**Pyelonephritis**  :

Is an ascending [urinary tract infection](http://en.wikipedia.org/wiki/Urinary_tract_infection) that has reached the  pelvis of the kidney. It is a form of [nephritis](http://en.wikipedia.org/wiki/Nephritis) that is also referred to as **pyelitis**. Severe cases of pyelonephritis can lead to [pyonephrosis](http://en.wikipedia.org/wiki/Pyonephrosis" \o "Pyonephrosis) (pus accumulation around the kidney), **[urosepsis](http://en.wikipedia.org/wiki/Sepsis" \o "Sepsis)** (a [systemic inflammatory response](http://en.wikipedia.org/wiki/Systemic_inflammatory_response_syndrome) of the body to infection), [kidney failure](http://en.wikipedia.org/wiki/Acute_kidney_injury) and even death.

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Most cases of pyelonephritis start off as lower urinary tract infections, mainly [cystitis](http://en.wikipedia.org/wiki/Cystitis) and [prostatitis](http://en.wikipedia.org/wiki/Prostatitis" \o "Prostatitis).

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| Bovine cystitis is an inflammation of the urinary bladder of cattle that may ascend the ureters to cause infection of the kidneys (pyelonephritis). A similar condition is seen in sheep. . It is most often seen after parturition . | |

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| **Etiology and Pathogenesis:** |
| |  | | --- | | The most common causative agents are the *Corynebacterium* *renale* *,C* *renale* , *C* *cystitidis* &, *Escherichia* *coli*(70–80%) .  Pyelonephritis may caused by bowel organisms that enter the urinary tract  e.g : *[Enterococcus faecalis](http://en.wikipedia.org/wiki/Enterococcus_faecalis" \o "Enterococcus faecalis)*. [coliform bacteria](http://en.wikipedia.org/wiki/Coliform_bacteria" \o "Coliform bacteria) ,  and *[Klebsiella](http://en.wikipedia.org/wiki/Klebsiella" \o "Klebsiella)* also uncommon bacteria e.g. [*Pseudomonas aeruginosa*](http://en.wikipedia.org/wiki/Pseudomonas_aeruginosa), staphylococci and streptococci. |  |  | | --- | | The most common causative agents, *Corynebacterium* spp and *E* *coli*  are common inhabitants of the vagina and prepuce.  Pyelonephritis develops from an ascending infection from the bladder.  The predisposing factors that lead to occurance of infection:   1. The stresses of parturition. 2. Abnormal deformity of the vaginal tract 3. Peak lactation 4. High-protein diet (which increases the pH of the urine and is therefore conducive to colonization of the attacking organisms. | | [Back to top](http://www.merckvetmanual.com/mvm/htm/bc/130302.htm) |

