ABDOMINAL COMPARTMENT SYNDROME IN DOGS

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Preface

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Summary

Intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) are terms originating in human medicine. Extensive research has been performed on IAH and ACS in human patients, but via experiments on dogs as well.

In veterinary medicine, the concepts of IAH and ACS are not commonly used so far and no attention has been given to their meaning either. Consequently, few scientific publications on IAH and ACS in diseased dogs exist so far. Therefore, one should use a number of data resulting from the canine experiments performed in human medicine.

When intra-abdominal pressure (IAP) rises above the reference values, IAH exists. However, no consensus has been reached so far on the reference interval of normal IAP in dogs. When IAP attains abnormally high levels, it can be associated with symptoms of organ failure. This is considered as ACS in human patients. The aetiology of IAH and ACS in dogs is probably comparable to that in humans. Several conditions entailing a) impairment of the adaptability of the abdominal wall, b) a mass effect in the abdominal cavity, c) free fluid or gas in the abdomen or d) capillary leakage, can trigger IAH. The pathophysiology of IAH shows similarities between dogs and humans as well. Particularly, cardiovascular consequences for the organs are important. The symptoms of ACS are described in only one case report of a dog suffering from babesiosis. However, it has not been established with certainty that these symptoms can be attributed to an increase in IAP, since this dog’s IAP was not measured objectively.

Different techniques are available to measure IAP directly and indirectly. The intravesical method is used most of the time in the canine experiments. If it would be clinically relevant to measure IAP in dogs, the need for a standardized measurement protocol exists. So far, however, it remains unclear whether ACS in dogs occurs as a clinical problem.
Samenvatting

Intra-abdominale hypertensie (IAH) en abdominaal compartiment syndroom (ACS) zijn begrippen die afkomstig zijn uit de humane geneeskunde. Er is reeds veel onderzoek gebeurd naar IAH en ACS bij humane patiënten, maar ook via experimenten op honden.

In de diergeneeskunde zijn de begrippen IAH of ACS tot nu toe nog niet gebruikelijk en evenmin werd aandacht gegeven aan wat zij inhouden. Bijgevolg bestaan er ook nog niet veel wetenschappelijke publicaties over IAH en ACS bij de zieke hond. Daarom moeten een aantal gegevens gebruikt worden die voortvloeien uit de hondenexperimenten, die uitgevoerd werden ter ondersteuning van de humane geneeskunde.

Men spreekt over IAH wanneer de intra-abdominale druk (IAD) boven de referentiewaarden stijgt. Bij de hond bestaat echter nog geen consensus over het referentie-interval van de normale IAD. Wanneer de IAD abnormaal hoog oploopt, kan dit gepaard gaan met symptomen van orgaanfalen. Bij de mens spreekt men dan van ACS. De etiologie van IAH en ACS bij de hond is vermoedelijk vergelijkbaar met de mens. Allerlei situaties waarbij a) het aanpassingsvermogen van de buikwand is aangetast, b) waarbij er een massa-effect bestaat in de buikholte, c) waarbij er vrij vocht of gas in de buikholte zit of d) waarbij er capillaire lekka ge bestaat, kunnen aanleiding geven tot IAH. De pathofysiologie van IAH vertoont eveneens gelijkenissen bij zowel de hond als de mens. Vooral de cardiovasculaire gevolgen voor de organen zijn belangrijk. De symptomen van ACS zijn slechts beschreven in één casus van een hond met babesiosis. Toch is het niet helemaal zeker of deze symptomen wel toe te schrijven zijn aan een verhoogde IAD, aangezien de IAD van deze hond niet objectief werd gemeten.

Er zijn verschillende technieken voorhanden om IAD op een directe en indirecte manier te meten. Tijdens de hondenexperimenten is de meest gebruikte manier de intravesicale methode. Mocht het klinisch belangrijk zijn om de IAD te meten bij de hond, dan is er wel nog nood aan een gestandaardiseerd protocol om dit te doen. Het blijft tot nu toe evenwel onduidelijk of ACS bij de hond voorkomt als een klinisch probleem.
Introduction

In human medicine, the development of abdominal compartment syndrome (ACS) due to rising intra-abdominal pressure (IAP) is a feared complication in patients. In particular, ACS tends to affect people who had recent abdominal surgery or who suffer from abdominal trauma. An extensive amount of literature covers the different aspects of pathophysiology, diagnosis and treatment with regard to ACS. It is clear that ACS is recognized as a dangerous, potentially fatal, condition in the human patient that needs to be approached as an emergency.

A lot of data from human medicine result from experiments conducted on animals, mostly dogs. The first goal of the experiments is gaining an insight into the pathophysiology of IAH and ACS in people. Especially the cardiovascular consequences are of great importance (Barnes et al., 1985). Another group of experiments aims to establish an ideal measurement technique to estimate IAP reliably. A whole array of methods has been investigated. The intravesical method, in particular, has been adapted and fine-tuned over the years (Smith and Sande, 2012).

However, the amount of veterinary literature on intra-abdominal hypertension (IAH) and ACS is clearly smaller, including experiments on pigs, horses and companion animals. There are only a few actual cases of dogs suffering from IAH, i.e. an IAP of more than 5.5 mmHg. They do not exhibit symptoms of ACS (Conzemius et al., 1995; Kirby, 2011). One case report describes a clinical example of supposed ACS. In this article, the dog showed symptoms which were similar to those in human patients with ACS (Joubert et al., 2007). The question remains whether this is really a case of ACS similar to the condition in humans. The ultimate goal of this master thesis is to ascertain if, in dogs, an IAP of more than 5.5 mmHg is a serious, life-threatening condition as it is in human medicine.
I. Definitions and basic concepts

First of all, it is important to set up a framework for the subject of IAH and ACS. In this chapter, we aim to define certain terms, in order to render the subsequent use of them clear throughout the manuscript.

In human medicine, the complications of IAH and ACS have been thoroughly investigated in recent years. There has been a lot of discussion about the normal values of IAP, i.e. the constant pressure within the normal, closed abdominal cavity (Kirkpatrick et al., 2013). Eventually, the World Society for the Abdominal Compartment Syndrome (WSACS) proposed a number of definitions and guidelines for human medicine. Normal IAP in adults ranges from 0 to 5 mmHg, whereas in children, the normal values range from 0 to 4 mmHg. These intervals should not be interpreted too strictly, as there are healthy people with an IAP slightly higher than 5 mmHg (Kirkpatrick et al., 2013).

In adult people, the WSACS defines IAH more specifically as “a sustained or pathological elevation in IAP” above 12 mmHg for at least 12 hours. A grading system for adults has been introduced to divide IAH in four stages. Grade I consists of an IAP of 12-15 mmHg, whereas grade II shows an IAP of 16-20 mmHg. Grade III is reached, when IAP rises to 21-25 mmHg. Finally, grade IV comprises an IAP of more than 25 mmHg. In children, IAH occurs from 10 mmHg upwards (Kirkpatrick et al.; 2013).

When IAP keeps rising, it will lead to ACS, a life-threatening situation. Typically, it occurs in critical patients with an abdominal pathology or who underwent recent abdominal surgery (Malbrain et al., 2006). Whereas IAH is defined by the WSACS as “a sustained or pathological elevation in IAP above 12 mmHg”, ACS is defined by the WSACS as “a sustained IAP above 20 mmHg”, leading to organ failure (Kirkpatrick et al., 2013). The interval 15-20 mmHg is considered as critical (Sugrue et al., 1995). In children, ACS occurs when IAP increases above 10 mmHg and is “associated with new or worsening organ dysfunction” (Kirkpatrick et al., 2013).

