BLOOD PRESSURE MEASUREMENTS BY USE OF DOPPLER EFFECT IN INTERNAL DISEASES OF SMALL ANIMALS

THESIS PRESENTED
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قـرار لجنة الحكـم و المناقشـــــه

قررت لجنة الحكم و المناقشه ترشيح السيد ط.ب / جمال السيد محمد أبو العينيـــــن المدرس المساعد بقسم طب الحيوان و الطب الشرعى للحصول على درجة دكتور الفلسفـه فى العلوم الطبيه البيطريه "تخصص الطب العام و العلاجى"

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INTRODUCTION

Arterial blood pressure must be at an adequate level in order to ensure adequate blood flow to all parts of the body. If it falls too low, symptoms may occur owing to ischemia of various organs. Profound decrease in blood pressure and blood flow culminate in a form of circulatory collapse referred as"shock", which, unless quickly reveresed, progresses to death. On the other hand, if the blood pressure rises too high, it places damaging strain on the cardiovascular system with acceleration of arteriosclerosis, cardiac hypertrophy, and risks of congestive heart failure and cerebral hemorrhage.

Blood pressure determination has not become a routine procedure in small animal practice, though its value is generally recognised. Chief among the reasons for this is the lake of convenient and accurate noninvasive methods. Recently, a technique based on the ultrasonic Doppler principle has come into use. This method, which is a modification of the auscultatory technique, is extremly sensitive and uniquely suited to blood pressure measurement in difficult situations without vascular invasion or pain.

Although hypertension has been reported mainly in conjunction with renal disease, it undoubtedly exists in dogs, as in human beings, associated with a variety of other conditions.

This work was completely done in the Department of Internal Medicine, Faculty of Veterinary Medicine, Ludwig Maximillian University, Munich, West Germany, to survey blood pressure in relation to arterial blood pH and oxygen tension before treatment on dogs hospitalised because of a wide variety of clinical disorders using Doppler technique. These clinical disorders include: anemia, diabetes mellutus, heart diseases, acute pancreatitis, gastroenteritis including paravovirus infection, respiratory diseases including canine distemper, uremia, and leukemia.

REVIEW OF LITERATURE

BLOOD PRESSURE AND ANEMIA:

In anemia, the mean and diastolic areterial pressures fall only moderately even at low hemoglobin levels (Brannon et al., 1945; Graettinger et al., 1963, Duke et al., 1964).

Hatcher et al.,(1954) found the decrease in total peripheral resistance which accompanies the rise in cardiac output might represent a homeostatic mechanism which maintained a normal nlood pressure during the period of elevation in cardiac output. Also a normal systolic pressure level was reported by Bishop et al.(1955) and Wheatherall et al.,(1981).

Anemia is the most common disease that increase the cardiac output at rest. The studies by Richardson and Guyton (1959), and Clarke et al.,(1978) have supported a role for the lowered viscosity of blood in high cardiac output of anemia.

Conditions that lower peripheral vascular resistance are among the most important factors augementing the venous return and therefore elevating the cardiac output. The finding of Liang and Huckabee (1973) that

tissue hypoxia associated with anemia can lead to an autonomic reflex response resulting in reduced arteriolar resistance is of considerable interest.

Grossman and Braunwald (1980) reported that the consequences of anemia depend to an important extent on its rate of development. When it occurs rapidly, as in hemorrhage, blood volume is not maintained, and the picture of hypovolemic shock predominantes. If anemia develops more slowly, so that blood volume is maintained, cardiac output rises predominantly as a result of tachycardia with a little change in strok volume. He also added that in anemia a local tissue hypoxia, lactic acidemia, and accumulation of bradykinin and vasodilator metabolites such as adinosin may lower the peripheral vascular resistance.

BLOOD PRESSURE AND DIABETES MELLITUS:

The relation between diabetic nephropathy and hypertension was discussed by many workers. In young diabetic patients, White (1956) observed that the clinical evidence of diabetic nephropathy manifested by protienuria was rarely observed until diabetes had been present

for 10 years. The hypertension almost always accompanies the syndrome of diabetic nephropathy caused by intercapillary glomerusclerosis and may reflect both advancing renal insufficiency with inability to handle volume loads, extensive structural narrowing of the peripheral vasculature that is the hallmark of longstanding diabetes (Gellman et al., 1959; Kuhlman and Mehnert, 1969; Christlieb, 1980; Kaplan, 1980; Parving, 1981; Mogensen 1982).

Bell (1960) reported hyalinization of renal afferent arterioles in diabetic patients studied postmortem. He concluded that it was produced by the diabetic state and was largely responsible for the development of hypertension.

Factors which may have contributed to the exaggerated pressor responses in mildly to moderatelly hypertensive diabetic patients include hypertropic changes of the resistance vessels (Folkow, 1971), normal catecholamines and plasma renin activity levels in the presence of a hypertensive cardiovascular system (Chinn and Dusterdieck, 1972; Philipp et al., 1978), this mechanism may complement the excess body sodium as a concomitant

factor favoring the development or maintenance of hypertension in diabetes mellitus (Weidmann et al., 1979).

Christlieb (1973) reported that hypertension occurs with greater frequency in diabetic than nondiabetic patients.

Diabetics without perceptible renal disease were reported to have normal blood pressure (Oakly et al., 1974; Keen et al., 1975). It is of interest that patients with uncomplicated diabetes were reported to have a normal left ventricular function, and a normal cardiovascular sensitivity to norepinephrine or angiotensin (Christlieb, 1976; Seneviratne, 1977). Piccoli et al., (1979), and Williams and Braunwald (1980) reported that the diabetics may have a normal or a high blood pressure level.

BLOOD PRESSURE AND HEART DISEASE:

The presence of a hyperkinetic heart and an abnormally elevated cardiac output may be responsible for the elevation of arterial pressure in some patients with hypertension (Gorlin et al., 1959; Eich et el., 1962 ;

Gorlin, 1962; Julius and Conway, 1968), although this is generally associated with early borderline hypertension, an elevated cardiac output has been observed in some patients with left ventricular hypertrophy, fixed and severe hypertension as well (Ibrahim et al., 1975). He also reported that the close association of augmented cardiac activity and increased arterial pressure suggested that the heart might be involved in the genesis and maintenance of systemic hypertension in these patients.

Since hypertension is the most common cause of left atrial and ventricular hypertrophy (Tobian et al., 1960; Tarazi et al., 1966; Benznak et al., 1969; Kannel et al., 1969), the clinical diagnosis of hypertensive heart disease is made by the combined presence of hypertension and left ventricular hypertrophy (LVH) when other causes of LVH are resonably excluded (Dustan,1986).

It has been pointed out that valvular lesions that overload the left ventricle (aortic stenosis, aortic regurgitation, and mitral regurgitation) can over time, produce severe left ventricular hypertrophy, which eventually is associated with left ventricular dysfunction

(Ross and Braunwald, 1968; Schwarz et al., 1981).

Many investigators had been impressed that arrythmia, either a severe tachycardia or a bradycardia with or without acute myocardial infarction, may cause a decrease in cardiac output, and hypotension (Ruskin et al., 1970; Linden, 1973; Esente et al., 1983; Ross, 1983 ; Fergusen and Abboud, 1986).

Mason et al.,(1971) claimed that in cardiac hypertrophy without failure, the absolute increase in total muscle mass, and the elevated ventricular end-diastolic pressure maintain cardiac output at rest, while in hyper trophy with failure, despite the striking increase in ventricular end-diastolic pressure and muscle mass, cardiac output falls as a result of the markedly reduced level of contractile state. In chronic overload ., such as that due to valvular heart disease or a large left - to- right shunt, adaptations occur primarily through the development of concentric or eccentric hypertrophy, which compensates for the overload and prevent overall cardiac failure. In most of these conditions, heart failure does not occur until myocardial damage supervenes due to long-standing hypertrophy (Ross, 1974; Sasayama et al., 1976).

Braunwald (1980) reported that in mild or moderately heart failure, the stroke volume is normal at rest, in severe heart failure, it reduced. With very severe failure, systolic blood pressure may be reduced, particularly if the cardiac output drops acutely. Also Schlant and Sonnenblick (1986) supported the view that in mild heart failure, the compensatory mechanisms which include: (1) reflex increase in autonomic sympathetic excitation to the heart and to the most of arteries and viens, (2) renal retension of sodium and water, and (3) myocardial hypertrophy with or without cardiac chamber dilatation, are often able to restore to normal or near normal the arterial blood pressure, and the cardiac output at rest or even during mild exercise.

