Pseudolactation in companion goats in the Netherlands

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Abstract

Pseudolactation can be a very disturbing condition for both the goat and the owner. It causes the udder of a non bred goat to develop at a moment in time which it is not physiological or functional. The udder can increase significantly and it can start producing milk. As a result of the enlargement the udder and teat can be damaged by constant contact with the legs of the animal and by contact with the ground. Common reported complications are mastitis and difficulty with normal movement. Reports on pseudolactions in the literature is very limited and the information is often obsolete. The knowledge about pseudolactation in the field is confined and the treatments are various. The pathogenesis of precocious udder in goats is still unknown and as a consequence an ideal therapy is not available. Interviews with patient owners were held to acquire insight into the current situation of pseudolactation in companion goats in the Netherlands. The symptoms differed very much, especially the severity of the disease. A common used manner of treating patients is the use of hormonal injections. A strict ration was also often subscribed. In the absence of satisfying results drastic measures sometimes have to be taken. Amputation of the udder can be a final solution. The condition can cause both the owner and the animal much distress. Veterinarians involved do not have answers to the question of the owner and are not in possession of evidenced-based therapy. The outcome of this research demands continuation to untangle the pathogenesis of pseudolaction in companion goats in order to provide evidenced-based therapy.

Contents

Introduction

Pseudolactation is not well understood. This condition can cause serious harm to the animal involved. The biggest problem is with companion goats. In professional held dairy goats the problem does not stand out as much, the patients start to lactate and get milked with the other dairy goats.

Without a reasonable cause the goat starts to develop a large udder and even starts to lactate. The growth of the udder can be out of proportion and can compromise normal movement. The teat can be damaged by being low to the ground and mastitis can occur, often inflicted by wounds on the teat or open teat canals. The cause of this problem is unknown though there are several theories to be found in literature. Without knowing the aetiology of pseudolactation, developing a preventive and/or curable evidence-based treatment is not possible (amputation not taken into account). Because this condition seriously affects the well-being of the animal and causes the owners a lot of stress we decided to look further into pseudolactation and its aetiology.

Questions/Problems

Pseudolactation is also named ‘maiden milker’ or wrongly pseudopregnancy. This is an example of the mixed information that is available of this condition. There is some literature to be found but a lot of it is very dated or very limited in content. There are several different opinions on the cause of pseudolactation and therefore also several different theories about how to treat the patient. In practice veterinarians are confronted with goats that developed large udders which appear to have happened without an assignable cause. Because there is not a cause known for this problem, the way of treating these patients differs widely. The purpose of this research is getting answers to some questions that were raised during the research prior to starting this inquiry. We want to find out what the aetiology of pseudolactation is. To get to know the cause of this disease it is important to understand the complete process of mammogenesis because it is presumed that somewhere in this process an error occurs after which the udder starts to develop. It is also important to see into the symptoms and side effects it precisely causes the patients. It is interesting to get to know how this problem is handled in practice at the moment, which therapies are used and what are their results. Eventually the purpose will be to find a cure or at least a satisfying solution for the goats affected by pseudolactation.

Materials and methods

A survey was used to get answers to the questions raised. It consisted of 12 questions, five of them were strictly multiple choice, another five of them were multiple choice but with the option to give more specific information. The other two were open questions and there was a part in the survey were interviewed could describe which symptoms the goat expressed during the time. Also the age, the breed and the approximate date the problems started was asked at the beginning of the survey. The vast majority of the owners of patients were questioned over the phone by the same person. A few have been send a copy of the survey and filled in the answers them selves. Because of the large amount of open questions it was necessary to divide the answers into groups so that it became possible to process the information that was obtained from the survey. The groups were formed using the majority of the answers obtained. While arranging the answers it also became clear that a few questions did not have the results hoped for. The owners did not have satisfying information about details like the length of the lactation period if the goat experienced one. They were marked as irrelevant and were not used in the rest of the process.

The answers where put in the right categories and then further processed. Percentages where calculated using excel-spreadsheets. It became clear that a few questions had so many different answers that it was not possible to make connections between them. This involved mostly the various symptoms the patients showed and the equally various therapies the goats where subscribed. To get further insight into the information obtained the percentages and amounts where visualized in charts using excel.