Canine reference values of IAP are still open to debate, since IAP in dogs has barely been investigated. The pressure measured clinically in the abdominal cavity of normal dogs was between 1.5 and 5.5 mmHg (Conzemius et al., 1995) or between 0 and 7.4 mmHg (Drellich, 2000), depending on the study methodology. Both studies were pioneering works, that have been supported by several other authors afterwards (Smith and Sande, 2012). However, a variety of upper levels can be encountered in later works. For instance, normal IAP in dogs measured up to 3.75 mmHg (Joubert et al., 2007) or up to 5 mmHg (Land et al., 2009). In short, there is no consensus in veterinary literature about the exact values constituting normal IAP in dogs. Theoretically, values above the upper reference values in humans and in dogs constitute IAH (Conzemius et al., 1995). What is interesting, though, is the fact that higher values are sometimes considered normal in dogs. For instance, an IAP of 6.6 mmHg can be physiological in a dog. This can be due to IAP measurement performed on animals that are awake and contract their abdominal muscles (Land et al., 2009).
II. Aetiology of intra-abdominal hypertension

IAH and ACS can be caused by a variety of pathological conditions. These potential aetiologies have been extensively investigated in human medicine for decades and are summarized in concise lists of causes by Malbrain et al. (2006), Cheatham et al. (2007) and Malbrain et al. (2008). We will use the authors’ subdivision of aetiologies here, paying extra attention to causes that have been proven to exist in dogs. A number of conditions in human patients have not been documented in dogs or other domestic animals so far, but are thought to have the potential to trigger IAH and ACS in animals as they do in humans. These conditions will be discussed under their specific headings. At the moment, veterinary research is catching up on this topic. Still, many human medicine studies include experiments on dogs, from which can be gleaned useful knowledge for veterinary purposes.

Potential causes for IAH/ACS can be assigned to four pathological conditions, i.e. a) decreased abdominal wall compliance, b) elevated intraluminal contents in the abdomen, c) free fluid, air or blood in the peritoneal cavity and d) capillary leakage (Cheatham et al., 2007; Malbrain et al., 2008). Some overlap exists between these four categories and IAH/ACS is often found in patients suffering from more than one of these conditions. If the patient suffers from pre-existing disorders (heart, lungs, kidneys), IAH can further increase or ACS can manifest itself more easily. Chronic IAH will lead more swiftly to clinical ACS (Papavramidis et al., 2011).

In general, causes defined in humans can predispose for higher IAP in dogs as well. It was observed in a clinical study that a number of dogs suffering from these risk factors did not have an elevated IAP. Due to the small sample size, the authors were not able to draw conclusions about whether the risk factors are as important in dogs as in people (Fetner and Prittie, 2012).

Furthermore, ACS (and IAH) in humans can be categorized into a primary, secondary and recurrent form. Primary ACS results from pathologies of the abdominal and pelvic organs, e.g. acute pancreatitis or splenic rupture. Secondary ACS originates from injuries or diseases not affecting the abdominal and pelvic organs, e.g. sepsis, burns, capillary leak. Tertiary or recurrent ACS occurs when the medical or surgical treatment of primary or secondary ACS leads to a new bout of ACS (Malbrain et al., 2006). A supposed example of secondary ACS in a dog is shown in a case of babesiosis. The animal suffered from a distended abdomen, renal failure and respiratory distress, which were probably attributed to an increased IAP of 17.6 mmHg (Joubert et al., 2007).

a) Decreased abdominal wall compliance

In situations that require mechanical ventilation, a lot of stress is placed upon the abdominal wall. This happens especially when positive end expiratory pressure (PEEP) is used or when auto-PEEP occurs (e.g. air-trapping in the airways of patients with bronchospasms, mucosal oedema...) (Corona and Aumann, 2011). In one study, a mechanically ventilated dog with a high IAP of 15.3 mmHg is described as an example (Fetner and Prittie, 2012).

In fact, even normal inspiration puts a physiological pressure on the abdominal wall. The diaphragm is important in this respect. During inspiration the abdominal cavity is compressed by the expanding thorax, thus putting an outward strain on the abdominal wall (Dörfelt et al., 2012). Related to ventilation- and inspiration-triggered pressure increase, airway conditions...
that entail difficult removal of air from the lungs, also give rise to higher pressures inside the body cavities. An increase in intrathoracic pressure is partly transmitted to the abdomen, leading to an elevation in IAP (Barnes et al., 1985). For instance, (pleuro)pneumonia is combined with high intra-thoracic pressure, especially in cases of acute respiratory failure (Kirkpatrick et al., 2013).

Further, recent abdominal surgery with tight (fascial) closure can put a severe strain on the abdominal wall. It has been shown that dogs have IAH at least 24 hours after elective ovariohysterectomy. However, the elevated IAP did not have any clinical significance in these healthy animals (Conzemius et al., 1995). In another study, five dogs underwent one or more laparotomies. Three dogs suffered from high IAP (10.6, 15.3 and 9 mmHg), whereas two dogs' IAP was rather normal (5.3 and 3.6 mmHg) (Fetner and Prittie, 2012). Especially when the surgical site is oedematous, the abdominal wall is not able to adapt to changing pressures (Carlotti and Carvalho, 2009). If there is also oedema of the intestinal organs due to manipulation and evisceration during surgery, IAP will increase even more (Burch et al., 1996). When primary closure of the abdominal wall was performed, 52 % of the human patients showed symptoms of ACS. Primary closure is not considered the best option in all cases. When the abdomen was closed with an abdominal wall-sparing technique, only 24 % of the patients showed symptoms. A clinical study in dogs presented for mastectomy showed that the highest IAP was recorded 4 hours post-operatively (Land et al., 2009). Afterwards, the values diminished again, until pre-operative values were reached again. At the time of mastectomy, a lot of tissue was removed in order to obtain tumour-free margins. This led to a substantial skin loss and the wound was closed under considerable tension. The effect was that of a corset, leading to a rise in IAP (Land et al, 2009). Another study in dogs has nuanced the association between recent abdominal surgery and an increase in IAP. No difference was observed between IAP before and IAP after surgery in dogs undergoing elective ovariohysterectomy. Thus, IAP is not influenced by elective abdominal surgery in normal, healthy dogs. When IAP is increased post-operatively, this is likely to be a transient situation, without clinical significance. This may not be the case in diseased patients (Way and Monnet, 2014).

Correction of abdominal and diaphragmatic hernias can lead to similar consequences for IAP (Conzemius et al., 1995; Dörfelt et al, 2012). Similarly, when external counterpressure is placed upon the abdomen, IAP elevates (Kirby, 2011). This can happen iatrogenically, when patients are treated with abdominal compressive bandages. One dog in a study was treated with such an abdominal compressive bandage after liver lobectomy and this led to an IAP of 15.3 mmHg. This example supports that the same risk factor applies to humans and dogs (Fetner and Prittie, 2012).