Adaptation of the heart to an abnormal burden depends not only on the baseline state of myocardial function when the addition of burden is imposed and on the magnitude of the burden, but also on the rate at which the new burden is added. Gradual imposition of the hemodynamic load allows the appopriate compensatory mechanisms to come into play. Thus, it is well established that cardiac compensation may be maintained in the presence of severe aortic regurgitation if this lesions

develops gradually, allowing marked hypertrophy to take place. On the other hand, sudden regurgitation of comparable severity, as in acute infective endocarditis or trauma, will often produce left ventricular failure (Grossman and Braunwald, 1980).

Sobel and Roberts (1980) mentioned that the cardiac causes of sudden hupotension related to a sudden reduction in ventricular rate, as occurs with atrioventricular block, or in strok volume, as may occur in hypovolemia, or to massive myocardial infarction. In this connection the report of Wynne and Braunwald (1980) that in congestive cardiomyopathy, the systolic blood pressure is usually normal or low is of considerable interest.

BLOOD PRESSURE AND ACUTE PANCREATITIS:

Shock complicating the course of acute pancreatitis is a serious and ominous prognostic sign. In Siler and Wulsin study (1951), fifteen patients had the classic features of acute circulatory failure. A decrease in total blood volume has previously been regarded as a primary mechanism accounting for the reduction in arte-

rial blood pressure and shock complicating pancreatitis (Keith and Watman, 1955; Dos Reis, 1963; Facey et al., 1966; Geokas et al., 1972).

Thal et al.,(1963) do not regard plasma volume reduction by peripancreatic loses as the sole explanation of the shock state. They suggested the diffuse vascular injury caused by the systemic actions of pancreatic enzymes will lead to capillary leakage and generalized vasodilatation. Moreover, in other studies, it has been shown that the pancreatic exudate contains hypotensive agents (Amudsen et al., 1968), and histamine releasing agents (Ofstad et al., 1969).

During acute hemorrhagic pancreatitis, hypotension is a prominant feature and may be due to plasma loss,and the presence of plasma kinins (Katz et al., 1964; Nuget and Atendido, 1966; Andersonet al., 1967 a; Rogers,1983).

It has been pointed out that many of the local and systemic features of acute pancreatitis were due to the releasing of pancreatic factors, including enzymes, kallikrein, bradykinin, and to myocardial depressant factors. All of these agents play a part in the develo-

pment of hypovolemic shock, and are capable to produce severe hypotension (Beck et al., 1965; Creutzfeldt and Schmidt, 1970; Bocjman et al., 1973; Colman, 1974; Hardy and Stevens, 1975).

RELATIONSHIP BETWEEN PARVOVIRUS, NONPARVOVIRUS GASTROENTRITIS AND BLOOD PRESSURE:

Bacterial endotoxins are constantly entering the body via the intestinal circulation and are ordinarily inactivated by the reticuloendothelial system (RES) which becomes depressed during the course of all types of shock. Thus as the RES becomes depressed, endotoxins rise sufficiently to produce peripheral circulatory failure, and thus an intensification of the hemodynanic changes characteristic of shock (Fine et al., 1959,1965). In this connection, the findings of Priano et al. (1971), and Erasmer (1972) that gram-negative or positive septicaemia or endotoxins absorption will cause low cardiac output and hypotension is of considerable interest.

McDonell (1974) claimed that in hypovolemic shock which is the most common shock syndrome seen in small animal practice, central venous pressure, cardiac outrut

and blood pressure fall. Ross (1975) reported that among the numerous unsults which are capable of initiating circulatory shock in the dog and cat are sepsis due to conditions such as peritonitis, and fluid loss (hypovolemic) due to severe burns, hemorrhage, and severe diarrhea, or vomiting. Smith and Hamlin (1977), and Sleisenger (1981) like other authors supported the view that in shock, with the exception of severe primary heart disease, the main reason for hypotension is a significant reduction in the effective intravascular volume.

Canine parvovirus in now know to be a cause of both of enteritis and myocarditis (Appel et al., 1979; Hayes et al., 1979), and death due to parvoviral enteritis result from dehydration, endotoxic shock (Green, 1984).

THE RELATION BETWEEN ARTERIAL BLOOD PRESSURE AND ARTERIAL PH, OXYGEN TENSION

The myocardium of intact subject is fairly resistant to acidosis, alkalosis, and the depression of cardiac contractility, or depression of responsiveness to catecholamines is minimal until the pH decreases to 7.0,

7.1 (Gertler et al., 1946; Terroux et al., 1947; Brown and Miller, 1952, Boniface and Brown, 1953; Clowes et al., 1961; Downing et al., 1965; Anderson et al.,1967b).

In intact animals, moderate metabolic alkalosis, and respiratory alkalosis or acidosis are generally associated with increased cardiac output, slight decrease in blood pressure, and peripheral vascular resistance, while moderate metabolic acidosis is usually associated with decreased cardiac output, and a slight decrease in blood pressure (Terroux et al., 1947; Boniface and Brown, 1953; Goodyer et al., 1961; Carson et al., 1965; Kittle et al., 1965; Mitchell et al., 1972 ; Cullen and Eger, 1974). Haskins (1977 a) reported that the magnitude of these changes is generally mild within a pH of 7.2 or 7.6.

In the intact animal, the activation of sympathetic nervous system in response to acidosis causes increased cardiac output and counteracts much of the direct depressant effect of acidosis (Brown and Miller, 1952; Nahas and Cavert, 1957; Thrower et al., 1961; Cullen and Eger, 1974).

With regard to hypoxia, it has been pointed out that in systemic hypoxia, the cardiovascular responses include an elevation of blood pressure, tachycardia, increased cardiac output, and increased ventricular contractility. These responses are the result both of hypoxic stimulation of the peripheral chemoreceptors, and of direct hypoxic stimulation of CNS (Gorlin and Lewis, 1954; Nell, 1956; Gross et al., 1959; Downing and Siegel, 1962; Downing et al., 1962; Downing et al., 1963; Smith and Hamlin, 1977).

Stone et al.,(1958), Clowes et al.,(1961), and Richardson et al. (1961) found that the direct effect of acidosis on local vasculature is generally vasodilation, although in the intact animal the increasing in the sympathetic tone may cause vasoconstriction. In the denervated heart, acidosis generally depresses cardiac contractility and cardiac output, whereas alkalosis generally increases cardiac contractility and cardiac output (Thrower et al., 1961; Kerpel, 1967; Cingolani et al., 1969; Cingolani et al., 1970).

Goldblatt and Braunwald (1962) mentioned that in dogs, the direct effect of hypoxia is to produce net

arterial dilatation, and to depress myocardium contractility but this response is opposed by arteriolar constriction, and augmentation of myocardium contractility mediated through the chemoreceptor refles arc. This report was supported by Abboud (1986) who also added that despite the compensatory mechanisms that permit delivery of oxygen to the vital organs during hypoxia, myocardial contractility may be impaired as arterial Po_2 declines and the severity of the decline in the arterial Po_2 reflects the extent of myocardial infarction.

RELATIONSHIP BETWEEN RESPIRATORY DISEASES, CANINE DISTEMPER (with respiratory disturbances) AND BLOOD PRESSURE.

Hilton (1933), and Abboud (1986) claimed that mechanical compresion of the heart may prevent its filling, resulting in a fall in cardiac output and this may be seen with tension pneumothorax, and positive pressure breathing.

Pulmonary hypertension is the leading etiological factor responsible for the right side heart failure (McGinn and White, 1935; Gazes, 1957; Dalen et al., 1969; Murray, 1981; Kuida, 1986), and it is a consequence to (1) the interaction of hypoxia and acidemia (Enson et al., 1964; Fishman, 1976), (2) mechanical obstruction of the

pulmonary circulation due to pulmonary embolism which is sufficient to obstruct more than 60 to 75 percent of pulmonary circulation (Dalen et al., 1969; Mctntyre and Sasahara, 1971; Murray, 1981), (3) emphysema, is typically associated with resting pulmonary hypertension and cor pulmonale only late in its course (Thurlbeck et al., 1970; Bishop, 1973), (4) most of disorders involve lung parenchyma and produced by physical, chemical, infectious agents, tumor cells, and ova or parasites (Kuida 1986).

Alveolar hypoxia is the most important cause of pulmonary hypertension, it is the most potent such stimulus that has yet been identified, and acute pulmonary vasoconstriction appears when alveolar Po_2 is 60 mm Hg (Fowler and Read, 1963; Fishman, 1976; Bohr, 1977).