Etiology pseudolactation

There are several different terms for this disease; it is called 'maiden milkers', precocious udder and falsely pseudopregnancy. [1, 2, 3, 4] The goats that are affected with this condition develop a large udder and often start producing milk. The animals are unbred, they can even be maidens. The udder enlargement is mainly because of deposition of adipose tissue in some animals. In others there will be milk production from one or both glands. [2, 3, 4] The symptoms will develop spontaneous and at a moment in time that it isn't physiological or functional. [1]

There are signs that the goats that get a precocious udder are often from genetic stock with high production potential. So there may be a hereditary trait. [1,3]

There are several theories for the etiologic basis of this disease. One theory states that it is possible that some goats are very sensitive to prolonged exposure to progesterone from a persistent corpus luteum or do to elevated prolactin levels in the spring. [3]

Treatment

Mostly it is not advised to start milking the goat. The majority doesn't require milking because they do not produce enough milk to make them uncomfortable. If you would start milking the goat it would stimulate production of more milk which will make it necessary to milk the animal even more often. Also the teat seals will be broken which will increase the risk of mastitis. [3] Pseudolactation in young animals has another complication; the production of milk will act as a nutritional drain on a growing animal. When it is decided to milk the goat it is very important to empty the udder completely as partial milking will predispose to infection. The teats should be dipped after milking. [2, 4]

It is possible to reduce the feed intake in well fed, well grown animals, this may reduce the milk production. [2, 3, 4] Reducing the grain ration and increasing the dry hay uptake could decrease the amount of milk formed. Water deprivation could have the same effect but is not animal-friendly and therefore dissuaded. If it really is necessary to milk the goat because the udder is painfully engorged, or the owner already started milking, a bovine dry period injector could be used to diminish the risk of mastitis. This injector should be administered after fully emptying the udder. If these treatments fail to stop lactation it is possible to keep on milking the goat for a while until a dry-off period can be attempted again. The best period

in the year for this attempt would be the fall because this is the season in which the production naturally decreases. If everything fails it could be the best option to amputate the udder. [3]

Drugs:

Cabergoline, 5 ug/kg, 0,1 ml/kg, orally could be tried, it will inhibit the prolactin secretion. [4] If the pseudolactation is due to elevated progesterone levels because of an persistent corpus luteum a possible treatment would be a luteolytic dose op prostaglandin (2,5-2 mg PgF2Alfa). A treatment with bromocryptine (5 mg bromocryptine mesylate/day/14 days orally), which is an antiprolactin drug, could have a positive effect. There is not enough evidence for this treatment yet and it is very expensive. [3]

Mammogenesis

The development of the udder starts in the foetal stadium in the uterus. In the pre-adolescent period there will be growth of the udder tissue, normally this growth is linked to the growth of the rest of the body. There will develop more fat deposit and connective tissue. Normally there will not be any development of secretion tissue. When the animal starts to get cyclic a fast development of ductuli will start. This growth will now be allometric. After five to six cycli some alveoli will develop. The biggest part of lobulo-alveolair tissue will develop in the last part of a pregnancy. In goats this last stage starts at day 70 and will be finished around day 120. There will be additional growth in the first few days post partum. [1,10]

There are several different hormones involved in the process of mammagonesis.

Progesterone

Normally optimal udder growth starts when both oestrogen- and progesterone-levels are high enough. When a goat is pregnant these concentrations are both high. When a goat is cyclic there should not be any udder development because progesterone and oestrogen concentrations are not high at the same time. [1,10]

Progesterone is a major factor inhibiting the onset of lactation during pregnancy in sheep. Lactogenesis starts when progesterone concentrations have fallen to a very low level at the end of the pregnancy because of regression of the corpus luteum. [8,9,10] Just the drop in progesterone concentration alone is not enough to trigger normal, complete lactogeneseis. [1,7]

Progesterone inhibits the action of prolactin on the induction of milk protein synthesis. It blocks the ability of prolactin to induce prolactin receptors. [1,7,8] It also inhibits the formation of alpha-lactalbumin which is the B protein subunit of lactose syntheses. [1,7]

Then there is the effect of progesterone competing with glucocorticoids for binding on the glucocorticoid receptors. Progesterone is produced by the corpus luteum on one of the ovaries. [1,7,8] During pregnancy the receptors of progesterone disappear. This way, even when progestron concentrations rise, they will not have a negative effect. After involution of the udder the receptors will reappear. [1,10]

Estrogens

Estrogens cause growth of the ductuli in the udder, together with progesterone they cause lobuloalveolar development. [1,8,10] If oestrogen concentrations are sufficiently high they can cause lobuloalveolar development without progesterone. [1,9]

