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INTERRELATIONSHIPS OF THE PORCINE ELECTROCARDIOGRAM WITH ERYTHROCYTE AND PLASMA ELECTROLYTES

by

Dwight Bernard Coulter, $\mathcal{D} \vee \mathcal{M}$

A Dissertation Submitted to the Graduate Faculty in Partial Fulfillment of The Requirements for the Degree of DOCTOR OF PHILOSOPHY

Major Subject: Veterinary Physiology

Approved:

Signature was redacted for privacy.

In Charge of Major Work

Signature was redacted for privacy.

Head of Major Department

Signature was redacted for privacy.

Deah of Graduate Collegé

Iowa State University Ames, Iowa

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INTRODUCTION

With the introduction of a new subject a student may pass through three phases: the introductory phase where the new material seems overwhelming, the understanding phase where the information falls into a neat pattern, and the questioning phase where the student suddenly realizes that the information is incomplete and that available information is only as reliable as the method used to obtain the data from which his concepts were conceived.

The understanding of the body fluid electrolyte dynamics and the relationship of these dynamics to the electrocardiogram has not, in the case of the author, avoided the first of the above phases. Indeed the progress of the mental processes has varied with the particular aspect of the subject and after a review of the literature, the author perceives that he is not alone in his varied success.

An understanding of physical chemistry is necessary to develop concepts of electrolyte dynamics in a living organism. However, body fluids are not ideal solutions and there exists a lack of homogeneity due to the forces of metabolism which places every cell in a unique environment. Despite the problems, techniques useful to physical chemistry have lent themselves to eloquent investigations of electrolyte dynamics in various living cells. There should be some reservations as to the

meanings of these investigations, but the concepts that have evolved from such investigations have prepared the way for experimental work involving the intact animal with the aid of techniques that are repeatable in any well equipped clinical laboratory.

The experimental animal chosen for this investigation was the pig. The utilization of swine in biological research has increased in recent years. The increase has resulted largely from a growing awareness that swine and men have many biological similarities. The electrolyte composition of plasma and erythrocytes is similar in man and swine, thereby, further justifying the use of swine for this work.

Somatic cells, for instance those found in muscle, contain a greater amount of electrolytes than that found in plasma. Thus, a small fluctuation in somatic cell electrolyte content could greatly influence the plasma electrolyte content if it were not for the kidney and the fact that the electrolyte content for each type of cell is genetically set within certain limits.

Plasma, because of its availability and because it has been shown to be subject to variation of electrolyte concentration in certain pathological conditions, has been the fluid of choice in the study of electrolyte imbalance. Despite its usefulness, the use of plasma does have certain limitations. There exist ranges of electrolyte levels that are compatible with normal function and that are found in animals not in electrolyte

imbalance. Extremes, which lie outside these ranges, are often found only in the terminal stages of pathogenesis. As the plasma electrolyte concentrations approach these extremes, changes in electrocardiograms may warn of impending danger.

Is it possible that the electrolyte concentrations of some cells are a better indicator of impending electrolyte imbalance than plasma electrolyte concentrations or the electrocardiogram? Such cells ought to be readily available and of a homogeneous genetic makeup. Erythrocytes are as available as plasma and because they are not involved in impulse conduction, they might possibly be subject to a wider range of electrolyte content which could reflect electrolyte imbalance.

Experimental work involving the relationship of plasma to erythrocyte electrolytes has been done for some time and the relationship of plasma electrolytes to the electrocardiogram has been studied. Work involving the former has not been extremely rewarding. The erythrocyte electrolyte concentration values in the literature are variable for a given species and most reports involve a small number of samples. What is needed is a simple and precise method of determining the electrolyte concentrations in erythrocytes. Such a method would encourage the inclusion of erythrocyte electrolyte concentrations in clinical and research determinations. The author is not aware of investigations of the temporal relationships of plasma

electrolyte concentrations, erythrocyte electrolyte concentrations, and the concurrent electrocardiogram.

It is teleologically reasonable that the plasma, its composition influencing extracellular fluids, should be maintained within certain electrolyte levels by the kidney for normal function of cardiac cells. Therefore, plasma electrolyte concentrations do not necessarily reflect changes in total body content of electrolytes. On the other hand, the erythrocytes could undergo electrolyte changes without affecting cardiac cells and consequently, without affecting the electrocardiogram. There also are times, other than during electrolyte imbalance, when plasma electrolytes are not at their normal adult concentrations. During porcine fetal development the fetal plasma concentrations of sodium and chloride are lower and potassium higher than the corresponding maternal concentrations. In such a plasma environment, the fetal erythrocyte potassium concentration is lower than the maternal concentration. It is known from in vitro studies that the outside electrolyte concentrations affect the erythrocyte electrolyte pump. Do erythrocytes, when subjected to a changing electrolyte environment, adjust their intracellular electrolyte concentrations or maintain their electrolytes as genetically programed? If erythrocytes adjust to a changing environment in the plasma, erythrocyte electrolyte concentrations may give a better indication, at certain times,

of electrolyte distribution in swine than do the more routine plasma and electrocardiographic values.

This dissertation is concerned with the investigation of the temporal relationships in swine of changing plasma electrolyte concentrations, the concurrent electrocardiograms, and erythrocyte electrolyte concentrations. Normal values for erythrocyte electrolyte concentrations determined by an uncomplicated indirect method as well as the effects of fetal development and anemia on these values are established for comparative purposes.

LITERATURE REVIEW

Electrolytes in Erythrocytes

Methods and units

Erythrocyte electrolyte concentrations have been determined by gravimetric and volumetric means (Abderhalden, 1898), by flame spectrophotometry (Overman and Davis, 1947), and by emission spectrography (Valberg, 1964). The determination of erythrocyte water and electrolyte concentration is fraught with uncertainties, mainly due to the variations in the amount of trapped plasma (Czaczkes et al., 1967).

Basically there are two methods for determination of erythrocyte electrolyte content. The two methods are direct and indirect (Streef, 1939). In the indirect determination, plasma and whole blood are analyzed separately. The concentration of electrolytes in the cells is calculated from the packed cell volume and the difference between the analytical results yielded by the whole blood and plasma.

When the electrolyte concentration in the erythrocytes compared with plasma is small, the indirect method has the serious drawback that a slight error in the estimation of cell volume or in the electrolyte concentration in plasma or whole blood gives a considerable error (Streef, 1939). In addition, this method does not allow for the presence of white blood cells (Beilin et al., 1966). Also the use of serum values for the

indirect estimation of calcium is inadmissible, as there is likely to be a small difference in calcium content between serum and plasma (Streef, 1939).

The direct method may be subdivided into two types of procedures. One direct procedure involves determining the amount of trapped plasma and the other requires washing the erythrocytes. Czaczkes <u>et al</u>. (1967) point out that if a direct procedure is used, the packed erythrocytes should be sampled by weight, not volume, because of the viscosity of erythrocytes.

To avoid errors due to trapped plasma, erythrocytes may be washed prior to a determination (Beilin <u>et al.</u>, 1966). Washing may also introduce errors (Maizels, 1936). Czaczkes <u>et al</u>. (1963) concluded that the water and electrolyte content of the erythrocytes can be accurately determined by measuring the amount of trapped plasma in each blood sample.

Beilin <u>et al</u>. (1966) concluded that human erythrocyte sodium concentration can be measured with accuracy by the direct method if trapped plasma sodium is estimated with radioisotopes of sodium and a correction made for entry of sodium into the erythrocytes. The erythrocytes and plasma must be separated rapidly. Sucrose is used as a standard plasma marker, and the erythrocytes are washed with sodium-free solutions.

The use of a plasma trapping correction factor (Chien et al., 1965) would appear to increase accuracy. This would most likely not be true. A number of methods have been devised to

measure trapped plasma, but with ¹³¹I-labelled serum albumin as a marker, cell packing is not uniform in the individual, nor constant between individuals; even the simultaneous use of two marker substances does not give identical results for trapped plasma in the same erythrocyte mass (Coldman and Good, 1967). The most refined techniques are, therefore, not without inherent error.

Coldman and Good (1967) have been able to obtain comparative values by the use of indirect determinations of erythrocyte electrolytes. Because water bound to proteins is an integral component of the erythrocyte membrane ultrastructure, it is impossible to determine what portion of the apparent trapped water is properly a constituent of the cell surface. The practice of expressing erythrocyte electrolyte concentrations in terms of total cell water is also subject to the error of hydration (Coldman and Good, 1967).

Comparison of the data available in the literature is a difficult task. There is little uniformity in the number of determinations, breed and state of the animals, electrolytes determined, method of determination, and the units of measurement. Nevertheless, values are available for normal erythrocytes of man and animals (Kramer and Tisdall, 1922; Hoffman and Jacobs, 1934; Oberst, 1935; Maizels, 1936; Crabtree and Maizels, 1937; Kerr, 1937; Raszeja, 1937; Streef, 1939; Overman and Davis, 1947; Hutt, 1952; Bernstein, 1954; Evans and King, 1955;

Keitel et al., 1955; Barlow et al., 1956; McCance and Widdowson, 1956; Behrendt, 1957; Evans and Phillipson, 1957; Czaczkes, 1963; Freeman and Spirtes, 1963; Roughton, 1964; Duggan et al., 1965; Valberg et al., 1965a,b; Beilin et al., 1966; Gross and Wendel, 1966; Coldman and Good, 1967; Coulter and Swenson, 1967; Valberg et al., 1967).

In general the erythrocytes of mammals contain more potassium than sodium. There are, however, certain important exceptions (Abderhalden, 1898). The dog and cat erythrocytes contain much more sodium than potassium. Cattle, sheep, and goats may also have more sodium than potassium, but not to the extent seen in the dog and cat (Kerr, 1937). It is now known that the sodium to potassium ratio in sheep is genetically controlled and erythrocytes from an individual sheep are classified as low potassium or high potassium erythrocytes (Evans and King, 1955). It was first noted by Kerr (1937) that these two types of erythrocytes correlated with the breed of sheep. Low potassium erythrocytes contain more sodium than potassium and high potassium erythrocytes contain more potassium than sodium (Duggan et al., 1965). Little work has been done in birds, but it appears that the erythrocyte potassium content exceeds the sodium content (Kerr, 1937; Valberg et al., 1965a).

It is known that water bound to proteins (nonsolvent water) varies in amount for each erythrocyte constituent (Cook, 1967).

The relationship of erythrocyte osmolarity with variance in nonsolvent water and hydration makes interpretation of electrolyte values difficult.

The units in which erythrocyte electrolytes are expressed varies somewhat with the method. Some workers prefer analytical data expressed as weight per unit weight of dry matter; others prefer moles per wet weight or a moles per volume basis of measurement (Coldman and Good, 1967). Valberg <u>et al</u>. (1967) has expressed concentration per unit weight, per cell, per unit volume of cells, per unit volume of cell water, and per mole of hemoglobin. Coldman and Good (1967) find it inadvisable to claim exceptional precision for analytical data that do not allow for hydration and its consequences. As for most data, a given set of results refers only to the specific conditions under which they were obtained.

Man and animals

Work on the quantitative distribution of inorganic ions between red blood cells and blood plasma of man and animals has been done since the middle of the nineteenth century according to Kramer and Tisdall (1922) and Raszeja (1937). Abderhalden (1898) was the first to run detailed determinations of the contents of erythrocytes and serum for domestic animals. He found no calcium in the erythrocytes of domestic animals nor sodium in the erythrocytes of the horse, pig, or rabbit. While Abderhalden's data are similar to data from later

investigations, later investigations have been more accurate and values for calcium and sodium have been determined for most domestic animals and man. Abderhalden's work should be admired considering the techniques that were available to him at the time.

Fetal erythrocyte composition differs from adult erythrocyte composition. The sodium concentration is higher and the potassium concentration is lower in fetal erythrocytes than in adult erythrocytes, at least in man and pig (McCance and Widdowson, 1956) and in guinea pigs (Widdas, 1954). The concentration of chloride changes in the same direction as that of sodium. In sheep the fetal erythrocytes contain more potassium and less sodium than those of an adult (Widdas, 1954). For lambs whose mothers have a high sodium concentration in the erythrocytes, the transition to the erythrocyte electrolyte pattern of the adult is complete at 7-8 weeks postpartum (Blechner, 1961). The fact that new cells formed after bleeding are relatively high in potassium indicates, in sheep with high sodium erythrocytes, that the high sodium membrane develops relatively late in the cytodifferentiation (Tosteson, 1966). Karvonen (1958) discusses the effect of age of the animal on its erythrocyte electrolyte composition.

Equally important is the difference in electrolyte composition among erythrocytes within a given sample (Keital <u>et al</u>., 1955). A detailed analysis of the erythrocyte cation levels

during the development and maturation of rabbit erythrocytes has been made by Valberg et al. (1967).

Even more dynamic are the kinetics involving the chloride shift or Hamburger phenomenon. The rate of penetration of chloride and bicarbonate ions across the erythrocyte membrane is very rapid, so that the transfer of these ions is completed during each phase of the respiratory cycle (Van Slyke, 1926). Swine

Abderhalden (1898) cites Bunge, in whose laboratory Abderhalden worked, as having analyzed porcine blood in the year 1876. Bunge failed to find either sodium or calcium in porcine erythrocytes, as was the case with Abderhalden in later work. Bunge analyzed swine blood, serum, and erythrocytes for water, solids, hemoglobin, protein, potassium, sodium, iron, calcium, magnesium, chloride, phosphoric acid, as well as other inorganic and organic materials not identified specifically. Abderhalden analyzed porcine blood, serum, and erythrocytes for the above constituents plus sugar, cholesterol, lecithin, fat, fatty acids, nucleic phosphoric acid, and inorganic phosphoric acid.

Rona and Takahashi (1911) reported values, in contrast to Bunge and Abderhalden, for calcium in porcine erythrocytes. Their values are probably the first established for calcium in the porcine erythrocyte.

Kerr (1937) and Raszeja (1937) determined the amount of sodium in porcine erythrocytes. Kerr found 10.8 mM/1000 gm of corpusles and Raszeja reported 0.2059 mg/cm³ of corpusles (approximately 9 mM/1000 gm of erythrocytes). Thus, values for sodium in the porcine erythrocyte, which Bunge and Abderhalden failed to find, were established. Kerr used four pigs and Raszeja one. Kerr measured potassium and phosphorus in addition to sodium. Dill <u>et al</u>. (1930) had previously reported sodium in human erythrocytes as had other workers (Kramer and Tisdall, 1922).

Streef (1939) was the next to offer new values. He analyzed washed porcine erythrocytes for both sodium and calcium. Erythrocyte sodium ranged from 15 to 43 mg% (7 to 19 mEq/L), and erythrocyte calcium content averaged 0.95 mg% (0.5 mEq/L) when erythrocytes were washed in different solutions. Unwashed cells gave higher values.

Rapaport and Guest (1941) analyzed erythrocytes for acidsoluble phosphorus. Porcine erythrocytes were included in the different erythrocytes analyzed.

McCance and Widdowson (1956) studied the effects of age and severe undernutrition on the composition of porcine erythrocytes. As pigs pass through the fetal, newborn, and adult stages, McCance and Widdowson found that the concentration of water, sodium, and chloride fall as development occurs and

the concentration of nitrogen, hemoglobin, iron, potassium and phosphorus rise.

The distribution of sodium, potassium, and glucose in porcine erythrocytes has been reported by Coldman and Good (1967). In seven pigs they reported a mean of 15.6±1.8 mEq/L for sodium and 105.9±12.7 mEq/L for potassium in the erythrocytes.

Kirschner and Harding (1958) studied the effect of adenosine on the retention of phosphate esters and on sodium extrusion from porcine erythrocytes. They found that the incorporation of adenosine into swine blood during storage definitely increases both sodium extrusion and maintenance of labile phosphate esters. Further studies on sodium fluxes in swine erythrocytes were made by Sorenson et al. (1962). They compared swine erythrocytes (low sodium content) with bovine erythrocytes (intermediate sodium content) and with canine erythrocytes (high sodium content). Unidirectional sodium fluxes are slowest in swine erythrocytes, fastest in canine erythrocytes, and between these extremes in bovine erythrocytes. Pump to leak ratios are about 21 for swine and 3 for bovine erythrocytes, a difference that probably accounts for the higher erythrocyte sodium content in cattle. It was quite clear to Sorenson et al. (1962) that the sodium-potassium pump is most effective in swine erythrocytes and ineffective in canine

erythrocytes. Bovine erythrocytes are intermediate in their effectiveness.

Sheep, whose potassium content in erythrocytes vary from high to low depending upon the breed, have been studied in respect to cation transport (Tosteson and Hoffman, 1960). Both types of erythrocytes have a cation pump which exchanges one sodium ion from inside the cell with one potassium ion from outside the cell, but the pump is working approximately four times faster in the high potassium erythrocyte. The characteristics of the cation leak are also very different in the two erythrocyte types since the high potassium erythrocytes leak more sodium than potassium than is the case in low potassium erythrocytes. Porcine erythrocytes resemble high potassium sheep erythrocytes with regard to cation transport.

From an antomical point of view, little work has been done on porcine erythrocytes. Hoffman <u>et al</u>. (1956) found high density particles in electron micrographs of porcine red blood cell ghosts. High density particles are present in bat, camel, ox, mouse, sheep, and pig erythrocyte ghosts as well as in human erythrocyte ghosts with certain types of anemia, but not from normal human, groundhog, rat, and rabbit erythrocyte ghosts. Hoffman (1956) found that the surface ultrastructure of the erythrocyte ghosts of man, mouse, camel, ox, and pig are characteristically different from each other.

Electrolytes in Plasma

Man and animals

A review of serum electrolytes in man and their relationship to acid-base balance has been compiled by Nuttal (1965). A number of texts are available (Elkinton and Danowski, 1955; Davenport, 1958; Robinson, 1962; Weisberg, 1962; Christensen, 1964). As with many subjects, there exists a confusing array of terms.

The physiology of fluids and electrolytes in domestic animals has been discussed by Kare (1955). Fluids and electrolyte values in animals have been reviewed by Meir (1963). His chapter is a guide and a source of references in respect to animal body fluids. Methodology is discussed as related to the value of results.

Thalme (1966) has reviewed work concerning electrolyte and acid-base balance in fetal and maternal blood of animals and man. Thalme's experiments with rats are compared with previous findings in the literature. In rats the maternal potassium concentration in serum remains constant and significantly below the fetal level for the periods observed except on the last day of gestation when the maternal concentration reaches the fetal level. The level of sodium concentration is fairly constant and of the same magnitude both in rat fetuses and in their mothers. The fetal rat serum chloride levels are below the maternal level. Swine

A comprehensive review of the composition of body fluids in swine has been compiled by Swenson (1964). Changes in composition and content of body fluids are discussed in relation to the age of swine. The pig has one of the most rapid growth rates when compared with birth weight and this affects changes in body fluids.

Hewitt (1932) made certain chemical determinations, including the electrolytes calcium and phosphorus, on porcine serum from normal swine. His work has an extensive review of the literature up to that time.

Meyer <u>et al</u>. (1950) reported data on the sodium, potassium, and chloride content of plasma from pigs fed rations low in sodium and/or chloride. Their studies reveal that plasma sodium cannot be used positively to detect a sodium deficiency. The individual variation is so great that the differences are not statistically significant. Chlorine deficiency causes a significant decrease of plasma chloride. Coincident with severe sodium deficiency there is a significant rise of potassium in the plasma.

Widdowson and McCance (1956) compared the composition of serum and extracellular fluids in fetal, newborn, and adult swine. During fetal (40-50 days of gestation) and post-natal development, serum potassium falls and a small increase in serum sodium occurs. Extracellular potassium in the body decreases

from 20-30% in the fetus to 2% in the adult. In younger fetuses the extracellular potassium forms a still higher proportion of the potassium in the body.

Garner <u>et al</u>. (1957) described blood changes in piglets associated with weaning. Weaning is followed by a well-marked but transient increase in serum sodium concentration. This may be due in part to a sudden decrease in the water intake.