The right ventricular response to pulmonary hypertension varies depending on a number and variety of variables, including (1) rapidity of progression of hypertension, (2) severity of vascular obstruction. Acute increased pressure load on the right ventricle is poorly tolerated, leading to decreased cardiac output, hypotension, and possible cardiac arrest (chidsey et al., 1964, Chadler et al., 1967; Dalen et al., 1969; Bermis, 1974), while patients with relatively mild obstructive lung disease without severe hypoxemia, generally have

low to normal cardiac output (Burrows et al., 1972). In chronic cor pulmonale, the response may include increased protein synthesis and right ventricular hypertrophy (Morkin, 1974).

Fishman (1971), and Murphy et al., (1974) claimed that the direct effects of hypoxia, and acidosis arising from primary lung disease may precipitate left ventricular failure.

The nose and larynx form parts of the conducting system of the airway. Ohnishi et al., (1972), Obrien and Harvey (1983) mentioned that nasal or laryngeal obstruction can result in alteration in pulmonary function, respiratory acidosis, and severe hypoxia.

Mcfadden and Ingram (1980) said that, because of the integrated nature of the function of the heart and the lungs, it is difficult for one component to be compromised without altering the physiology of the other.

Steffey and Robinson (1983) reported that in lung disease Po_2 is often reduced, but unless Po_2 is less than 75mm Hg, there will be little change in O_2 content or percentage saturation of hemoglobin because the horizontal portion of oxyhemoglobin dissociation curve

provides a protection.

BLOOD PRESSURE AND UREMIA:

Though high blood pressure in dogs has been given little attention in clinicalliterature, it has been suggested as an explanation of the characteristic pathological changes often accompanying renal failure (Goldblatt et al., 1934; Katz et al., 1954; Anderson, 1968; Anderson and Fisher 1968; Vertes et al., 1969; Tobian and Azar, 1972; Vollonen and Oksamen, 1972; Spangler et al., 1977).

Renal parenchymal disease is often accompanied by hypertension (renoparenchymal hypertension), and the most common types of primary kidney disease associated with hypertension are chronic glomerulonephritis, diabetic nephropathy, polycystic kidney disease, chronic pyelonephritis, and interstilial renal disease (Hamilton et al., 1939; Weidmann and Maxwell, 1975).

In kidney disease, the elevated blood pressure appears to be mediated primarily by increased total peripheral resistance, in addition, plasma renin activity is usually high (DelGreco et al., 1967; Neff et al., 1971).

In dogs with renal failure, renal vascular lesion have been observed, but their relationship to hypertens-

ion has not been well decumented (Anderson and Fischer, 1968; Weiser et al., 1977).

In man, renal artery stenosis due fibrous dysplasia or atherosclerosis is the most common potentially curable cause of renovascular hypertension (Maxwell et al.,1972).

The study of Onesti et al., (1975) have provided a clear picture about the hemodynamics of the hypertension of uremia. They claimed that people with renin dependent and severe hypertension usually had a low cardiac output with extremely high vascular resistance, while those with salt and water dependent hypertension presented a different hemodynamic pattern characterised by an elevated cardiac output with a slightly elevated vascular resistance.

Dustan (1986) reported that renoprival hypertension is the term frequently used for the hypertension of uremia, and it implies a failure of salt and water homeostasos, and some is renin dependent.

BLOOD PRESSURE AND LEUKEMIA

The heart may be the site of primary tumor or be invaded by malignancies that arise in other organs. In the case of leukemia, the leukemic infelteration of the heart is characteristically confined to acute leukemia,

with few reports to chronic type, usually found at postmortem study, and generally is not suspected before death (Bierman et al., 1952; Robert et al., 1968; Terry and Kilgerman, 1970; Suryaprosad et al., 1972; Bergeran and Datnow, 1974; Applefeld et al., 1980; Schwartz and Shamsuddin, 1981; Hall and Cooley, 1986).

It has been pointed out that leukemia has caused congestive heart failure (Bregani and Perrotta, 1960; Moller et al., 1975; Kubac et al., 1980), and the lymphatic obstruction by tumor will lead to myocardial interstitial edema, and the secondary pressure on the myofibers may contribute to the eventual cardiac decompensation (Kline, 1972). In this connection, the findings that leukemia has caused conduction disturbances (Redwine, 1971), as well as mitral valve dysfunction, regurgitation (Meltzer et al., 1975), and the association of large quantities of pericardial fluid with tumor encasing the heart frequently results in persistant cardiac constriction (Mann et al., 1978) are of considerable interest.

Darsee and Brauwald pointed out in 1980 that the incidence of systemic hypotension in patients with pericardial neoplastic disease ranged between 5 to 10%. This hypotension may be due to massive pericardial eff-

usion (Casis and Porterfield, 1982), and it was supported by the view of McDonell et al., 1982).

NORMAL ARTERIAL PRESSURE, Po_2, AND OXYGEN TENSION:

Many investigations were carried out by several workers to determine the normal value of arterial blood pressure for the dogs. Allen (1941), and Shingatgeri et al., (1963) reported an average value of 139/79 mm Hg for indirect brachial artery pressure, while an average of 132/79 was reported by Spangler et al.,(1977).

A systemic blood pressure of 160/90mm Hg was established as the upper limit of normotension in a series of clinically normal dogs (Anderson, 1968; Anderson and Fisher, 1968).

Werner (1972) claimed that the indirect blood pressure values for normal dogs were found to be 120 ± 10 mm Hg for the systolic, 75 ± 10 mm Hg for systolic, and 105 mm Hg for the mean blood pressure were found by Kuhn et al.,(1979) who added also that the lower and upper limit of systolic pressure was 110 and 150 mm Hg respectively.

The indirect tibial artery pressure was found to be 155 ± 26/74 ± 14mm Hg (Weiser et al., 1977), or

158 ±30/61±12 mm Hg (Dwight et al., 1981).

Normal arterial Po_2, and pH values for dogs were estimated by many workers. Feigl and Louis (1972) found values of 89 ± 4.9 mm Hg and 7.414 ± 0.032. Similar values of 90.9 ± 5.7 mm Hg, and 7.453 ± 0.023 were reported by Wise (1973), and value of 88.1 ± 1.82 mm Hg, 7.404 ± 0.006 also reported by Klingstrom et al., (1976).

A value of 101.3 ± 5.6 mm Hg for Po_2 was reported by Clark et al., (1977). Haskins (1977 b) found values of 98 ± 6 mm Hg, and 7.402 ± 0.028 for Po_2 and pH respectively. A similar values of 99.7 ± 1.7 mm Hg, 7.41 + 0.00 were also reported by Thompson et al., (1977).

Cornelius and Rawlings (1981) estimated the mean values of arterial Po_2, and pH for 38 healthy dogs and reported to be 90.7 mm Hg, 7.45 respectively. A lower values of 79.7 ± 1.1 mm Hg, and 7.386 ± 0.005 were reported by Wingfield et al., (1982).

MATERIAL AND METHODS

Animals:

A total of 164 unanesthetized, pure and mixed breeds dogs of both sexes have been utilized in the present study and were in 2 categories. The first comprised 15 healthy dogs with ages ranged from 2 months to 12 years. The second consisted of 149 dogs (3 months to 14 years of age), had a wide variety of medical problems and fall into 8 groups based upon the type of clinical situations as follows:

Group 1: included 11 anemic dogs.

Group 2: included 13 diabetic dogs.

Group 3: (Heart Disease) and comprised 35 dogs.

Group 4: consisted of 7 dogs with acute pancreatitis.

Group 5: comprised 18 dogs with nonparvovirus, and 8 dogs with parvovirus gastroenteritis.

Group 6: included 22 dogs had respiratory disease, and 7 dogs with canine distemper (respiratory disturbances).

Group 7: consisted of 22 dogs, had uremia.

Group 8: included 5 leukemic dogs.

MAKING THE DIAGNOSIS (Kraft and Durr, 1981; Schaer and Ackerma, 1982; Kraft, 1984; Dennis, 1985, Kirk and Bistwer, 1985).

1. Routine examination: included history, and physical examination.

2. Routine laboratory procedures: the screening tests that will be described here were usually done. They included determination of packed-cell volume, hemoglobin, RBC, WBC count, and differential leukocytic count. Blood glucose, urea nitrogen serum amylase, lipase, arterial oxygen tension, and arterial pH estimations were routine.

3. Specialized examination:
   a. Radiography; mainly for dogs in group 3 , 6 , 7 (Fig. 1 , 2).
   b. Electrocardiography: mainly for the dogs in group 3.
   c. Endoscopy; laryngoscopy,bronchoscopy,and/or rhinoscopy was ancillary method for further evaluation of the dogs in group 6.
   d. Renal ultrasonography: for the dogs in group 7 (Fig. 3).
   e. In the feces of dogs with enteritis; parvovirus was detected by the electron microscope.
   f. Dogs that died or euthanitized were necropsied and examined histologically (group 3, canine distemper, group 8).

g. For healthy dogs, a complete hemogram, serum electrolytes (calcium, phosphorus, magnesium, sodium, chloride, potasium), serum lactic dehydrogenase, glutamic oxaloacetic transaminase, glutamic pyruvic transaminase, alkaline phosphatase, beside total serum proteins, blood glucose, urea nitrogen, serum amylase, and lipase determinations were a useful diagnostic tests that assist in confirming the healthy condition.