Estrogens stimulate the release of thyroid-stimulating hormone and prolactine. Estrogens are produced by the ovaries and also by the udder and placenta. [1]

Estrogens can be used to induce lactation in goats. [7,8] Hexoestrol 90,25 mg/d) promoted udder growth an initiated the secretory process in goats. When it was combined with progesterone lobuloalveolar development was normal, without progesterone abnormally large cystic alveoli formed. When excessively high doses (1 mg/d) hexoestrol where administered the lactogenesis was delayed or prevented. [7]

Oestrogen has mitogenic effects on the mammary gland [1,7] They stimulate secretion of apha-lactalbumin together with prolactin. When they are combined with glucorticoids the number of prolactin receptors is increased. They also stimulate prolactin secretion directly.

Adrenal corticoids

Glucocorticoids are produced in the adrenal gland, the most important hormone is cortisol. Glucocorticoids bind to specific receptors in mammary cells. They potentiate prolactine induction of milk synthesis, while being inactive alone. In cows progesterone blocks binding of cortisol at the cortisol receptor. This can be the mechanism behind progesterone being able to inhibit lactogenesis. [1,7] Cortisol plays a role in the maximal growth of the ductuli, it is nt essential in this process though. [1]

If you administer exogenous glucocorticoids to a pregnant goat the partus will be induced. In cows large doses of glucocorticoids have been used to induce lactation (Cowie 1969; Kuhn 1977). The mechanism behind this induction is not completely clear. It can be because it triggers release of other hormones, whether there are direct effects on lactogenesis or not is not clear. [7]

Prostaglandin F2Alpha

Prostaglandins cause luteolyse, the result is a drop in progesterone in the blood. When progesterone concentrations lower there inhibitory effect also diminishes. [1,8] It is said that after induced mamamogenensis, prostaglandin f2Alpha aids the onset of lactation. Since prostaglandin concentrations peak during labour the timing of this peak would be ideally suited to enhance lactogenesis. Prostaglandins are produced by the mammary gland, prior

to parturition this synthesis and secretion raises. There is very little evidence that prostaglandins have a direct effect on lactogenenesis. It still is very well possible that prostaglandins are a part of the lactogenic complex which initiates lactation. It helps fast releases of other lactogenic hormones like prolactin, growth hormone en glucocorticoids. [1,7,8,10]

Oxytocin

Oxytocin plays a role in the milk-ejection reflex, it is secreted into the blood when the young suckles or the animal is being milked. Also oxytocin is released during second stage labour in the goat especially during vaginal distension.

Prolactin

Prolactin is produced by the acidophiles cells in the adenohypofyse. [1] Longer photo-period gives increased levels of prolactine during milking in goats. The amount of prolactin in the circulation rises rapidly during the last 2-3 days of pregnancy to peak around the time of parturition. This is followed by a decline during early lactation.

Suckling evokes a release of prolactin, this can be reinforced by visual, olfactory and auditory stimuli. Cowie (1969) did a study with hypophysectomised goat in which he demonstrated the major role of prolactine in lactogenesis. After hypophysectomy milk production reduced 95%. The milk yields rose with substitution of glucocorticoid, triiodothyronine and growth hormone to 28% of normal, when prolactin was also added the milk yields were restored completely. [1,7,10]

With decryption (a dopamine antagonist) it is possible to block secretion of prolactin which leads to a smaller udder and lower milk yield. [1]

Prolactin induces casein synthesis by stimulating effects on the mRNA. This way it stimulates the cellular biosynthesis of lactose, alpha-lactalbumine and fatty acids. When this effect is suppressed by bromocryptine the amount of udder cells stay even but the synthesis within these cells drops. Prolactin receptors are located on the cell membranes and the golgi-complexes. [1,8] When prolactin concentrations rise the amount of receptors will rise too, this will be the same the other way around. This up-regulation is blocked by progesterone. Glucocorticoids can have a stimulating effect. [1]

Growth hormone

Growth hormone can potentiate the effects of prolactin and cortisol. It is produced in the adenohypofyse. This hormone is mostly important for sustaining sufficient milk yield, when removed from the animal the milk yield will drop. [1,10]

Placental lactogen

This hormone is synthesized by the foetal site of the placenta, it is transported actively over the placental-foetal barrier. It works like protactinium and growth hormone. It is not certain that placental lactogen acts like prolactin, it is thought that it binds to prolactinreceptors. In the liver placental lactogen acts like growth hormone, but the effect here is very small. The more foetuses there are in the uterus the higher the concentration of placental lactogen in the maternal blood gets. This then leads to the development of more udder tissue.

