and is treated with difficulty. TOA is associated with IUD, microbe association, chronic salpingoophoritis.

Intoxication in case of TOA leads to liver disorders. Decreasing of albumin-globulin index is observed while the level of general proteins is normal for a long time. The degree of these disorders depends on the time of duration of the process.

**Clinic.** Clinic of TOA depends on the volume of purulent damage of adnexa, duration of the process, disorders of adjacent organs. There are some syndromes which are divided into local syndrome (pain, purulent discharge, peritoneal symptoms and palpation of tuboovarian mass).

Inflammatory-intoxicative syndrome includes fever, tachycardia, nausea, vomiting. Luecocytosis, decreasing of albumin-globulin index, C-reactive protein are observed in blood. Immune syndrome (decreasing of lymphocytes and mono-cytis in blood) is found.

Syndrome of adjacent organs disorders (dysuria, urinary frequency, menstrual disorders) is also possible.

Severe lower abdominal pain occurs, pelvic peritonitis may be present. Pain can irradiate to back, pelvic bottom, in the chest. In such cases the examinations should be performed to exclude pneumonia, pancreatitis, cholecystitis. Musclar defance which prevents abdominal palpation in the lower quadrants, adnexa are tender to various degrees and cervix movement may cause pain in case of bimanual examination. The adnexa often are either adherent to the posterior aspect of the uterine or prolapsed in cul-de-sac, which may pull the uterine into a retroverted position. TOA is characterized by pain and tenderness, fever or chills, temperature rises up 39°C, blood pressure decreases. Abdomen takes part in breathing, and it is painful in lower parts. In blood analysis elevated white blood count (9-10x107 1) erythrocytes' sedimentation rate more than 30mm/hour, positive C-reactive protein, decreasing of albumin-globulin index till 0,8 are observed.

Sometimes there can be urinary syndrome with proteinuria, leucocyturia. There may be disorders of filtrative kidney' function, even unuria. Changing of albumin-globulin index and hypofybrinogenemia characterizes the liver dysfunction.

**Diagnosis** is based on clinic, bimanual examination, laboratory analyses and additional methods of investigation (ultrasound, laparoscopy).

**Treatment.** Tuboovarian abscess is treated by antibiotics, desensibilisative and nonsteroidal antiinflammatory medicines, detoxication and immunostim-mulation. Best of all one should combine taking of penicillin with tetracyclins. When anaerobic infection is suspected metronidazole is used. Daily punctions of tuboovarian abscesses are indicated to remove purulent containts.

***Indications to surgical removal of tuboovarian abscess are:***

* abscence of efficiency of complex treatment with usage of punctions during 2-3 days
* suspicion on tuboovarian abscess perforation; volume of surgical intervention depends on process' spreading, woman's age and extragenital pathology

**Peritonitis**

Pelvioperitonitis is an inflammation"of pelvic peritoneum.The polymicrobial infection such as Escherichia coli and other aerobic, enteric, gramnegative rods, group of ?-hemolytic staphylococci, anaerobic, streptococci, Bacteroides species, staphylococci, mycoplasms cause the process. Pelvioperitonitis occurs secondary. Primary process is in uterine tubes, ovaries, uterus and parametrium. In most cases purulent damage of uterine adnexa lasts with pelvioperitonitis. Infection can be spread by limphogenic or blood vessels, and from uterine tubes in case of salpingitis, especially gonococcial infection.

**Clinic**characterizes the acute inflammation. High temperature, severe lower abdominal pain, fever or chills, tachycardia are common. There can be nausea and sometimes vomiting. Muscular defence and rebound tenderness are the symptoms of peritoneal irritation. Anterior abdomen wall takes part in breathing act.Tender adnexa are present at bimanual examination. Cervical motion causes pain. Posterior fornix is painfull.

Laboratory tests reveal increasing of white blood cell count and erythrocyte sedimentation rate. C-reactive protein levels may appear. Generall blood test should be done 4-5 times per day to diagnose transformation of pelvioperitonitis to peritonitis.

**Treatment.** All the patients should be hospitalized. Ideally, the antibiotic should be selected according to the organism present in the fallopian tube or uterus, but in most cases empiric therapy must be used. Treatment includes intravenous doxycycline and either cefoxitin or cefotetan or intravenous clindamycin and gentamicin for at least 4 days followed by oral clindamicin or tetracyclin for 10-14 days. Hospitalized patients who have peritonitis but do not have adnexal abscess usually respond rapidly to the regimens. In the presence of an adnexal abscess, even if the systemic manifestations are mild, antibiotics which eliminate B.fragilis should be selected because most pelvic abscesses contain this organism. Clindamycin, Metronidazol, Cefoxitin, or Impinem should be used to treat pelvic abscess. If there is an intrauterine device it should be removed as soon as therapy is started. Surgery is indicated in the case of ruptured pyosalpinx or ovarian abscess. Colpotomy drainage usually is preferable when unruptured midline cul-de-sac abscess is present. Laparotomy is required for such problems as unresolved abscess or adnexal mass that does not subside, surgery should be limited to the most conservative procedures that will be effective. Unilateral abscess respond to unilateral salpingoophorectomy.