Another cause of decreased abdominal wall compliance is major trauma. The abdominal wall layers are severely damaged and cannot cope with the excessive strain of bleeding and hematomas. The same happens with excessive burns along the surface of the abdomen. The abdominal wall is not able to accommodate pressure changes, when there are extensive eschars on the abdomen. For example, two dogs suffering from major trauma had both an IAP that was higher than normal (11 and 9 mmHg) (Fetner and Prittie, 2012).

Contraction of the abdominal muscles can lead to an increase in IAP (Smith and Sande, 2012). This occurs especially in conscious patients suffering from pain and in coughing, vomiting, panting and whining patients (Smith and Sande, 2012). Indeed, it has been proven that there is a significant increase in IAP in conscious, struggling cats compared to sedated cats (Rader and Johnson, 2010). A clinical study in healthy cats undergoing ovariohysterectomy has proven that, immediately postoperatively, IAP rises significantly (Bosch et al., 2012). This increase can be attributed to contracted abdominal muscles due to
pain and hypothermia. Similarly to humans, shivering can raise IAP temporarily because the abdominal wall muscles contract (Bosch et al., 2012). In another study, two dogs were presented with severe pain in the abdomen. Only one of them, however, had an elevated IAP of 10.6 mmHg (Fetner and Prittie, 2012).

In spontaneously breathing human patients, it has been proven that the use of opioids (fentanyl) stimulates active expiration and increases muscle tone (Drummond and Duncan, 2002). This results in an increase in IAP above the recommended value for laparoscopy (14.7 mmHg). The ideal value for laparoscopy in dogs is 10 mmHg, which combines good visualization and minimal cardiovascular stress (Maiti et al., 2013). An older study advises that CO₂ insufflation can range from 8 to 12 mmHg (Ishizaki et al., 1993). However, a study in 16 young, medium large dogs shows no elevation of IAP following the intra-operative use of fentanyl. There was no extreme muscle rigidity observed in dogs during anaesthesia. Possibly, maintenance of anaesthesia with isoflurane decreased active expiration and muscle use. Another viable explanation for the difference with human and canine patients is a different activity of fentanyl in the central nervous system. Further, the effective drug dose might vary between humans and dogs. Nevertheless, one dog in this study did show an increase in IAP during expiration. The cause of this is not clear. Possibly, some form of airway obstruction (e.g. bronchoconstriction) lay at the root of this deviant patient (Dörfelt et al., 2012).

Obesity or high body mass index (BMI) is also likely to elevate IAP by putting excessive strain on the abdominal wall. In people, a BMI of more than 30 predisposes the patient for IAH (normal BMI ranges from 18.50 to 24.99 (WHO, 2000)). Possibly, IAP is even chronically increased in patients with obesity (Smith and Sande, 2012). A study in cats has shown that animals with a body condition score (BCS) of 7/9 (Purina)¹ are indeed prone to have a significantly higher IAP (Rader and Johnson, 2010). This seems to apply to dogs as well: a morbidly obese dog had a high IAP (9.3 mmHg) (Fetner and Prittie, 2012).

Lastly, sternal position in humans causes higher IAP compared to supine position, when the abdominal wall is relieved from the weight of the organs and spine. The difference between sternal and supine positions has not been investigated in dogs and cats (Smith and Sande, 2012). In any case, lateral recumbency is more comfortable for small animals in hospitalization. In cats, lateral recumbency leads to higher IAP than sternal position. However, the values of IAP in these cats are not clinically significant (Rader and Johnson, 2010).

b) Elevated (intraluminal) contents in the abdomen

Hypomotility of the gastrointestinal tract or ileus can lead to an accumulation of digested material in the abdomen. An example of raised IAP due to hypomotility is reported in one dog suffering from ileus and high IAP (11 mmHg) (Fetner and Prittie, 2012). Along the same lines, (sub)obstruction and intussusception of the gastrointestinal tract, especially of the colon, can give rise to increased IAP (Beck et al., 2001). Similarly, gastric distention and volvulus entail a severely increased volume in the stomach. Even a moderately increased amount of gas in the gastrointestinal tract can elevate IAP (Conzemius et al., 1995; Joubert et al., 2007).

¹ UNC research body condition scoring feline (https://research.unc.edu/files/2012/11/CCM3_032386.pdf), consulted in May 2018.
Intra-abdominal inflammation, infection or abscesses trigger an elevation of IAP. Examples of dogs suffering from acute pancreatitis, vasculitis and enterocolitis have been documented. Not all of them, however, did have an increased IAP (Beck et al., 2001; Fetner and Prittie, 2012).

Intra-abdominal and retroperitoneal tumours and hematomas are space-occupying processes, contributing to a rise in IAP (Kirkpatrick et al., 2013).

Iatrogenically induced IAH can occur due to abdominal packing. This technique can be used for the treatment of severe trauma and uncontrolled haemorrhage of abdominal organs (Burch et al., 1996).

A physiological reason for an increase in IAP is pregnancy. The reproductive organs occupy more space in the abdomen, sometimes even in the postpartum period (Sugerman et al., 1999). Additionally, pathologies of the abdominal organs are frequently encountered in dogs. Pyometra remains a common affliction of the intact female dog. A clinical study in dogs has shown that pyometra is a cause of an increase in IAP. In dogs with a closed pyometra, IAP can rise above 12 mmHg. When the uterus was removed in these patients, IAP had decreased dramatically post-operatively. This difference was even more marked when the uterus has reached a large size and weight pre-op. None of the dogs showed symptoms of ACS (Land et al., 2009).

c) Free fluid or air in the peritoneal cavity

Peritonitis can cause the accumulation of free fluid in the abdominal cavity (Malbrain et al., 2006). Primary or spontaneous peritonitis, in which case there is no contamination or trauma of the abdominal cavity, is not common in dogs. Secondary peritonitis, however, is seen more often and presupposes a concurrent abdominal pathology (Swann and Hughes, 2000; Doom and De Rooster, 2016). The secondary form can be aseptic or septic. Tertiary peritonitis results from uncontrolled or recurrent secondary peritonitis and has not yet been found in dogs (Chromik et al., 2009; Doom and De Rooster, 2016). Indeed, a significantly higher IAP has been documented in dogs with peritonitis (Fetner and Prittie, 2012).

Another reason for abdominal fluid accumulation is liver dysfunction (cirrhosis), resulting in ascites. A dog with ascites suffered from high IAP (10.6 mmHg) (Fetner and Prittie, 2012). Interestingly, liver disease and ascites do not always lead to IAH. If the liver pathology is chronic, the abdominal wall and organs can adapt to the increasing pressure, without the development of real IAH (Burch et al., 1996).

After trauma, hemoperitoneum, pneumoperitoneum and uroabdomen influence IAP significantly (Conzemius et al., 1995; Burch et al., 1996; Joubert et al., 2007). Indeed, a dog with hemoperitoneum is reported suffering from high IAP (15.3 mmHg) (Fetner and Prittie, 2012).

Iatrogenically induced IAH can occur when laparoscopy with high inflation pressure is performed, thus releasing a huge amount of free gas in the abdomen. Additionally, when peritoneal dialysis is performed, IAP can sustain an acute increase (Malbrain et al., 2006).

