SEXUALLY TRANSMITTED DISEASES
<table>
<thead>
<tr>
<th>Agent</th>
<th>Relevant human disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Viruses</strong>&lt;br&gt;Human immune deficiency virus (HIV 1 and 2)&lt;br&gt;Papillomavirus&lt;br&gt;Hepatitis B virus&lt;br&gt;Hepatitis C virus&lt;br&gt;Herpes simplex virus&lt;br&gt;Cytomegalovirus</td>
<td>Acquired immune deficiency syndrome (AIDS)&lt;br&gt;Ano-genital warts&lt;br&gt;Liver disease&lt;br&gt;Cold sore, genital infection, cervical dysplasia, carcinoma of the cervix&lt;br&gt;Generalized infection</td>
</tr>
<tr>
<td><strong>Bacteria</strong>&lt;br&gt;Neisseria gonorrhoeae&lt;br&gt;Haemophilus ducreyi&lt;br&gt;Calymmatobacterium granulomatis&lt;br&gt;Treponema pallidum&lt;br&gt;Chlamydia trachomatis&lt;br&gt;Mycoplasma hominis&lt;br&gt;Ureaplasma urealyticum</td>
<td>Urethritis, vaginitis, cervicitis, proctitis, etc.&lt;br&gt;Chancroid&lt;br&gt;Granuloma inguinale&lt;br&gt;Syphilis&lt;br&gt;Lymphogranuloma venereum&lt;br&gt;Non-specific urethritis&lt;br&gt;Reiter’s syndrome&lt;br&gt;Non-specific urethritis</td>
</tr>
<tr>
<td><strong>Fungi</strong>&lt;br&gt;Candida albicans</td>
<td>Vaginal discharge, balanitis</td>
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<tr>
<td><strong>Protozoa</strong>&lt;br&gt;Entamoeba histolytica&lt;br&gt;Trichomonas vaginalis</td>
<td>Balanitis&lt;br&gt;Vaginal discharge, balanitis</td>
</tr>
<tr>
<td><strong>Ectoparasites</strong>&lt;br&gt;Crab lice/pubic lice</td>
<td>Infestation</td>
</tr>
</tbody>
</table>
1.252 Trichomoniasis
Typical vulvovaginitis, with profuse, foul-smell purulent frothy discharge.

1.177 Vaginal thrush is a common problem in adult women during or after antibiotic treatment, in pregnancy and during oral contraceptive use. The curdy white discharge is characteristic.

1.178 Vulvovaginitis in a baby, caused by Candida albicans. Infection at this age is not unusual, but in older children and adults it may suggest an underlying disorder such as diabetes.
1.231 Florid labial, perineal and perianal warts. Associated cervical lesions may put this patient at increased risk of carcinoma of the cervix.

1.230 Plane warts of the prepuce and glans penis. Genital warts vary greatly in appearance. They may be sessile, filiform or hyperplastic.
1.232 Gonorrhoea. The typical purulent urethral discharge can often be demonstrated during examination by ‘milking’ the urethra. The patient also has associated meatitis.

1.233 Gonococcal proctitis as seen through a proctoscope. Note the erythematous mucosa and the profuse purulent exudate.

1.234 Gonorrhoea in a symptomatic woman. Note the purulent discharge, which usually indicates the presence of cervicitis.

1.235 Ophthalmia neonatorum. Purulent conjunctivitis follows 2–5 days after birth in the infected infant, and may be associated with septicaemia.
1.251 Typical non-gonococcal urethritis, with mucopurulent discharge. Although the discharge is often more watery than that in gonorrhoea (1.232), gonorrhoea must always be excluded by Gram stain and culture.
1.239 Syphilis — typical primary chancre in the coronal sulcus. A small red macule enlarges and develops through a papular stage, becoming eroded to form a typical round, painless ulcer. If untreated, the ulcer usually heals after 4-8 weeks.

1.240 Syphilis — typical chancre of the labium majus.

1.236 Chancroid of the prepuce, showing typical multiple ulcerating lesions.
1.78 Primary genital herpes. Note the numerous lesions on the penis and the associated tissue reaction.

1.79 A primary herpetic dendritic ulcer, stained with fluorescein. Herpes simplex virus proliferates in the epithelial layer of the cornea. Urgent treatment with antiviral drops or ointment is indicated.

1.80 Recurrent herpes simplex on the cervix. The ulcers have recurred in a common primary site for genital infection. Other common sites include the external genitalia and the lips.
1.241 **Secondary syphilis.** This patient has a very typical papulosquamous rash (syphilide). Note the facial lesions, the colour and the symmetrical distribution of the rash.