This hormone stimulates the alveolar growth and differentiation of the cells, steroid production, growth of the foetus and redirection of nutrients between mother and foetus. [1,7]

Placental lactogen is not absolutely necessary for normal udder development, it is possible to induce normal udder development in non pregnant animals. [1]

Mastitis

Cattle is much more often affected with contagious mastitis than goats. There are several different bacterial agents that can cause mastitis in goats. Mastitis is an important sign in infectious diseases associated with My*coplasma agalctiae* and *Mycoplasma mycoides* var. *mycoides*. Coliform bacteria are mentioned but the opinions about the presence of this agent associated with mastitis are very wide spread. Some say it never occurs, others say it is the most common agent. Coagulase-negative staphylococci are found in udders that do not show any visual symptoms, they seem to cause persistent infections. Other bacterial agents are: *Pseudomonas* spp., *Streptococcus dysgalactiae*, *Streptococcus pyogenes*, *Streptococcus intermedius*, *Arcanobacterium pyogenes*, *Bacillus coagulans*, *Bacillus licheniformis*. Also *Klebsiella pneumoniae*, *Corynebacterium pseudotuberculosis*, *M. haemolytica* and *Actinobacillus equuli* have been found in association with mastitis in goats. Then there is *Nocardia asteroids* which causes a systemic reaction with granulomatous lesions in udder and lungs. [11]

Clinical findings

There is al lot of variation in symptoms caused by the different bacterial agents. Staphylococci can produce enterotoxins and the toxic shock syndrome toxin. They are also likely to cause food poisoning in humans. Streptococci can be transferred from one goat to another by the milker's hand or milking machines. Pseudomonas can cause acute mastitis with extensive necrosis and fatal septicaemia. [11]

In cattle there are a few different forms of mastitis: clinical, subclinical, chronic, acute and peracute, these forms all also occur in goats. Milk samples are often used for diagnostic measures in cattle. In goats there are a few difficulties with diagnostic milk samples. Goat's milk appears often visually quite normal even when there are severe inflammatory changes in the udder. Somatic cell counts is another way of diagnosing problems in the udder. These cell counts vary widely in goats, they are not as reliable as they are in cattle. The cell counts always increase with stage of lactation, also when there is not an infection present. During October, December and January the cell counts are the highest. A lower mature equivalent milk production can also be associated with higher cell counts, as can increased parity. Goats without an infection can have cell counts up to 1.000.000 cells/ml or more. These wide

variations make the reliability of somatic cell counts in milk of goats disputable. The cut-off value in the cell counts is difficult to set. In different studies there is a variation between 500.000 and 1.000.000 cells/ml, above these values it is said that the goat is positive for mastitis. [11, 13, 14, 15, 17] With subclinical mastitis the milk yield can be significantly lower in infected halves. [12] The California Mastitis Test (CMT) is a test that can be used in cattle to get an indication of the severity of mastitis that is present in each udder halve. This CMT could be used on goat milk too. One article states that scores 2 and 3 discriminated between infected and uninfected udder glands. The results showed this test could be used to detect a high percentage of true uninfected glands, the test was not as reliable as hoped to detect effected glands. A lot of them, up to 25 %, where false positive because of high somatic cell counts without an infection in the udder halve tested. [14, 15, 16, 18]

Results

There where nineteen patient owners interviewed. Most have them owned a few companion goats in a field near there home or in their backyard. There was also one petting zoo with a patient and a care institution for disabled people with a goat affected with pseudolactation. In most cases the owners where quite troubled by the problems the goats displayed. The veterinarians involved did not have satisfying answers to their questions and could not solve the symptoms of the goats. In a few cases the owners became desperate and took radical decisions like an udder amputation ore were even advised to have the goat euthanized. On the contrary there where also a few goats with relatively little discomfort from their illness. They just displayed an enlarged udder and some minor difficulty walking normally. In a few cases the owner did not assess the situation to be serious enough to consult a veterinarian.

Age distribution

<1 yr	1-3 Yr	4-6 yr	6-9 yr	9-13 yr	>13 yr	Unknown
0	2	4	4	4	3	2
0 %	10,53%	21,05%	21,05%	21,05%	15.79%	10,53%

Table 1.1

In between the age of four to thirteen years old are the majority of patients. The distribution is fairly even.