An experimental study in medium-sized dogs has shown how IAP can be increased by fluid infusion into the abdomen, as an example of a volume-occupying process (Barnes et al., 1985). The animals were in supine position, while some important vessels and the atria of the heart were cannulated for placing pressure and flow probes. The infusion of fluids at a rate of 0.15-0.30 L/min into the abdominal cavity in dogs led to an increase in IAP from 0 to
40 mmHg. The rapid infusion rate was used here to prevent absorption of the fluids into the blood and stress relaxation of the abdominal wall. So, an IAP elevation of 40 mmHg was caused by a rise in intra-abdominal volume of 240 mL/kg body weight. This had a lot of consequences. The compliance of the abdominal wall decreased from 10.8 to 0.56 mL.mmHg\(^{-1}.kg\(^{-1}\) body weight when the IAP elevated from 0 to 40 mmHg. Initially, there was extensive compliance of the abdominal wall, which tended to decrease rapidly from a certain point during the increase of IAP. In short, this study showed that the abdominal wall can compensate for a certain amount of free fluid in the abdomen, until a maximum volume is reached. If this volume becomes too high, IAP rises until IAH occurs (Barnes et al., 1985).

d) Capillary leakage

When major trauma or external burns have occurred in humans, the inflammatory response increases capillary leakage in the abdomen due to higher vascular permeability (Malbrain et al., 2006). The situation can be aggravated when a great amount of fluid is administered to stabilize the patient. Massive fluid resuscitation overfills blood and lymphatic vessels, leading to ascites and visceral oedema (Kirkpatrick et al., 2013). Crystalloids are more dangerous than colloids in this respect (Joubert et al., 2007). Rising IAP hampers venous return, causing even more fluids to stay in the abdomen. This results in still higher IAP and even more decreased venous return (Carlotti and Carvalho, 2009; Papavramidis et al., 2011). This is documented in dogs as well. Two dogs suffering from major trauma and one dog receiving a high-volume fluid therapy all suffered from high IAP (11, 9 and 10.3 mmHg respectively) (Fetner and Prittie, 2012). Similarly, when people receive a polytransfusion (more than 10 U packed red blood cells per 24 hours), such as in trauma cases, the risk of capillary leakage is significant (Malbrain et al., 2006). This has not been studied in dogs so far.

Other causes that trigger the inflammatory response cascade are several serious systemic infections. A case report has described a dog suffering from complicated babesiosis. The dog was resuscitated with crystalloids, aggravating the increase in IAP. Ultrasonography of the abdomen showed thickening of the intestinal loops, pointing to intestinal oedema. The IAH in this dog originates possibly from the inflammatory response to Babesia combined with excessive crystalloid oedema (Joubert et al., 2007).

Coagulopathy can trigger capillary leakage. In people, the cut-off values are the following: the platelet count is less than 55,000/mm\(^3\) or activated partial thromboplastin time is at least double the normal time or prothrombin time is less than 50% or the international standardized ratio is higher than 1.5 (Malbrain et al., 2006). Sometimes, coagulopathy is associated with sepsis, which in itself can entail capillary leakage. However, in a study two dogs suffer from sepsis, but only one of them has a significantly higher IAP (10.6 mmHg) (Fetner and Prittie, 2012).

Acidosis can lead to capillary leakage. This means that pH decreases below 7.2 in people (Malbrain et al., 2006). This condition occurs in two dogs with higher than normal IAP (+/- 10 mmHg) (Fetner and Prittie, 2012).

Oliguria is another cause of capillary leakage (Cheatham et al., 2007). Two dogs suffering from oliguria both had high IAP (10.6 and 10.3 mmHg) (Fetner and Prittie, 2012). Interestingly, oliguria is also a symptom of ACS, so it seems to be a chicken-and-egg problem.

Other causes of capillary leakage are hypothermia, bacteraemia (Malbrain et al., 2006), hypotension (Cheatham et al., 2007), shock (Kirkpatrick et al., 2013), thrombosis and central
nervous system disorders (Beck et al., 2001). All of these have not been investigated in dogs so far.
III. Pathophysiology of intra-abdominal hypertension

The pathophysiology of IAH and ACS is roughly based on a direct and an indirect mechanism (Barnes et al., 1985; Malbrain et al., 2006; Land et al., 2009). An increase in IAP influences the abdominal organs directly due to compression. Typically, the kidneys suffer greatly from their parenchyma being compressed, which contributes to renal failure. Indirectly, the organ systems are damaged because of severe changes in cardiovascular and hemodynamic parameters. Dogs were used as models in a lot of experiments to study IAH and ACS in humans. Clinical veterinary data are still relatively scarce, rendering it sometimes necessary to use facts from human medicine (Land et al., 2009).

The pathophysiology of IAH and ACS is strongly related to regional blood flow in the abdomen. High pressure on the blood vessels gives rise to severe cardiovascular changes (Barnes et al, 1985). Preload of the heart diminishes (due to decreased venous return), whereas afterload increases (due to pressure on the abdominal arteries). This may lead to left-sided heart insufficiency. Consequently, cardiac output lowers, vascular resistance rises and venous return decreases (due to compression of the vena cava and the portal vein). Blood pools in the abdominal veins and organs because of impaired venous return (Joubert et al., 2007). Intracranial pressure increases due to obstructed venous outflow from the brain. Mesenteric lymph flow diminishes or disappears even because of compression of the abdominal lymph vessels (Nielsen and Whelan, 2012). The blood flow towards the abdominal organs diminishes due to a reduction in perfusion pressure. Portal blood flow is already inhibited at an IAP of 10 mmHg and mesenteric and mucosal blood flow is impaired from 20 mmHg onwards (Joubert et al., 2007). All this is particularly pronounced in hypovolemic patients (Land et al., 2009). The lack of sufficient blood supply and outflow leads to intestinal wall damage, giving rise to oedema, capillary leakage and even bacterial translocation (Nielsen and Whelan, 2012). Visceral blood flow is further impaired, such as in the kidneys, where the high vascular resistance and the reduced blood flow are associated with a decreased glomerular filtration rate. In dogs, a decrease in glomerular filtration rate occurs at an IAP of 9.75-15 mmHg, which possibly leads to prerenal azotaemia and oliguria/anuria. This happens in dogs when IAP reaches above 18.75 mmHg (Joubert et al., 2007). In a certain way, this cardiovascular response is physiological.

As already mentioned, vascular resistance increases when IAP elevates. Vasoconstriction leads to increased vascular resistance and can occur in four ways. First, angiotensin is released as the kidney is compressed and less perfused than in a normal pressure range. A second potential mechanism consists of the atrial receptors signalling a smaller pressure build-up in the heart. Thirdly, vasoconstriction can be triggered by chemoreceptors detecting arterial hypoxemia at a pressure above 30 mmHg. Lastly, the intrinsic response of the vessel walls to the elevated IAP can lead to vasoconstriction of the abdominal vasculature. On the other hand, tissue pressure is increased because of organ compression, which should lead to an intrinsic response of vasodilatation. As a result, the role of the vessel wall is ambiguous in the context of high IAP (Barnes et al., 1985).