1.242 **Secondary syphilis.** Gross condylomata lata of the vulva and anus. Note the resemblance to warts (condylomata acuminata).

1.243 **Secondary syphilis** – classic 'snail-track' ulcer of the buccal mucosa. Other mucosal lesions at this stage may be round or oval in shape.
1.244 Tertiary syphilis – gummata of the skin. The lesions start as subcutaneous masses, which increase in size before breaking down to form typical gummata ulcers. The ulcers are painless, and have sharply defined ‘punched out’ edges and an indurated base that is occupied at this stage by a slough of necrotic tissue. In contrast to the ulcerating lesions in primary and secondary syphilis, Treponema pallidum organisms cannot be found.

1.245 Tertiary syphilis – a large aortic aneurysm on chest X-ray. The aneurysm results from vasculitis affecting the vasa vasorum of the aorta.

1.246, 1.247 Tertiary syphilis – Charcot joints. In tabes dorsalis, impaired pain and position sensation, combined with muscular hypotonia, often lead to the destruction of joints and inappropriate new bone formation, as seen in these clinical and radiological examples. Charcot joints may also occur in patients with diabetes, leprosy and syringomyelia.
1.249 Congenital syphilis.
Treponemal infection of bone leads to epiphysitis, retardation of bone formation and separation of the epiphyses, with resulting interference with growth. In the nasal bones, the infection results in destruction of the nasal septum and the classic 'saddle nose', giving the characteristic facies of congenital syphilis.
1.237 Granuloma inguinale, showing a typical ulcer on the inner thigh. The ulcerated lesion is deep and its floor is covered by a thick, offensive, purulent exudate. Bilateral inguinal lymphadenopathy is present.

1.238 Granuloma inguinale of the vulva. The disease often runs a chronic course and is associated with mutilating ulceration, chronic swelling and ultimately extensive scarring.

1.250 Lymphogranuloma venereum showing superficial ulceration and enlarged lymph nodes (buboes) in the left inguinal region.
1. Causes:
   a) *Chlamydia trachomatis* and *Neisseria gonorrhoeae* are associated with a purulent discharge.
   b) *Ureaplasma urealyticum* and noninfectious causes are non-purulent.

2. Symptoms and signs:
   a) Burning on urination, worse with concentrated urine after alcohol consumption
   b) Staining of underwear, mucous in the urine.
   c) Meatus erythematous, milky discharge from penis

3. Diagnosis:
   a) Primarily by DNA probes
   b) Gram stain—In gonorrhea, intracellular gram-negative diplococci almost always found; negative Gram stain indicates non-gonococcal disease (NGU)
   c) Culture of *N. gonorrhoeae* using 5% CO₂ has to be planted immediately

4. Treatment:
   a) Third-generation cephalosporin or a fluoroquinolone for gonorrhea
   b) Macrolide, tetracycline, fluoroquinolone, or sparfloxacin for NGU
### About the Causes and Pathogenesis of Pelvic Inflammatory Disease

1. Primarily caused by *Neisseria gonorrhoeae* and *Chlamydia trachomatis*. Other, less common, pathogens include
   a) *Streptococcus pyogenes* and *Haemophilus influenzae* (most frequently accompany gonorrhea and chlamydia).

2. Cervical canal usually prevents vaginal flora from invading the endometrium.
   a) Menstruation allows bacteria to bypass the cervix, with pelvic inflammatory disease (PID) usually beginning 7 days after menstruation.
   b) Delayed treatment of urethritis leads to PID (15% of cases progress to PID).

3. Risk factors for PID:
   a) Young age (sexually active teenagers at highest risk)
   b) Multiple sexual partners
   c) Past history of PID

### About the Clinical Manifestations of Pelvic Inflammatory Disease

1. Lower abdominal pain during or immediately following menses,
   a) made worse by jarring motions,
   b) accompanied by vaginal bleeding (one third of cases), and
   c) commonly presenting with vaginal discharge.

2. On physical exam,
   a) only half of patients have fever.
   b) bilateral lower quadrant tenderness and cervical, uterine, and bilateral adnexal tenderness are present.
   c) right upper quadrant tenderness indicates Fitz–Hugh–Curtis syndrome.
   d) localized tenderness to one adnexa suggests tubo-ovarian abscess.
About the Diagnosis and Treatment of Pelvic Inflammatory Disease

1. No specific test is available for pelvic inflammatory disease (PID); diagnosis is usually clinical:
   a) Erythrocyte sedimentation rate and C-reactive protein are elevated. Normal values make the diagnosis unlikely.
   b) On examination of vaginal exudate, more than 3 white blood cells per high-power field is 80% sensitive and 40% specific.