Cummings and Kaiser (1959) studied blood gases, pH, and plasma electrolytes of sows and fetal pigs at 106 days of pregnancy. Fetal plasma chlorides are lower than maternal values and fetal plasma bicarbonate exceeds maternal values. The sodium and potassium concentrations are about equal, according to Cummings and Kaiser (1959), in fetal and maternal plasma. On the other hand, Widdowson and McCance (1956) found higher potassium in the fetal plasma and lower sodium in fetal plasma as compared with adult plasma. Both pairs of authors agree upon the chloride relationship.

Hackett <u>et al</u>. (1960) have reported serum calcium and phosphate values. The pigs involved were newborn and up to four years of age.

Ullrey <u>et al</u>. (1967) have accumulated values for serum taken from swine from the time of birth to maturity. They reported values for calcium, magnesium, sodium, potassium, copper, zinc, and inorganic phosphorus. Sodium and potassium concentrations tend to vary together from one sampling to the

next. Sodium and potassium concentration curves, when plotted against age, are very irregular. Within age correlations are low and nonsignificant.

Effects of Disease on Blood Electrolytes

Erythrocytes

Hoffman and Jacobs (1934) investigated the distribution of potassium between serum and erythrocytes in health and disease. In general, they felt that the levels of both serum and erythrocyte potassium are constant in health and disease for man.

Maizels (1936) found that cation, anion, and water contents are near normal in macrocytic anemias and in myelosclerosis. In microcytic anemias, the cation and water contents are increased, but the cation concentration is only slightly raised. The concentration of erythrocyte cations is thus relatively constant in spite of great variation in concentration of hemoglobin and despite wide variation in blood regeneration and destruction. In acholuric family jaundice, Maizels (1936) found erythrocyte potassium and water low, hemoglobin per unit volume increased, and sodium content normal.

Hutt (1952) came to the conclusion that the erythrocyte potassium level appears to reflect the direction of change in body stores of potassium. In nineteen patients with disturbed potassium metabolism, Hutt noted that the plasma potassium

level does not necessarily reflect erythrocyte potassium concentration. Also some patients had normal potassium levels in erythrocytes and plasma, yet had abnormally low serum sodium levels.

McCance and Widdowson (1956) investigated anemia in man and found potassium, water, and phosphorus content elevated, a lowered concentration of nitrogen, hemoglobin, and iron, but no alteration of sodium concentration in the erythrocyte.

Behrendt (1957) summarized the clinical aspects of erythrocyte chemistry. He states that no general rule can be laid down for the behavior of erythrocyte sodium under pathological conditions. Erythrocyte sodium concentrations, in his opinion, do not respond in uniform fashion to sodium imbalance or to a rise or fall in plasma sodium levels. However, abnormal variations in sodium levels of erythrocytes may be reasonably examined on an empiric basis in relation to a specific condition. Behrendt discusses the relationship of erythrocyte electrolytes to some conditions such as anemia, diabetic acidosis, familial periodic paralysis, and infant diarrhea.

Behrendt (1957) did not consider any range of erythrocyte potassium as an attribute of clinical disease states. However, certain distributions of potassium between erythrocytes and plasma are frequently associated with certain metabolic pathology. In studies of anemia, Behrendt states that the calculation of the mean erythrocyte potassium and sodium content is mandatory.

Spurr and Barlow (1959) studied plasma and erythrocyte sodium, potassium, chloride, and water in hypothermic and hyperthermic dogs. Induced respiratory alkalemia in both normothermic and hypothermic dogs appears to result in a shift of sodium from plasma into erythrocytes. The plasma and erythrocyte electrolyte changes in hyperthermic dogs are all in the direction of an increase. These increases are probably the result of reduction in plasma and erythrocyte water content.

Czaczkes et al. (1967) determined the quantity of erythrocyte sodium, potassium, and water in human patients suffering from cirrhosis of the liver, from chronic renal failure, and congestive heart failure. Deviations from the normal values are found especially in erythrocyte sodium content. Most of the patients with chronic renal failure and with cirrhosis of the liver had a lower erythrocyte sodium content. In patients suffering from congestive heart failure, an increased frequency of deviation of the erythrocyte sodium values in either direction is observed in severe stages of the condi-In mild and moderate congestive heart failure there is tion. an increased frequency of low erythrocyte sodium. In severe congestive heart failure normal results were obtained in only 2 of 29 cases. In 16 cases the sodium content was low and in 11 cases high. No correlation is found between sodium content of erythrocytes and that of plasma. Czaczkes et al. (1967)

stated that it remains doubtful to what extent the total intracellular sodium is represented by the erythrocyte sodium content.

Blunt and Evans (1965) investigated the sodium and potassium content of sheep erythrocytes during an experimental anemia. In anemic sheep the concentration of potassium in the erythrocytes increases. The sodium content of erythrocytes decreases slightly. These changes occur in sheep with low potassium levels in the erythrocytes as well as those with high potassium levels in the erythrocytes. In all probability, the increases in the concentration of potassium in the total cell population is due to an influx of younger cells. When sheep with low potassium concentrations in their erythrocytes are subjected to massive hemorrhage, the cells formed during subsequent rapid hematopoiesis contain substantially more potassium than do the cells which are released into the circulation under normal conditions (Evans and Blunt, 1963).

Apparently, the only work done with erythrocyte electrolyte content in the pig during a pathological condition was by McCance and Widdowson (1956). Young pigs subjected to prolonged undernutrition have a lower concentration of hemoglobin, nitrogen, and iron in their erythrocytes than do animals of a comparable age, and they also have a higher concentration of water and to a certain extent potassium.

Plasma

Bland (1963) has published a text on clinical metabolism of body water and electrolytes in man. The main aim of the

text is to emphasize clinical medicine as it relates to research involving water and electrolyte metabolism. Statland (1963) has published a text that encompasses the abnormalities of body fluid volume and electrolyte concentrations.

Roy <u>et al</u>. (1959) investigated the effect of bacterial diarrhea on the sodium and potassium concentration in the serum of newborn calves. With increased occurrence of diarrhea, serum sodium levels decrease and there is a slight increase in potassium levels.

Dalton <u>et al</u>. (1965) investigated the effects of diarrhea in 60 experimental calves. Changes in the packed cell volumes occurred infrequently. There is a significant incidence of hyponatremia, hypokalemia, and hypochloremia in diarrheic calves. A few calves had raised plasma potassium concentrations.

Fitzgerald (1967) found that in calves with coccidiosis the changes in serum sodium and potassium concentration are minor unless there are severe signs of coccidiosis. About 6 to 8 hours before death, the potassium levels rise and the sodium levels decrease.

Tasker (1967) found that in horses with diarrhea the plasma sodium is reduced and the plasma potassium is markedly reduced. Most of the sodium is lost in the feces. The kidney is unable to conserve potassium.

Relatively little clinical work has been done with water and electrolyte metabolism in swine. The values of serum

calcium and phosphorus in hog cholera-infected swine have been reported by Hewitt (1932). Serum calcium is increased in hog cholera-infected swine.

The effect of transmissible gastroenteritis upon feed consumption, water, nitrogen, sodium, and potassium balance of 6 young pigs was investigated by Reber and Whitehair (1955). Serum sodium is not altered by transmissible gastroenteritis. Infected pigs appear to be able to retain sodium, but serum potassium levels are lowered in infected pigs. Infected pigs appear to have a negative potassium balance.

McCance and Widdowson (1958) investigated the effects of potassium chloride in solution, administered by stomach tube, on newborn piglets. When water and potassium chloride are administered together, the potassium is progressively retained and paralysis occurs. In addition, the blood sugar rises. Piglets given potassium chloride in milk retained a mild excess of potassium, but pigs growing normally with their concurrent rapidly expanding water compartments are completely protected from toxic effects.

Coulter and Swenson (1968) studied serum sodium and potassium in iron-injected and iron-deficient swine at one, two, and three weeks of age. Serum sodium is at the same level in both groups, but a significant difference, not out of the physiological range, is apparent for serum potassium at one and three weeks of age with the iron-deficient group being lower.

Coulter and Swenson (1968) also followed serum potassium and sodium levels in three week old pigs injected intravenously with endotoxin. A decrease in serum potassium occurs during the first hour after injection of endotoxin. An increase in serum potassium occurs eight hours after injection of endotoxin. The only effect of endotoxin injection on serum sodium is a decrease in concentration fifteen minutes after injection. The fluctuations are in the same direction for both iron-injected and iron-deficient swine. All of the fluctuations are within the range of concentrations of the normal control swine. Thus, no pathological signs can be attributed to fluctuations of serum sodium and potassium after injection of endotoxin.

Changes in electrolyte levels, blood pH, packed cell volume, and blood urea nitrogen were studied in baby pigs with transmissible gastroenteritis by Cornelius <u>et al</u>. (1968). Plasma sodium decreases in those pigs left with their dams, but there is no significant change in the plasma sodium levels of isolated pigs. The only significant changes in serum potassium levels occur in those pigs which die, where a marked rise occurs on the day of death. This hyperkalemia is probably coupled with abnormal cardiac function. Plasma chloride levels rise only in isolated pigs. Availability of drinking water greatly influences electrolyte levels in the plasma.

S.,
Electrocardiography

Swine

Wintrobe et al. (1943) recorded the electrocardiographic changes associated with thiamine deficiency in pigs. Normal electrocardiograms were included in his study. Thiamine deficiency is associated with pronounced electrocardiographic changes. The electrocardiographic changes are probably the expression of disturbance in metabolism which is caused by a lack of thiamine. Inanition alone causes electrocardiographic changes but not to the extent seen in thaimine deficiency. Ellis and Madsen (1944) also conducted electrocardiographic studies on a few pigs that were thaimine-deficient. The electrocardiogram of one pig indicated hypertrophy of the right ventricle. Miller et al. (1957) recorded electrocardiograms weekly from birth through 5 weeks of age on 35 pigs receiving various subminimal and adequate levels of thiamine in the diet. Duration of electrocardial intervals were established in normal and thaimine-deficient pigs. Statistically significant increases in P-R time, Q-T time, and cycle length occur in deficient Thiamine-deficient pigs consistently exhibit sinus pigs. arrhythmia, and there is evidence of cardiac hypertropy from the electrocardiograms.

Platner <u>et al</u>. (1948) made an effort to establish "normals" for electrocardiographic values of domestic animals. Six pigs,

60-90 days of age, were recorded from in a standing position. In swine the T wave is inverted in about 80% of the instances in the limb leads.

Hamlin (1960) established the ventricular activation process of normal pigs by estimating qualitatively body surface potentials and epicardial electrograms. The ventricular activation process in the pig is similar to that accurately described in the dog.

Engelhardt (1963) recorded the systolic blood pressure, heart sounds, and electrocardiograms in pigs at rest and after exercise. Diastole-systole time ratios fall after exercise. These ratios are correlated with heart rate.

Thielscher (1966) studied the influence of controlled exercise on the electrocardiogram of pigs of different breeds. A significant lengthening of almost all sections of the electrocardiogram, with reduced heart rate, occurs as bodyweight is increased. The effect of training is best seen by an increased diastole-systole quotient. It is possible that differences between families of the same breed might exceed those between different breeds.

Relationship to electrolytes

Winkler et al. (1938) recorded serial electrocardiograms in dogs injected intravenously with potassium chloride in isotonic solution. Blood samples were withdrawn at intervals throughout the experiments to permit determination of potassium

concentration in the serum. Alterations in waveforms were recorded at increasing concentrations of potassium in the serum. There is a critical concentration of potassium in the plasma at which the heart stops. If the concentration of potassium in the plasma rises and then falls, there is no delayed toxic action. Cardiac arrest should occur independently of rate of injection or the total amount of potassium given, except insofar as these factors combine to produce the necessary rise in the potassium concentration in the plasma.

Winkler <u>et al</u>. (1938) noted high toxicity of potassium administered intravenously as compared with its benign character when given orally. Laragh and Capeci (1955) found that in sodium-depleted dogs, the capacity to remove potassium given orally is limited. Potassium chloride given orally is not diuretic.

Ono <u>et al</u>. (1964) studied the effects of different levels of dietary potassium on the electrocardiogram and serum electrolytes in young dogs. When serum potassium levels decreased to 4.0 mEq/L or less, the T wave is usually decreased in height or inverted, and the U wave is frequently broadened. On the other hand, when the serum level rises above 6.0 mEq/L, the T wave is consistently peaked and increased in height. There is divergence of opinion regarding the degree of correlation between the electrocardiographic changes and the serum potassium levels.

Surawicz <u>et al</u>. (1967) studied the hemodynamics and electrocardiographic effects of hyperpotassemia in dogs. Differences in response of the electrocardiogram to slow and rapid increases in concentration of plasma potassium as a result of intravenous infusion were noted. Changes in intracardiac pressures, cardiac output, and left ventricular contractile force were also noted. A threefold increase in the QRS duration accompanying hyperpotassemia has no deleterious effect on the pressure, stroke volume, and the force of left ventricular contraction.

Surawicz (1967) reviewed the relationship between the electrocardiogram and electrolytes. Abnormal concentrations of sodium, magnesium, and hydrogen ions are seldom recognizable in the electrocardiogram. The electrocardiogram is a fairly sensitive indicator of changes in plasma concentration of potassium and calcium. The electrocardiogram does not reflect the changes in total body potassium unless the plasma potassium concentration is altered. Patients with large deficits of total body potassium have normal electrocardiograms as long as their plasma potassium concentration is normal. Despite the difficulty of predicting concentrations of plasma potassium and plasma calcium from the electrocardiogram, the electrocardiogram can be used to detect electrolyte abnormalities, especially when control tracings are available and the patient can be monitored with serial tracings.

Bradlow and Levin (1969) discussed the effects on the electrocardiogram of combined abnormal serum potassium and calcium levels in human patients. They claim that the effect of combinations of moderate to severe serum abnormalities of both electrolytes can frequently be diagnosed on the electrocardiogram. Marked changes in serum levels of either electrolyte may obscure electrocardiographic changes due to minor serum changes of the other electrolyte.

Jensen <u>et al</u>. (1961) studied the response of young pigs to different levels of dietary potassium. The electrocardiogram indicated marked cardiac impairment even though the necropsy revealed no pathological lesions directly attributable to the potassium deficiency. The electrocardiogram showed changes in the intervals and amplitudes of electrocardiographic waves. Changes in the conduction time of impulses within the heart need not be accompanied by histological lesions.

Cox <u>et al</u>. (1966) evaluated electrocardiograms of young pigs on potassium-deficient diets. Pigs receiving a potassiumdeficient diet have electrocardiographic abnormalities within 2 weeks. Abnormalities include reduced heart rate, increased wave intervals, arrhythmia, and atrioventricular heart block. Pigs fed an adequate diet in an amount equal to the voluntary intake of pigs on the deficient diet also exhibited a reduction in heart rate. Electrocardiograms of pigs receiving an adequate diet showed little change over a period of 4 weeks.

MATERIALS AND METHODS

Experimental Design

Swine

Comparisons of maternal and fetal blood electrolyte concentrations were made in eight sows and their fetal litters. The sows, which were 1/4 Landrace, 1/4 Yorkshire, and 1/2 Poland China and bred by boars of the same hybrid, weighed approximately 160 kg. The sows were selected at 67, 77, 86, 87, 91, 109, 110, and 111 days of gestation and were 32, 29, 24, 38, 22, 24, 24, and 24 months of age, respectively. Surgery was performed in the months of February and March, 1968. They had been fed the following ration:

ground corn	82.85%
50% soybean meal	13.50%
calcium carbonate	0.80%
dicalcium phosphate	1.75%
iodized salt	0.50%
trace mineral premix	0.10%
vitamin premix	0.50%

When added at 0.1% of the diet the trace mineral premix provides the following in ppm: manganese, 100; zinc, 100; iron, 100; copper, 10; cobalt, 1; and iodine, 3. When added at 0.5% of the diet the vitamin premix provides 750 IU of vitamin A, 300 IU of vitamin D, 2.0 mg of riboflavin, 4.0 mg of pantothenic

acid, 9.0 mg of niacin, 10 mg of choline, and 10 ug of vitamin B_{12} per pound of feed. The ration was fed at 5 pounds per sow per day.

Pigs used to establish representative values of hematologic and electrolyte data were 1/4 Landrace, 1/4 Yorkshire, and 1/2 Poland China. The sixty pigs weighed 18-24 kg and were 9 weeks old. The first thirty pigs listed in Tables 2 and 3 were bled in August, 1968 and the last thirty pigs were bled in January, 1969. They were fed the following ration:

ground corn	78.25%
50% soybean meal	18.50%
calcium carbonate	0.90%
dicalcium phosphate	1.25%
iodized salt	0.50%
trace mineral premix	0.10%
vitamin premix	0.50%

The trace mineral premix and vitamin premix were the same as those in the ration described for sows in the maternal and fetal blood electrolyte study.

Pigs used to compare electrolyte and hematologic data in anemic and normal pigs were Yorkshire and Hampshire hybrids. They weighed 5-18 kg and were 7-8 weeks old. The normal pigs had been injected with 150 mg of iron-dextran intramuscularly at 2 days of age and again at 12 days of age. Seventeen of the 31 pigs were anemic (Table 4). The anemic and normal pigs were fed the following ration:

ground corn	46.5%
ground oats	20.0%
wheat bran	5.0%
44% soybean meal	16.5%
dried whey	10.0%
dicalcium phosphate	1.0%
iodized salt	0.5%
vitamin premix	0.5%

The vitamin premix contained the following per pound of premix:

vitamin A	200,000	USP units
vitamin D	50,000	USP units
riboflavin	500	mg
calcium dl-pantothenate	2,400	mg
niacin	2,000	mg
vitamin B ₁₂	2	mg

Pigs used for infusion of isotonic KCl solutions were 1/4 Landrace, 1/4 Yorkshire, and 1/2 Poland China. They weighed approximately 16 kg and were about 8 weeks old when purchased. Upon arrival they were placed in a 10 by 40 by 20 inch crate with a wire floor. Pigs spent 7-10 days in the crate prior to infusion. The ration was the same as that fed anemic and normal pigs. Prior to purchase the pigs were fed the ration described for the pigs used to obtain representative values. Male pigs in the representative group, the anemic and normal group, and those used for infusion of isotonic KCl solutions had been castrated at 3-5 days of age.

Pigs for the infusion experiments were numbered 1 through 18. Pigs 1-16 were infused with an isotonic KCl solution. Pig number 16 was infused with an isotonic NaCl solution 2 days following the KCl infusion. Pig number 12 was investigated for the effects of time and blood sampling 5 days prior to KCl infusion. Pig number 17 was infused with an isotonic glucose solution on December 4, 1968. The effects of sodium pentobarbital on the electrocardiogram were investigated on pig number 18. The infusion dates of pigs 1-16 are listed in Table 7.

Infusion of isotonic potassium chloride

Pigs were placed in a canvas sling for infusion. An initial blood sample was drawn if the venous cannula (femoral vein) was patent, and an electrocardiogram recorded. The femoral cannulas often became plugged upon aspiration. This was due to fibrin curtains at the end of the cannulas, but the fibrin curtains always allowed injection or infusion into the vein. The pig was then lightly anesthetized via the femoral cannula with sodium pentobarbital, a venous cutdown performed (external juglar vein), and another blood sample drawn followed by the recording of an electrocardiogram. Infusion was then begun.

An isotonic solution of potassium chloride (11.2 gm KCl per liter of ion-free water) was injected into the femoral vein via a cannula at a rate of 3.82 ml per minute in 50 ml

portions. The solution contained 150 mEq/L of potassium. Infusion took 13.1 minutes, followed by five minutes which allowed for blood sample withdrawal, recording of the electrocardiogram, and refilling of the infusion pump for the next infusion. These 16-18 minute cycles were repeated until cardiac arrhythmia (partial heart block), at which time infusion was discontinued and a blood sample withdrawn and an electrocardiogram recorded. Cardiac arrhythmia was detected by monitoring the electrocardiogram at 1.5 mm per second. Five minutes after cessation of infusion the electrocardiogram was monitored for return of the P wave. Blood samples were drawn every 15-20 minutes until the P wave returned at which time a blood sample was withdrawn and an electrocardiogram recorded.

There were a few occasions during infusion when the electrocardiogram indicated, confirmed later by plasma K determinations, that the plasma K concentration was dropping from previous increments from prior infusions at 3.82 ml per minute. When this occurred, the infusion rate was doubled (7.64 ml/minute) until the plasma K concentration, as indicated by the electrocardiograms, began to elevate.