In human patients undergoing laparotomy, IAH occurs in association with renal failure (Sugrue et al., 1995). A study cohort was monitored postoperatively for IAH and increasing creatinine values. Postoperative serum creatinine increased to more than 130 µmol/L (upper reference limit in humans is around 110 µmol/L, thus a 1.2 fold increase) or serum creatinine elevated in a leap of more than 100 µmol/L the first 72 h after surgery. One third of the patients exhibited renal failure, while 68 % of this group also had a high IAP (a mean of 21
Renal failure and increased IAP occurred both within 48 h, leading to oliguria. In some patients, sepsis of the abdomen further complicated their condition, until even death occurred. It is not clear what happened first in these cases: IAH or renal failure. Some patients developed renal failure before, others during and others after the occurrence of IAH. In addition, there was no straightforward explanation as to why renal failure occurred in these surgical patients (Sugrue et al., 1995). It is plausible that the renal failure did not result from IAH in all patients with increased IAP.

An experimental study in dogs under general anaesthesia showed the interactions between IAP and cardiovascular mechanisms (Barnes et al., 1985). The animals’ abdomen was infused with a large volume of fluid, while pressure probes and flow probes in the major abdominal blood vessels monitored cardiovascular parameters. Interestingly, heart rate did not change when IAH was present. However, cardiac output and stroke volume decreased by 36 %, when IAP reached 40 mmHg. Consequently, blood flow in the important arteries diminished significantly: i.e. a decrease of 42 % in the celiac artery, of 61 % in the superior mesenteric artery, of 70 % in the renal artery and of 65 % in the femoral artery. Vascular resistance increased in the same arteries, by 26 %, 90 %, 136 % and 103 %, respectively, once IAP reached 40 mmHg. This experiment clearly documented that the renal blood flow was compromised the most (Barnes et al., 1985). Blood flow is impaired until the point of ischemia. When afterwards reperfusion occurs, tissues are damaged further, contributing to general organ dysfunction (Nielsen and Whelan, 2012). Furthermore, pressure rose in the veins, e.g. pressure in the femoral vein elevated from 4.4 mmHg to 46 mmHg, when IAP was 40 mmHg. Pressure in the femoral and brachial arteries stayed constant (125 mmHg-130 mmHg) at an IAP of less than 30 mmHg. However, when IAP rose further, pressure in the femoral artery dropped, whereas the brachial artery experienced a pressure peak. The reason for this phenomenon is not clear (Barnes et al., 1985).

Similar findings are reported in an experimental study on anesthetized dogs. IAP was elevated by air insufflation into the abdominal cavity until 29.4 mmHg was reached. During the rise in IAP, several parameters were monitored. Cardiac output decreased from 22.1 mmHg onwards. Mean arterial pressure increased at first and later decreased when 29.4 mmHg was attained. Central venous pressure increased all the time. This study included the influence of IAH on central nervous system parameters as well. Indeed, intracranial pressure elevated continuously, whereas cerebral perfusion pressure increased at first until 7.4 mmHg was reached and then decreased again beyond 22.1 mmHg and upwards. All values returned to normal after the abdomen was deflated again (Pinheiro Villaça and Mantovani, 2006). These data support the idea that a rise in IAP has a profound influence on hemodynamics in all systems of the body.

In addition, there is a mechanical interaction between the different body compartments of the peritoneal cavity, the thoracic cavity, the forebody and the hindquarters. When hydrostatic pressure elevates in the peritoneal cavity, this pressure is transferred to the blood vessels, the organs and the abdominal wall. The large blood vessels responsible for nutrition and drainage of the forequarters and hindquarters pass through the abdominal and thoracic cavities that are suffering from elevated pressure. Consequently, the increased pressure is transmitted to these vessels, which leads indirectly to changed hemodynamical parameters in the limbs and the head. For instance, high intrathoracic pressure compresses the cranial vena cava. The pressure difference between the arteries and veins decreases, potentially leading to a lessening of the carotid blood flow (Barnes et al., 1985). In other words, there is a decrease in the arteriovenous pressure gradient (Nielsen and Whelan, 2012). However, resistance is higher in the femoral vessels due to vasoconstriction, possibly as a vessel wall response to an increasing pressure in the femoral vein. The elevated femoral pressure is a
result of the pressure transfer from the abdomen. This means that all the smaller vessels in the hindlimb will experience higher pressures as well, which encourages the leakage of fluids out of the vessels, resulting in oedema of the hindlegs and potentially an increase in haematocrit. At an IAP of 70 mmHg or less, the abdominal aorta is compressed as well, but not to the extent that the femoral perfusion pressure is significantly decreased. When IAP reaches 80 mmHg or more, femoral perfusion pressure diminishes distinctly. This mechanism does not occur in the brachial arteries. In general, IAP below 20 mmHg does not have major adverse effects. Once the IAP crosses this threshold, the negative effects quickly reach grave dimensions (Barnes et al., 1985).

An important fact to mention is that IAP is transferred in part to the thoracic cavity. High IAP leads to bulging of the diaphragm, which in turn results in an increase in intrathoracic pressure. Around 20 % of the increase in IAP is passed on to the thoracic cavity, as appears from the aforementioned experimental study in anesthetised dogs after fluid infusion in the abdomen (Barnes et al., 1985). As pressure inside the thorax increased, this led to compression of the heart, lungs and vessels of the thorax. Pressure measurement in the left atrium showed a rise from 3 to 6 mmHg when IAP elevated from 0 to 40 mmHg. Within the same increase of IAP, pressure in the right atrium varied a bit more widely, e.g. from 0.8 to 6.3 mmHg. There was a generally smaller elevation in intracardiac pressure than in intrathoracic pressure, measured by an oesophageal probe. When IAP reached 40 mmHg, pressure in the oesophagus rose by approximately 7.5 mmHg. This pressure difference hints at a diminished expansion of the heart. The heart can no longer distend as well as before (diminished diastolic ventricle filling), which means that it cannot produce the same pressure and stroke volume either. This leads to a smaller cardiac output, increased central venous pressure and a diminished venous return (Barnes et al., 1985).

Furthermore, a pressure rise in the thoracic cavity can result in arterial hypoxemia (Barnes et al., 1985). The lungs become compressed, atelectatic and oedematous, losing part of their compliance in the process. Their volume and functional residual capacity is decreased, so functional dead space is elevated, creating hypoxia. This triggers the hypoxic reflex in the lungs. Ultimately, this leads to pulmonary hypertension, vessel collapse and reduction of the ventilated alveoli (Joubert et al., 2007). As a consequence, not enough O\textsubscript{2} reaches the arterial blood. Combined with decreased regional blood flow, the oxygen does not reach all organs and body parts. In this way, tissues are injured and release metabolic waste products, which leads to acidosis (Joubert et al., 2007).

Besides, a rise in IAP leads to the activation of the renin-angiotensin-aldosterone system and an increase in anti-diuretic hormone, plasma renin and adrenalin/noradrenalin levels. Anti-diuretic hormone lowers organ perfusion, which is already less than ideal due to organ compression and hemodynamic alterations. Microcirculation in the liver and the adrenal glands, in particular, are seriously compromised, which contributes to swift organ failure (Land et al., 2009).
IV. Symptoms of intra-abdominal hypertension

The symptoms of ACS in people are variable and related to the failure of the (abdominal) organs. In children, signs of ACS include respiratory distress, oliguria/anuria, hypotension, shock and metabolic acidosis. During laparotomy, the children's bowels were ischemic or necrotic (Beck et al., 2001). In adults, oliguria and a tense abdomen are observed (Nielsen and Whelan, 2012). Further symptoms in adults are similar to those in children (Beck et al., 2001).