2. Definitive diagnosis can be made by
   a) Laparoscopy (low sensitivity; should be reserved for the seriously ill patient).
   b) Histologic evidence of endometritis on biopsy.
   c) Imaging revealing thickened, fluid-filled oviducts with or without free pelvic fluid or tubo-ovarian swelling.

3. To prevent infertility and chronic pain, the threshold for treatment should be low:
   a) Outpatient treatment—ofloxacin or levofloxacin, plus metronidazole for 14 days or 1 dose of ceftriaxone, plus doxycycline with or without metronidazole for 14 days.
   b) Inpatient treatment—cefoxitin or cefotetan, plus doxycycline, or clindamycin plus gentamycin.
   c) Laparoscopy to rule out tubo-ovarian abscess; laparotomy to rule out ruptured abscess.
<table>
<thead>
<tr>
<th>Disease</th>
<th>Number</th>
<th>Location</th>
<th>Tenderness</th>
<th>Ulcer characteristics</th>
<th>Appearance</th>
<th>Adenopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Herpes simplex virus</td>
<td>Clusters</td>
<td>Labia, penis</td>
<td>Tender</td>
<td>Uniform size, clean base,</td>
<td>Clean base, indurated border</td>
<td>Very tender inguinal nodes</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>erythematous border</td>
<td></td>
<td></td>
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<tr>
<td>Syphilis</td>
<td>1–2</td>
<td>Vagina, penis</td>
<td>One third</td>
<td>Clean base, indurated border</td>
<td>Rubbery, mildly tender</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>tender</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Chancroid</td>
<td>—</td>
<td>Labia, penis</td>
<td>Tender</td>
<td>Can be large; ragged and necrotic base,</td>
<td>Very tender fluctuant inguinal nodes</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>undermined edge</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lymphogranuloma venerium</td>
<td>—</td>
<td>Labia, penis</td>
<td>Painless</td>
<td>Ulcer lasts 2–3 weeks, spontaneously</td>
<td>Fluctuant inguinal nodes, “groove sign”</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>heals at time of fluctuant adenopathy</td>
<td></td>
<td></td>
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<tr>
<td>Donovanosis</td>
<td>Kissing lesions</td>
<td>Labia, penis</td>
<td>Painless</td>
<td>Clean, beefy red base; stark white</td>
<td>Nodes usually firm, can mimic lymphogranuloma venerium</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>heaped-up edges</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Behcet’s syndrome</td>
<td>—</td>
<td>Mouth, scrotum or vulva</td>
<td>Painful</td>
<td>Yellow necrotic base</td>
<td>Adenopathy minimal</td>
<td></td>
</tr>
</tbody>
</table>
About Genital Ulcers

1. Genital ulcers have five major causes: herpes simplex virus (HSV), syphilis, chancroid, lymphogranuloma venerium (LGV), donovanosis, and Behcet's syndrome.

2. Diagnosis is usually made by the clinical characteristics of the ulcer (not always reliable):
   a) Size and location
   b) Pain and tenderness
   c) Appearance of base and edges
   d) Lymphadenopathy

3. Laboratory studies include VDRL (Venereal Disease Research Laboratory), HIV antibody, Gram stain (for suspected chancroid), viral culture for HSV, LGV serum titers, dark-field exam for syphilis.

4. Treatment:
   a) HSV—acyclovir, valacyclovir, or famciclovir
   b) Syphilis—penicillin
   c) Chancroid—azithromycin or ceftriaxone
   d) Donovanosis—trimethoprim-sulfamethoxazole or doxycycline
   e) LGV—doxycycline or erythromycin
### About the Pathogenesis and Manifestations of Primary Syphilis

1. *Treponema pallidum* is a very thin, long bacterium that moves by flexing. Doubling time is very slow (30 hours); it cannot be grown by conventional methods.

2. Able to penetrate skin; initially multiplies subcutaneously.

3. Stimulates acute inflammation, followed by cell-mediated and humoral immunity.

4. Inflammation leads to tissue destruction. The resulting painless skin ulcer teams with spirochetes that can readily be seen with darkfield microscopy.
About Secondary Syphilis

1. After skin penetration, *Treponema pallidum* enters the lymphatics and bloodstream, and disseminates throughout the body.

2. Pink to red, macular, maculopapular, or pustular rash, begins on trunk and spreads to extremities, palms, and soles. Less commonly seen are a) condyloma lata in moist groin areas, and b) areas of alopecia in eyebrows and beard.