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| **Clinical Findings:** |
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| **Diagnosis:** |
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| **Treatment:** |
| |  | | --- | | 1-Early diagnosis is needed for a successful recovery. 2-A catheterized urine sample should be taken for culture and antibiotic sensitivity.  3- The treatment of choice for pyelonephritis due to the *C* *renale* group is penicillin (22,000 IU/kg, IM, bid)  4-Trimethoprim-sulfadoxine (16 mg combined/kg, IM,bid) for ≥ 3 wk.  5-Adequate precautions must be taken to prevent antibiotic residues from entering the human food chain.  6- *E* *coli* infections require a broad-spectrum antibiotic. Gentamicin (2.2 mg/kg, IM, bid) for 4 wk has been used successfully in some cases.  **strangles:** |  |  | | --- | | Strangles is a highly contagious and serious infection of horses and other equids caused by the bacterium*,* *Streptococcus equi*. The disease is characterized by severe inflammation of the mucosa of the head and throat, with extensive swelling and often rupture of the lymph nodes, which produces large amounts of thick, creamy pus.  Strangles is caused by *Streptococcus equi* subspecies *equi*, better known as *Streptococcus equi (S. equi)*.    **Susceptility:**  1-Horses of all ages are susceptible.  2-Animals less than 5 years of age.  3-Foals under 4 months of age are usually protected by colostrum-derived passive immunity.  **Transmission**:  1- Transmission is either by direct or indirect contact of susceptible animals with a diseased horse.  2- Direct contact includes contact with carrier horse that is incubating strangles or has just recovered from the infection.  3-Indirect transmission occurs by contaminated stable (buckets, feed, walls, doors) or pasture environment (grass, fences, but almost always the water troughs), or through flies.  **Clinical signs:**  1- Incubation period is 3–14 days.   2-Animals show depression, inappetence**,** and fever of 39°C–39.5°C).  3-Horses develop a nasal discharge firstly mucoid then thickening and purulent.  4-Soft cough and slight but painful swelling between the mandibles.  5-Swelling of the submandibular lymph node.   1. Horses are often seen positioning their heads low and extended to relieve the throat and lymph node pain. 2. Abscesses develop in the submandibular lymph nodes. 3. The lymph nodes become hard and very painful, and may obstruct breathing ("strangles"). 4. The lymph node abscesses will burst in 7–14 days, releasing thick pus heavily contaminated with *S. equi*. 5. The horse will usually rapidly recover once abscesses have ruptured. 6. Older horses develop mild infection without or with minor lymph node abscessation..   **complications**   * Bastard strangles, which is the dissemination of infection to abdominal or lung lymph nodes and abscess may develop . A brain abscess may rupture causing sudden death . * Purpura haemorrhagica, which is an immune-mediated acute inflammation of peripheral blood vessels that occurs within 4 weeks of strangles. It results from the formation of immune complexes between the horse's antibodies and bacterial components trapped in capillaries and cause inflammation , mucous membranes pinpoint haemorrhages and severe edema of the head, limbs, and other parts of the body. * myocarditis . * Purulent cellulitis . * Laryngeal and throat paralysis . * Anaemia . * Guttural pouchinfection.   **Diagnosis and Treatment**  Diagnosis can be confirmed by culturing pus from the nose, from abscessated lymph nodes or from the throat of clinically affected horses.   * 1. Many veterinarians think that treatment will impair the development of immunity and may predispose an animal to prolonged infection and to bastard strangles.   2. Treatment of a horse in the early stages of strangles is usually effective.   3. The causative agent is highly susceptible to penicillin G.   4. If the disease is more advanced, then most veterinarians will not use antibiotics but drain the abscess.   5. Antibiotics may be used if complications arise.     **Prevention of Strangles**  **Detection of carriers**  A series of 3 nasopharyngeal swabs will result in the detection of about 60% of carriers using isolation and identification of the organism, or of about 90% of carriers using PCR. For the detection of carriers, the laboratory should use a selective medium (Columbia blood agar with nalidixic acid and colistin).  Investigation of carriers should be done either before a new animal is introduced into a stable or herd, or at least 30 days following recovery of a horse from strangles. Animals should be isolated until there have been 3 consecutive negative cultures and/or PCR reactions.  **Vaccination**  1-Both a killed and a live vaccine are available for the control of strangles. In general killed vaccines, are administered of intramuscular injections followed by an annual booster(painfuland may lead to purpura haemorrhagica).  2- The killed vaccines do not provide complete protection  3-More recently, a live, attenuated *S. equi* vaccine available as an intranasal vaccine for the prevention of strangles(MORE PROTECTIVE). It administered twice, at an interval of 1–2 weeks.  **Immunity**  Approximately 75% of horses develop a solid immunity to strangles following recovery from the disease.   However, individual animals may remain carrier and may secrete the organism in nasal exudate or saliva for months or years.  **Control of Strangles**  Isolate clinically affected animals or identified carriers immediately in a quarantine area,  and clean and disinfect their water buckets or feed containers daily.  Bedding can be burned to prevent spread by flies. | | [Back to top](http://www.merckvetmanual.com/mvm/htm/bc/130302.htm) |

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