There are only a few case reports about IAH and ACS in dogs available. A supposed example of ACS in a dog with babesiosis illustrates potential symptoms related to the aforementioned pathophysiological changes (Joubert et al, 2007). The animal was presented with abdominal distension, respiratory distress (dyspnoea), cardiac dysfunction, sudden oliguria and a rise in IAP of 17.3-18.4 mmHg. Radiography of the thorax showed pulmonary oedema. On ultrasound, the patient displayed oedema of the gastro-intestinal organs, hepatomegaly and splenomegaly. Blood tests highlighted azotaemia, hinting at failing renal function. All signs were similar to those of ACS described in children (Beck et al., 2001). The dog had also increased liver enzymes; this could be due to Babesia. When there is liver failure in humans, though, the risk of IAH increases (Malbrain et al., 2006). Obviously, the dog could also have suffered from myocardial insufficiency due to babesiosis, instead of from ACS. Babesiosis is in certain ways similar to sepsis and may lead to similar cardiac problems. This may explain the diminished renal function and the pulmonary and intestinal oedema. No conclusive evidence for a heart problem was possible in this patient, because no cardiac ultrasound was performed. However, the intestinal oedema and venous congestion of the abdominal organs, whether due to myocardial insufficiency or not, may have led to IAH (Joubert et al., 2007).
V. Measurement of intra-abdominal pressure

Concerning IAP measurement, there are a number of methods that are used for human and veterinary patients in clinical conditions. The techniques that will be discussed, have been adapted from human medicine in order to measure IAP in small animals.

The direct way to measure IAP can be performed by inserting a catheter attached to a saline manometer or a pressure transducer into the abdominal cavity. Otherwise, abdominal drains have been used as well for this procedure. Intermittent and continuous measurements are possible. This technique is often performed during laparoscopy in humans and animals. For critical hospitalized patients, however, it is not advisable to use direct IAP measurement. The method is invasive, expensive and carries risks, such as infection (Smith and Sande, 2012).

The classic way to measure IAP in a non-invasive, indirect way is the intravesical method, developed in human medicine. The original, intermittent measuring technique was elaborated by Kron in 1984. The urinary collection system is open and, thus, not sterile, entailing a considerable risk of infection. The Kron technique was revised in 1998 by Cheatham and Safcsak. Since then, the urinary system remains connected to the urinary catheter, thus lowering risk of infection. The pressure measurement apparatus, however, is serially linked by two three-way stopcocks and is not attached to the urinary collection system by a three-way stopcock. This means that the procedure of pressure measurement remains inconvenient and slow. To improve the ease of use, the measurement technique was revised again by Malbrain, connecting all measurement apparatus via 3 serial three-way stopcocks to the urinary collection system. The procedure is still laborious and only applicable to intermittent IAP measurement (Smith and Sande, 2012).

In order to measure IAP continuously, the three-way Foley catheter technique was developed. The three-way catheter is attached to an irrigation port that links both the saline and the pressure transducer to the animal. The other two exits of the catheter are connected to the urinary collection system and the balloon inflation device. Measurement of IAP can be performed without interruption and without interfering with urinary outflow, thus rendering the whole procedure more efficient. However, it is not often used in animals due to availability of the material and financial considerations. Another way to measure IAP is the U-tube technique by Harrahill. In this case, the patient’s urine column itself is used to gauge IAP. The urinary catheter tube must drop below the level of the bladder in a U-shape and then rise against a measuring stick. The distance from the meniscus of the urine column to the pubic symphysis is the measured IAP in cm H₂O. This method dispenses with stopcocks and pressure transducers, making it a simple, inexpensive and fast procedure. However, the technique is prone to inaccuracy and needs to be elaborated further to ensure efficient usage. Lastly, the Foleymanometer technique can be used to measure IAP. A 50 mL container is attached between the Foley catheter and the urinary collection system. The tube between the catheter and the container serves as the manometer and has markings to measure IAP. Urine collects in the container until the container is elevated. Thus, a urine column is created to measure IAP as the distance from the urine level in the manometer tube to the pubic symphysis. This procedure is fast and uses a standardized volume instilled in the bladder, i.e. 50 mL, in contrast to the U-tube technique (Smith and Sande, 2012).

In small animals, the intermittent intravesical measuring procedure is used most of the time in experimental and clinical conditions. The basic technique was developed in a clinical study in dogs (Conzemius et al., 1995). First, a Foley urinary catheter is placed, to which a sterile urinary collection system with two 3-way stopcocks is attached. Then, a water manometer is
placed at the level of the first stopcock, situated at the height of the patient’s pubic symphysis (the zero level). A 250 mL bag of 0.9 % NaCl and a 20 mL syringe are connected to the second stopcock. Before each measurement, the bladder must be emptied. Then, a volume of saline, usually 0.5 to 1 mL/kg, is infused into the bladder and the manometer must be filled with saline. IAP is registered as the difference between the zero level and the level of the saline (Nielsen and Whelan, 2012). This method has also been investigated in cats (Rader and Johnson, 2010). Instead of a water manometer, a special monitoring system can be directly connected to the Foley catheter without three-way stopcocks, to measure IAP via a pressure transducer (Fetner and Prittie, 2012). In any case, body positioning, reference point and other factors influencing correct intravesical IAP measurement are not yet standardized in small animals and need further research (Smith and Sande, 2012).

Another approach to measure IAP indirectly is the intragastric method, originating in human medicine. This can be useful when the patient has a condition affecting the bladder or the pelvic region, such as fractures or hematomas (De Waele et al., 2007). Then, it is preferable to measure IAP via the stomach, because pressure measurement via the bladder would overestimate IAP. The first option is the Collee intragastric technique originating in 1993. A nasogastric tube is linked to a 60 mL syringe and a water manometer or a pressure transducer by a three-way stopcock. The stomach is drained first and then 50 mL of sterilized water is infused via the tube. The IAP measuring procedure is similar to the intravesical method. The Collee technique is easy, inexpensive and does not interfere with urine outflow, as is a disadvantage of some intravesical techniques. However, this method cannot be used in patients with gastrointestinal conditions or needing oral medications and tube feeding. Besides, stomach contractions can interfere with correct measurements. There is also still debate about the amount of fluid and gas that should be removed before infusing the sterile solution. Lastly, this method may pose a risk of aspiration pneumonia, as is generally the case with nasogastric tubes (Smith and Sande, 2012).

The Collee technique was altered into the balloon-tipped catheter technique in 1994 (Sugrue et al., 1994). This special gastric tonometry catheter can serve as a pressure transducer and registers $P_{CO_2}$ as well. In addition to IAP, the catheter gives information about the perfusion of the stomach and, indirectly, about the effect of IAH on the abdominal organs. The advantages of this technique are that the contents of the stomach do not influence the measurements and there is no need to instil fluid into the stomach. When IAP measurement need to be continuous, the oesophageal balloon catheter technique by Malbrain can be used. The catheter is attached to a pressure transducer by a three-way stopcock. All air must be removed from the balloon by a syringe, after which 1 mL of air must be put back in and IAP can be measured. Measurements can be performed without interruption during 2 hours, because the air in the balloon remains present during this time. A really continuous intragastric technique with special air-filled balloon catheters connected to automated IAP measurement equipment can be used for monitoring IAP during a longer period. The apparatus is very expensive, but can be combined with tube feeding (Smith and Sande, 2012).