3. Lymphadenopathy is generalized, and enlarged epitrochlear nodes suggests the diagnosis.

4. Basilar meningitis can cause ocular motor, pupillary, facial, and hearing deficits.

5. Anterior uveitis, glomerulonephritis, hepatitis, synovitis, and periostitis can result.

About Late Neurosyphilis

1. Meningovascular syphilis causes arteritis and cerebral infarction. Can be a rare cause of stroke in younger patients. Occurs within 5 to 10 years of primary disease.

2. General paresis arises from direct damage to the cerebral cortex by spirochetes, 15 to 20 years after primary disease. Includes
   a) emotional lability, paranoia, delusions, hallucinations, megalomania; and
   b) tremors, hyperreflexia, seizures, slurred speech, Argyll Robertson pupils, optic atrophy.

3. Tabes dorsalis is caused by demyelination of the posterior column, 15 to 20 years after primary disease. Includes
   a) ataxic gait, loss of position sense, lightening pains, absence of deep tendon reflexes, loss of bladder function; and
   b) Charcot’s joints, skin ulcers.

About Cardiovascular Syphilis and Late Benign Gummas

1. Arteritis of the vasa vasorum causes damage to the aortic vessel wall, 15 to 30 years after primary disease. Includes
   a) dilatation of the proximal aorta, leading to aortic regurgitation and congestive heart failure; and
   b) saccular aneurysms, primarily of the ascending and transverse aorta.
   c) Chest radiographs may demonstrate linear calcifications of the aorta.

2. Gummas are granulomatous-like lesions, rare today, except in patients with AIDS.
   a) Skin gummas can break down, forming a chronic ulcer.
   b) Lytic bone lesions can cause tenderness and draining sinuses.
   c) Mass lesions of cerebral cortex, liver, and gastric antrum.
About Testing for Syphilis

1. Non-treponemal tests: The VDRL (Venereal Disease Research Laboratory) and RPR (rapid plasma reagin) test the ability of serum to flocculate a cardiolipin–cholesterol–lecithin antigen.
   a) Modern tests produce only occasional false positive results, usually connective tissue disease.
   b) Prozone phenomenon observed in 2% of cases.
   c) Can be used as a marker of response to therapy.

2. Treponemal tests measure antibody directed against the treponeme.
   a) Specific and sensitive, but antibody titers may persist for life.
   b) Not useful for assessing disease activity, used to verify a positive VDRL or RPR.

3. Tests of cerebrospinal fluid (CSF):
   a) A VDRL of the CSF is positive in one half of neurosyphilis cases.
   b) A peripheral VDRL is positive in three quarters of cases.
   c) Specific treponemal test is positive in all cases, should be ordered when considering neurosyphilis.

About the Treatment of Syphilis

1. Penicillin is the drug of choice.
   a) Therapy must be prolonged (2 weeks) because of the slow rate of growth of the treponeme.
   b) Jarisch-Herxheimer reaction is common: 10% to 25% at most stages, 70% to 90% in secondary disease.

2. Primary or secondary syphilis. Intramuscular benzathine penicillin or, for the penicillin-allergic patient, doxycycline for 2 weeks.

3. Early latent syphilis (within 1 year of exposure). Intramuscular benzathine penicillin or, for the penicillin-allergic patient, doxycycline for 4 weeks.

4. Late latent syphilis. Intramuscular benzathine penicillin for 3 weeks, or, for the penicillin-allergic patient, doxycycline for 4 weeks.

5. Neurosyphilis. Intravenous aqueous penicillin G for 2 weeks, or intramuscular procaine penicillin plus probenecid for 2 weeks.

6. Late syphilis (other than neurosyphilis). Intramuscular benzathine penicillin for 3 weeks, or, for the penicillin-allergic patient, doxycycline for 4 weeks.
About Venereal Warts

1. Condyloma acuminata (anogenital warts) are caused by the human papilloma virus (HPV).
2. The papules vary in size and can be visualized by treatment with 3% to 5% acetic acid.
3. Genital warts predispose to epithelial cell cancers by altering the function of the p53 protein.
4. Palliative treatment is available:
   a) Cryotherapy with liquid nitrogen
   b) Laser surgery
   c) Topical therapy with 10% podophyllum, 0.5% podophyllotoxin (podofilox), or 5% 5-fluorouracil cream
   d) Intralesional interferon
5. A quadrivalent vaccine against HPV types 6, 11, 16, and 18 is efficacious and recommended for girls and women 9 to 26 years of age.
6. Molluscum contagiosum is a rarer form of venereal warts resulting from a poxvirus (seen mainly in patients with advanced AIDS).