THE SAMPLES

1. **Blood:**
   Two blood samples were collected as follows:
   a. Venous blood for serum, and hematological examination.
   b. Arterial blood was collected from the femoral artery, and immediately analysed for pH, and oxygen tension using gas analyser (Instrumentation Laboratory System 1302).

2. **Feces:** analysed for the presence of parasites and parvovirus.

BLOOD PRESSURE DETERMINATION:

In a quite room, the instrument used for indirect or noninvasive blood pressure determinations was an

ultrasonic Doppler sensing device (Blutdruch-Monitor Bp -103 N, HOYER-BREMEN), the dog was placed in right recumbency and the brachial artery was found suitable for the application of the cuff that has different sizes. The dogs were not restrained, and the mean of three consecutive readings was taken to avoid errors.

STATISTICAL ANALYSIS:

Statistical analysis of the results was performed at the Computer Centere of the Faculty of Veterinary Medicine, Ludwig Maximillian University, Munik, West Germany.

RESULTS

BLOOD PRESSURE AND ANEMIA:

Table (1) revealed that the anemic dogs had a normal blood pressure of 133/79 mm Hg, also a normal arterial Po_2, and pH of 85 mm Hg, 7.414 respectively.

BLOOD PRESSURE AND DIABETES MELLITUS:

Based on their blood pressure, the dogs fall into 2 subgroup. Subgroup 1 (Table 2) with a normal blood pressure (142/83 mm Hg), a normal arterial Po_2 of 94 mm Hg, and with a slight decrease in pH(7.325) Subgroup 2 with a higher blood pressure of 175/100 mm Hg, slightly decrease in Po_2(85 mm Hg), and a normal pH of 7.346 (Table 3).

BLOOD PRESSURE AND HEART DISEASES:

Based on blood pressure, this group was further stratified into 3 main subgroups.

Subgroup 1. In this subgroup, 6 dogs had a normal arterial blood pressure, and Po_2 of 140/80,

92 mm Hg respectively, as compared to 134/76, 94 mm Hg, in ormal subjects. Also this subgroup had a normal pH value of 7.404 (Table 4).

Subgroup 2. For this subgroup, the arterial blood pressure was very high (172/104), as compared with (134/76) in normal dogs. The arterial Po_2 was slightly lower (83 mm Hg), with a normal pH of 7.388. The electrocardiographic findings were useful in many of our dogs. The frequency of cardiac enlargement in the 14 canine patients in this subgroup was 78% (11 of 14 dogs). Of the 11 dogs, 6 (42%) had right atrium enlargement, 5 (35%) had right and left ventricular enlargement (Table 5).

Subgroup 3. It is evident from the statistical data in table (6) that this subgroup exhibited a very low arterial blood pressure, and Po_2, as the mean values were 101/59 and 74 mm Hg respectively. The arterial pH value was similar to that in normal control dogs. The electrocardiographic changes, and pathological findings indicated that cardiac arrthmia, and congestive cardiomyopathy were the most frequent abnormalities, beside myocardial infaction mitral, and tricuspid valvular fibrosis.

BLOOD PRESSURE AND ACUTE PANCREATITIS:

This group, in which acidemia (7.246) had been observed, showed a fall in blood pressure (97/58 mm Hg), with a normal arterial Po_2 of 93 mm Hg when compared with the control dogs (Table 7).

RELATIONSHIP BETWEEN PARVOVIRUS, NONPARVIRUS GASTROENTERIEIS.

Based on the detection of parvovirus in the feces by electron microscope, the gastroenteritic group was further stratified into parvovirus infected, and parvovirus noninfected subgroup. Results obtained in table (8) revealed that blood pressure in the two subgroups was (96/58, 100/60 mm Hg respectively) significantly lower than in normal dogs, with a normal arterial Po_2 of 90, 89 mm Hg. The parvovirus infected subgroup had acidemia with a pH of 7.307, while the other did not (pH = 7.373).

RELATIONSHIP BETWEEN RESPIRATORY DISEASES, CANINE DISTEMPER (with respiratory disturbances) AND BLOOD PRESSURE

Based upon the arterial blood pressure, the dogs with respiratory diseases were further stratified into 2 main subgroups.

Subgroup 1. In this subgroup, 13 dogs had a normal arterial blood pressure, pH of 137/77 mm Hg, 7.374 respectively, as compared to 134/76 mm Hg, 7.376 in normal subjects. A significant decrease in arterial Po_2 was noticed (Table 9).

Subgroup 2. It is evident from the statistical data in table (9) that this subgroup exhibited a very low arterial blood pressure, and Po_2, as the mean values were 110/64, 60 mm Hg respectively. The arterial pH value was similar to that in normal dogs (7.348).

Dogs with canine distemper showed a very low arterial blood pressure, Po_2, and a normal pH of 101/58, 78 mm Hg, and 7.384 respectively (Table 9).

BLOOD PRESSURE AND UREMIA:

In this group, arterial blood pressure, Po_2, and pH values were compared with the normal values (Table 10). The former had a higher blood pressure of 154/91 mm Hg, a normal values for Po_2 (90 mm Hg), and for pH (7.334) when compared with a normal values of 134/76 mm Hg, 94 mm Hg, and 7.376 respectively.

BLOOD PRESSURE AND LEUKEMIA:

For this group, the arterial blood pressure was very low (106/63mm Hg), as compared with (134/76) in normal dogs, beside a normal arterial Po_2, and pH of 86 mm Hg, 7.405 respectively. The pathological findings indicated infilteration of the heart, and lymph nodes of most dogs with the leukemic cells (Table 11).

NORMAL ARTERIAL BLOOD PRESSURE, Po_2, AND pH VALUES:

Table (12) revealed that the healthy dogs had a normal arterial blood of 134 $\pm$ 7/76 $\pm$ 4 mm Hg, with a minimum value of 123/70 mm Hg, and maximum value of 149/88 mm Hg. The normal arterial oxygen tension was 94 $\pm$ 3 mm Hg, with a minimum of 91 mm Hg, and a maximum of 106 mm Hg.

The normal average of arterial pH was 7.376 $\pm$ 0.028 with a minimum, and maximum values 7.323, 7.426 respectively.

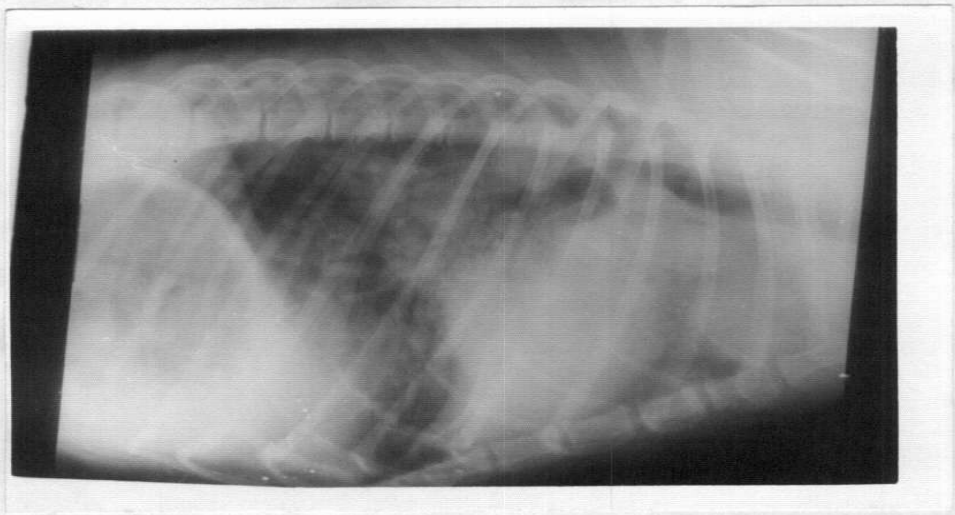

Fig. (1): Lateral radiograph of 6 years old male poodle with a chronic, nonproductive cough of over two month's duration. The dog had a lower arterial blood pressure, Po_2 of 112/60, 70mm Hg respectively. A needle biobsy from the lung was done, and malignant tumor cells were found. The dog was euthanitized, and the pathological report was cor pulmonale due to primary lung tumor.