For comparative purposes isotonic NaCl (140 mEq/L of Na) and isotonic glucose (54 gm/L) solutions were infused. Isotonic NaCl was infused into one pig at 7.64 ml per minute in 50 ml portions with 4 minutes allowed for blood sample withdrawal, if drawn, and recording of the electrocardiogram after each

infusion. Eight hundred fifty milliliters were infused. Blood samples were drawn every other infusion cycle. Ten blood samples were drawn. Isotonic glucose was infused into one pig at 3.82 ml per minute in 50 ml portions with 5 minutes allowed for blood sample withdrawal and recording of the electrocardiogram after each infusion. Twelve blood samples were drawn. Five hundred fifty milliliters of isotonic glucose were infused.

The effects of time and sampling on the hematologic, electrolyte, and electrocardiographic data of a pig were investigated. Blood samples were drawn at 20 minute intervals accompanied by the recording of electrocardiograms. Twelve blood samples were drawn.

Surgical Procedures

Pregnant sows used in the comparative studies of maternal and fetal blood were fasted 12 hours prior to surgery, and anesthetized with thiamylal sodium (50 mg/ml) injected into an ear vein. An anesthetized sow was placed on her back in a metal sling and prepared for surgery. A surgical level of anesthesia was maintained with halothane in a closed inhalation system¹ (model #412A modified for halothane). A face mask was used. A midline abdominal incision was made and the uterus

¹Ohio-Heidbrink, Ohio Chemical & Surgical Co., Division of Air Reduction Co. Incorp., Madison, Wisc.

isolated. Blood samples were drawn from the uterine artery and vein. The uterus was then incised and as the pigs were removed from the uterus, blood samples were taken from each pig's umbilical artery and vein.

Pigs to be prepared for infusion were anesthetized, after a 12-hour fast, with sodium pentobarbital injected into the anterior vena cava and then injected with atropine sulfate (0.65 mg) subcutaneously. Anesthesia was maintained with ether in a veterinary anesthesia apparatus¹ (model #950). A face mask was used.

The cannulas were made from silicone rubber tubing² with an inner diameter of 0.050 inches, outer diameter of 0.094 inches, and a wall thickness of 0.022 inches. The tubing was cut into 50 cm lengths and a stainless steel suture tied around each piece of tubing and cemented³ in place 13 cm from one end. A cannula was placed 13 cm into the femoral vein and triple 0 silk ligatures tied around the vein and cannula and around the distal portion of the vein. The stainless steel suture that had been wrapped around the cannula and cemented in place was passed through muscle adjoining the vein and tied. The portion of the cannula distal to the stainless steel suture

¹Ibid.

²Ronthor Silatube, Ronthor Reiss Corp., 111 Fourth Ave., New York, N.Y.

³Silastic Medical Adhesive, Silicone type A, Dow Corning Corp., Midland, Mich.

was passed under the skin, with the aid of a large needle, and brought to the outside on the pig's side about 10 cm anterior to the tuber coxa. The cannula was filled with sodium heparin (200 units per ml) prior to the cannulation. A small metal plug was placed in the cannula's open end to prevent a back flow. Adhesive tape was placed over the exposed cannula and plug. This cannula was used for subsequent anesthetizing and potassium chloride infusion.

Prior to infusion the pig was anesthetized, after a 12hour fast, with sodium pentobarbital through the cannula in the femoral vein and a cutdown performed on the external juglar vein. A sodium heparin (200 units per ml) filled cannula placed in the external jugular vein was used for the withdrawal of blood samples during infusion with potassium chloride. The cannula placed in the external jugular was withdrawn after infusion; whereas, the femoral vein cannula, which was placed in the vein 3-10 days prior to infusion, remained in the pig.

Electrocardiography

Electrocardiograms were recorded with a Grass model 7 Polygraph.¹ Three electrocardiogram pre-amplifiers (model 7P6A) were used. The pre-amplifiers were calibrated at one millivolt per centimeter. The time constant was set at 2.5 seconds and a

¹Grass Instrument Co., Quincy, Mass.

half-amplitude low frequency of 0.04 cycle per second.

The driver amplifiers were calibrated for 50 millivolts per centimeter with a half-amplitude high frequency of 75 cycles per second. Driver amplifiers were model 7DAB.

Leads I, II, III, aVR, aVL, aVF, and a precordial lead were recorded at a paper speed of 30 and 60 mm per second. Respiration was recorded from the electrocardiogram at 1.5 mm per second paper speed or actually counted and recorded if it did not show on the electrocardiogram. Leads I, II, and III were recorded simultaneously and the others separately to prevent reduction in amplitude.

Electrodes were Grass¹ E-2 platinum needle electrodes placed subcutaneously approximately one centimeter anterior to the tuber calcis on the lateral side of the hindlegs and approximately one centimeter anterior and one centimeter ventral to the olecranon on the lateral side of the forelegs. The precordial electrode was placed approximately four centimeters posterior and four centimeters dorsally of the olecranon on the left side of the chest. The ground electrode was placed dorsally on the neck just posterior to the ears. The pig was suspended in a canvas sling during the recording. Sixty cycle per second filters on the driver amplifiers were used only in the event of 60 cycle interference. Interference was infrequent.

l_{Ibid}.

Diphasic waves in Table 11, usually the P wave in swine, are not designated as such. The larger of the two waves was measured and reported as a monophasic wave.

Analytical Procedures and Hematology

Blood from sows and their fetal pigs was taken from uterine and umbilical vessels, respectively. When insufficient blood was obtained from individual fetal pigs (67, 77, and 91 days of gestation) the blood was pooled into one arterial and one venous sample (Table 1). Blood samples were taken from the anterior vena cava of representative pigs, anemic pigs, and normal pigs. Blood samples were taken from cannulated external jugular veins of anesthetized pigs in the infusion experiments. Blood was drawn from the cannula in the femoral vein in unanesthetized pigs in the infusion experiments. Three milliliters of blood were drawn and disposed of prior to drawing of the blood sample from cannulas. Twelve milliliters of blood were drawn for each sample. Disposable plastic syringes were used in all experiments.

Ten microliters of sodium heparin solution (1,000 units/ ml) were used per ml of freshly drawn blood. The packed cell volume was determined by the microhematocrit method. Plain capillary tubes were employed. Samples were run for five minutes at 12,500 revolutions per minute. The packed cell volume was read by means of a light under a frosted glass, a

millimeter ruler, and calculated with a slide rule. Three hematocrit tubes were run per sample. It two of the three tubes did not match, another was run. When two tubes matched, that value was recorded. Two readings were recorded per hematocrit tube, one was read at the top of the erythrocytes, and the other at the top of the leukocytes.

The hemoglobin determinations were done in duplicate by the cyanmethhemoglobin method.¹ The average of the two determinations was recorded if the two readings were within 0.2 gm hb/100 ml. Further determinations were made, if necessary, until two readings matched within the tolerance.

Enumeration of erythrocytes was accomplished by using pipettes with 1% accuracy and a glass counting chamber. Saline (0.9%) was used for the diluting fluid. Two counts were made per sample. If the counts did not match within 200,000 cells per cubic ml, further counts were made until two readings matched within the tolerance.

Plasma and whole blood samples were covered with mineral oil and frozen in plastic culture tubes for later analyses. Tubes were agitated thoroughly after being thawed.

Plasma sodium and potassium were estimated on a twochannel AutoAnalyzer² simultaneously from the same sample. The

¹Method described in pamphlet revised 1962, Hycel Inc., Houston, Tex.

²Technicion Corp., Tarrytown, N.Y.

samples were not diluted or otherwise prepared before starting the determinations. The proportioning pump automatically made the correct dilution. Lithium was used as an internal standard. Two light-sensitive cells, located in the flamephotometer, measured sodium on one channel and potassium on the other. Lithium was measured on a third light-sensitive cell and the ratio of sodium or potassium to lithium, depending upon the channel, was recorded on paper by pens driven by voltage changes from the light-sensitive cells.

In the procedure the undiluted sample merged with an airsegmented stream of acid lithium nitrate diluent and was dialysed. The recipient stream from the dialyzer was debubbled and entered the flame of the flamephotometer. Standards were made from sodium chloride and potassium chloride.

Total carbon dioxide and chloride in plasma were simultaneously estimated with a two-channel AutoAnalyzer. Plasma samples were diluted by the proportioning pump with an acid solution to liberate carbonate, bicarbonate, and dissolved carbon dioxide into a carbon dioxide-free air segment. The diluted sample stream entered the dialyzer and chloride passed into the recipient stream.

After leaving the dialyzer, the sample stream was mixed with an anti-foam agent. This stream then entered a liquid-gas separator. The gas phase was resampled and used to segment a weakly alkaline buffered phenolphthalein indicator solution.

As the gas was absorbed into the solution the pH decreased and the indicator color decreased as a result. The color change was measured in a colorimeter and recorded on the recorder chart (Skeggs, 1960). Total CO₂ was recorded in mEq of bicarbonate.

The recipient stream from the dialyzer was mixed with a chloride color reagent. The chloride ion reacted with the mercuric ion in $Hg(SCN)_2$ to form $HgCl_2$ and released (SCN)⁻ which reacted with Fe⁺⁺⁺ to form the red complex Fe(SCN)₃ (Zall <u>et al.</u>, 1956). The developed color was recorded from the colorimeter in the recorder channel opposite the channel recording total CO₂.

Simultaneous determinations of plasma calcium and inorganic phosphorus were made with the AutoAnalyzer also. Plasma samples were diluted by the proportioning pump and dialyzed into an air-segmented stream of HC1. Phosphorus was determined on the first dialysate by adding an acidic solution of ammonium molybdate. The phosphomolybdic acid formed was reduced by 1amino-2-naphthol-4-sulfonic acid. The reaction mixture was passed into a heating bath. The resultant blue color, proportional to the amount of inorganic phosphate present, was measured by a colorimeter and recorded on the recorder chart (Fiske and Subbarow, 1925). Calcium was determined from the second dialysate. Calcium was dialysed against an air-segmented stream of cresolphthalen dye solution. A colored complex of

calcium and dye was formed. The color was measured by a colorimeter and recorded (Kessler and Wolfman, 1964).

The automated glucose procedure was adapted from the method of Hoffman (1937). Samples of plasma were diluted by the proportioning pump with saline and dialyzed into an airsegmented recipient stream of alkaline potassium ferricyanide. Glucose was determined by passing this recipient stream through a heating bath and then measuring the loss of color when yellow potassium ferricyanide was reduced to colorless potassium ferrocyanide. Color remaining after the reaction was measured in a colorimeter and recorded on the recorder chart.

Sodium and potassium measured for calculation of erythrocyte sodium and potassium was measured in diluted¹ whole blood (1 part blood and 19 parts ion-free water). One and nine-tenths milliliters of ion-free water were added to 0.1 ml of whole blood.

Chloride measured for the calculation of erythrocyte chloride was measured in diluted whole blood (1 part blood and 1 part ion-free water). One milliliter of whole blood was added to 1 ml of ion-free water.

Calculations for erythrocyte sodium and potassium were made as described by Coulter and Swenson (1967). Calculations for chloride concentrations in the erythrocytes made use of

¹Auto-Spenser, Warner-Chilcott Laboratories Instrument Div., Morris Plains, N.J.

the same equation. Calculations for erythrocyte sodium and chloride included the packed cell volume value read at the top of the white blood cells, except in Table 1, where erythrocyte potassium content calculations utilized the reading taken from the top of the red blood cells. The equation is:

$$\left[a - \left(\frac{100 - PCV}{c \times 100} \times d\right)\right] \quad \frac{c \times 100}{PCV} = \begin{array}{c} \text{electrolyte content} \\ \text{of the erythrocyte} \\ \text{in mEq/liter of cells} \end{array}\right]$$

a = mEq/L of electrolyte in diluted whole blood

- c = dilution factor
- d = mEq/L of electrolyte in plasma
- PCV = packed cell volume

Diluted whole blood sodium was estimated with 2, 4, 6 and mEq/L standards on a channel set for potassium, but with a sodium filter. Diluted whole blood potassium required no adjustment of the AutoAnalyzer. Diluted whole blood chloride was determined with 25, 40, 55, and 70 mEq/L standards instead of the standards covering the plasma range of chloride values. Determination of diluted whole blood chloride required no adjustment of the AutoAnalyzer.

Statistical Analysis

The variables in Table 1 were used to compute means and standard errors for each variable for the sows (arterial and venous blood data combined), for pigs (arterial and venous blood data combined), for sow arterial blood, for sow venous blood, for pig arterial blood, and for pig venous blood.

The data were then computed into means and standard errors for each separate litter. Again the data were separated for computation into sow and pig values and also by vessels (arterial and venous values).

Correlation coefficients were computed for the first 7 variables in Table 1 and the 3 erythrocyte electrolyte values were individually correlated with the first seven variables. Regression coefficients, with time as the dependent variable, were computed for all variables, The data were separated for computing into two groups, one for sows and the other for pigs.

For comparison of sows with pigs and arteries with veins within pigs an analysis of variance was performed. This analysis yielded standard deviations for each variable for all data and as separated for computation for sows, for pigs, and for vessels within sows and pigs. F tests (variance ratios) were employed to test for significant differences between sows and pigs and between arteries and veins.

Data from representative pigs were used to establish means and standard deviations for normal pigs. The data were determined for comparison with data from other pigs in this investigation. Correlation coefficients were computed for all the variables measured in Tables 2 and 3. Plots were made

when the correlation coefficients indicated that the plots would illustrate an important relationship between two variables.

Data from anemic and normal pigs were used to determine the effect of anemia on electrolyte values. Means and standard deviations were computed for the two groups and tested for significant differences. Correlation coefficients were computed for all the variables measured in Tables 4 and 5. Plots were made when the correlation coefficients indicated that the plots would illustrate an important relationship between variables.

Correlation coefficients were computed for all variables in the data for infused pigs (Tables 9, 10, and 11). Plots were made when the correlation coefficients indicated that the plots would illustrate an important relationship between variables. The mean concentrations and their standard deviations of plasma potassium were recorded for the different electrocardiographic changes (Table 12).

RESULTS AND DISCUSSION

Electrolytes in Porcine Erythrocytes

Sow erythrocytes (arterial and venous) had a mean erythrocyte sodium (RBC Na) concentration of 19±7 mEq/L; whereas, the fetal RBC's had a mean RBC Na concentration of 15±9. There were 12 samples from 6 sows and 64 samples from 6 pooled samples (3 litters) and 26 fetal pigs (Table 1).

Sow RBC's (arterial and venous) had a mean RBC K concentration of 124±12 mEq/L; whereas, the fetal RBC's had a mean concentration of 115±14. There were 15 samples from 8 sows and 66 samples from 6 pooled samples (3 litters) and 30 fetal pigs.

Sow RBC's (arterial and venous) had a mean RBC Cl concentration of 37±5 mEq/L; whereas, the fetal RBC's had a mean RBC Cl concentration of 43±5. The same number of samples were involved as for RBC Na.

There were no significant differences detected in RBC electrolyte concentrations between arteries and veins for either sows or pigs. The umbilical arteries of fetal pigs contain venous blood. Significant differences in RBC electrolytes were detected between sows and pigs. Erythrocyte K and Na concentrations were lower in the fetal RBC's. The probability of a larger F value for the difference in RBC K between sows and pigs was 0.10 and for RBC Na 0.025. Erythrocyte Cl concentrations

were significantly higher for fetal RBC's. The probability of a larger F value was 0.005.

McCance and Widdowson (1956) have published data on fetal pig erythrocytes. After transposing their data into comparable units, the following comparisons can be made between their data and the combined fetal data from Table 1:

	fetal pig e	rythrocytes	adult swine ery	throcytes
	mEq/L c	f cells	mEq/L of cells	
	from data of Table 1	McCance and Widdowson (1956)	from data of Table 1	McCance and Widdowson (1956)
K	115	90	124	121
Na	15	26	19	7
C 1	43	71	37	46

Because of differences between gravimetric and volumetric measurements, there is a small error in reporting McCance and Widdowson's work in mEq/L of cells.

McCance and Widdowson (1956) did not compare fetal blood with maternal blood but with blood from other adult swine. McCance and Widdowson also drew the fetal blood after the uterus had been removed from the sow and transported to the laboratory. All the blood collected from each fetal litter was pooled by McCance and Widdowson. The most outstanding deviation in the comparison above is that McCance and Widdowson's results agree with the supposed theory that the mechanism which extrudes Na

from the erythrocytes matures less rapidly than the one which takes up K; whereas, the data from Table 1 does not follow that concept. In addition, the RBC Cl concentration would be expected to follow the direction of change of the RBC Na.

The individual results of maternal and fetal swine RBC electrolyte concentrations can be found in Table 1. Standard deviations of non-pooled fetal litters for RBC K varied from 1 to 4 mEq/L of cells, for RBC Na 5 to 17, and for RBC Cl 4 to 10.

McCance and Widdowson (1956) collected blood from fetal pigs earlier in gestation (42-49 days) than the pigs in Table 1. This could account for the difference in the RBC Na relationship. The linear regression equation for the fetal RBC Na values in Table 1 against time is

mEq/L of RBC Na = 6.3 + 0.088 (days).

Thus, RBC Na would have equaled about 10 mEq/L of cells at 42-49 days of gestation. Obviously, correction for the difference in time does not resolve the discrepancy.

The linear regression equation for fetal RBC K values in Table 1 against time is

mEq/L of RBC K = 100.0 + 0.152 (days).

Thus, RBC K would have equaled about 107 mEq/L of cells at 42-49 days of gestation. The 107 mEq/L would be a more valid

comparison with the 90 mEq/L reported by McCance and Widdowson (1956) than the 115 mEq/L.

The linear regression equation for fetal RBC Cl values in Table 1 against time is

mEq/L of RBC C1 = 37.7 + 0.057 (days).

Thus, RBC Cl would have equaled about 41 mEq/L of cells at 42-49 days of gestation. Extrapolation of this equation beyond parturition is certainly not justifiable. A positive slope is not consistent with the fact that adult RBC Cl is lower than fetal RBC Cl. This is not surprising when one considers that cytodifferentiation with regard to electrolytes can occur after birth (Blechner, 1961). Figure 1 is an example of how variance in maternal RBC K content is mimicked by fetal pig RBC K content.

The means and standard deviations of RBC electrolyte concentrations in a group of representative pigs are given at the end of Table 3. The following is a comparison with some of the literature. The number of pigs is in parenthesis. McCance and Widdowson's data have been transposed into comparable units. Comparisons are best made with the data of Coldman and Good as their method is similar (indirect) and their data were reported in the same units. Their standard deviation for RBC Na is quite small considering they used the indirect method. The standard deviation for RBC Na from Table 3 indicates the existence of

	from data of Table 3	Coldman anā Good (1967)	McCance and Widdowson (1956)	Kerr (1937)
	mEq/L	mEq/L	mEq/L	mM/1,000 gm
Na	2±7 (60)	16±2 (7)	7 (9)	11 (4)
K	132±8 (60)	106±13 (7)	121 (9)	100 (4)
Cl	32±8 (43)	-	46 (9)	-

some negative values after calculation of RBC Na. These values must be included if the method is to be honestly evaluated. The method is obviously not accurate, but may have sufficient precision to evaluate RBC changes. Negative values are probably the result of small errors in estimation of the packed cell volume, plasma Na content or whole blood Na content (Streef, 1939).

If the packed cell volumes used to calculate RBC K had included the white cells, the RBC K values would be about 4 mEq/L lower. Coldman and Good (1967) make no mention as to whether white cells were included in their calculations. They probably were not included.

No significant differences in RBC electrolytes due to sex were evident in the group of representative pigs. When anemic pigs were compared statistically with normal pigs, no significant differences were found in regard to RBC Na or RBC K. The means and standard deviations for the two groups of pigs are recorded at the end of Table 5. The mean corpuscular volume is much larger in fetal pigs than in their mothers (Figure 5). The mean corpuscular hemoglobin concentration is lower in fetal pigs than in sows and mean corpuscular hemoglobin is higher in fetal pigs than in sows. The erythrocytic indexes can be calculated from the data in Table 1. McCance and Widdowson (1956) also found the mean corpuscular hemoglobin concentration to be lower in fetal pigs. They did not report the other two erythrocytic indexes.