With regard to the intragastric IAP measurement, there are very few studies on animals in general. Experiments in dogs have shown promising results. An experiment in puppies has shown that the intragastric method correlates well with direct IAP measurement (Engum et al., 2002). However, clinical studies need to be conducted to gain more information about this approach to IAP measurement in canine patients (Smith and Sande, 2012).

A third way to approach IAP measurement is the transrectal method, used in humans and animals. An open rectal catheter can be used in combination with a continuous fluid infusion of 1 mL/min. Otherwise, a closed system with a fluid-filled balloon catheter can serve to
measure IAP. However, transrectal IAP measurement is not easy to perform and is contraindicated in patients with gastrointestinal conditions, such as bleeding or diarrhoea. The catheter can cause damage to the rectum and the anus. Besides, overestimation of IAP can occur when there is still faecal mass present in the rectum (Smith and Sande, 2012).

A last approach to IAP measurement is the placement of a central venous catheter in the caudal vena cava via the femoral vein. The catheter is attached to a three-way stopcock, in turn linked to a pressure transducer and a 1 L bag of saline solution. The measurements done in this way are not always reliable, because they are heavily dependent on vessel muscle tone, blood volume and filling pressures of the heart. Furthermore, a central venous catheter entails the risk of infection and thromboembolism. In contrast, this continuous method does not pose problems with urine output, oral drug administrations and fluid aspiration. There is, however, not enough evidence of the technique’s reliability and efficiency in patients (Smith and Sande, 2012).
VI. Discussion of intra-abdominal hypertension

The issue of IAH and ACS is still not generally recognized as a potential problem in veterinary medicine. Therefore, canine data on IAH and ACS are scarce and need to be supplemented by results from animal experiments in human medicine. Veterinary literature does not provide sufficient material so far to establish the existence of ACS in dogs. Further research is warranted to find evidence of the (lack) of ACS in dogs. Some veterinary studies discuss the applicability of human IAP measurement methods in small animals. In this respect, it remains to be seen whether there is a clinical need for IAP measurement in dogs.

IAP is not often monitored in veterinary patients due to limited awareness of the problem of IAH, supposed absence of clinical significance and increased financial commitment (Smith and Sande, 2012). In veterinary medicine, the intravesical method has been investigated in dogs and cats with average success. However, as already mentioned, there is no consensus as to the exact protocol of IAP measurement (e.g. when measured, how often measured, in what body position measured…) (Nielsen and Whelan, 2012). The indications for measuring IAP are most of the time related to abdominal pathologies. Its use in dogs may be helpful in the case of an acute abdomen, severe ascites, abdominal trauma, ileus, abdominal bleeding, severe pancreatitis… Ideally, IAP should be monitored every 4 hours in critically ill animals, when using an intermittent technique (Smith and Sande, 2012). Indeed, individual IAP values are important to monitor critical patients, but trends in their IAP values can be more relevant for assessing their clinical condition in general (Way and Monnet, 2014). The optimal parameters to measure IAP correctly have yet to be determined in dogs (Smith and Sande, 2012).

The intravesical technique is the most accurate and consistent way to measure IAP non-invasively and has been accepted in human medicine as the gold standard to monitor critical patients and to decide on surgical intervention. Certain factors can alter measurement values. It is important to take these influences into account, because they have repercussion on the subsequent management of the patient (Smith and Sande, 2012). Therefore, the World Society of the Abdominal Compartment Syndrome (WSACS) has formulated guidelines for standardized IAP measurement in people via the intravesical method (Malbrain et al., 2006; Cheatham et al., 2007).

With regard to the reference point to be used in IAP measurements, the pubic symphysis has been used as the zero level before the WSACS guidelines were introduced. Due to the patients’ different physique, the exact place of the pubic symphysis remains subjectively determined and, therefore, dependent on the person performing the measurements. Thus, IAP measurements are not accurate enough with the pubic symphysis as the reference point (Smith and Sande, 2012). The WSACS recommends using the mid-axillary line of the iliac crest as the ideal reference point in people (Malbrain et al., 2006). It is not yet clear if the mid-axillary line as the point of reference is also applicable to dogs (Smith and Sande, 2012). Several reference points have been used in small animal studies, such as the pubic symphysis (Rader and Johnson, 2010), the middle of the pelvis (Joubert et al., 2007) and the level of the vulva (Conzemius et al., 1995).

As for body positioning, it has been advised that supine position is applied, as it gives the most accurate IAP measurement results in people (Malbrain et al., 2006). It has been proven that head of bed elevation increases IAP in people, thus overestimation of the measurement values will follow (Kirkpatrick et al., 2013). Supine positioning has not been studied in dogs, as it is not a natural position for an animal in hospital conditions. Lateral recumbency is a
more comfortable position for dogs and has been used in some studies (Conzemius et al., 1995; Joubert et al., 2007). In cats, sternal recumbency has been compared with lateral recumbency with regard to influence on the accuracy of IAP measurement, but no significant differences were documented (Rader and Johnson, 2010).

Concerning the volume of saline to be infused in the bladder, a lot of studies have been conducted in human medicine. If too much saline is instilled, the bladder will react by detrusor muscle contractions, resulting in an overestimation of IAP (Smith and Sande, 2012). The WSACS has recommended using a maximum of 25 mL of saline infusion in people (Malbrain et al, 2006). In veterinary literature, debate is still going on about the volume of fluid to be infused in dogs. It has been advised to instil 0.5-1 mL/kg in dogs (Drellich, 2000). This recommendation was applied to measure IAP in a case report of a dog with babesiosis (Joubert et al., 2007). A clinical study in dogs used 1 mL/kg of fluid, again within the boundaries of the advice by Drellich (Fetner and Prittie, 2012). In another study, no fluid was infused at all (Conzemius et al., 1995). In cats, a marked difference was found between IAP measurement values using 0.5 mL/kg and 1 mL/kg of fluid. The difference was not clinically relevant, although it is recommended to use 0.5 mL/kg of solution in cats (Rader and Johnson, 2010).

A study in 15 research dogs tried to determine the ideal volume of saline to be instilled in the bladder, in order to obtain correct measurements. The IAP was measured before and after ovariohysterectomy in one group and before and during laparoscopy in another group. In the laparoscopy group, intra-operative IAP measurements were performed in a direct way via a pressure transducer connected to the abdominal cavity. All measurements were executed with 4 different volumes of saline to be instilled, on both moments of recording the IAP. The volume of saline influenced IAP measurement significantly in both groups of dogs. The best correlation between IAP measurement and laparoscopic insufflator pressure was obtained when a volume of 1 mL/kg was used. In other words, when this volume was used, the indirect IAP measurement via the bladder matched the direct IAP measurement via laparoscopy. Thus, 1 mL/kg of saline infused in the bladder is ideal to measure IAP in dogs reliably in experimental laparoscopic conditions. However, some remarks should be made regarding the laparoscopy experiment. The dogs in this experiment were very similar with regard to weight and size. A volume of 1 mL/kg was ideal for this sample population, but not necessarily so for other groups: there is more variation in canine patients than in humans. (Way and Monnet, 2014).