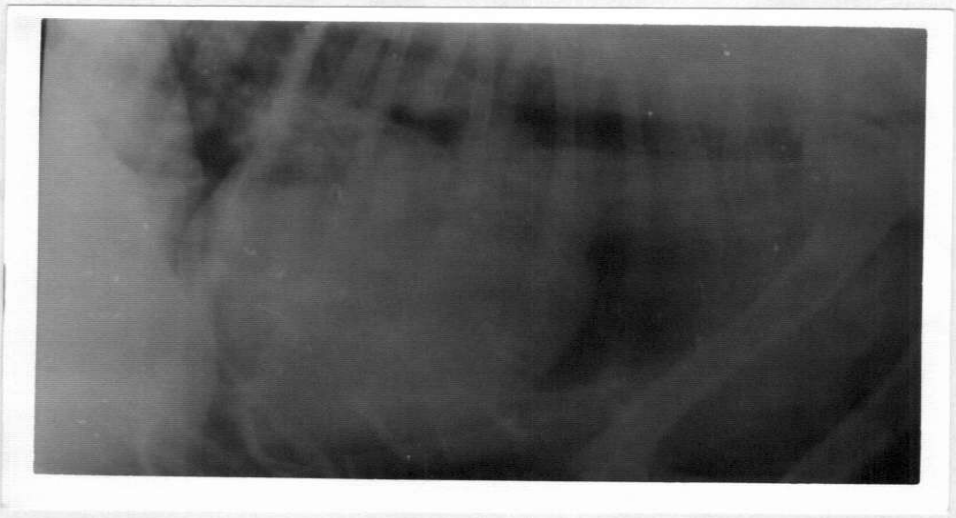

Fig. (2): Lateral radiograph of 12 years old, female Cocker Spaniel that had Arterial blood pressure of 115/65 mm Hg. the dog had generalized cardiac enlargement, died several hours after the radiograph was made. The pathological report indicated myocardial infarction.

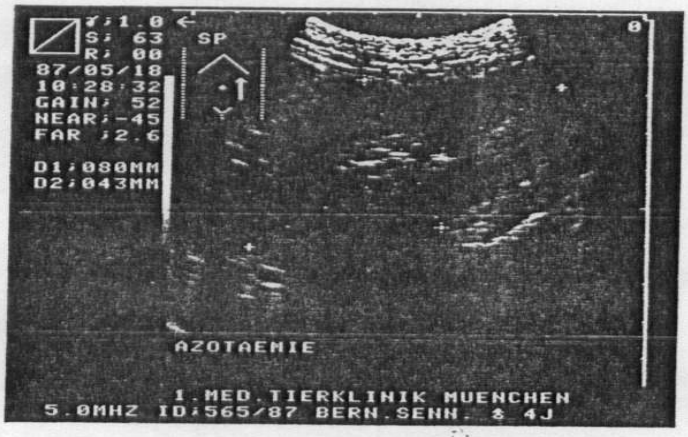

Fig. (3): Longitudinal ultrasonograph (5 MHz) through the left kidney of 4 years old male dog with blood urea nitrogen of 300 mg/dL, blood pressure of 159/91 mm Hg. The sonograph shows dimensions of 080 X 043 MM, and the pathological examination of the kidney showed chronic membranous glomerulosclerosis.

Table (1): Compartive statistical analysis of arterial pressure, Po_2, pH, Hb, and PCV in normal dogs and those with anemia.

Condition of dogs	No.of dogs	Systolic / diastolic blood pressure (mm Hg)	Po_2 (mm Hg)	pH	Hb g/100 ml	PCV %
		Mean	Mean	Mean	Mean	Mean
Healthy	15	134/76	94	7.376	16	47
Anemic	11	133/79 *	85**	7.414 *	5.9 **	19.7**

* Indicates values similar to the values found in healthy dogs ($P < 0.05$).

** = Significant decrease ($P < 0.05$).

Table (2): Comparative stastistical analysis arterial pressure, Po_2 pH, and blood glucose in normal dogs and those with diabetes mellitus (subgroup I).

Condition of dogs	No.of dog	Systolic / diastolic blood pressure (mm Hg)	Po_2 (mm Hg)	pH	Blood glucose (mg/dL)
		Mean	Mean	Mean	Mean
Healthy	15	134/76	94	7.376	60
Diabetic dogs (subgroup I)	5	142/83*	94*	7.325**	322***

* Indicates values similar to the values found in healthy dogs ($P < 0.05$).

** = Significant decrease ($P < 0.05$).

*** = Significant increase ($P < 0.05$).

Table (3): Comparative statistical analysis of arterial pressure, Po_2, pH, and blood glucose for normal dogs and tjose with diabetes mellitus (subgroup 2).

Condition of dogs	No. of dogs	Systolic/ diastolic blood pressure (mm Hg)	Po_2 (mm Hg)	pH	Blood glucose (mg/dl)
		Mean	Mean	Mean	Mean
Healthy	15	134/76	94	7.376	60
Diabetic dogs (subgroup 2)	8	175/100 xxx	85 xx	7.346 x	293 xxx

x Indicates values similar to the values found in healthy dogs ($P < 0.05$).

xx = Significant decrease ($P < 0.05$).

xxx = Significant increase ($P < 0.05$).

Table (4): Comparative statistical analysis of Arterial blood pressure, Po_2, and pH of normal dogs, and those with heart diseases (subgroup 1). The table also illustrated the electrocardiographic changes for this subgroup.

Dog No.	Systolic/ diastolic (mm Hg)	Po_2 (mm Hg)	pH	Electrocardiographic changes
1	145/85	94	7.395	Englargement of both right and left ventricles, and also right atrium.
2	142/88	93	7.400	Left ventricular enlargement.
3	144/84	102	7.395	Enlargement of the right ventricle.
4	140/80	103	7.390	Left atrium enlargement.
5	138/84	83	7.327	Second degree of atrioventricular block.
6	134/74	80	7.517	Right bundle branche block.
6	140/80 *	92 *	7.404 *	Mean values for diseased dogs.
15	134/76	94	7.376	Mean values for healthy dogs.

* Indicates values similar to the values of controle ($P < 0.05$).

Table (5): Comparative statistical analysis of arterial blood pressure, Po_2 and pH of the normal dogs and those with heart diseases (subgroup II). Thetable illustrated also the electrocardiographic changes for this subgroup in number and percent.

Dog Nos	Systolic / diastolic (mm Hg)	Po_2 (mm Hg)	pH	Electrocardiographic changes	No. %
1	204/113	92	7.426	Right atrium enlargement.	
2	167/127	103	7.426	Right atrium enlargement.	
3	169/ 97	86	7.401	Right atrium enlargement.	6
4	175/108	54	7.358	Right atrium enlargement.	42%
5	168/104	93	7.433	Right atrium enlargement.	
6	165/ 98	97	7.402	Right atrium enlargement.	
7	198/113	98	7.304	Elevated Twave in lead II, III.	1 7%
8	187/106	99	7.399	Hypovoltage.	1 7%
9	178/108	92	7.314	Normal.	1 7%
10	146/ 93	82	7.328	Enlargement of both right and left ventricles.	
11	173/104	66	7.388	Enlargement of both right and left ventricles.	5
12	154/ 94	54	7.473	Enlargement of both right and left ventricles.	35%
13	163/ 97	92	7.405	Enlargement of both right and left ventricles.	
14	173/104	66	7.388	Enlargement of both right and left ventricles.	
14	172/104***	83**	7.388*	Mean values for diseased dogs	
15	134/ 76	94	7.376	Mean values for healthy dogs	

* = Indicates values similar to the values found in healthy dogs ($P < 0.05$).
** = Significant decrease ($P < 0.05$).
*** = Significant increase ($P < 0.05$).

Table (6): Comparative statistical analysis of arterial blood pressure, Po_2 and pH of normal dogs and those with heart diseases (subgroup III). The table also illustrated the electrocardiographic changes, outcore, and the pathological findings for the dogs in this subgroup.