There were no significant correlations between erythrocyte electrolytes and the erythrocytic indexes in the group of anemic However, in the group of 60 representative and normal pigs. pigs, the mean corpuscular volume and the mean corpuscular hemoglobin were positively correlated (P<0.01) with RBC Na concentrations. The relationship of RBC Na concentration with erythrocytic indexes is not consistent from group to group which indicates that a relationship exists occasionally or a lack of precision is inherent in the method of determination of RBC Na concentrations and no relationship exists. In 43 pigs of the representative group the RBC Na was positively correlated with RBC Cl. The correlation coefficient was 0.666 and this is significant at the 1% level. In the KCl infused pigs, the RBC Na was also positively correlated with RBC Cl. The correlation was 0.375 and this is significant at the 1% level. Throughout all the experiments where correlations were calculated, the RBC Cl followed the RBC Na as is expected physiologically.

Electrolytes in Porcine Plasma

Analysis of the data in Table 1 gives a mean plasma Na concentration of 145±2 mEq/L for sows and 142±3 for fetal pigs. There were no significant differences between arterial and venous plasma Na for sows or fetal pigs. A significant difference existed between sows and fetal pigs. The probability of a larger F value is 0.005. The significant difference between sow and fetal pig plasma Na concentration fails to point out that the significant differences only occur early in gestation. The true relationship is shown in Figure 2. The relationship late in gestation is reflected by how close the two means for the entire gestation period are to each other.

Widdowson and McCance (1956) reported a mean plasma Na concentration of 144±5 mEq/L for adult swine and 122±10 for fetal pigs at 40-50 days of gestation. Cummings and Kaiser (1959) reported a mean plasma Na concentration of 146 mEq/L for sows and 143 for pigs at 106 days of gestation. The linear regression equation for fetal pig plasma Na against time from the data of Table 1 is

mEq/L plasma Na = 125.4 + 0.171 (days).

A correction for time would make the data from Table 1 closer to the findings of Widdowson and McCance for fetal plasma Na.

The increase of fetal plasma Na levels to the adult level near birth (Figure 2) is in agreement with Widdowson and McCance (1956).

Analysis of the data in Table 1 results in a mean plasma K concentration of 4.2±1.0 mEq/L for sows and 5.4±1.3 for fetal pigs. There were no significant differences between arterial and venous plasma K for sows or fetal pigs. A significant difference existed between sows and fetal pigs. The probability of a larger F value is 0.025.

Widdowson and McCance (1956) reported a mean plasma K concentration of 6.0±0.6 mEq/L for adult pigs and 17.5±2.4 for fetal pigs at 40-50 days of gestation. Cummings and Kaiser (1959) reported a mean plasma K concentration of 4.7 mEq/L for sows and 4.1 for fetal pigs at 106 days of gestation.

The linear regression equation for fetal pig plasma K against time from the data of Table 1 is

mEq/L of plasma K = 1.85 + 0.035 (days).

Obviously, correction for time does not bring the data from Widdowson and McCance close to the data from Table 1. The data for plasma K are plotted in Figure 3. Note that at 111 days when the sow plasma K is elevated that the fetal pig plasma K is also elevated.

Analysis of the data in Table 1 gives a mean plasma Cl concentration of 103 ± 2 mEq/L for sows and 96 ± 3 for fetal pigs.

There were no significant differences between arterial and venous plasma for sows or fetal pigs. A significant difference existed between sows and fetal pigs. The probability of a larger F value is 0.005.

Widdowson and McCance (1956) reported a mean plasma Cl concentration of 106±4 mEq/L for adult pigs and 98±3 for fetal pigs at 40-50 days of gestation. Cummings and Kaiser (1959) reported a mean plasma Cl concentration of 101 mEq/L for sows and 93 for fetal pigs at 106 days of gestation.

The linear regression equation for fetal pig plasma Cl against time from the data of Table 1 is

mEq/L of plasma Cl = 85.3 + 0.111 (days).

The equation is not particularly valuable in the interpretation of other data. The data from Table 1 for plasma Cl are plotted in Figure 4.

The data from Table 3 for plasma electrolytes in a representative group of pigs is compared with previous findings in Table 22. Most of the data in Table 22 agrees with the ranges given by Meir (1963). In Table 22 Coldman and Good reported a comparatively high plasma K concentration. Values higher than those usually expected in mammalian plasma or serum have been reported in young pigs by Garner <u>et al</u>. (1957) and Coulter and Swenson (1967). Ullrey <u>et al</u>. (1967) have recorded data that would suggest that these high plasma or serum K concentrations

are not due to age differences. The low plasma Na levels reported by Coldman and Good (1967) may account for their low standard deviation in their calculated RBC Na concentrations. The plasma Na concentration reported by Birkeland (1968) is also relatively low.

Table 5 gives the plasma electrolyte data for anemic and normal pigs. The anemic pigs had a plasma Na concentration of 134±7 mEq/L as opposed to normal pigs in the same group with a plasma Na concentration of 140±3. The difference is significant (P<0.01). The anemic pigs had a plasma Cl concentration of 97±6 mEq/L as opposed to normal pigs in the same group with a plasma Cl concentration of 100±10. This is a significant difference (P<0.1). The anemic pigs had a plasma Ca concentration of 10.3±0.5 mg/100 ml as opposed to normal pigs in the same group with a plasma Ca concentration of 11.1±0.7. This difference is significant (P<0.001). The anemic pigs had a plasma P concentration of 7.0 ± 0.9 mg/100 ml as opposed to normal pigs in the same group with a plasma P concentration of 7.9±0.5. This is a significant difference (P<0.01). No significant differences between anemic and normal pigs was noted with respect to plasma K and CO, concentrations. There was no significant difference between the two groups with respect to plasma glucose.

The plasma Ca concentration was not significantly correlated with plasma inorganic P concentration in the group of 60 pigs;

whereas, in the group of 31 pigs with 17 anemic and 14 normal pigs, the Ca was significantly (P<0.01) correlated with plasma inorganic P. The correlation coefficient was 0.7871.

Ullrey <u>et al</u>. (1967) found within age correlations of serum calcium and inorganic P levels generally low and insignificant. They found it difficult to ascribe particular significance to significant correlations that they did find at 1 and 3 weeks of age. They point out the conflicting reports in the literature on the influence of the diet on inorganic P.

The above differences in plasma electrolyte levels could possibly be due to differences in food consumption, but the lack of a significant difference in plasma glucose between anemic and normal pigs would lend evidence that such is not the case. Talbot (1963) was able to demonstrate that the reduction in red cell volume in anemic swine is compensated by a comparable increase in plasma volume. Perhaps dilution of some plasma electrolytes occurs.

Plasma Na and Cl concentrations were significantly correlated at the 1% level in the fetal pigs, the representative group of pigs, the normal and anemic group, and in the infused pigs. The correlation coefficients were 0.676, 0.852, 0.626, and 0.662, respectively. Thus, in the above experimental situations, it appears that the chloride anion is following the sodium changes in the plasma.

Silvette <u>et al</u>. (1938) investigated the effect of raising the potassium in the body on blood sugar. Injected potassium in rats, cats, and opossums produces glycogenolysis and/or the inhibition of glycogen formation with hyperglycemia resulting. Correlations between plasma glucose and plasma K concentrations were not significant for the group of anemic and normal pigs nor for the pigs infused with isotonic KCl solutions. Plotting plasma glucose (ordinate) against plasma K (abscissa) results in the following linear equation:

mg/100 ml plasma glucose = 81.8 - 0.95 (mEq/L of plasma K). The data are from Table 10.

Porcine Electrocardiograms

Electrocardiograms were recorded from five unanesthetized pigs (numbers 11, 13, 14, 15, and 16 in Table 7) prior to being anesthetized for KCl infusion. The following data recorded from lead II are compared with data on unanesthetized pigs from the literature. For the data following, Platner <u>et al</u>. (1948) recorded electrocardiograms from 6 pigs 60 to 90 days of age. Miller <u>et al</u>. (1957) recorded electrocardiograms from 14 pigs 35 days of age for the data following and Thielscher (1966) recorded electrocardiograms from 38 pigs weighing about 80 kg. Platner et al. (1948) reported Q-T intervals that were corrected

for heart rate. The Q-T interval from Platner <u>et al</u>. listed below has been transposed to a value not corrected for heart rate.

	Platner <u>et</u> <u>al</u> .	Miller <u>et</u> <u>al</u> .	Thielscher
	(1948)	(1957)	(1966)
heart rate			
beats/minute 156±15	182	180	132
P-wave duration			
$second 0.04\pm0.01$	0.05	-	0.06
P-Q interval			
second 0.08±0.01	0.07	0.08	0.10
QRS duration			
second 0.04±0.01	0.04	0.04	0.04
Q-T interval			
second 0.21±0.02	0.19	0.20	0.24
T-wave angle			
degrees 59±14	_	-	-
P-wave amplitude			
mV 0.14±0.04	0.06 to 0.12	-	-
T-wave amplitude			
mV 0.29±0.08	-0.30 to 0.32	_	_

Electrocardiograms were recorded after injection of sodium pentobarbital, and in some cases atropine sulfate, prior to KCl infusion from pigs 1-16 (Table 11). The following data were collected from lead II.

heart	P-wave	P-Q	QRS
rate	duration	interval	duration
beats/minute	second	second	second
170±26	0.04±0.01	0.07±0.01	0.04±0.01
Q-T	P-wave	T-wave	T-wave
interval	amplitude	amplitude	angle
second	second	second	degrees
0.22±0.03	0.15±0.08	0.24±0.18	50±14

Sodium pentobarbital usually increases the heart rate when injected intravenously into an unanesthetized pig (Table 11, pigs 11, 13, 15 and 16).

Respiration affects the porcine electrocardiogram. The respiratory rate can be determined from the electrocardiogram with a paper speed of 1.5 mm per second (Figure 6). The effects of respiration were evident in one, two, or three of the standard limb leads most of the time. The effects of respiration were most evident in lead I.

Sinus arrhythmia is nonrespiratory in the resting pig (Figure 6). Figure 7 is an electrocardiogram recorded at 30 mm per second and contains one arrhythmic beat. The
electrocardiogram is from pig number 18. This was the only arrhythmia recorded in a resting pig throughout the experiments. Buchanan (1965) reviewed spontaneous arrhythmias in domestic animals. The pig was not included in the discussion of sinus arrhythmia. It is difficult to classify spontaneous arrhythmias in pigs from one recording.

The T wave is altered by many physiologic states other than those found in the presence of cardiac disease (Burch and Winsor, 1966). Figures 7 and 8 were recorded from pig number 18. Fifteen minutes later Figure 9 was recorded. The electrodes were left in place from one recording to the next. The pig was restless as Figure 9 was being recorded; whereas, the pig appeared to be content during the recording of Figure 7. Thus it is important to control the physiologic state when evaluating changes in the T wave. Sodium pentobarbital was chosen, despite its shortcomings (Priano, 1969), to control the pigs throughout the infusion experiments. Table 6 gives the P and T wave configuration in lead II before and after being anesthetized with sodium pentobarbital.

Figure 10 is a recording of an electrocardiogram of pig number 18 after being anesthetized with sodium pentobarbital. The initial effect of sodium pentobarbital on the heart rate is one of quickening the rate. Figure 19 includes a recording of the effect of additional sodium pentobarbital after a pig had been anesthetized for some time. In Figure 19 the pig had been

anesthetized 2 1/2 hours when additional sodium pentobarbital was administered.

Figure 22 is a recording of the initial effect of injection of sodium pentobarbital on the electrocardiogram and the subsequent effect on the electrocardiogram after 3 1/2 hours of anesthesia. Blood samples were drawn every 20 minutes during anesthesia. Nothing was infused during the 3 1/2 hour period, except sodium pentobarbital injected to maintain anesthesia.

Interrelationships of Blood Electrolytes and Electrocardiograms

Fetal pigs have a different plasma electrolyte pattern than their mothers. The plasma Na and Cl concentrations are lower for fetal pigs and plasma K and CO_2 concentrations are higher in fetal pigs. Does the plasma electrolyte pattern have any influence on the RBC electrolyte pattern? Fetal pig RBC's have a lower concentration of K and a higher concentration of Cl than do adults. As previously noted, the RBC Na concentration data in Table 1 is not in agreement with McCance and Widdowson (1956) who reported a higher Na concentration in fetal pig erythrocytes.

Anemic pigs have a plasma electrolyte pattern resembling fetal pigs with respect to plasma Na and Cl concentrations as compared to normal pigs, but not with respect to plasma K and CO_2 concentrations. The comparison of anemic pig RBC electrolyte concentrations (Na and K) with normal pig RBC electrolyte

concentrations did not reveal any significant differences. It would appear then that the plasma electrolyte pattern of anemic pigs does not affect the RBC electrolyte pattern.

In pigs infused with isotonic KCl, the plasma Na and Cl concentrations were lowered and the plasma K concentration was raised. The plasma CO₂ was unaffected. This pattern is similar to the plasma Na, K, and Cl pattern seen in fetal pigs. When RBC K (ordinate) is plotted against plasma K (abscissa) for the KCl infused pigs (Table 10), the resultant line is

mEq/L of RBC K = 127 + 0.16 (mEq/L of plasma K).

The correlation between RBC K and plasma K was not significant. Thus, within a few hours, a change in the plasma electrolyte pattern does not affect RBC K. This is in agreement with the work of Ginsburg and Wilde (1954). They found in rats that the rate of potassium exchange between plasma and erythrocytes is slow as compared with other tissues. In the rat, changes in RBC K concentration occur in a matter of days rather than hours. In the dog, however, Spurr and Barlow (1959) measured changes in erythrocyte Na and K concentrations due to hypothermia and hyperthermia during 1 to 4 hour experiments. The RBC Na and Cl concentrations were not correlated with plasma K in the infused pigs. It would appear then that a change in plasma electrolytes of the pig is not followed by a sudden shift in erythrocyte electrolytes.

Significant (1% level) correlations existed between diluted whole blood K and packed cell volume and between diluted whole blood K and hemoglobin. These positive correlations reflect the difference in K concentrations of cells as opposed to plasma. The correlations of packed cell volume and hemoglobin with diluted whole blood Na are negative as expected when an electrolyte concentration is quite low in the cells as compared with plasma. Diluted whole blood Cl was not significantly correlated with packed cell volume or hemoglobin.

Diluted whole blood electrolyte or whole blood electrolyte values have little meaning by themselves. The packed cell volume and the plasma electrolyte concentration must also be considered. The correlation coefficient between diluted whole blood Na and plasma Na in normal and anemic pigs was -0.454. This is a significant correlation (P<0.05). The negative correlation is a result of the plasma Na concentrations being higher in normal pigs whose packed cell volumes are also higher than anemic pigs. The low Na concentration in the larger packed cell volume results in lower diluted whole blood Na concentrations.

From the preceding discussion of whole blood and RBC electrolytes, the conclusion can be made that swine erythrocyte electrolytes are not affected to an appreciable extent by changes in plasma electrolytes. The lower RBC K concentration found in fetal pigs could be expected in anemic pigs due to the

many immature erythrocytes in anemic pigs. Immature erythrocytes are prevalent in anemic pigs (Coulter and Swenson, 1968). However, anemic pigs do not have a lower RBC K concentration as compared to normal pigs (Table 5). Evans and Blunt (1963) found in anemic sheep with many immature erythrocytes, that the RBC K concentration was higher than with mature erythrocytes. In fetal sheep the RBC K concentration is higher than in adults. In swine the electrolyte pattern of erythrocytes must not be correlated with the age of the erythrocytes, but must be subject to some influences outside the erythrocyte. The larger standard deviation for RBC K concentrations (Table 5) in anemic pigs could be attributed to either a breakdown in the indirect method with variable packed cell volumes (the expected correlations of involved data do not indicate this) or it could be attributed to an actual increase in variation of RBC K concentration in anemic pigs.

Because the swine erythrocyte electrolyte concentration is not a sensitive index of electrolyte imbalance, the discussion of blood electrolytes as related to the porcine electrocardiogram will involve only plasma electrolytes.

Previous clinical investigations and research, mostly in man and dogs, have shown that the electrocardiogram is a fairly sensitive indicator of changes in plasma K and Ca and usually insensitive to abnormal concentrations of Na and hydrogen ions (Surawicz, 1967). In the experiment with pigs infused with

isotonic KCl solutions, the plasma Ca concentrations must be considered in the interpretations of the electrocardiograms. As infusion proceeded and the plasma K rose, the plasma Ca concentration was not affected to any extent. The correlation coefficient (0.104) between plasma K and Ca concentrations is not significant. The equation of the line when plasma Ca is plotted against plasma K is

mg/100 ml plasma Ca = 9.95 + 0.058 (mEq/L of plasma K).

The plasma Ca concentrations were well within the range set by Bradlow and Levin (1969) which have no effect on the human electrocardiogram.

Infusion of isotonic KCl solutions did, however, affect the plasma Na concentration. The correlation coefficient between plasma K and Na concentrations was -0.525 and is significant at the 1% level. Thus, as the plasma K concentration rose, the plasma Na concentration dropped. However, hyponatremia by itself cannot be recognized in the electrocardiogram (Surawicz, 1967). The effects of hyponatremia in conjunction with hyperpotassemia on the electrocardiogram reported by García-Palmieri (1962) occurred with plasma Na levels lower than occurred in the infused pigs. Therefore, in Figures 11, 12, 13, 14, 15, 16, 17, 18, and 19 the effects of changing plasma K concentrations on the porcine electrocardiogram are probably little affected by other ions.

As the plasma K concentration rose, the heart rate decreaced. The correlation coefficient between plasma K and beats per minute was -0.209 and is significant at the 1% level. The equation for the line when beats per minute is plotted against plasma K is

beats per minute = 195 - 4.8 (mEq/L of plasma K).

The equation applies to pigs lightly anesthetized with sodium pentobarbital. The slowing of the heart by increased plasma K is due to an increased responsiveness of the heart to vagal tone as well as the direct action of K on cardiac conduction (Hoff et al., 1944).

Tables 11 and 12 contain the data for the electrocardiographic responses of the KCl infused pigs. Table 10 contains the blood electrolyte data for the corresponding data from Table 11.

As the plasma K concentration rose, the P-wave amplitude in lead II decreased. Small P waves are the result of a displaced pacemaker and/or inexcitable atrial muscle fibers (Surawicz, 1967). The correlation coefficient between the two was -0.582 and is significant at the 1% level. As the plasma K concentration rose, the T-wave amplitude in lead II usually increased. The correlation coefficient between the two was 0.144 and is significant at the 5% level. The correlation was less than expected from visual monitoring of the

electrocardiograms. Many of the amplitude increases were small and 3 of the 16 pigs had no increase. In humans, the characteristic tall, steep, narrow, and pointed T waves are present in only 22% of patients with hyperpotassemia (Braun <u>et al.</u>, 1955). Pig number 7 had a negative T wave which increased in the negative direction. In 15 dogs anesthetized with sodium pentobarbital Greenspan <u>et al</u>. (1965) found that with KCl infusion that negative and biphasic T waves became upright.

As the plasma K concentration rose, the P-wave duration increased. The correlation coefficient between the two was 0.308 and is significant at the 1% level. Increased P-wave duration can be expected and has been reported in the literature (Braun et al., 1955).

As the plasma K concentration rose, the P-Q interval lengthened. The correlation coefficient between the two was 0.232 and is significant at the 1% level. In dogs injected intravenously with KCl Winkler <u>et al</u>. (1938) found no changes in the P-R interval up to the time of extinction of the P wave. Lengthening of the P-R interval is documented in the literature as a sign of hyperpotassemia in man (Braun <u>et al</u>., 1955). That potassium slows conduction in the porcine ventricle is shown by the lengthening of the QRS complex as plasma K rose. The correlation coefficient between the QRS complex and plasma K was 0.330 and is significant at the 1% level. This lengthening occurs just prior to and during heart block (Table 10, 11, and

12). There was no correlation between plasma K and the Q-T interval. The lack of correlation of the Q-T interval with plasma K helps verify that the plasma Ca concentration was not influencing the electrocardiogram as a result of infusion.

There was a correlation between the angle of the T wave and plasma K. The correlation coefficient was 0.327 and is significant at the 1% level. When the two are plotted, the linear equation is

degrees of T-wave angle = 41.5 + 2.74 (mEq/L of plasma K).