Breed distribution

Pygmy goat	Dairy type goat	Landgoat	Dutch white goat	Saanengoat	Unknown
7	5	2	2	1	2
36.84%	26,32%	10,53%	10,53%	5,26%	10,53%

Table 1.2

It is said that patients are often from a genetic stock with high production potential. [1,3]

The results from the questionnaire are not so defined. Dairy goat breeds represent 42,11 %, pygmy goats and land goats which are not bred for milk production represent 47.37 %. There is also a part of the owners who did not know the breed of there goats, mostly this seemed to be crossbreds.

Duration of symptoms

< 1 yr	1-2 yr	3-4 yr	>4 yr	Unknown
6	4	6	2	1
31,58%	21,05%	31,58%	10,53%	5,26%

Table 1.3

A large part of the goats where displaying symptoms for quite some time. Above one year of experiencing problems were 63,16% of the nineteen interviewed patients. One of the goats was displaying symptoms for over eight years, the symptoms where mild. The problems started with an enlarged udder and the animal suffered from some skin damage on the udder in the beginning. The last few years it displayed only the enlarged udder, which did not seem to interfere significantly with the goats well being.

Start symptoms

2010-heden	2008-2009	2006-2007	< 2006	Unknown
7	3	6	2	1
36,84%	15,79%	31,58%	10,53%	5,26%

Table 1.4

The goats differed a lot in age at which the problems started. A few of them started displaying problems at a young age, under two years old. There was a larger part which started at three-four years old. Between five and eight years old there where the same amount of patients as between three and four years of age. Three patients developed symptoms at a higher age. Two where fourteen when problems started and one was eleven years old.

Amount of gestation(s)

None	1 x	2 x	> 2 x	Unknown
12	4	2	0	1
63.12%	21.05%	10.53%	0%	5.26%

Table 1.5

The majority had never been pregnant, 63,12%. Approximately one third had carried a pregnancy to term one or two times. In a few cases a normal lactation period followed but in another few cases the young died and the lactation period was disrupted. This post partum disturbance can possibly be linked to the cause of pseudolactation.

Relatives with similar problems

Unknown	Sister	Mother	Grandmother	Sister & grandmother	None
15	1	1	0	1	1
78,95%	5,26%	5,26%	0%	5,26%	5,26%

Table 1.6

Because there are signs that the goats that develop a precocious udder are often from genetic stock with high production potential, there may be a hereditary trait. [1,3] Therefore one of the questions was set up to get some information about a possible genetic component. In the majority of the cases the owners of the animals did not have any information about the relatives of there goat. They bought one ore two goats some time ago without a genealogical register of any kind. It was notable that in cases that the owners did have knowledge about the origin of there animals, there were often one or more relatives with similar problems. The group of patients in which answers on this question where obtained included only 4 animals. In which 3 of them possibly had a genetic component in there condition.

Veterinarian consult

Yes	No
17	2

Table 1.7

As mentioned before there were a few cases in which the enlarged udder was about the only symptom the animals displayed. In two of those cases the owners found it unnecessary to consult a veterinarian. In most of the cases one veterinarian, or a few times even several veterinarians, paid one ore more visits to the patients.

Symptoms

Udder enlarged both sides	Udder enlarged one side	Compromised walk	Aggressive behaviour	Maternal behaviour	Udder damaged
17	4	14	1	1	5
89,47%	21,05%	73,68%	5,26%	5,26%	26,32%

Damaged teat, standing on teat	Mastitis	Abscess formation	Time bound symptoms	Lumpy udder	Pus from vagina	Kicks at udder
2	7	1	1	1	1	2
10,53%	36,84%	5,26%	5,26%	5,26%	5,26%	10,53%