Dog No.	Systolic/ diastolic (mm Hg)	Po_2 (mm Hg)	pH	Electrocardiographic changes.	Outcome	Pathological findings.
1	115/65	50	7.431	Atrial fibrillation	died	Myocardial infarction.
2	91/55	83	7.465	Enlargement of both right, and left atrium, and also left ventricle.	Euthanasia	Dilatation of hearts chambers-congestive cardiomyopathy.
3	74/43	57	7.442	Enlargement of both right, and left atrium.	Euthanasia	Dilatation of hearts chambers-congestive cardiomyopathy.
4	88/50	69	7.411	Atrial fibrillation	Euthanasia	Dilatation of both ventricles-congestive cardomyopathy.
5	107/64	46	7.386	Enlargement of the right ventricle.	Euthanasia	Mitral, tricuspid valvular fibrosis and dilatation of right ventricle.
6	111/65	95	7.220	Enlargement of both right and left ventricles.	Euthanasia	Autopsy not performed.
7	81/55	76	7.411	Enlargement of both right atrium; and ventrile.	Alive	
8	102/59	90	7.426	Second degree of atrioventricular block	Alive	
9	117/68	93	7.470	First degree of atrioventricular block	Alive	
10	107/61	63	7.471	Atrial fibrillation	Alive	
11	120/71	64	7.386	Sinus tachycardia	Alive	
12	92/56	68	7.422	Sinus tachycardia + S-T segment depression.	Alive	
13	97/54	71	7.450	Sinus tachycardia + S-T segment depression.	Alive	
14	118/68	95	7.345	Ventricular extrasystol.	Alive	
15	98/52	97	7.374	Enlargement of the right ventricle.	Alive	
15	101/59 *	74 **	7.399 *	Mean values for diseased dogs.		
15	134/76	94	7.376	Mean values for normal dogs.		

* Indicates values similar to those found in healthy dogs ($P < 0.05$).

** = Significant decrease ($P < 0.05$).

Table (7): Comparative statistical analysis of Arterial blood pressure, Po_2, pH, serum amylase, and lipase of normal dogs and those with acute pancreatitis.

Condition of dogs	No.of dogs	Systolic/diastolic blood pressure (mm Hg)	Po_2 (mm Hg)	pH	Serum amylase IU/I	serum lipase IU/I
		Mean	Mean	Mean	Mean	Mean
Healthy	15	134/76	94	7.376	1065	170
Pancreatitic	7	97/58 **	95 *	7.246 **	3211 ***	1590 ***

* Indicates values similar to the values found in healthy dogs ($P < 0.05$).

** = Significant decrease ($P < 0.05$).

*** = Significant increase ($P < 0.05$).

Table (8): Comparative statistical analysis of arterial blood pressure, Po_2, and pH of normal and those parvovirus infected, and noninfected subgroups.

Condition of dogs	No. of dogs	Systolic/diastolic blood pressure (mm Hg)	Po_2 (mm Hg)	pH
		Mean	Mean	Mean
Healthy	15	134/76	94	7.376
Parvovirus infected.	8	96/58 xx	90 x	7.307 xxx
Parvovirus noninfected	10	100/60 xx	89 x	7.373 x

x Indicates values similar to the values found in healthy dogs ($P < 0.05$).

xx = Significant decrease ($P < 0.05$).

Table (9): Comparative statistical analysis of arterial blood pressure, Po_2, and pH of normal, respiratory diseases (subgroup 1 and 2), and those with canine distemper(with respicatory disturbances).

Condition of dogs	No. of dogs	Systolic/diastolic blood pressure (mm Hg)	Po_2 (mm Hg)	pH
		Mean	Mean	Mean
Healthy	15	134/76	94	7.376
Respiratory diseases (subgroup 1)	13	137/77 *	84 **	7.374 *
Respiratory diseases (subgroup 2)	9	110/64 **	60 **	7.348 *
Canine distemper	7	101/58 **	78 **	7.384 *

* Indicates values similar to the values found in healthy dogs ($P< 0.05$).

** =Significant decrease ($P< 0.05$).

Table (10): Comparative statistical analysis of arterial blood pressure, Po_2, and pH of normal dogs, and those with uremia.

Condition of dogs	No of dogs	Systolic/diastolic blood pressure (mm Hg)	Po_2 (mm Hg)	pH	Blood urea nitrogen. mg/dL
		Mean	Mean	Mean	Mean
Healthy	15	134/76	94	7.376	30
Uremic	22	154/91**	90 *	7.334*	260**

* Indicates values similar to the values found in healthy dogs ($P < 0.05$).

** = Significant increase ($P < 0.05$).

Table (11): Comparative statistical analysis of arterial blood pressure, Po_2, and pH of leukemic dogs and healthy dogs. The table also illustrated the outcome, and the pathological findings in leukemic dogs.

Dog No.	Systolic/ diastolic (mm Hg)	Po_2 (mm hg)	pH	Outcome	Pathological findings
1	110/67	83	7.495	Euthanasia	Lymphocytic infilteration of different organs, including the heart and the liver (Lymphocytic leukemia)
2	85/51	98	7.438	Euthanasia	Lymphocytic infilteration of different organs, including the heart and the liver (lymphocytic leukemia)
3	103/62	93	7.387	Euthanasia	Enlargement of the peripheral, organs lymph nodes due to leukemia (Leukemia).
4	117/65	59	7.358	Euthanasia	mast-cell infilleration of the organs, peripheral lymph nodes, spleen, and the heart (mast-cell leukemia).
5	115/71	98	7.348	Euthanasia	Generalized enlargement of lymph nodes (Lymphocytic leukemia)
	106/63**	86*	7.405*	Mean values for leukemic dogs	
	134/76	94	7.376	Mean values for healthy dogs	

* Indicates values similar to those found in healthy dogs ($P < 0.05$).

** = Significant decrease ($P < 0.05$).

Table (12): Arterial blood pressure, oxygen tension, and pH values for healthy dogs.

	No. of dogs.	Minimum	Maximum	Mean	S.d.
Systolic/diastolic (mm Hg)	15	123/70	149/88	134/76	7/4
Arterial oxygen tension (mm Hg)	15	91	106	94	3
Arterial pH	15	7.323	7.426	7.376	0.028

DISCUSSION

BLOOD PRESSURE AND ANEMIA

The results obtained and recorded in Table(1) revealed a normal blood pressure for the anemic dogs with a normal arterial pH and normal value for the oxygen tension. The normal systolic blood pressure level is agreement with the findings of Bishop et al.,(1955) and Weatherall et al.,(1981) whereas a moderate decrease in diastolic blood pressure was reported by Brannon et al. (1945), Graettinger et al.,(1963) and Weatherall et al.,(1981).

Anemia is the most common condition that increase the cardiac output (Richardson and Guyton, 1959; Clarke et al., 1978; Grossman and Braunwald, 1980), and decrease the total peripheral vascular resistance (Liang and Huckabee, 1973; Grossman and Braunwald, 1980).

Richardson and Guyton (1959) found an increase in cardiac output of 26% in anemic dogs with a parallel decrease in the total peripheral resistance of 25.6%.

In our anemic dogs, the arterial pH, and oxygen tension play no role because this group had a

normal arterial pH, Po_2, and the oxygen tension, pH values reported to depress cardiac contractility, decrease blood pressure, and cardiac output are 75mm Hg, and 7.2 respectively (Haskins, 1977 a; Steffey and Robinson, 1983; Abboud 1986).

It appears from the above discussion that the high cardiac output and the parallel decrease in the total peripheral resistance of the anemic dogs are the main cause for the normal blood pressure obtained in our study because the mean arterial pressure = cardiac output X total peripheral ristance (Dustan, 1986).

BLOOD PRESSURE AND DIABETES MELLITUS

In this study, blood pressure determinations in dogs with diabetes mellitus were compared with those in nodiabetic dogs Table (2,3). The former group had either normal or high blood pressure when compared with the latter group. It is more surprising that similar results have been observed in man by Piccoli et al.,(1979), and Williams and Braunwald (1980).

It has been reported repeatedly that in diabetic patients, the hypertension is almost always accompanies the syndrome of diabetic nephropathy (Keen et al., 1975; Kaplan, 1980; Parving et al., 1981; Mogensen, 1982), and normal catecholamine and plasma renin activity levels in the presence of a hypersensitive cardiovascular system (Chinn and Dusterdieck, 1972; Philipp et al., 1978). In contrast, Diabetics without pereceptible renal disease were reported to have a normal blood pressure (Oakly et al., 1974; Keen et al., 1975), and a normal cardiovascular sensitivity to norepinephrine or angiotensin (Christlieb, 1976; Seneviratne, 1977).

In our diabetic dogs either with a normal, or a high blood pressure (Table 2,3) the arterial pH, Po_2 play no role because the pH, and Po_2 reported to decrease blood pressure, cardiac output, and depress cardiac contractility are 7.2, 75 mm Hg respectively (Haskins, 1977 a; Steffey and Robinson, 1983; Abboud, 1986.

BLOOD PRESSURE AND HEART DISEASES:

Based on the blood pressure, this group was further stratified into 3 main subgroups.

Subgroup I

Although there was a clear radiographic and electrocardiographic evidences of heart diseases, this group had a normal blood pressure (Table 4). One possible explanation for this observation is suggested by the fact that all dogs in this subgroup had a compensatory mechanisms which are often able to restore to normal or near normal the arterial blood pressure, and the cardiac output at rest. This in agreement with a previous findings regarding normotension and compensatory mechanisms (Mason *et al.*, 1971; Ross, 1974; Schlant and Sonnenblick,1986).