Further, it is important to decide the clinical conditions in which the animal will be subjected to IAP measurements, in order to obtain reliable results. In conscious dogs, muscle contractions due to a variety of reasons, such as pain and anxiety, influence the accuracy of the IAP measurements significantly. Therefore, precautions are necessary to ensure that the animals are not in pain and are as relaxed as possible under the circumstances (e.g. analgesics, light sedation) (Smith and Sande, 2012). Indeed, it has been proven in cats that IAP measurement differed significantly between conscious and sedated animals. In this case, the conscious cats were struggling so much during measurements that IAP values were not reliable (Rader and Johnson, 2010).

There are a number of works trying out different protocols to measure IAP in small animals accurately. A clinical study in healthy cats, in right lateral and sternal recumbency, used a specific feline urethral catheter without balloon, instead of a Foley catheter. The catheter was connected to a urine bag and a water manometer. The bladder was emptied and then filled with 0.5 mL/kg of saline solution. The zero point was at the level of the pubic symphysis. The values thus measured were consistent and reproducible in hospital conditions (Bosch et al., 2012). In another case, a female 9-year-old Bull Terrier with babesiosis and supposed ACS
was subjected to IAP measurement with the intravesical technique. A urinary catheter was placed, the bladder was emptied and filled with 50 mL of saline. IAP was measured via a water column or a pressure transducer. The zero point was situated halfway through the pelvis of the animal in lateral recumbency. In this dog, a range from 17.63-18.75 mmHg was measured (Joubert et al., 2007).

The effect of surgery and anaesthesia on the accuracy of the measured IAP values was studied in 41 female dogs before and after mastectomy and/or ovariohysterectomy. The intravesical technique was used again. The animals had a Foley catheter placed and were then allowed to rest for 48 hours. To determine normal IAP values first, measurements were performed in 26 healthy anesthetized female dogs without the risk of IAH. This baseline value was 3.6 mmHg. The dogs needing surgery were subjected to IAP measurement before and after surgery, when they were still anesthetized, and during recovery, when they were already awake. Some IAP measurements were obtained in conscious dogs, so when the animals were tense and contracted their abdominal muscles, the recorded values were not reliable. The appropriate volume of saline was calculated per kg body weight (0.5-1 mL/kg). The dogs were placed in lateral recumbency, so the abdominal organs did not put pressure on the bladder, leading to accurate measurements (Land et al., 2009). In contrast, a study in cats showed that measurement in sternal recumbency results in the most reliable values (Rader and Johnson, 2010). IAP was then measured for 48 hours. After mastectomy, IAP elevated in all dogs of this group. In 75 % of this group, IAP rose further when the dogs were awake 4 hours after the surgery. The animals’ mood had a significant influence on the measurements in this stage. The most accurate and reproducible values were obtained with animals that were awake and relaxed during the measurement (Land et al., 2009). Indeed, tension of the abdominal muscles due to stress and struggling can diminish abdominal wall compliance and thus increase IAP (Rader and Johnson, 2010). Twenty hours after surgery, IAP decreased again in 79 % of the dogs, and even further after 48 hours, until IAP was only slightly above the pre-operative level. This could be due to the dogs’ mood again: they were by then possibly more relaxed than the first hours after surgery. In the dogs who had mastectomy combined with ovariohysterectomy, post-operative IAP was higher than in the dogs that only had ovariohysterectomy. Probably, this was due to the compressive effect of wound closure under tension (in general, the mastectomies led to substantial skin loss). There was no post-operative IAP difference between the dogs that underwent mastectomy and those that underwent mastectomy and ovariohysterectomy. Thus, laparotomy did not influence IAP and IAP measurement (Land et al., 2009).

A potential disadvantage of intravesical IAP measurement is an iatrogenic urinary tract infection. In catheterized dogs, the risk of infection becomes significant (50 %) after four days. Even when antibiotics are used in these dogs, bacteriuria can develop after 12 days or more. Haematuria can be the only clinical symptom of a UTI. Nine percent of the dogs in a study on IAP after mastectomy and/or ovariohysterectomy developed haematuria immediately after placement of the Foley catheter, fifty-eight percent after 24 hours and all dogs were affected after 48 hours. Probably this was due to an infection, but no other symptoms were observed (e.g. strangury, pollakisuria) (Land et al., 2009).

Measuring abdominal circumference is not reliable to estimate IAP. In humans (especially children), it is used to diagnose IAH. In dogs, measuring abdominal circumference can be used because it is an easy way to obtain a general idea of the patient’s IAP. It shows clearly how much abdominal distension is present. However, diameter and pressure do not relate to each other. A change in diameter shows a change in content volume in the abdomen. This may be associated with an altered IAP, but there is no strict evidence available using this method (Joubert et al., 2007).
Lastly, a useful method for triage and monitoring of critical patients and tracking of abdominal pathologies in general is discussed. Abdominal FAST ultrasonography (Focused Assessment with Sonography for Trauma) can contribute indirectly to the diagnosis of high IAP in dogs, such as in the case of hemoperitoneum, uro-abdomen, pneumoperitoneum and peritonitis. In this respect, FAST is used to screen for free fluid in the abdominal cavity and the retroperitoneal space. This technique is very specific and sensitive, i.e. comparable to CT imaging. The advantages of FAST are numerous, such as short duration and non-invasiveness. In this way, it should be less stressful for the patient and it can be combined with other diagnostic procedures (e.g. abdominocentesis) (Lisciandro et al., 2009). However, CT remains the gold standard for detection of abdominal pathologies (Kirkpatrick, 2007). It is not ideal for critical patients, though, because sedation or anaesthesia is needed. When FAST is used, an abdominal fluid scoring (AFS) system should be applied. The application of AFS in humans has been shown to improve the prognosis due to a quick diagnosis. A veterinary AFS is not yet widely established. In dogs, the AFS is correlated with the degree of anaemia, thus expressing a rough quantification of the degree of abdominal pathology (e.g. the degree of (occult) bleeding) (Lisciandro et al., 2009). The AFS ranges from 0 to 4, dependent on the number of FAST-positive sites. The location of these FAST-positive sites is important to detect the source of the free fluid (e.g. haemorrhage originates most of the time in the spleen or liver) and to decide further diagnostic tests. However, FAST is not reliable enough in dogs as a screening method for injuries of the abdominal organs, so other diagnostics are needed, such as abdominocentesis and fluid characterisation. When there is very little free fluid or it is situated in a difficult location, diagnostic peritoneal lavage is necessary (Lisciandro et al., 2011).
Conclusion

In veterinary literature, only very few case reports, reviews or other scientific writings present dogs suffering from (supposed) ACS. The dogs generally show symptoms which are very alike to those in humans with ACS. In addition, the animals showing clinical symptoms have an elevated IAP. Sometimes, IAP is not even measured, because awareness of IAH in dogs is not widespread. There are probably indications to measure IAP that are not yet known to veterinarians (Way and Monnet, 2014). Possibly, ACS may be underdiagnosed as a consequence. Furthermore, ACS is not as straightforward to define in dogs as it is in humans. In consequence, it is still debatable whether ACS even occurs in dogs. Whether high IAP has a clinical significance has not been proven so far. It has not been shown in dogs suffering from IAH that organ pathologies really originate as the consequence of the elevated IAP. Thus, the need for diagnosis itself could be questioned, as it is not yet clear whether ACS is a real problem in dogs. This might be attributed in part to anatomical differences (e.g. dogs have a looser skin than humans) and normal walking posture (e.g. walking upright, people put a lot of weight from the upper body on the abdomen).
References