Table 1.8

The symptoms and mostly the combination of symptoms varied a lot. Not one of them was the same. An important discrimination to make was whether the condition occurred on both sides of the udder or unilaterally. In almost 90% of the cases both sides of the udder became enlarged. In approximately 20% only one halve of the udder displayed symptoms. The majority showed, at any level, some difficulty with normal locomotion. This is a quite logical result of an enlarged udder between the legs. As was to be expected there was a reasonable amount of patients (36.9%) who developed wounds on the teat and/or udder because of contact with the ground or there hind legs. In over 35% of the cases this and the tension on the udder, sometimes in combination with an open teat canal, led to mastitis. In one case the wounds even led to a chronic abscess. Also seen once was a lumpy udder. The veterinarian involved told the owners of the goat that it was due to the formation of a lot of connective tissue in the udder. Two cases stood out in there peculiarity. The goats in these cases started to express abnormal behaviour. One goat started expressing aggressive behaviour after developing pseudolactation. It attacked people entering the field she was housed in, the owners described here manners as male goat behaviour. In another interesting case the patient started expressing maternal behaviour towards a Brahma rooster present at his pasture. It had the same colour as the goat and the patient seemed to have adopted the rooster as here young. The goat tried to groom the rooster and pushed it towards here udder the same way a goat who just kidded of would do. In this last case the owners decided to let the udder be amputated to stop the symptoms of pseudolactation. After this operation the maternal behaviour towards the rooster stopped.

One theory on the cause of the formation of a precautious udder states that it is possible that some goats are very sensitive for prolonged exposure to progesterone from a persistent corpus luteum or do to elevated prolactin levels in the spring. [3] This means that the symptoms should be time bound. Only in one case there was a clear time bound pattern. The goat started developing symptoms in January and in July. The symptoms returned every year at least once, sometimes twice, in both months. This pattern started in 2006.

It was not always clear when the symptoms exactly started in a lot of the patients. Some times because it was simply a long time ago, sometimes because the supervision over the animal was not very regular. In a few cases the time of year when the problems started was noted. Two times the problems started in the spring, two times in the summer and once

around Christmas in the winter. The answers obtained in this survey are not very conclusive on the theory on hormonal changes in the spring.

Self suckling

Never noticed	occasionally	regularly
16	1	2
84,21%	5,26%	10,53%

Table 1.9

One of the risk factors to develop pseudolactation is thought to be self suckling of the goat. By sucking on the teat of her own udder it is possible that she stimulates milk production. It is often not clear whether the milk production or the self suckling was the first to start. It is known that stimulation of the udder can stimulate milk production. In the majority of the cases the goat was never caught self suckling, sometimes the surveillance of the goats was not very regular so this kind of behaviour could be missed. Two goats expressed self suckling regularly and one goat sucked on her own teat only once in a while.

Irritation of the udder

Thistles/nettles	High grass	Unknown
5	7	7
26,32%	36,84%	36,84%

Table 1.10

Another form of stimulation of the udder is an irritating environment. Thistles and nettles in the field can tickle or lightly scratch the udder and teat. This irritation can cause milk production stimulation. In over one third of the fields the goats lived in the grass was, in some areas of the field, so high that it reach the udder. In over a quarter of the pastures there were irritating plants like thistles present. The goats could get in contact with these plants and the areas of high grass and this way stimulation of the udder could have an effect on the severity of the condition. It is not possible to tell from this results how much influence the presence of this kind of stimulation and irritation has. Also it is still possible that this effect plays a role in the development of pseudolactation.

Therapies

Single hormone injection	Series hormone injections	Regimen short < 4 weeks	Regimen long > 4 weeks	To mate
8	4	2	6	1
42,10%	21,05%	10,53%	31,58%	5,26%

Dry-treatment antibiotic	Regular milking	Milking once	Mastitis treatment	Udder amputation
4	2	4	3	2
21,05%	10,53%	21,05%	15,79%	10,53%

Minimize manipulation of the udder	Pain relief	Mowing grass short	Puncture/ echo; for diagnosis	No therapy
1	1	1	1	3
5,26%	5,26%	5,26%	5,26%	15,79%

Table 1.11

Because of the lack of information and knowledge on pseudolactation in literature there is consequently also a lack of knowledge on this condition in the field. This is the reason the therapies advised differ so much. Almost al the patients where treated differently. There was a tendency towards hormone therapy. Over 40% administered a single hormone injection, above 21% used series of multiple hormone injections. Hormone injections could not be further defined because the interview was held with the owners of the animals which were not aware of the precise drug used by the treating veterinarian. Secondarily a ration was often advised (42,11%). In literature it is one of the treatments advised. [3] Mostly the owners were told to diminish the amount of protein intake by feeding less concentrates and more hay. The way the owners followed this advice differed widely. The animals involved where exclusively pet animals, this consequently caused that the owners soon started to feel sorry for the goat being fed just hay and water. The rations were not as strict and as long as was advised most of the time. The owners soon started to administer concentrates and other treats which means the effects of the rationing where offset.