Because hypoxemia and/or acidosis were reported to depress myocardial contractility (Abboud, 1986), and may precipitat left ventricular failure (Fishman, 1971; Murphy *et al.*, 1974), it is very important to mention also hier that, this subgroup had a normal arterial pH and PO_2 (Table 4).

Subgroup 2

All dogs in this subgroup, in which a normal arterial pH and a slight decrease in PO_2 had been observed, showed a high blood pressure level (Table 5).

The presence of hyperkinetic heart syndrome may explain the high blood pressure where this syndrome was responsible for the high pressure reported in some patients (Eich et al., 1962; Julius and Conway, 1968), also Ibrahim et al. (1975) reported that the heart may be involved in the genesis and maintenance of hypertension in these patients. In contrast, hypertension may be the cause of electrocardiographic and radiographic changes associated this subgroup (Tobian et al., 1960; Tarazi et al., 1966; Beznak et al., 1969; Kannel et al., 1969).

Subgroup 3

It is evident from the statistical data that this subgroup had a very low arterial blood pressure and PO_2, as the mean values were 101/59 and 74 mm Hg respectively. The arterial pH value was similar to that in normal control dogs (Table 6).

The cardiac causes of hypotension were reported to be: arrythmia, either tachycardia or bradycardia (Ruskin et al., 1970; Linden, 1973; Sobel and Roberts, 1980; Esente et al., 1983; Ross, 1983; Ferguson and Abboud, 1986), acute myocardial infarction (Linden, 1973; Sobel and Roberts, 1980; Esente

et al., 1983), mitral and aortic regurgitation (Ross and Braunwald, 1968; Schwars et al., 1981), and congestive cardiomyopathy (Wynne and Braunwald, 1980).

Hypoxemia was reported to depress myocardial contractility and the severity of the decline in arterial PO_2 reflects the extent of myocardial infarction (Abboud, 1986), also hypoxemic may precipitat left ventricular failure (Fishman, 1971; Murphy et al., 1974).

The above mentioned discussion, the electro cardiographic and necropsy findings (Table 6) indicate that, this hypotension condition is mainly due to cardiac arrythmia, congestive cardiomyopathy, myocardial infarction, and hypoxemia.

BLOOD PRESSURE AND ACUTE PANCREATITIS

Table (7) lists the results from 7 cases of acute pancreatitis. The dogs had acidosis, and a blood pressure below the lower limit observed in normal dogs. The PO_2 lay within normal limit.

Previous findings regarding hypotension and acute pancreatitis have been reported (Katz et al., 1964; Anderson, 1967; Rogers, 1983), and the hypotension was a consequence to hypovolemia (Dos Reis, 1963; Facy et al., 1966; Ofstad et al., 1969; Geokas et al., 1972), and to the vasodilator effect of bradykinin and kalejrein (Thal et al., 1963; Beck et al., 1965; Creutzfeldt and Schmidt, 1970; Hardy and Stevens, 1975; Rogers, 1983).

Our dogs in this group had acidosis. It has been pointed out that acidosis reduces left ventricular contractility, decreases myocardial responsiveness to catecholamines constricting the venous system and hypotension (Carson et al., 1965; Kittle et al., 1965; Mitchell et al., 1972; Abboud, 1986). This observation has to some extent indicated the participation of acidosis in the pathophysiology of hypotension in acute pancreatitis.

RELATIONSHIP BETWEEN PARVOVIRUS, NONPARVIRUS GASTROENTERITIS.

Based on the detection of parvovirus in the feces by electron microscope, the gastroenteritic group was further stratified into two subroups:

1. Parvovirus infected.
2. Parvovirus noninfected.

The blood pressure was significantly lower in both than it was in the control dogs.

Canine parvovirus is now know to be a cause of both of enteritis and myocarditis (Appel et al., 1979; Hayes et al., 1979), and death due to parvoviral enteritis results from dehydration, and endotoxic shock (Green , 1984).

A decrease in total blood volume has previously been regarded as a primary mechanism accorunting for hypotension complicating gastroenteritis (McDonell, 1974; Ross, 1975; Smith and Hamlin, 1977; Sleisenger, 1981), also impaired (RES) function, septicaemia, and endotoxins absorption are additional contributary factors (Fine et al., 1959; 1965; Priano et al., 1971; Brasmer, 1972).

The parvovirus infected subgroup represented acidemia with a pH of 7.30, while the noninfected subgroup did not.

The finding observed in Table (8) have demonstrated that in both of the subgroups, the arterial oxygen tension, and pH play no role in this hypotensive condition, because the two subgroups had a normal PO_2 and the pH reported to decrease blood pressure was 7.2 or less (Haskin, 1977a).

RELATIONSHIP BETWEEN RESPIRATORY DISEASES, CANINE DISTEMPER (with respiratory disturbances) AND BLOOD PRESSURE

Table (9) lists the results obtained from clinical cases of respiratory disease. Those with normotension (subgroup 1) had a normal blood pressure of 137/77 mm Hg, a normal pH of 7.374, while those with hypotension (subgroup 2) exhibited blood pressure level of 110/64 mm Hg, and a normal pH of 7.348. The canine distemper group had also a lwer pressure of 101/58 mm Hg, and a normal pH of 7.384 (Table 9).

It has been reported repeatedly that pulmonary hypertension is the leading factor responsible for righ side heart failure (McGinn and White,1935; Gazes, 1957; Dalen et al., 1969; Murray, 1981; Kuida, 1986), and that disorders of the right vent-

ricle may result in left ventricular failure leading to decreased cardiac output, hypotension and possible cardiac arrest (Chidsey et al., 1964; Chadler et al., 1967; Dalen et al., 1969; Bermis et al.,1974).

Alveolar hypoxia is the most important cause of pulmonary hypertension and acute pulmonary vasoconstriction appears when alveolar PO_2 is 60 mm Hg (Fowler and Read, 1963; Fishman, 1976; Bohr, 1977), also the direct effect of hypoxia may impair myocardial contractility, leading to left ventricular failure (Fishman, 1971; Abboud, 1986).

Our dogs in subgroup 2, and those with canine distemper had severe hypoxemia with arterial PO_2 of 60, and 78 mm Hg respectively, while those in subgroup 1 exhibited a very slightly decrease in arterial PO_2 which of no clinical significance (Steffey and Robinson, 1983). It therefore appears that the hypotension that was observed in lung disease (subgroup 2), and canine distemper related to the effect of hypoxemia associated these contitions.

BLOOD PRESSURE AND UREMIA

In this study, blood pressure determination in dogs with uremia was compared with those in nonuremic dogs (Table 10). The former group had a higher blood pressure when compared with the latter group. This is in agreement with a previous findings regarding hypertension and renal failure (Katz et al., 1957, Anderson, 1968; Anderson and Fisher, 1968; Weidmann and Maxwell, 1975, Kaplan, 1980).

The pathogenesis of renal hypertension is unclear (Weiser et al., 1977). In early studies in dogs, it was demonstrated that renal ischemia resulted in increased plasma renin activity, which resulted in increase of angiotensin II, a potent vasoconstrictor (Goldblatt et al., 1934). It is now know that the hypertension is either renin-dependent or salt-and-water-dependent, or due to both lechanisms (Vertes et al., 1969). Another mechanism may involve the interstitial cells of the renal medulla (Tobian and Azar, 1972). These cells are thought to produce prostaglandins that posses antihypertensive properties, and a deficiency of these prostaglandins may facilitate elevation of blood pressure.

It is very important to mention heir that this group had a normal arterial pH and PO_2 (Table 10).

BLOOD PRESSURE AND LEUKEMIA:

It is evident from the statistical data in Table (11) that this group exhibited a very low arterial blood pressure, with a normal Po_2, and pH, as the mean values were 106/63 mm hg, 86 mm hg, and 7.405 respectively.

This hypotension condition is in agreement with a previous findings reported by Darsee and Braunwald (1980), and McDonnell et al., (1982) in human beings.

Since our dogs in this group had a normal arterial Po_2, pH, and leukemic infilteration of the heart (Table 11), and this infilteration was reported to cause heart failure, and conduction disturbances (Bregani and Perrotta, 1960, Roberts et al., 1968; Moller et al., 1975; Applefeld et al., 1980; Kuboc et al., 1980; Hall and Cooley, 1986); it seemes possible, therefore that in this group, the hypotension is mainly due to leukemic infilteration of the heart.

NORMAL ARTERIAL BLOOD PRESSURE, OXYGEN TENSION, AND PH VALUES:

It is evident from the obtained results (Table 12) that the normal nonivasive brachial artery pressure value was 134/76 mm Hg. A similar value of 139/79 mm Hg was reported by Allen (1941), Shingatgeri et al.,(1963).