The earliest electrocardiographic evidence of potassium intoxication in man is peaking of the T waves (Levine <u>et al.</u>, 1952). For comparison, the linear equation for plotting the T-wave angle against the plasma Na to K ratio is

degrees of T-wave angle = 73.6 - 0.63 (plasma Na/K).

Complicated formulas have been compiled to evaluate the T wave in hyperpotassemia (Braun <u>et al.</u>, 1955). Table 12 reveals that the simple angle between the isoelectric line and the positive deflection of the T wave is an early warning of hyperpotassemia. Figure 14 is a series of T-wave angles of lead II as the plasma K concentration rises. As with most electrocardiographic changes in hyperpotassemia, the change in the T-wave angle cannot be expected in all cases. Vagal activity probably affects ventricular repolarization and the effect is exaggerated as the plasma K level is elevated. The variation in vagal activity affects ventricular repolarization with resultant variation in T-wave changes (Greenspan <u>et al.</u>, 1965). Peaked T waves can be found in electrocardiograms of swine with normal plasma K levels (Figure 18).

Table 8 contains the data on KCl and K dosage. Variations among the 16 pigs are lessened when the data is on a body weight per time basis. Table 12 contains data in plasma K concentrations at which certain electrocardiographic (lead II) changes appear. Winkler et al. (1938) reported the concentration of K in the serum at which certain electrocardiographic changes (lead II) occur in the dog (4 dogs) and Burch and Winsor (1966) have illustrated some expected changes in the electrocardiogram at various serum K levels in man. Table 12 illustrates that certain changes never occur, the sequence of events is not constant, that the P wave returns at a lower plasma K concentration than the concentration at which it disappears, and that the T-wave angle should be included in the monitoring of porcine electrocardiograms for hyperpotassemia. Some similar experiments have been done in a few intact dogs and the multiplicity of reported work in man is clouded by clinical situations where time and other metabolic malfunctions have not been controlled. Thus, comparison of the data in Table 12 with other work must be done with some reservations. However, the data should be

useful in the monitoring of porcine electrocardiograms for electrolyte imbalances which involve mainly hyperpotassemia. Research involving the cardiovascular system of the pig must include control tracings and serial electrocardiograms to monitor plasma K concentration changes. The administration of electrolytes to swine in experimental situations should be monitored by electrocardiograms in addition to plasma electrolyte determinations. Examination of Figure 11, 12, 13, 14, 15, 16, 17, 18, and 19 gives some insight for monitoring the electrocardiogram of swine for hyperpotassemia.

Figure 11 is the electrocardiograms of pig number 12 recorded as the plasma K concentration was elevated. All of the electrocardiographic changes listed in Table 12 occurred. Figure 12 is the electrocardiograms of pig number 16 recorded as the plasma K concentration was elevated. The S-T depression did not occur in the electrocardiograms of this pig.

Pig number 9 had a negative QRS complex as is evident from Figure 13. The increased plasma K concentration appears to have reversed the direction of depolarization of the ventricle which returned to its original direction upon the decrease in plasma K concentration.

Figure 15 is the electrocardiograms of pig number 15 recorded as the plasma K was elevated. The augmented limb leads and the precordial lead are included. No particular advantage was evident from the inclusion of leads other than lead II in

the electrocardiographic monitoring of hyperpotassemia. Finch <u>et al</u>. (1946) observed that in potassium intoxication of humans, peaked T waves are apt to be most prominent in the precordial leads. Optimal placing of the precordial lead for detecting electrocardiographic changes in swine was not investigated.

Figure 16 is the electrocardiograms of pig number 13 recorded at a paper speed of 60 mm per second as the plasma K concentration was elevated. All of the electrocardiographic changes listed in Table 12 occurred.

Figure 17 is a recording of pig number 3 going into heart block during isotonic KCl infusion. The infusion pump was shut off where the artifacts (changes in baseline) appear. In the few seconds that it takes to change the paper speed, the heart block had disappeared. Heart block, as a result of KCl infusion, is a very ephemeral phenomenon after infusion has stopped.

It is difficult to characterize the blocking of impulse conduction in the heart resulting from hyperpotassemia in the pig because the P wave is usually missing and because conduction may be blocked in different parts of the heart at the same time. The term heart block is commonly thought of as partial or complete atrioventricular block. The electrocardiographic change associated with cardiac conduction disturbances in hyperpotassemia involves more than atrioventricular block. Winkler et al. (1938) described intraventricular block in dogs starting at about 10 mEq/L of plasma K. Burch and Winsor (1966) have

shown an illustration of auricular standstill and intraventricular block at about 9 mEq/L of plasma K in humans. In the pig the type of impulse conduction disturbance (heart block) is variable from one pig to the next. The electrocardiograms were monitored for arrhythmia resulting from disturbances in the conduction of impulses in the heart (Figure 17). In pig number 5, where the P wave did not disappear, a dropped beat was followed by a P-QRS-T sequence. This would be classified as a partial sinoatrial block. Figure 11 (I) is an indication of intraventricular block. Figure 15 (G) and Figure 16 (G) are recordings of bigeminy (leads I, II, and III). Bigeminy and/or trigeminy occurred in some pigs for 10 to 20 seconds. Bigeminy and trigeminy may be caused by premature contractions (Burch and Winsor, 1966). Premature beats of ventricular origin were not seen in the electrocardiogram of pigs with elevated plasma K concentrations. This agrees with the electrocardiographic observations made by Winkler et al. (1938) of KCl infused dogs. Bigeminy and trigeminy may also be caused by the presence of an atrioventricular block (Burch and Winsor, 1966) and this is probably the case in some pigs with elevated plasma K concentrations. The lengthening of the P-Q interval, without dropped QRS complexes, just prior to arrhythmia in some pigs (Table 11) is indicative of partial atrioventricular block in a mild form (Burch and Winsor, 1966).

Figure 19 (B) is a typical example of arrhythmia in pigs due to elevated plasma K concentrations. The absence of the P wave makes differentiation between partial sinoatrial block and partial atrioventricular block difficult, if not impossible. Precordial leads might be useful in the differentiation of the different types of conduction disturbances. In hyperpotassemia partial sinoatrial block could result from the increased sensitivity of the heart to vagal impulses (Hoff <u>et al</u>., 1944). Sinus arrest is usually brought on by increased vagal stimulation (Burch and Winsor, 1966). Sinus arrest was not discernible in the electrocardiograms of the infused pigs.

If the hearts of fetal pigs are no more resistant to potassium toxicity than adult hearts, the values reported by Widdowson and McCance (1956) for plasma K in fetal pigs are incompatible with proper cardiac function. They reported a plasma K concentration of 17.5±2.4 mEq/L in fetal pigs.

Figure 18 is the electrocardiograms of pig number 5 recorded to show that T waves may be peaked prior to an elevation in plasma K levels. Figure 19 is the electrocardiograms of pig number 10 and illustrates S-T depression due to an elevated plasma K concentration. Within minutes after heart block, the heart rate elevated. Recording C of Figure 19 illustrates the effect of the intravenous injection of additional sodium pentobarbital on heart rate.

Figure 20 is the electrocardiograms of pig number 16 before and after infusion of isotonic NaCl (140 mEq/L of Na). The T wave in lead II decreased in amplitude. It would be difficult to attribute any changes in the electrocardiograms to electrolyte changes. Plasma Na was lowered slightly by the infusion. The hematologic, electrolyte, and electrocardiographic changes during infusion are recorded in Tables 13, 14, and 15.

Figure 21 is the electrocardiograms of pig number 17 before and after infusion of an isotonic glucose solution. Infusion had little effect on the electrocardiograms. The hematologic, electrolyte, and electrocardiographic data during infusion are recorded in Tables 16, 17, and 18.

Figure 22 is the electrocardiograms of pig number 12 and illustrates the effects of the initial intravenous injection of sodium pentobarbital. As the rate increased, the P wave is superimposed upon the downstroke of the preceding T wave because of a shortening of the T-P interval. The electrocardiographic change after 3 1/2 hours of sodium pentobarbital anesthesia and blood sampling was a slowing of the heart rate. No other distinguishable changes occurred. The hematologic, electrolyte, and electrocardiographic data during the 3 1/2 hours are recorded in Tables 19, 20 and 21.

The heart rate of the pig infused with isotonic NaCl changed very little during infusion (Table 15). The heart rate of the pig infused with isotonic glucose was faster at the

beginning and at the end of infusion (Table 18). The effect of anesthesia with no infusion and blood sampling on heart rate was a slowing of the heart at the end of the experiment (Table 21). Obviously, the heart rate of pigs anesthetized with sodium pentobarbital under the above conditions is variable. Gilmore (1965) in ten unanesthetized dogs recorded a heart rate of 113±30, after 1 hour of sodium pentobarbital anesthesia the heart rate was 155±25, after 2 hours 156±29, and after 4 hours 168±24. The packed cell volumes decreased in dogs after intravenous sodium pentobarbital (Gilmore, 1965). Such is usually the case in swine (pigs 11, 13, 15, and 16 in Table 9). Despite the added variables introduced by sodium pentobarbital anesthesia, economy and convenience would indicate its continued use in some experimental conditions.

SUMMARY AND CONCLUSIONS

Hematological and electrolyte data were measured from blood of 8 pregnant sows, one selected at 8 different days of gestation, and their fetal pigs, of a representative group of 60 pigs ⁹ weeks old, and a group of 31 normal and anemic pigs about 8 weeks of age. The blood electrolyte distribution in the above pigs was compared with the blood electrolyte distribution in 16 anesthetized pigs monitored with electrocardiograms and infused with an isotonic KCl solution.

By the use of an automated indirect method of erythrocyte electrolyte determination, the relationship previously described in the literature between maternal and fetal swine erythrocytes was verified with respect to potassium and chloride, but not with respect to sodium. By the same method, normal values for erythrocyte electrolytes (Na, K, and Cl) were established from a group of representative pigs. The erythrocyte electrolytes (Na and K) of anemic pigs were compared to the erythrocyte electrolytes of normal pigs.

The plasma electrolyte pattern of fetal pigs was compared with maternal plasma and the maternal-fetal relationship compared with previous findings in the literature. Plasma electrolyte concentrations of anemic pigs were compared to those of normal pigs. The relationships of erythrocyte electrolytes to erythrocytic indexes and plasma electrolytes were statistically analysed.

It was found that the erythrocyte electrolyte pattern (Na and K) of anemic pigs does not resemble that of fetal pigs and despite significant plasma electrolyte differences between anemic and normal swine, anemic pig erythrocyte sodium and potassium concentrations are not significantly different from normal pigs. No constant relationship was evident between erythrocytic indexes and erythrocyte electrolytes.

Infusion of isotonic KCl solutions significantly raised plasma K and lowered Na and Cl, but had no significant effect on erythrocyte electrolytes (Na, K, and Cl). The effects of elevated plasma K concentrations on the electrocardiograms of pigs anesthetized with sodium pentobarbital were established.

In most cases, as the plasma K rose, the P-wave amplitude decreased, the T-wave amplitude increased, the T-wave angle increased, the P-Q interval lengthened, the QRS duration lengthened prior to heart block, the Q-T interval was not affected, and heart block, characterized by arrhythmia, occurred.

In conclusion, the above experiments with the resultant data suggest that:

1. The automated indirect method of determination of erythrocyte electrolytes is accurate and precise enough to demonstrate the difference between maternal and fetal erythrocyte concentrations of K and Cl. The method lacks accuracy in the determination of erythrocyte sodium in swine and the precision is questionable.

2. There are plasma electrolyte concentration differences between porcine maternal and fetal plasma and between anemic and normal pig plasma. Maternal plasma contains a higher concentration of Na and Cl and a lower concentration of K. Anemic pig plasma contains less Na, Cl, Ca, and P than normal pig plasma.

3. Changes in red cell size or hemoglobin content as estimated by the erythrocytic indexes have no significant effect on erythrocyte Na or K concentrations.

4. Changes in plasma electrolyte concentrations have no discernible effects on erythrocyte electrolyte concentrations in swine.

5. Porcine electrocardiograms are good indicators of porcine plasma K concentration, but certain expected electrocardiographic changes may not occur, an expected change does not always occur at the same plasma K concentration, and the sequence of events is not constant from pig to pig.

6. The T-wave angle should be included as an expected change in the porcine electrocardiogram when hyperpotassemia is suspected.

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APPENDIX A. TABLES

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	RBC's 10 ⁶ /cmm	PĊV %	Hb gm%	plasma Na mEq/L	plasma K mEq/L	plasma CO mEq/L	plasma Cl mEq/L	RBC*** Na mEq/L	RBC K mEq/L	RBC Cl mEq/L	gestation days
sow A	8.1	40	13.5	141	3.4	15	103	24	127		67
** V woe	8.6	41	13.5	141	3.5	15	102	15	122	-	67
pigs A	4.0	34	9.0	138	4.4	18	95	18	100	-	67
pigs V	4.0	35	9.0	137	3.9	19	95	11	109	-	67
sow A	6.9	38	12.0	148	4.2	21	104	0	114	-	77
sow V	6.7	38	12.0	145	3.8	22	106	-5	115	-	77
pigs A	4.5	39	10.8	141	4.5	22	99	-5	101	-	77
pigs V	5.4	39	11.0	139	4.6	22	97	-5	101	-	77
sow A	5.5	30	9.2	147	3.9	26	101	-3	151	34	86
sow V	5.5	30	9.7	147	4.0	27	100	5	143	42	86
pig A	-	39	-	137	4.4	30	92	52	132	47	86
pig V	3.9	39	9.6	143	5.3	28	95	12	125	39	86
pig A		36	-	137	5.2	28	92	12	135	45	86
pig V	-	36	-	138	5.1	26	92	5	130	45	86
pig A	4.4	44	10.3	145	5.5	29	95	6	125	41	86
pig V	4.5	44	10.3	136	4.7	29	91	18	122	44	86

Table 1. Sow and fetal pig hematologic and electrolyte data at various stages of gestation

*A (artery, uterine for sow, umbilical for pig).

** V (vein, uterine for sow, umbilical for pig).

*** negative values are due to inherent error of the indirect method.

Tab:	le	1	•	C	0	n	t	i	n	u	e	d	

	RBC's 10 [°] /cmm	PCV %	Hg gm%	plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L	gestation days
pig A	3.9	43	10.1	138	4.7	28	92	17	124	45	86
pig V		43		140	5.1	31	93	10	123	44	86
pig A	-	40		141	4.9	29	97	14	128	32	86
pig V	3.7	40	9.2	140	4.7	30	93	10	128	66	86
pig A	3.7	38	9.0	141	4.4	30	95	2	130	42	86
pig V	4.0	40	9.5	140	4.4	29	97	-		40	86
pig A	4.1	41	10.0	141	4.8	29	96	12	125	44	86
pig V	4.2	41	10.0	146	4.8	30	100	6	130	36	86
sow A	5.6	37	8.8	143	3.8	26	104	19	113	34	87
sow V	5.8	37	8.8	143	3.8	26	104	11	113	36	87
pig A	4.1	41	8.2	138	5.1	28	93	6	100	44	87
pig V	4.0	41	8.2	137	5.1	26	93	13	100	36	87
pig A	3.9	39	7.7	139	5.0	28	94	19	95	49	87
pig V	3.8	38	8.0	140	4.6	28	95	8	103	39	87
pig A	4.5	43	8.5	139	4.0	30	91	20	102	39	87
pig V	4.5	42	8.4	138	4.0	30	90	14	99	36	87
pig A	5.6	53	9.8	140	5.2	29	93	12	101	49	87
pig V	5.6	54	9.8	140	5.8	28	94	18	102	48	87
pig A	3.9	35	6.7	143	4.4	28	96	3	106	42	87
pig V	3.8	37	6.0	141	4.6	27	97	19	95	46	87
Table	9 1.	Cont	inued								
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	RBC's 10 ⁶ /cmm	PCV %	Hb gm&	plasma Na mEq/L	plasma K mEq/L	plasma Co mEq/L	plasma Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L	gestation days
sow A	7.1	44	11.4	147	3.5	27	102	22	118	32	91
sow V	7.5	47	13.2	146	3.9	27	100	27	119	25	91
pigs A	4.5	41	9.2	142	6.5	28	97	15	98	36	91
pigs V	4.8	41	8.3	139	7.4	27	96	24	97	39	91
sow A	5.6	34	9.7	146	5.6	28	106	-	-	35	109
sow V	5.8	36	9.7	144	3.7	29	102	22	138	44	109
pig A	4.5	37	8.8	143	4.0	31	99	27	123	50	109
pig V	4.3	34	8.6	145	4.5	30	102	13	132	40	109
pig A	4.9	35	8.8	142	4.4	32	96	16	135	45	109
pig V	4.8	34	8.4	146	4.7	30	100	5	132	35	109
pig A	4.6	36	9.0	148	5.0	31	101	9	130	40	109
pig V	4.6	35	9.0	144	4.3	29	100	18	135	38	109
pig A	4.4	32	8.2	142	4.9	32	97	17	133	44	109
pig V	4.4	31	7.9	145	4.9	30	101	6	137	39	109
pig A	3.9	35	8.0	147	4.6	31	100	1	134	38	109
pig V	4.5	36	9.1	145	4.7	29	99	15	136	41	109
pig A	4.1	34	8.0	143	4.4	31	96	34	133	48	109
pig V	3.8	32	7.5	142	4.2	30	97	6	135	37	109
sow A	6.2	34	11.6	144	3.9	22	103	32	122	38	110
sow V	6.2	34	11.6	144	4.1	24	102	21	122	43	110

Table 1. Continued

	RBC's 10 ⁶ /cmm	PCV %	Hb gm%	plasma Na mEq/L	plasma K mEq/L	plasma CO mEq/L	plasma Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L	gestation days
pig A	4.3	35	10.6	144	4.6	27	98	7	105	47	110
pig V	4.5	35	10.8	143	4.8	25	99	20	111	42	110
pig A	3.8	33	10.1	142	5.1	28	96	21	105	47	110
pig V	3.6	32	9.5	141	5.1	27	97	30	102	44	110
pig A	4.9	36	11.6	146	5.0	27	98	18	108	48	110
pig V	5.3	38	11.8	144	4.9	26	99	23	108	46	110
pig A	4.9	36	11.1	145	4.8	27	96	20	108	43	110
pig V	4.7	37	10.9	145	4.5	28	95	23	106	49	110
pig A	3.8	32	9.5	143	4.7	28	98	27	103	45	110
pig V	3.8	31	9.3	143	4.6	26	98	17	106	46	110
pig A	4.1	32	9.7	142	4.7	27	97	17	109	44	110
pig V	3.9	32	9.5	142	4.8	26	97	23	109	50	110
sow A	6.5	31	10.0	144	7.4	27	101	15	119	39	111
sow V	6.6	33	10.5	144	6.0	28	102	17	121	38	111
pig A	4.1	33	8.9	145	7.0	31	96	15	107	44	111
pig V	4.0	32	8.5	144	7.5	30	97	19	109	44	111
pig A	4.9	38	10.5	144	8.0	31	96	12	108	43	111
pig V	4.9	37	10.6	145	8.0	30	97	18	105	43	111
pig A	3.8	30	7.0	145	7.5	31	96	8	-	46	111
pig V	3.6	30	6.9	144	8.0	30	96	4	108	42	111

Table	1.	Continued

	RBC's 10 ⁶ /cmm	PCV %	Hb gm%	plasma Na mEq/L	plasma K mEq/L	plasma CO mEq/L	plasma Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L	gestation days
pig A	3.8	29	6.9	144	7.5	30	95	13	106	47	111
pig V	3.1	27	6.0	143	7.5	30	97	21	106	49	111
pig A	4.3	37	10.0	146	8,6	31	96	22	104	47	111
pig V	4.2	35	9.9	145	8.7	31	96	22	104	48	111
pig A	5.4	37	11.2	147	7.6	29	95	15	112	52	111
pig V	5.7	39	11.3	146	9.0	29	97	13	109	50	111

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PCV [*]	PCV %	RBC's 10 ⁶ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %
42.7	40.3	6.6	12.4	61	19	31
34.8	33.6	5.7	10.0	59	18	30
36.0	35.0	6.5	10.7	54	17	31
35.2	33.4	6.5	10.4	51	16	31
35.6	34.0	7.2	10.4	47	14	31
35.8	34.6	6.2	10.7	56	17	31
33.8	32.4	7.6	10.1	43	13	31
35.2	33.7	7.4	10.7	46	15	32
36.5	34.9	6.4	10.7	55	17	31
36.9	35.1	7.3	11.0	48	15	31
36.3	35.2	7.2	11.4	49	16	32
37.0	35.1	7.6	10.7	46	14	31
35.2	34.2	6.6	10.4	52	16	30
33.6	32.1	5.1	9.8	63	19	31
36.2	33.8	5.5	10.4	62	19	31
32.0	30.8	6.0	8.9	51	15	29
33.3	32.6	6.7	9.8	49	15	30
33.5	32.4	5.4	9.5	60	18	29
34.6	32.7	5.3	9.8	62	19	30
34.0	32.5	4.9	9.8	66	20	30
32.6	31.4	5.9	9.8	53	17	31
33.8	32.6	5.1	9.8	64	19	30
34.4	33.2	5.1	10.1	65	20	30
31.9	30.5	5.8	9.2	53	16	30

Table 2. Hematologic data from a group of representative pigs 9-10 weeks of age

*includes leukocytes.