Mastitis was a quite frequently seen complication, the animals diagnosed received a mastitis treatment. This consisted mostly of antibiotics in the udder after completely emptying the udder. Sometimes a dry-treatment antibiotic was used without signs of mastitis, just to try and stop lactation and prevent mastitis from developing. Start milking the goat to relief the pressure and sometimes to later try and attempt to induce a drying off period, was advised a few times (10,53%). Most of the time the animals where just milked once by the veterinarian to rule out mastitis (21,05%).

Udder irritation can cause stimulation of milk production. This is why in a few cases it was advised to minimize stimulation of the udder by mowing long grass, keeping the animal inside to diminish contact with irritating insects and by minimizing manipulating the udder.

In two cases the symptoms where so severe and the therapy results so minimal, the owners decided to go for a more radical treatment and had the udder of the goat amputated. The goats where healthy afterwards and did not express any problems anymore.

Another advice given was to mate the goat. The intention of this was to reset the hormonal balance and induce a normal lactation and dry-off period after kidding. The goat that was bred did not yet give birth and the owner was unsure whether the goat got pregnant ore not.

Milking therapeutically

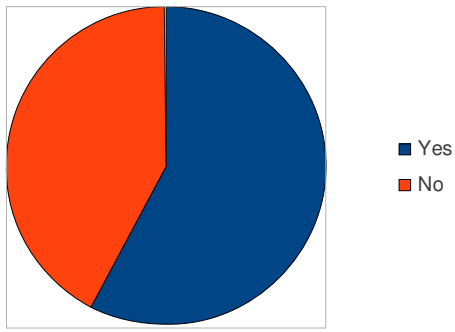

Fig. 1

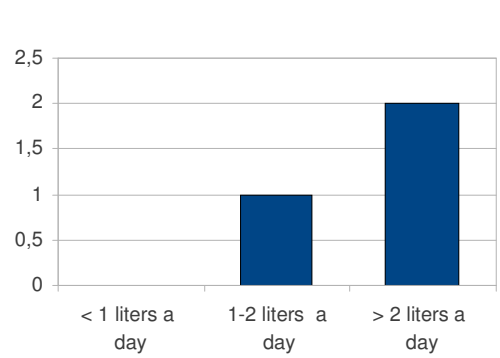

Fig. 2

Once	2-4 days	Weekly	Daily	Occasionally
5	2	2	1	1
26,32%	10,53%	10,53%	5,26%	5,26%

Table 1.12

One of the measures taken by the owners and veterinarians involved was starting to milk the goat. Mostly the goat was milked only once to rule out mastitis and to be sure that there was milk production. Therefore udder development and not just an udder enlarged by a tumour of some sort could be made plausible. Sometimes the milk production and udder development was so severe that tension on the udder caused serious discomfort. A few times the goat had to be milked regularly to relief some of the pressure. This could be every few days (10,53%), weekly (10,53%) ore in one case occasionally. One time it was decided to start and milk the goat daily to relief the pressure and later try to attempt an dry-off period to stop the symptoms. It was notable that the milk yield in all regular milked goats was

considerable. One goat produced one to two liters a day, two goats produced over two liters a day.

Therapy results

Improved momentarily	Recovered	No effect	Slight improvement	No therapy used
0	4	6	7	2
0%	21,05%	31,58%	36,84%	10,53%

Table 1.13

The therapies used differed a lot, also the combination of therapies used was very variable. In the majority off cases the effects were unsatisfactory. In 36,84% there was slight improvement noticeable but the residual symptoms where still problematic. In 31,58% there was no difference between the situation before therapy and afterwards.

Four goats recovered completely after treatment. Two of them underwent amputation of the udder. Two of them recovered with therapy. Both of these two goats received a strict diet for a sustainable amount of time.

Discussion

The surveys were taken from owners of companion goats. Their vision on what happened to there animal can be coloured with emotions. We tried to avoid this effect by taking the surveys personally spoken over the phone and all by one person. When asked about the treatment a veterinarian started for the animal involved, we received very different answers and most of them were not sufficient enough. Answers like ‘a hormone injection’ are not specific and because of this it did not always became clear what the veterinarian exactly administered.

It became clear that some questions resulted in so many different answers that it was not possible to use them for analysis. These questions mostly concern the various symptoms and the equally various therapies the goats were subscribed

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