The normal arterial Po_2, and pH values were 94 ± 3 mm Hg, and 7.376 ± 0.028 respectively. The Po_2 value agree with the views of Feigl and Louis (1972), Wise (1973) and Thompson et al. (1977). And the pH value agree with the finding of Feigl and louis (1972), Klingstron et al., (1976), and Wingfield et al.,(1982).

SUMMARY

The aim of this study was to survey blood pressure in relation to arterial oxygen tension, and pH on dogs hospitalised because of a variety of clinical disorders. Blood pressure was determined from the brachial artery using ultrasonic Doppler technique.

The dogs utilized in the present study were in 2 categories. The first comprised 15 healthy dogs, and the second consisted of 149 dogs with a wide variety of medical problems, and fall into these groups:

Group 1:

Included 11 anemic dogs, with a normal blood pressure, pH, Po_2, and a severe decrease in hemoglobin and packed-cell volume.

Group 2:

had 13 diabetic dogs, and fall into 2 subgroups. Subgroup 1, with a normal blood pressure, Po_2, and a slight decrease in pH. Subgroup 2, had a higher blood pressure, normal pH, and a slight decrease in Po_2.

Group 3:

with heart diseases, and was further classified to 3 subgroups based on blood pressure as follow: Subgroup, had a normal blood pressure Po_2, and a normal value for pH . Subgroup 2, with a very high blood pressure, slightly lower Po_2, and a normal pH. The electrocardiographic findings indicated cardiac enlargement to 78% of the total number of dogs. Subgroup 3, had a very low arterial blood pressure, due to hypoxemia, mitral, tricuspid valvullar fibrosis, cardiac arrythmia, and congective cardiomyopathy.

Group 4:

had pancreatitis, acidemia which play a role in the hypotension condition found in this group.

Group 5:

based on the detection of parvovirus in the feces of gastroenteritic dogs, this group was further stratified into: parvovirus infected subgroup, and parvovirus noninfected subgroup. The two had a very low arterial pressure, nor-

mal Po_2, but the parvovirus subgroup showed acidemia which plays no role in this hypotension condition.

Group 6:

With respiratory disease, and fall into 2 subgroups: with a normal blood pressure, pH, and a slight decrease in Po_2. Subgroup 2, had a very low arterial pressure due to respiratory failure characterised by hypoxemia that plays a role in this hypotension. Also this group included dogs with canine distemper (with respiratory disturbances), and had a very low arterial pressure which may be due to hypoxemia.

Group 7:

Uremic dogs, exhibit high blood pressure, normal Po_2, and pH.

Group 8:

The leukemic dogs with hypotension, normal arterial Po_2, and pH.

The healthy dogs were found to have arterial blood pressure of 134 ± 7/76 ± 4 mm hg, Po_2 of 94 ± 3 mm hg, and a pH of 7.376 ± 0.028.

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الملخص العربى

كان الهدف من هذا البحث الذى أجرى بالكامل بقسم الأمراض الباطنه بكلية الطب البيطرى – جامعة ميونيخ – ألمانيا الغربيه هو قياس ضغط الدم فى أمراض الكلاب المختلفه . وقد تم قياس ضغط الدم لعدد ١٦٤ حيوان حيث كان عدد الكلاب المريضه ١٤٩ والسليمه ١٥ .

بالنسبه للكلاب السليمه : فقد تم أنتقاؤها بعد تحليلات كامله للدم شملت وظائف الكليه والكبد والقلب والجهاز التنفسى والهضمى .

أما الكلاب المريضه : فبعد اجراء الفحص الطبى الشامل والأختبارات المعمليه والتى شملت تعيين البولينا والسكر والنزيم الاميلاز والليباز وفحص كامل لصورة الدم بالاضافه الى فحص الكليه عن طريق الاشعه والموجات فوق الصوتيه، فحص القلب عن طريق الاشعه ورسم القلب وفحص الجهاز التنفسى عن طريق الاشعه والمنظار . بالنسبه للكلاب المصابه بالاسهال فقد تم فحص البراز عن طريق الميكروسكوب الالكترونى لمشاهدة وجود فيروس البارفو . بالنسبه للكلاب التى نفقت فقد تم فحص الجثه بعد النفوق بقسم الباثولوجى بكلية الطب البيطرى – جامعة ميونيخ وذلك لتأكيد التشخيصات السابقه وقد قسمت هذه الكلاب الى المجموعات التاليه :

١) المجموعه المصابه بالانيميا
٢) المجموعه المصابه بمرض السكر
٣) المجموعه المصابه بأمراض القلب
٤) المجموعه المصابه بألتهاب البنكرياس الحاد
٥) المجموعه المصابه بالاسهال وفيروس البارفو
٦) المجموعه المصابه بأمراض الجهاز التنفسى والديستمبر (خلل بالجهاز التنفسى)
٧) المجموعه المصابه بالهولينا
٨) المجموعه المصابه بالليوكيميا

وقد تم دراسة تأثير كلا من ضغط الاكسجين الشريانى وكذلك التأين الا بدروجينى الخاص بالشريان على ضغط الدم فى تلك المجموعات وكانت النتائج كما يلى

١٠ تميزت المجموعه الاولى بضغط دم عادى ولا يوجد تأثير لضغط الأكسجين وكذلك التأين الايدروجينـــى .

٢٠ قسمت المجموعه المصابه بمرض السكر الى جزئين :
أ٠ الجزء الاول : وقد تميز بضغط دم عادى
ب٠ الجزء الثانى : وقد تميز بضغط دم مرتفع

ويجدر الاشاره هنا بأن هذه النتيجه مشابهه تماما لما وجد فى كثير من المراجع البشريه .

٣٠ المجموعه المصابه بأمراض القلب والتى قسمت الى ثلاث أجزاء :
أ٠ الجزء الاول : وقد تميز بضغط دم عادى بالرغم من وجود ما يثبـــت أصابتها بأمراض القلب وذلك بعد فحص رسم القلب .
ب٠ الجزء الثانى : وقد تميز بأرتفاع ضغط الدم .
جـ٠ الجزء الثالث : وقد تميز بأنخفاض فى ضغط الدم .

٤٠ المجموعه المصابه بالتهاب البنكريا س الحاد تميزت بأنخفاض حاد فى ضغط الدم .

٥٠ المجموعه المصابه بالاسهال وفيروس البارمو تميزت بأنخفاض حاد فى ضغط الدم .

٦٠ المجموعه المصابه بأمراض الجهاز التنفسى والتى قسمت الى جزئين .
أ٠ الجزء الاول وقد تميز بضغط دم عادى .
ب٠ الجزء الثانى وقد تميز بضغط دم منخفض .

أما الكلاب المصابه بالديستمبر فقد تميزت بأنخفاض فى ضغط الدم .

٧٠ المجموعه المصابه بالبولينا وقد تميزت بأرتفاع فى ضغط الـــدم .

٨٠ المجموعه المصابه بالليوكيميا وقد تميزت بأنخفاض حاد فى ضغط الدم .

ويجدر الاشاره هنا الى أن حموضة الدم قد لعبت دورا بارزا فى أنخفـــاض ضغط الدم فى حالة ألتهاب البنكريا س الحاد ، بينما لعب نقص ضغـــــط الاكسجين الشريانى نفس الدور فى حالة أمراض الجهاز التنفســـــــــى

(الجزء الثانى) وفى حالة الأصابة بالديستمبر • وقد كان لقصور القلب الأحتقانى والاهتزازات الأذنيه والسكته القلبيه بجانب نقص ضغط الأكسجين الشريانى أثر واضح فى أنخفاض ضغط الدم الخاص بأمراض القلب (المجموعه الثالثه) •

وقد تراوح ضغط الدم فى الكلاب السليمه ما بين ١٣٤ ± ٧/ ٧٦ ± ٤ وضغط الأكسجين الشريانى ٩٤ ± ٣ مم زئبق بينما كان التأين الأيدروجينى الشريانى ٧٠٢٧٦ ± ٠٠٢٨ •

الاشراف

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قياسات ضغط الدم باستخدام تأثير دوبلر
فى الامراض الباطنة للحيوانات الصغيرة

رسالة مقدمة من

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بكالوريوس فى العلوم الطبية البيطرية - جامعة الزقازيق ١٩٧٨م

ماجستير فى العلوم الطبية البيطرية - تخصص طب عام

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للحصول على

درجة دكتور الفلسفة فى العلوم الطبية البيطرية

"الطب العام والعلاجى"

الى

كلية الطب البيطرى

جامعة الاسكندرية

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