PCV*	PCV %	RBC's 10 ⁶ /cmm	HB gm%	MCV u ³	MCH uug	MCHC %
35.5	34.5	5.8	9.8	60	17	28
35.0	33.9	4.8	9.8	71	20	29
32.8	31.8	5.2	9.2	61	18	29
35.3	34.4	5.6	10.1	61	18	29
34.0	32.8	5.2	9.9	63	19	30
33.2	32.1	5.6	9.5	57	17	30
39.0	38.7	6.9	12.1	56	18	31
33.5	32.9	6.3	9.8	52	16	30
33.6	33.2	6.0	10.1	55	17	30
33.4	32.9	5.9	9.8	56	17	30
36.5	35.6	6.8	10.7	52	16	30
37.1	36.5	7.0	11.0	52	16	30
40.0	39.1	7.4	12.1	53	16	31
35.0	33.8	6.7	10.6	50	16	31
38.6	37.6	6.7	11.4	56	17	30
38.1	37.3	6.7	11.4	56	17	31
35.6	34.6	6.9	10.6	50	15	31
34.8	33.8	7.3	10.4	46	14	31
36.6	35.6	6.2	10.7	57	17	30
33.1	31.9	5.2	10.1	61	19	32
37.1	36.1	6.2	10.6	58	17	29
37.7	36.7	6.7	11.4	55	17	31
38.5	38.0	6.1	11.4	62	19	30
36.9	35.7	6.0	10.7	60	18	30
30.3	29.1	4.8	8.6	61	18	30
42.3	41.6	7.1	12.7	59	18	31
34.4	33.6	6.0	10.4	56	17	31
36.9	36.0	6.1	10.4	59	17	30

Table 2. Continued

¥ ع	PCV %	RBC's 10 ⁶ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %
37.0	36.2	6.9	11.0	53	16	30
38.1	37.3	7.3	11.4	51	16	31
34.0	33.4	6.6	10.4	51	16	31
36.2	35.3	6.0	11.4	59	19	32
38.4	37.4	6.2	11.0	60	18	29
34.8	33.6	5.6	10.4	60	19	31
33.1	32.3	5.9	10.1	55	17	31
31.6	30.9	5.5	9.5	56	17	31
moan				•		
35.5	34.8	6.2	10.5	56	17	30
±sđ					•	
2.9	2.4	0.8	0.8	6	2	1

Table 2. Continued

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plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
139	5.7	19	96	7.6	11.2	_
142	5.7	24	98	8.2	10.7	-
142	5.7	19	100	9.3	9.8	-
142	5.8	21	98	10.0	9.0	-
142	5.6	21	99	9.5	9.2	-
143	4.5	21	100	9.6	9.3	-
145	5.5	20	101	9.7	9.8	-
142	5.1	16	99	10.0	9.4	-
143	5.0	24	100	8.8	8.8	-
144	5.5	19	99	9.2	8.8	-
145	6.0	23	99	9.0	9.0	-
144	5.3	24	97	9.0	9.4	-
148	5.1	19	101	9.8	9.7	-
145	5.2	-	-	11.4	9.0	-
144	6.4	-	-	10.3	8.1	-
144	5.1	-	-	12.0	9.0	-
144	4.9	-	-	9.5	7.6	-
145	5.7		-	10.6	8.4	-
144	5.5	-	-	10.5	7.6	-
143	5.4	-	-	10.0	8.1	-
142	4.7	-	-	10.8	8.5	-
145	5.6	-	-	11.6	11.3	-
144	4.2	-	-	10.6	10.3	-

Table	3.	Blood electrolyte data from a group of representative
		pigs 9-10 weeks of age

*1 part blood and 19 parts H O, multiply by 20 to obtain whole blood value. *1 part blood and 1 part H₂O, multiply by 2 to obtain whole blood value.

diluted [*] whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted ** whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L
4.7	2.4	40	31	108	59
4.6	2.5	39	-2	130	57
4.6	2.4	38	1	126	36
4.8	2.4	40	4	132	44
4.7	2.4	39	3	134	33
4.7	2.5	40	6	130	41
4.8	2.5	39	-5	140	33
4.6	2.4	39	-3	135	39
4.8	2.5	39	1	126	42
4.8	2.5	41	14	129	52
4.8	2.5	41	10	131	53
4.6	2.5	38	4	133	42
4.8	2.5	39	-3	136	37
4.8	2.5	35	0	125	-
4.7	2.8	37	6	154	-
5.0	2.4	38	6	138	-
4.8	2.5	38	0	141	-
4.9	2.4	40	6	136	-
4.8	2.6	39	6	147	-
4.8	2.5	39	6	142	-
4.8	2.4	38	0	140	-
5.0	2.4	41	13	135	-
5.3	2.6	39	27	151	_

Ta	ble	è 3	. C	ont	inue	d
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plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
146	5.0			9.7	10.1	
148	5.6	-	_	10.1	10.9	-
144	5.2	-	-	10.1	9.5	-
144	5.2	-	-	10.0	10.4	-
147	5.2	-	-	9.8	10.9	-
144	6.4	-	-	10.3	11.1	-
143	5.4	-	-	9.8	9.0	-
141	4.8	23	100	12.2	9.6	103
143	5.1	28	99	10.7	8.4	107
141	5.1	25	100	12.1	8.9	102
141	4.6	26	99	11.7	8.8	103
142	4.9	21	101	12.5	9.8	112
143	4.7	19	100	12.3	10.0	133
148	5.7	21	102	12.5	10.3	117
143	5.1	24	102	11.8	8.9	118
147	5.8	25	100	11.8	10.4	106
146	5.2	22	101	11.7	10.3	117
146	5.2	23	99	12.4	9.4	107
145	4.6	26	99	11.5	9.1	93
147	5.1	17	102	11.8	9.8	127
145	4.9	22	103	11.0	10.2	117
151	4.7	19	103	12.0	10.7	114
149	6.1	21	104	12.0	10.5	103
147	6.0	24	102	12.4	10.6	106
145	4.6	24	100	10.4	10.7	108
144	5.0	25	100	10.7	9.9	100
150	6.5	25	102	11.5	11.0	110

diluted [*] whole	diluted [*] whole	diluted** whole	RBC	RBC	RBC
blood Na mEq/L	blood K mEq/L	blood Cl mEq/L	Na mEq/L	K mEq/L	Cl mEq/L
4.9	2.3	41	0	138	
4.9	2.6	40	6	139	-
4.8	2.5	38	6	136	_
5.0	2.3	41	12	132	-
4.9	2.3	41	11	140	-
4.9	2.5	39	6	140	-
4.9	2.4	40	6	137	-
4.3	2.5	38	0	124	37
4.7	2.3	37	-6	128	22
4.6	2.3	38	-6	127	30
4.6	2.3	38	-6	128	31
4.5	2.5	37	0	129	27
4.4	2.5	36	-5	132	25
4.4	2.7	39	0	128	40
4.6	2.5	38	0	136	27
4.5	2.6	38	0	128	38
4.4	2.5	38	-5	123	35
4.7	2.5	39	0	133	38
4.7	2.4	38	0	130	33
4.6	2.4	39	-5	124	32
4.8	2.3	40	-6	132	33
4.7	2.5	40	0	127	41
4.3	2.3	40	-16	114	28
4.7	2.7	39	10	132	37
4.6	2.4	39	0	129	42
4.8	2.0	39	-1	124	24
4.4	2.8	38	5	125	41

plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
145	5.8	26	100	10.2	10.6	121
148	5.5	25	100	12.0	10.7	79
145	5.7	26	100	11.5	9.2	112
145	5.8	26	100	11.3	9.3	100
143	6.1	23	102	10.5	10.6	109
147	5.1	23	101	11.1	10.5	108
148	5.8	22	101	12.3	11.6	112
147	5.5	23	100	11.6	11.6	95
145	6.7	23	101	12.4	10.9	108
145	5.4	26	101	10.5	10.0	108
mean		<u></u>				
145	5.4	22	100	10.8	9.8	109
±sđ						
2	0.5	3	2	1.2	1.0	10
<u></u>					·····	

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Table 3, Continued

diluted [*] whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted ^{**} whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L
4.7	2.5	37	-6	137	24
4.7	2.5	38	0	128	35
4.5	2.5	38	-5	127	35
4.5	2.7	38	0	134	37
4.7	2.4	38	0	132	25
4.6	2.5	38	-6	130	32
4.6	2.7	38	0	134	35
4.8	2.5	38	0	137	31
5.0	2.5	39	6	142	32
4.9	2.2	39	-6	129	25
4.7	2.4	39	2	132	36
0.2	0.1	1	7	8	8

	anemic pigs								norma	l pig	s		
PCV [*]	PCV %	RBC's 10 ⁶ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %	PCV %	PCV %	RBC's 10 ⁶ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %
29.6 22.9 15.7 24.8 25.7 13.7 18.8 28.8 24.3 28.7 27.2 16.7 26.4 17.6 25.0 10.0 29.1	$\begin{array}{c} 28.0\\ 22.0\\ 15.0\\ 23.6\\ 24.6\\ 12.8\\ 18.2\\ 28.1\\ 23.1\\ 27.7\\ 26.0\\ 15.4\\ 25.7\\ 16.7\\ 24.0\\ 9.7\\ 27.9\end{array}$	7.9 7.3 6.5 6.4 6.3 4.0 4.7 8.2 6.2 7.6 4.8 4.1 7.4 6.5 4.3 1.7 6.0	6.3 5.5 6.3 6.5 2.8 4.3 7.8 5.7 7.8 8.9 3.8 7.3 3.6 7.5 2.2 8.3	35 30 23 37 39 32 39 34 37 36 54 38 35 26 57 47	8 6 10 7 9 10 9 10 19 9 10 6 17 13 14	23 25 24 27 26 22 24 28 25 28 34 25 28 25 28 25 28 21 23 30	35.6 39.4 38.4 31.3 37.2 34.1 34.6 32.5 32.7 32.6 36.8 29.8 34.0 31.5	34.2 37.9 37.2 33.5 36.6 33.0 33.8 31.9 31.9 31.9 31.4 35.9 29.0 32.7 30.4	5.3 6.1 6.7 6.6 8.9 6.5 6.2 5.5 7.6 5.5 7.7 7.4 6.7	9.2 10.4 11.0 10.4 10.4 10.0 9.8 10.1 9.5 10.1 11.0 9.2 10.1 9.8	65 62 56 51 55 58 42 57 47 39 45	17 17 16 12 15 16 18 13 18 14 12 15 15	27 27 30 31 28 30 29 32 30 32 31 32 31 32
mean 22.6	21.7	5.9	5.8	39	10	26	34.5	33.5	6.7	10.1	51	15	30
±sd 6.0	5.9	1.7	2.1	10	4	4	2.7	2.6	1.0	0.6	8	2	2

Table 4. Hematologic data of anemic and normal pigs 9 weeks of age

*includes leukocytes.

plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%	diluted [*] whole blood Na mEq/L	diluted whole blood K mEq/L	* RBC Na mEq/L	RBC K MEq/L
anemic	pigs									
135	6.5	15	99	10.6	7.7	150	4.8	2.0	7	129
119	4.7	26	77	10.0	5.9	104	4.9	1.4	26	109
144	5.2	26	102	11.1	8.2	104	4.8	2.4	-	-
121	4.7	18	91	9.7	5.7	109	5.1	1.0	40	68
136	5.2	23	100	10.7	6.6	100	5.2	1.6	8	1.1.4
122	5.9	15	90	10.0	5.9	124	5.3	0.9	0	94
137	5.3	18	102	10.4	6.5	108	5.4	1.3	-21	1.21
137	5.2	21	100	10.5	7.5	102	4.7	1.9	-14	121
137	6.3	19	100	10.5	7.5	127	5.0	1.7	-17	1.30
140	5.6	24	99	10.9	8.1	106	4.9	1.9	- 7	123
142	5.9	21	104	10.8	7.1	119	5.1	1.8	- 7	123
134	5.7	23	99	9.8	6.7	100	5.8	1.2	24	130
140	5.4	2 5	99	10.6	8.5	111	4.3	1.9	68	132
132	4.9	26	96	9.8	7.0	100	5.4	1.2	0	120
133	4.2	24	98	9.8	7.0	94	5.1	1.6	8	117
					(

Table 5. Blood electrolyte data of anemic and normal pigs 9 weeks of age

^{*}1 part blood and 1 part H_2O .

Table	5.	Continued	

	<u></u>		1				diluted*	diluted	*	
plasma	plasma	plasma	plasma	plasma	plasma	plasma	whole	whole	RBC	RBC
mEq/L	™Eq∕L	mEq/L	mEq/L	mg %	mgt	mg%	mEq/L	mEq/L	mEq/L	nEq/L
131	6.0	26	9 8	9.1	5.6	90	5.9	0.8	80	1.0 3
140	5.3	28	98	10.8	7.3	110	5.1	2.0	7	129
normal	pigs									
140	5.2	11	101	10.5	8.1	191	4.8	2.2	17	117
141	5.5	14	101	10.0	7.5	144	4.6	2.5	15	121
138	5.5	17	99	10.7	7.4	122	4.6	2.5	16	124
141	5.1	27	100	12.0	8.4	106	4.9	2.2	18	119
138	5.4	23	100	12.5	8.5	120	4.8	2.3	27	115
136	4.7	22	99	10.5	6.6	99	5.2	1.7	42	91
141	6.9	23	100	11.4	8.5	98	4.4	2.2	-12	118
142	5.2	26	100	11.3	8.0	110	4.8	2.1	0	119
144	5.5	22	102	11.3	8.1	107	4.8	2.2	0	125
142	5.8	21	102	11.9	8.2	107	4.7	2.1	- 6	121
145	6.3	25	100	11.5	8.3	110	4.7	2.4	5	123
137	4.9	25	100	10.6	7.3	112	5.1	2.1	20	131

Ti	ab]	Le	5	•	Con	ıt:	inue	d
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plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%	diluted whole blood Na mEq/L	diluted whole blood K mEq/L	* RBC Na mEq/L	RBC K n:Eq/L
137	4.9	26	99	10.8	7.6	118	4.8	2.3	18	128
138	5.0	25	101	10.8	8.0	120	4.9	2.1	13	125
mean (a	anemic pi	gs)								
134	5.4	22	97	10.3	7.0	109	5.1	1.6	12	127
±sd (ar	nemic pig	s)								
7	0.6	4	6	0.5	0.9	14	0.4	0.4	28	46
mean (r	normal pi	.gs)								
140	5.4	22	100	11.1	7.9	119	4.8	2.2	12	120
±sd (no	ormal pig	s)				,				
3	0.6	5	1	0.7	0.5	24	0.2	0.2	14	9

no Na pento	barbital	a.	Na pentoba	rbital	
P wave	T wave		P wave	T wave	
not record	ded		diphasic (-+ type)	monophasic	(+)
not recor	ded		monophasic (+)	monophasic	(+)
not recor	ded		diphasic (-+ type)	monophasic	(+)*
not recor	ded		monophasic (+)	monophasic	(+)*
not recor	ded		diphasic (-+ type)	monophasic	(+)*
not recor	ded		diphasic (-+ type)	monophasic	(+)*
not recor	ded		monophasic (+)	monophasic	(-)*
not recor	ded		diphasic (-+ type)	monophasic	(-)*
not recor	ded		monophasic (+)	monophasic	(+) *
not recor	ded		monophasic (+)	monophasic	(+)*
monophasic (+)	monophasic ((+)	diphasic (-+ type)	monophasic	(+)
not recor	ded		monophasic (+)	monophasic	(-)
monophasic (+)	monophasic ((+)	monophasic (+)	monophasic	(+)
diphasic (-+ type)	monophasic ((+)	monophasic (+)	monophasic	(+)
monophasic (+)	monophasic ((+)	monophasic (+)	monophasic	(+)*
monophasic (+)	monophasic ((+)	monophasic (+)	monophasic	(+)*
	no Na pentol P wave not record not record monophasic (+) monophasic (+)	no Na pentobarbital P T wave vave not recorded not recorded monophasic (+) monophasic diphasic (-+ type) monophasic monophasic (+) monophasic	no Na pentobarbitalPTwavewavenot recordednot recordedmonophasic (+)monophasic (+)	no Na pentobarbitalNa pentobarbitalP waveT waveP wavenot recordeddiphasic (-+ type) monophasic (+)not recordeddiphasic (-+ type) monophasic (+)not recordeddiphasic (-+ type) monophasic (+)not recordeddiphasic (-+ type) monophasic (+)not recordeddiphasic (-+ type) mot recordednot recordeddiphasic (-+ type) mot recordednot recordedmonophasic (+) diphasic (-+ type) mot recordednot recordedmonophasic (+) monophasic (+)monophasic (+)monophasic (+) diphasic (-+ type) mot recordedmonophasic (+)monophasic (+) monophasic (+)monophasic (+)monophasic (+) monophasic (+)	no Na pentobarbitalNa pentobarbitalP waveT waveP waveT wavenot recordeddiphasic (-+ type)monophasic: monophasic (+)monophasic: monophasic (+)not recordeddiphasic (-+ type)monophasic: monophasic (+)monophasic: monophasic (+)not recordeddiphasic (-+ type)monophasic: monophasic (+)monophasic: monophasic (+)not recordeddiphasic (-+ type)monophasic: monophasic (+)monophasic (+)not recordedmonophasic (+)monophasic (+)monophasic monophasic (+)monophasic (+)monophasic (+)monophasic (+)monophasic (+)monophasic (+)monophasic (+)monophasic (+)monophasic (+)

Table 6. Configuration of the P and T waves (Lead II) of pigs prior to KCl infusion

*also given 0.3 mg atropine sulfate after anesthetized.

pig #	infusion date 1968	age days	average time between samples minutes	total number of samples
1	27 Aug	73	19	14
2	4 Sep	85	18	10
3	26 Sep	63	16	15
4	7 Oct	88	19	18
5	8 Oct	69	16	15
6	14 Oct	74	16	17
7	17 Oct	89	17	16
8	29 Oct	70	14	16
9	13 Nov	56	16	14
10	19 Nov	75	17	14
11	25 Nov	73	18	16
12	26 Nov	64	17	11
13	3 Dec	62	18	8
14	9 Dec	60	18	11
15	10 Dec	60	17	9
16	16 Dec	68	18	10
mean		71	17	13
±sđ		10	1	3

Table 7. KCl infusion dates, age, average time between samples, and total number of blood samples from infused pigs

pig #	pig weight kg	period of infusion hours	total volume of isotonic KCl ml	mg/kg of pig (KCl)	mg/kg of pig per hour (KCl)	mEq/kg of pig (K)	mEq/kg of pig per hour (K)
1	18.1	3.2	500	309	97	4.1	1.3
2	23.1	2.8	450	218	80	2.9	1 . J.
3	15.9	2.4	385	271	112	3.6	1.5
4	21.8	5.3	850	437	83	5.9	1 , 1.
5	18.1	4.1	720	446	120	6.0	1.5
6	19.1	3.9	700	410	104	5.5	1.4
7	23.1	3.3	590	286	87	3.8	1,2
8	17.2	2.2	450	293	136	3.9	1.8
9	13.6	2.3	370	305	136	4.1	1.8
10	17.2	2.6	420	273	106	3.7	1.4
11	16.8	2.1	350	233	112	3.1	1.5
12	15.9	2.3	370	261	116	3.5	1.6
13	15.4	1.5	250	181	121	2.4	1.6
14	15.4	2.3	400	291	124	3.9	1.7
15	15.9	1.4	225	159	112	2.1	1.5
16	16.8	2.1	330	220	105	2.9	1.4
mean ±sd	17.7 2.8	2.7 1.0	460 173	287 83	109 17	3.8 1.1	1.5 0.2

.