**Septic shock**

Septic shock is associated with infection caused gram-negative aerobic coliform organisms those are producing endotoxins. In gynecological practice it may occur in case of septic abortion, localized or spreading peritonitis, thrombophlebitis. Septic shock is a special organism reaction that is expressed in development of severe systemic disorders. It may be caused by using of broad spectrum antibiotic in high doses, that results in releasing of great amount of endotoxin.

Endotoxin, a complex cell wall-associated lipopolysaccharide, is released into the circulation at the time of bacterial death, resulting in multiple hemodynamic effects. The subsequent activation of lymphocytic T-cells and mass cells results in histamine and kinin activation as well as the activation of kallikrenin and decrease in kallikreinogen and kallikrein inhibitor. These changes result in the release of bradykinin, a potent arterial dilator. Early septic shock is a classic example of distributive shock, related to a systemic maldistribution of relatively normal or even increased cardiac output. Clinical findings include hypotension, fever and chills. Initial hemodynamic findings include decreased systemic vascular resistance and high normal or elevated cardiac output. The continued maldistribution of cardiac output leads to local tissue hypoxia and to the development of lactic acidosis and organ dysfunction. This decrease in systemic vascular resistance is caused by the release of vasoactive substances, as well as by vascular endothelial cell injury, which promotes capillary plugging secondary to complement induced leukocyte aggregation. These factors lead to increased arteriovenous shunting.

If the process continues a second hemodynamic phase of septic shock is developed. The primary importance in this late phase is the development and progression of myocardial dysfunction leading to ventricular failure. Studies assessing stroke work index and ventricular ejection fraction have demonstrated depressed intrinsic ventricular function even in the early stage of septic shock. Pulmonary hypertension, another important hemodynamic alteration is often associated with septic shock, may have additional profound hemodynamic consequences. As the sequalae of renal kidneys filtration disorders — the shock kidney is formed and acute renal insufficiency is developed. Signs of liver disorders are hyperbilirubinemia, lipid metabolism abnormalities.

Patients who recover from the initial hemodynamic instability of septic shock may suffer prolonged morbidity secondary to endotoxin-mediated pulmonary capillary injury and noncardiogenic pulmonary edema. Such lung failure is a major cause of death in patients whose hypotension was prolonged and may experience acute tubular necrosis. Endotoxin mediated endothelial cell injury and associated thromboplastine-like activity as well as prolonged shock from any other cause may also lead to activation of the coagulation cascade and a clinical picture of disseminated intravascular clotting syndrome (DIC).

**Clinic and diagnosis.** The clinical manifestation develops just after surgical operation on infected organs. The body temperature rises till 39-40°C and is high during 1-3 days. Then the temperature decreases, chills is a characteristic feature of the septic shock.

Among clinical findings there are hypotension without bleeding or nonadequete to it, tachycardia, 120-140 per minute. Decreasing of blood circulative volume leads to rising of shock index till 1,5 (normally 0,5). Skin is pale and wet because of perspiration, later akrocyanosis can appear. Breath disorders, like tachypnoe till 30-60 per minute, is the sign of shock lungs. Skin may be colored in yellow, there may be blood vomiting.

The most dangerous complication of septic shock is kidney insufficiency. Clinical manifestation at the beginning is oligouria — less than 30 ml per hour. Later anuria is developed. All these changes in organism appear in very short time in 6-8 or sometimes 10-12 hours.

Diagnosis is based on the following signs:

* septic organ
* low blood pressure, nonadequate to blood loss
* nervoues system disorders
* pain of different parts of body
* decreasing of diuresis
* rash on the skin

The blood temperature should be taken every 3 hours, blood pressure is measured every 30 minutes, urine quantity must be measured. Bacterial culture from infected organ, blood analysis, coagulogram and biochemical tests are performed.

**Treatment of septic shock.** The treatment of septic shock involves optimising preload relative intravascular volume with crystalloid infusion as well as treating of the underlying infection. Although some authorities advocate the use of colloid solutions for volume replacement, there is noconvincing evidence that using of such solutions decreases the incidence of pulmonary edema or adult respiratory distress syndrome. In most cases the infection is polymicrobial and broad spectrum coverage for gram-negative and gram-positive aerobic and anaerobic organisms is most appropriate. If an abscess is involved, promt surgical drainage after initial resuscitation is mandatory. Patients in septic shock should be treated with dopamine hydrochloride. This agent in doses of less than 5mg/kg/minute improves renal blood flow by means of dopaminergic mesenteric vasodilatation; in doses of 5 to 30 mg/kg/minute, a positive inotropic effect is also seen. The hemodynamic manipulation of patients whose hypotension fails to respond rapidly to volume infusion may be assisted by pulmonary artery catheterization, allowing the clinician to achieve optimal preload before the institution of inotropic or vasoconstrictive therapy. High-dose corticosteroids are advocated (60-120 mg of prednizolone or 8-16 mg of dexamethazone). To renew Ph balance lactosol or bicarbonate natrii are indicated.