Table ⁸. Quantity of KCl and K given intravenously as isotonic^{*} KCl until hear: block

*11.2 mg/ml of KCl (150 mEq/L of K).

	allowed	between po	ortions for s	sampling)	I	
PCV [*] %	PCV %	RBC's 10 ⁶ /cmm	HB gm%	MCV u ³	MCH uug	MCHC %
pig #1 -	• Na pentob preceded	arbital pri samples 2-1	or to sample	el. Ini	Eusion	
30.2	29.3	5.1	9.2	58	18	31
30.5	29.4	5.5	9.2	54	17	31
30.5	29.1	5.4	9.2	54	17	32
31.7	30.5	5.3	9.3	58	18	31
32.7	32.2	5.2	9.8	62	19	30
34.3	33.2	5 .7	10.4	58	18	31
36.4	34.7	5.9	11.0	58	19	32
37.4	36.4	6.5	11.4	56	18	31
38.6	37.5	7.2	11.7	52	16	31
39.5	38.5	7.2	12.1	54	17	31
40.2	39.0	7.6	12.4	51	16	32
31.9	38.1	7.2	12.1	53	17	32
38.0	36.2	5.9	12.1	61	21	33
37.4	36.1	5.4	11.0	67	20	31
pig #2 -	- Na pentob preceded	arbital pri samples 2-1	ior to sample LO.	e l. In	fusion	
31.6	31.0	5.6	10.4	55	19	34
37.7	36.8	6.3	12.3	58	20	33
37.6	36.7	6.1	12.3	60	20	34
38.2	37.2	6.8	12.4	54	18	33
37.7	37.1	6.7	12.1	55	18	33
37.9	37.2	6.6	12.3	56	19	33
36.0	35.0	6.5	11.9	54	18	34

Table 9. Hematologic data of pigs infused intravenously with an isotonic KCl solution (Infused in 50 ml portions at 3.82 ml/minute until heart block. Five minutes allowed between portions for sampling)

*includes leukocytes.

PCV [*]	PCV %	RBC's 10 ⁶ /cmm	HB gm%	MCV u ³	MCH uug	MCHC %
38.8	38.3	6.9	12.9	56	19	34
39.9	39.1	7.5	13.4	52	18	34
38.0	37.3	6.9	12.4	54	18	33
pig #3 ·	- Na pentob prior to	arbital pr: sample 1.	ior to san Infusion	nple l. A preceded	Atropine samples	sulfate 2-9.
26.4	26.0	4.0	7.8	65	20	30
27.3	26.8	4.1	8.1	65	20	30
26.2	26.6	4.1	7.8	65	19	29
27.7	27.1	4.7	7.8	58	17	29
28.6	28.2	4.7	7.8	60	17	28
29.4	29.0	5.0	8.3	58	17	29
30.9	30.4	5.4	8.5	56	16	28
32.1	31.3	5.5	8.8	57	16	28
32.7	32.3	5.1	9.0	63	18	28
32.6	32.1	5.2	8.9	62	17	28
30.2	29.7	5.2	8.9	57	17	30
36.1	35.7	5.0	9.8	71	20	28
32.0	31.4	5.1	9.2	62	18	29
32.5	32.5	4.8	9.2	68	19	28
30.8	30.6	4.8	8.6	64	18	28
pig #4 ·	- Na pentob prior to	arbital pr: sample 1.	ior to san Infusion	nple 1. A preceded	Atropine samples	sulfate 2-18.
24.4	23.5	4.3	6.8	55	16	29
24.0	23.0	4.4	6.8	52	16	30
24.3	23.6	4.2	6.5	56	16	28
25.8	24.8	4.6	7.5	54	16	30
26.1	25.2	4.5	7.1	56	16	28
27.3	26.2	4.5	7.8	59	17	29

Table ⁹. Continued

PCV [*]	PCV १	RBC's 10 ⁶ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %
28.2	27.0	4.7	8.1	57	17	30
29.2	28.2	4.8	8.3	5 9	17	29
30.3	29.5	5.0	8.9	59	18	30
31.4	30.4	5.0	9.5	61	19	31
31.2	30.4	5.0	9.2	61	18	30
31.2	30.2	5.0	9.0	60	18	30
31.2	30.5	5.1	9.0	60	18	30
30.8	30.0	5.1	8.8	59	17	29
30.2	29.1	5.0	8.6	58	17	30
29.5	28.8	4.8	8.6	60	18	30
28.8	28.0	4.8	8.3	58	17	30
29.0	28.0	4.8	8.2	58	17	29
pig #5 ·	- Na pentok prior to	oarbital prio sample l. I	r to sampl nfusion pr	e l. Ati eceded sa	copine su amples 2-	ulfate -14.
25.4	24.7	4.0	7.8	62	20	32
25.8	25.1	4.2	7.8	60	19	31
26.0	25.4	4.5	8.1	56	18	32
30.2	29.5	5.0	9.2	59	18	31
27.1	26.3	4.9	8.3	54	17	32
32.1	31.5	5.3	9.7	59	18	31
30.3	29.7	5.5	9.8	54	18	33
31.8	30.9	5.4	10.0	57	19	32
33.1	32.4	5.8	10.1	56	17	31
33.6	32.9	5.8	10.1	57	17	31
33.8	33.0	5.9	8.9	56	15	27
33.4	32.6	5.6	9.2	58	16	28
33.2	32.5	5.9	10.1	55	17	31
32.8	32.1	5.7	9.5	56	17	30
32.0	31.3	5.6	9.5	56	17	30

Table 9. Continued

PCV %	PCV %	RBC's 10 ⁶ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %
pig #6	- Na pentok prior to	parbital pr sample 1.	ior to sa Infusi o n	mple 1. 2 preceded	Atropine samples	sulfate 2-15.
32.1	31.4	6.3	9.5	50	15	30
36.0	35.4	7.5	10.6	47	14	30
36.8	36.1	8.0	11.0	45	14	31
37.5	37.0	8.8	11.1	42	13	30
37.3	36.7	8.6	11.1	43	13	30
35.5	34.8	8.0	10.7	44	13	31
36.3	35.7	7.5	11.0	48	15	31
35.4	34.8	7.5	10.7	46	14	31
36.2	35.8	7.6	11.2	47	15	31
37.8	37.0	8.0	11.4	46	14	31
38.0	37.3	7.8	11.4	48	15	31
37.1	36.7	7.4	11.0	50	15	30
36.7	36.1	7.2	10.7	50	15	30
35 .9	35.2	6.5	10.4	54	16	30
35.0	34.4	6.4	10.5	54	16	31
35.1	34.2	6.4	10.4	53	16	30
33.3	32.6	6.2	9.8	53	16	30
pig #7	- Na pentok prior to	parbital pr sample 1.	ior to sa Infusion	mple 1 preceded	Atropine samples	sulfate 2-12.
32.4	31.4	5.4	9.8	58	18	31
32.3	30.9	5.5	9.5	56	17	31
31.8	30.7	5.5	9.5	56	17	31
32.4	31.4	5.7	9.8	55	17	31
32.5	31.6	5.8	9.8	55	17	31
33.4	32.4	6.2	10.1	52	16	31
33.9	33.0	6.5	10.1	51	16	31
34.4	33.4	6.8	10.4	49	15	31

.

Table ⁹. Continued

PCV %	PCV %	RBC's 10 ⁶ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %
34.8	34.0	6.8	10.7	50	16	32
36.4	35.6	7.0	10.7	51	15	30
36.6	35.7	7.2	11.0	50	15	31
37.8	36.9	7.1	11.2	52	16	30
36.2	35.4	6.5	11.0	55	17	31
36.4	35.4	5.8	10.7	61	18	30
36.4	35.5	5.8	11.0	61	19	31
36.1	35.2	5.8	10.7	61	18	30
	•					
pig #8 -	- Na pentol prior to	parbital prices sample 1. I	or to sampl infusion pr	e 1. At: eceded sa	ropine su amples 2-	lfate
26.0	25.1	4.0	7.9	63	20	32
25.4	24.5	4.2	7.3	58	17	30
25.2	24.3	4.1	7.1	59	17	29
25.7	25.0	4.1	7.4	61	18	30
26.8	25.8	4.5	7.7	57	17	30
28.1	27.2	4.4	8.3	62	19	31
28.6	27.7	4.4	8.2	63	19	30
29.9	29.2	4.7	8.9	62	19	31
31.3	30.4	5.0	8.9	61	18	29
33.2	32.3	5.5	9.5	59	17	29
33.2	32.3	5.5	9.6	59	18	30
32.9	31.8	5.5	9.2	59	17	29
31.1	30.4	5.5	8 .9	55	16	29
30.6	29.8	5.7	8.9	52	16	30
30.2	29.3	5.4	8.9	54	17	30
29.6	28.6	5.4	8.3	53	15	29

Table 9. Continued

PCV [*] %	PCV %	RBC's 10 ⁶ /cmm	Hp Hp	MCV u ³	MCH uug	MCHC %
pig #9 ·	- Na pentok prior to	oarbital pr sample l.	ior to sar Infusion	nple 1. <i>P</i> preceded	Atropine samples	sulfate 2-9.
29.8	29.3	5.2	8.6	56	17	29
30.4	29.6	5.6	8.6	53	15	29
29.7	29.3	5.4	8.1	54	15	28
30.6	30.2	5.3	8.6	57	16	29
31.8	31.3	5.4	8.8	58	16	28
30.7	30.3	5.3	8.8	57	17	29
31.9	31.4	5.1	9.2	62	18	29
31.9	31.5	5.0	9.2	63	18	29
34.4	34.0	5.5	9.8	62	18	29
33.5	33.2	5.6	9.7	59	17	29
36.0	35.4	5.8	10.1	61	17	29
33.8	33.3	5.4	9.5	62	18	29
34.3	33.8	5.1	9.6	66	19	28
30.9	30.2	5.4	8.9	56	17	30
32.0	31.3	5.8	9.5	54	16	30
29.5	28.5	5.2	8.6	55	17	30
pig #10	- Na pento prior to	obarbital p sample 1.	rior to sa Infusion	ample 1. n preceded	Atropine 1 samples	e sulfate s 2-10.
35.9	34.9	5.2	10.3	67	20	30
35.7	35.0	5.5	10.5	64	19	30
35.6	34.6	5.5	10.5	63	19	30
38.0	37.1	6.0	11.4	62	19	31
38.5	37.8	6.3	11.4	60	18	30
39.1	38.3	6.3	11.4	61	18	30
40.0	39.1	6.4	12.1	61	19	31
40.3	39.3	6.4	11.7	61	18	30

Table 9. Continued

PCV [*] §	PCV %	RBC's 10 ⁶ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %
40.8	40.1	6.4	11.7	63	18	29
40.2	39.2	6.3	11.7	62	19	30
39.5	38.7	6.0	11.4	65	19	30
38.9	38.3	6.1	11.4	63	19	30
39.1	38.2	6.0	11.4	64	19	30
38.1	37.6	5.8	11.4	65	20	30
pig #ll -	Na pent precede	obarbital prio d samples 3-9.	r to sam <u>r</u>	ple 2. In	nfusion	
35.7	35.0	6.2	10.7	57	17	31
32.9	32.4	6.1	10.2	53	17	32
32.3	31.9	5.9	9.7	54	16	30
32.9	32.5	5.9	9.8	55	17	30
33.1	32.8	5.9	10.0	56	17	31
33.6	33.2	5.9	10.4	56	18	31
34.4	34.2	6.0	10.4	57	17	30
35.7	35.3	6.0	10.7	59	18	30
36.1	35.8	6.0	11.0	60	18	31
36.7	36.2	6.0	11.9	60	20	33
35.6	35.0	5.9	10.4	59	18	30
34.6	34.3	5.9	10.4	58	18	30
35.0	34.6	5 .9	10.4	59	18	30
32.7	32.4	5.9	9.8	55	17	30
31.6	31.2	5.9	9.5	53	16	30
31.0	30.5	5.8	9.2	53	16	30
pig #12 -	Na pent precede	obarbital prio: d samples 2-9.	r to samp	ole 1. Ir	fusion	
25.9	24.9	3.8	7.4	66	20	30
26.4	25.3	3.8	7.4	67	20	29

Table 9. Continued

PCV [*] %	PCV %	RBC's 10 ⁶ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %
26.5	25.5	3.9	7.5	65	19	29
27.0	26.1	3.8	7.8	69	21	30
27.2	26.4	4.4	8.0	60	18	30
29.2	28.5	4.7	8.3	61	18	29
30.1	29.6	4.8	8.5	62	18	29
31.6	30.2	4.8	8.7	63	18	29
31.7	30.9	5.1	9.0	61	18	29
30.6	29.6	5.0	8.6	59 .	17	29
30.8	30.0	5.1	8.8	59	17	29
pig # 13	- Na pento precedeo	obarbital pri 1 samples 3-7	or to samp •	le 2. Ir	fusion	
33.0	32.2	5.7	9.8	57	17	30
30.6	30.0	5.5	9.4	55	17	31
31.9	31.2	5.5	9.5	57	17	30
32.8	32.4	5.7	10.1	57	18	31
30.7	30.2	5.6	9.2	54	16	31
32.2	31.5	5.8	9.8	54	17	31
38.0	37.7	6.5	12.1	58	19	32
35.5	35.0	6.0	10.4	58	17	30
pig #14	- Na pento prior to	obarbital pri sample 3.	or to samp Infusion p	le l. At receded s	ropine : amples :	sulfate 2-8.
36.6	36.1	5.7	11.0	63	19	31
36.1	35.7	5.7	11.0	63	19	31
37.2	36.6	6.1	11.1	60	18	30
36.7	36.4	6.0	11.1	61	19	31
38.0	37.7	6.2	11.7	61	19	31
39.3	38.7	6.2	12.0	62	19	31

Table 9. Continued

PCV [*]	PCV &	RBC's 10 ⁶ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %
40.9	40.5	6.4	12.4	63	19	31
41.8	41.4	6.5	12.4	64	19	30
42.7	42.4	6.7	13.1	63	20	31
42.3	42.9	6.6	13.0	65	20	30
42.5	42.0	6.6	12.7	64	19	30
pig #15	- Na pento prior to	barbital pri sample 2.	or to samp Infusion p	le 2. At	ropine samples	sulfate 3-7.
34.1	33.4	5.4	10.0	62	19	30
30.2	29.5	5.1	8.8	58	17	30
30.9	29.8	5.1	8.9	58	18	30
31.8	31.1	5.2	9.3	60	18	30
32.1	31.3	5.3	9.6	59	18	31
33.4	32.7	5.4	9.7	61	18	30
34.5	33.7	5.5	9.7	61	18	29
34.4	33.6	5.5	10.0	61	18	30
32.7	31.6	5.3	9.4	60	18	30
pig #16	- Na pento prior to	barbital pri sample 2.	or to samp Infusion p	le 2. At receded s	ropine amples	sulfate 3-9.
33.4	32.8	5.6	9.8	59	18	30
34.0	33.5	5.6	9.7	60	17	29
33.4	32.9	5.5	9.2	60	17	28
33.0	32.7	5.5	9.7	60	18	30
34.0	33.5	5.6	10.4	60	19	31
36.2	35.8	5.9	10.4	61	18	29
35.9	35.5	5.8	10.4	61	18	29
37.4	37.0	6.1	10.7	61	18	29
37.7	37.0	6.1	10.7	61	18	29
37.0	36.4	6.0	10.7	61	18	29

Table 10. Blood electrolyte data of pigs infused intravenously with an isotonic KCl solution (Infused in 50 ml portions at 3.82 ml/minute until heart block. Five minutes allowed between portions for sampling)

plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
pig #1 -	• Na pentob preceded	arbital pi samples 2	rior to sa -11.	ample 1.	Infusion	
141	3.9	26	96	9.0	7.5	79
140	4.7	26	97	9.0	7.2	78
139	5.1	26	97	9.3	7.1	78
138	6.0	26	96	8.9	7.1	73
137	6.5	25	96	9.6	7.1	72
136	7.5	25	96	9.3	7.0	69
133	8.3	25	96	9.9	6.7	63
134	8.8	25	95	9.9	6.2	59
133	8.7	25	96	8.7	6.1	57
133	8.8	25	96	9.7	6.2	53
132	9.0	25	96	9.8	6.7	52
135	8.1	24	96	9.8	6.7	56
134	7.3	23	96	9.5	6.7	66
134	7.8	23	96	8.3	8.0	74
pig #2 -	 Na pentob preceded 	arbital p samples 2	rior to sa -10.	ample 1.	Infusion	
140	3.9	30	97	9.5	7.5	-
139	5.8	28	96	8.8	8.8	-
138	4.2	28	97	8.6	8.4	-
····						

*1 part blood and 19 parts H₂O.

******1 part blood and 1 part H₂O.

diluted [*] whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted ^{**} whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L
		······································			
5.0	2.0	39	2	127	39
5.0	2.1	40	7	131	38
5.1	2.2	42	16	136	49
5.0	2.3	39	15	134	36
4.9	2.4	38	15	132	35
4.6	2.5	38	8	133	38
4.5	2.6	38	12	134	41
4.4	2.7	35	9	133	28
4.3	2.8	37	11	135	38
4.3	2.9	37	16	134	38
4.2	2.8	37	12	130	39
4.3	2.8	37	10	131	37
4.3	2.7	38	8	137	41
4.4	2.6	37	9	130	39
4.7	2.0	-	-6	121	-
4.3	2.4	-	-2	121	-
4.3	2.4	-	0	121	

plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
140	4.7	. 29	98	8.7	7.9	_
139	5.3	29	99	9.1	7.9	-
139	5.6	29	98	9.0	7.7	-
139	6.1	29	98	9.2	7.5	-
135	7.8	30	96	8.9	7.5	-
135	8.0	29	96	9.3	7.3	-
132	11.2	28	97	8.6	8.2	-
pig #3 -	Na pentob prior to	arbital p sample 1.	rior to sa Infusion	ample 1. n preceded	Atropine 1 samples	sulfate 2-9.

pig #3 -	Na pentoba prior to s	rbital pr ample 1.	rior to sa Infusion	ample 1. n preceded	Atropine I samples	sulfate 2-9.
135	4.3	23	99	9.5	7.3	80
134	4.7	22	100	10.2	7.5	88
134	5.4	23	100	8.7	7.0	80
133	5.7	24	99	9.9	6.6	76
133	6.1	24	99	10.6	6.9	73
131	6.7	24	98	10.9	6.7	69
131	6.9	24	98	10.2	6.5	64
129	8.2	24	97	10.4	6.6	62
127	8.4	24	97	10.0	6.7	60
128	8.4	23	97	10.8	6.7	60
129	7.7	23	97	10.5	6.9	61
130	7.1	21	96	11.4	8.0	54
131	5.4	22	96	10.7	7.6	73
131	5.0	22	97	10.1	7.6	75
132	5.0	22	96	11.3	8.3	79

diluted [*] whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted ^{**} whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/I
4.2	2.4		-6	118	-
4.6	2.5	-	14	126	-
4.4	2.5	-	3	130	-
4.6	2.4	-	8	126	-
4.2	2.5	-	4	118	-
4.1	2.7	-	3	126	-
4.1	2.7	-	1	126	-
5.0	17	_	0	115	_
5.0	1.8	_	7	119	
4.9	1.7	-	, 0	113	_
4.9	1.8	_	7	118	-
5.0	1.9	-	14	121	-
4.7	2.0	-	7	124	-
4.6	2.1	_	7	125	
4.4	2.0	-	0	109	-
4.4	2.2	-	6	118	-
4.3	2.2	-	0	118	-
4.4	2.1	-	-6	121	-
4.2	2.3	_	0	118	-
4.4	2.0	-	-6	115	-
4.5	2.1	-	6	117	-
4.6	2.0	_	0	118	-

plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
pig #4 -	Na pentob prior to	arbital pr sample 1.	ior to s Infusio	ample 1. n preceded	Atropine samples	sulfate 2-18.
140	3.8	28	97	11.1	7.4	78
140	4.3	28	98	11.4	7.4	74
139	4.7	28	98	11.8	7.7	74
139	5.0	28	97	11.9	7.5	73
139	5.2	28	97	11.8	7.5	67
138	5.5	28	97	11.6	7.5	70
137	5.7	27	97	11.8	7.6	64
136	6.1	27	96	11.3	7.4	63
136	6.5	27	96	11.7	7.6	61
135	6.5	26	95	12.0	7.8	55
135	7.1	26	96	11.6	7.8	54
134	7.0	25	95	11.2	7.6	54
134	7.3	24	96	10.7	7.7	60
134	7.5	23	96	11.0	7.7	64
134	7.0	23	96	10.4	7.7	69
134	7.0	22	96	10.3	7.6	72
133	7.4	21	97	10.3	7.8	75
133	8.3	21	97	10.2	7.5	76
pig #5 -	Na pentob prior to	arbital pr sample 1.	ior to s Infusio	ample l. n preceded	Atropine samples	sulfate 2-14.
142	3 .9	25	102	9.9	7.1	105
141	4.7	24	102	10.0	6.9	9 8
142	5.2	25	102	9.9	6.6	101
140	5.4	25	101	10.0	6.4	110
138	5.4	24	102	9.9	6.3	95

diluted [*] whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted ^{**} whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L
	 	· · · · · · · · · · · · · · · · · · ·			
6.2	1.6	-	74	128	-
6.1	1.6	-	67	122	-
5.5	1.7	-	17	127	-
5.7	1.8	-	39	129	-
5.3	1.8	-	15	127	-
5.1	1.9	-	7	128	-
5.1	2.0	-	14	133	-
5.0	2.0	-	14	128	-
4.8	2.0	-	7	122	-
4.8	2.1	-	13	125	-
4.6	2.1	-	0	118	-
4.8	2.1	-	13	126	-
5.0	2.1	-	26 ·	118	-
5.0	2.2	-	26	127	-
5.0	2.1	-	13	131	-
5.1	1.9	-	27	111	~
5.0	2.0	-	21	121	-
5.2	2.2	-	35	136	-
5.3	1.6	-	0	122	· _
5.2	1.6	-	0	122	-
5.1	1.8	-	-	126	-
4.9	2.0	-	0	122	-
5.1	1.8	-	7	122	-

plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
139	5.4	24	101	10.5	6.7	100
137	5.6	24	101	10.3	6.3	90
137	5.6	24	101	10.3	6.3	87
133	7.0	25	99	11.2	6.8	90
133	7.5	25	98	11.5	6.5	92
132	8.0	24	99	9.8	6.7	96
131	8.2	24	97	10.4	6.8	96
130	8.4	24	97	10.4	6.8	98
131	8.8	24	97	10.6	6.9	104
131	7.1	23	97	10.3	6.8	104
pig #6 -	Na pentob prior to	arbital p sample 1.	rior to sa Infusion	ample 1. n preceded	Atropine 1 samples	sulfate 2-15.
137	3.7	26	99	9.3	8.6	82
134	4.1	26	98	9.3	8.6	2 15
135	4.2	24	99	9.2	8.8	154
137	4.3	24	102	9.3	9.3	89
136	4.5	22	101	9.2	9.7	65
135	5.1	23	101	9.0	9.9	69
136	5.6	24	100	9.1	9.6	76
134	6.2	24	99	9.0	8.2	81
134	6.7	24	98	9.3	8.3	79
133	7.3	24	97	9.6	8.6	75
132	8.3	21	98	9.7	9.0	95
133	7.2	21	98	9.1	8.7	84
132	8.3	21	97	8.9	8.4	72
131	9.1	22	96	9.7	8.3	73

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diluted whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted ** whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L	
4.8	2.1	_	6	121		
4.7	1.9	-	-7	115	-	
4.8	2.0	-	6	117	-	
4.6	2.2	-	6	124	-	
4.5	2.2	-	6	116	-	
4.3	2.1	-	-6	109	-	
4.6	2.3	-	12	123	-	
4.5	2.2	-	12	117	-	
4.5	2.2	-	6	118	-	
4.8	2.2	-	13	128	-	
5.2	2.1	_	31	127	-	
4.6	2.3	-	17	124	-	
4.7	2.4	-	22	127	-	
4.6	2.5	-	16	130	-	
4.7	2.5	-	21	131	-	
4.7	2.4	-	17	126	-	
4.6	2.4	-	17	123	-	
4.3	2.2	-	0	115	-	
4.6	2.5	-	17	129	-	
4.3	2.4	-	11	119	-	
4.4	2.6	-	16	123	-	
4.4	2.5	-	11	125	-	
4.5	2.5	_ .	16	122	-	
4.4	2.5	-	11	125	-	
plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
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131	9.8	22	95	9.1	8.2	74
132	9.4	23	95	9.4	8.1	77
131	9.1	22	95	10.2	8.2	76
pig #7 -	- Na pentob prior to	arbital pr sample l.	tior to sa Infusion	ample 1. n preceded	Atropine samples	sulfate 2-12.
142	4.0	26	100	10.0	7.2	95
141	4.3	26	101	9.6	7.0	89
143	4.8	26	101	9.5	6.9	83
143	5.6	27	101	9.9	6.9	84
141	5.5	26	100	9.9	7.0	83
141	5.8	26	100	9.8	7.0	86
141	6.2	25	101	10.0	7.1	93
139	6.7	25	100	9.6	7.2	87
139	7.1	25	100	10.2	6.9	88
139	8.0	24	100	9.8	6.8	97
137	8.1	24	100	10.0	6.7	99
132	10.5	24	99	11.6	7.5	119
134	7.5	19	99	9.3	7.9	237
131	7.6	21	98	9.1	7.7	202
130	7.9	27	99	10.0	7.5	158
130	7.5	28	99	10.0	7.7	119
pig #8 -	– Na pentob prior to	arbital pr sample 1.	ior to sa Infusion	ample 1. n preceded	Atropine 1 samples	sulfate 2-10.
142	4.0	30	95	9.8	7.6	70
140	4.6	30	97	9.7	7.9	65
140	5.0	30	96	9.9	8.0	60

diluted [*] whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted ^{**} whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L
4.4	2.4	_	6	122	-
4.4	2.5	-	6	129	
4.5	2.3	-	6	123	-
5 0	2 2	20	10	124	20
5.0	2.2	39	12	102	29
4.9	2.1	39	6	123	27
5.0	2.2	39	6	130	29
4.9	2.2	39	6	127	30
4.9	2.2	40	6	127	35
4.8	2.2	39	6	124	31
4.8	2.3	39	6	127	30
4.7	2.4	39	6	132	33
4.7	2.4	39	12	129	34
4.5	2.5	38	6	124	34
4.5	2.5	38	11	123	34
4.3	2.6	40	11	125	46
4.5	2.5	38	11	130	33
4.4	2.5	38	11	130	35
4.5	2.5	39	22	121	39
4.6	2.5	39	22	131	39
5 3	1 -	20	•	100	<u>.</u>
5.1	1./	39	U	120	25
5.2	1.7	39	0	114	18
5.3	1.8	39	8	132	21

plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
138	5.2	30	97	10.2	7.5	56
132	5.2	30	96	10.0	7.1	52
136	5.8	30	96	10.3	7.1	47
136	6.6	30	96	10.4	6.9	49
135	7.5	30	96	10.7	7.2	50
134	8.3	29	95	10.6	7.1	50
131	8.7	28	96	11.3	7.1	51
130	8.4	28	95	10.7	6.9	51
133	7.7	28	95	11.0	7.2	53
134	7.0	28	95	10.8	7.4	53
133	6.5	28	94	10.6	7.5	59
135	5.9	28	94	10.8	7.6	58
134	5.5	27	95	9.4	7.4	63
pig #9 -	Na pentob prior to	arbital pr sample l.	ior to sa Infusion	ample 1. 1 precedeo	Atropine 1 samples	sulfate 2-9.
144	3.8	23	104	10.7	6.6	80
142	4.8	22	104	10.5	6.7	74
144	5.5	23	105	10.6	6.6	81
140	6.0	23	105	10.5	6.5	81
140	6.9	22	105	10.2	6.4	81
138	7.7	23	104	10.5	6.2	85
137	8.2	23	103	10.6	6.1	76
137	8.8	23	104	10.8	6.2	71
135	9.3	22	103	10.8	6.1	70
136	8.5	23	103	10.7	6.5	69
136	8.3	23	102	10.5	6.9	70

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diluted [*] whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L
5.2	1.8	38	8	128	19
4.8	1.6	38	0	109	22
5.1	1.9	38	14	125	21
4.9	1.9	38	0	123	23
4.8	2.1	38	7	123	26
4.6	2.1	37	0	118	28
4.5	2.3	37	6	123	27
4.5	2.2	38	12	118	35
4.6	2.2	37	6	120	28
4.7	2.1	37	6	125	24
4.8	2.1	37	13	128	26
4.8	2.2	38	7	137	31
4.9	2.1	37	14	133	24
5.0	2.0	41	-7	130	27
5.0	2.1	41	7	143	28
4.9	2.1	40	-13	130	18
4.9	2.2	40	0	132	24
4.7	2.3	40	-6	134	23
4.7	2.3	40	-7	132	23
4.7	2.4	39	0	134	25
4.8	2.5	39	6	140	27
4.3	2.4	39	-6	124	27
4.5	2.5	41	0	133	41
4.5	2.6	38	6	130	27

plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
136	8.2	23	100	10.6	7.1	82
135	7.7	23	99	10.4	7.7	83
135	7.0	25	97	9.9	8.2	95
141	4.1	26	103	10.1	8.0	95
145	4.4	24	104	10.0	7.9	86
pig #10	- Na pento prior to	barbital p sample 1.	rior to a Infusio	sample 1. on precede	Atropine d samples	sulfate 2-10.
139	4.0	25	100	11.4	8.9	99
140	5.0	25	100	11.2	9.0	99
138	5.5	24	101	12.1	9.3	95
138	6.2	23	101	12.0	9.0	99
137	6.6	23	100	11.7	9.0	96
134	7.3	22	100	12.3	9.2	99
134	7.6	22	100	11.9	9.1	95
133	8.0	21	100	11.7	9.1	102
132	8.0	20	100	12.2	9.2	97
134	8.2	20	101	11.6	9.5	96
134	7.1	20	100	11.7	9.5	97
133	6.9	20	100	11.6	10.0	96
133	6.8	21	99	11.8	10.3	93
133	6.9	20	99	10.6	10.0	95
pig #ll	- Na pento preceded	barbital p samples 3	rior to : -9.	sample 2.	Infusion	
148	3.7	23	109	11.0	6.8	84
150	3.7	24	109	11.2	6.9	88
150	4.5	23	110	11.5	6.8	93

diluted [*] whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted ^{**} whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/I
4.6	2.5	38	6	132	29
4.4	2.5	37	0	130	26
4.7	2.4	38	0	146	26
5.5	2.6	40	44	160	28
5.2	2.1	40	7	133	19
4.7	2.5	38	11	138	30
4.9	2.6	38	22	137	30
4.7	2.6	37	17	139	25
4.3	2.6	37	0	129	30
4.5	2.8	37	16	138	30
4.4	2.9	36	15	141	28
4.3	2.9	36	15	138	30
4.2	2.9	36	10	137	30
4.3	3.0	36	20	140	31
4.3	2.9	36	15	138	29
4.4	2.8	36	15	134	29
4.4	2.8	37	15	136	30
4.3	2.7	36	10	131	30
4.7	3.0	36	32	149	28
A Q	2 5	40	٤	127	25
*•J 5 3	2.5	40 A 1	10	112	20
5.5	4 • 4	±⊥ 4]	10	1 2 2	32 34

plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
149	5.1	23	110	11.2	6.6	81
147	5.4	22	111	11.3	6.4	77
147	6.0	23	110	11.2	6.6	75
146	6.4	22	110	11.2	6.6	77
145	7.3	22	110	11.2	6.6	80
143	8.0	21	111	10.5	6.4	83
144	8.5	21	110	11.5	6.4	80
143	8.8	21	111	11.5	6.5	80
142	8.8	21	110	11.2	6.4	79
141	10.0	22	111	11.1	6.2	85
144	7.7	21	111	11.7	6.5	92
144	7.3	21	111	11.5	6.5	90
144	7.0	21	109	10.9	6.4	87
pig #12	- Na pento precedeo	barbital j 1 samples	prior to 3-9.	sample 1.	Infusion	
141	3.6	23	99	9.8	7.5	66
138	4.6	22	99	10.1	7.6	66
139	5.5	22	99	9.9	7.3	69
137	5.9	22	100	9.8	7.2	73
134	6.4	21	100	9.9	7.3	65
132	7.2	21	98	10.2	7.2	71
132	7.9	21	100	10.2	7.0	76
131	8.2	21	99	9.9	6.9	77
131	9.9	20	100	10.2	7.4	73
132	6.4	15	98	9.7	8.3	208
132	6.1	18	98	9.4	7.2	144

Table 10. Continued

diluted [*] whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L
5.1	2.3	41	6	129	25
5.0	2.4	41	0	134	24
4.9	2.4	41	0	133	27
4.9	2.5	41	6	135	26
4.7	2.6	41	0	136	29
4.8	2.7	40	11	134	25
4.7	2.7	40	5	133	26
4.8	2.7	40	11	137	21
4.8	2.6	40	6	134	23
4.7	2.7	40	6	139	22
5.0	2.4	41	12	130	22
5.0	2.3	42	6	128	22
5.1	2.3	41	7	138	19
5.1	1.7	40	-8	129	26
4.8	1.7	41	-23	119	31
4.8	1.8	41	-23	125	31
4.7	1.8	41	-22	123	30
4.7	1.9	40	-15	129	23
4.6	2.0	39	-7	119	30
4.5	2.1	38	-7	122	20
4.5	2.2	38	0	126	26
4.4	2.3	39	-6	129	27
4.6	2.1	39	0	128	29
4.5	2.0	38	-7	120	27

plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
pig #13 -	- Na pento preceded	barbital p samples 3	rior to 3-7.	sample 2.	Infusion	an <u>, ,,,,,,,,,,,,,,,,</u> ,,,,,,,,,,,,,,,,,,,
139	3.5	20	102	10.3	9.7	97
140	3.5	22	102	9.7	10.0	84
140	4.4	21	102	10.3	10.2	87
141	5.7	22	103	10.5	10.4	82
139	6.2	22	103	10.8	10.5	82
138	7.3	22	101	11.0	10.4	80
135	9.9	21	102	11.9	9.9	75
138	7.6	22	105	10.1	9.6	69
pig #14 -	Na pento prior to	barbital p sample 3.	rior to Infusio	sample 1. on precede	Atropine d samples	sulfate 2-8.
141	3.6	23	105	10.9	8.7	91
141	4.6	22	106	11.2	9.1	93
140	4.8	22	106	11.2	8.7	91
140	5.3	21	107	11.3	8.6	81
140	5.8	21	107	11.5	8.5	68
140	6.3	21	107	11.6	8.3	71
138	7.2	21	106	11.5	8.1	73
138	7.3	21	105	11.7	8.1	71
137	9.0	19	105	11.4	7.5	93
138	7.7	20	106	11.4	7.6	77
138	7.3	20	107	11.1	7.3	65
pig #15 -	• Na pento prior to	barbital p sample 2.	rior to Infusio	sample 2. on precede	Atropine d samples	sulfate 3-7.
142	4.0	26	98	10.3	8.2	81
144	4.0	26	98	9.6	8.2	44

diluted [*] whole blood Na	diluted [*] whole blood K	diluted ^{**} whole blood K	RBC Na	RBC K	RBC C1
mEq/L	mEq/L	mEq/L	mEq/L	mEq/L	mEq/L
4.6	2.1	38	-6	124	23
4.8	2.0	39	-7	127	20
4.6	2.0	39	-13	115	24
4.6	2.1	39	-6	117	24
4.7	2.0	40	-7	119	25
4.6	2.2	39	-6	121	26
4.1	2.6	38	-5	122	33
4.3	2.4	38	-11	126	29
4.8	2.6	39	16	139	28
4.7	2.6	39	11	140	28
4.7	2.7	39	16	137	31
4.7	2.7	39	16	137	28
4.5	2.8	39	11	138	28
4.5	2.9	39	15	140	31
4.3	3.0	39	10	138	35
4.2	3.0	38	10	135	35
4.1	3.2	38	9	136	37
4.1	3.1	38	5	137	33
4.2	3.1	38	10	136	34
4.9	2.4	37	12	138	28
5.2	2.2	39	13	142	29

plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
143	4.9	26	98	9.3	8.4	31
143	5.4	26	100	9.4	8.0	24
142	6.2	25	99	9.3	7.9	19
139	7.2	25	98	9.2	7.7	18
139	8.3	24	9 9	9.3	7.8	20
139	8.0	22	99	9.1	8.3	16
136	6.5	22	96	8.9	7.9	67
pig #16	- Na pento prior to	barbital p sample 2.	prior to a Infusio	sample 2. on precede	Atropine ed samples	sulfate 3-9.
140	3.3	22	100	9.8	6.6	101
141	3.5	24	100	10.1	7.3	89
141	4.6	25	100	10.0	7.3	83
140	5.3	25	101	10,1	7.2	75
140	5.8	25	100	9.9	7.0	65
138	6.7	26	100	9.8	6.7	68
137	7.9	24	100	9.6	6.4	71
135	8.9	25	100	10.3	6.4	67
135	9.2	25	99	10.3	6.4	66
135	8.5	25	98	10.4	6.5	51

diluted [*] whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted ^{**} whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L
5.0	2.2	30	7	134	30
5.0	2.3	38	6	135	25
5.0	2.3	38	13	134	28
4.8	2.5	38	12	141	32
4.7	2.5	38	6	131	30
4.6	2.4	38	0	125	29
4.6	2.3	38	0	133	32
4.7	2.2	39	0	128	31
4.8	2.1	40	6	119	38
4.8	2.2	40	6	122	37
4.9	2.2	40	12	122	35
4.8	2.2	40	6	119	38
4.7	2.4	39	17	123	37
4.6	2.5	39	11	124	33
4.4	2.7	38	11	130	33
4.4	2.7	38	11	130	38
4.5	2.6	38	11	126	38

Table 11. Electrocardiographic data (Lead II) of pigs infused intravenously with an isotonic KCl solution (Infused in 50 ml portions at 3.82 ml/minute until heart block. Five minutes allowed between portions for recording)

heart rat per minute	te respirations per minute	P amplitude mV	T amplitude mV
pig #1 -	Na pentobarbital prior to preceded samples 2-11.	o sample l. Infu	ision
124	32	0.10	0.45
122	31	0.10	0.28
124	32	0.10	0.30
120	29	0.10	0.18
113	31	0.10	0.28
120	34	0.12	0.28
112	29	0.15	0.28
122	30	0.08	0.28
133	31	-0.10	0.32
189	30	0	0.65
200	36	0	0.62
150	39	0.10	0.25
144	38	0.20	0.22
144	43	0.10	0.18
pig #2 -	Na pentobarbital prior to preceded samples 2-10.	o sample l. Infu	ision
200	44	0.14	0.30
222	20	0.20	0.35
178	29	0.22	0.20
160	28	0.22	0.20
149	26	0.20	0.10
164	29	0.22	0.10
138	32	0.12	0.05

P duration second	P-Q interval second	QRS duration second	Q-T interval second	T-wave angle degrees
		<u></u>		, an
0.08	0.10	0.04	0.29	59
0.08	0.10	0.04	0.30	56
0.08	0.10	0.04	0.29	56
0.08	0.10	0.04	0.29	50
0.08	0.10	0.04	0.29	59
0.10	0.10	0.04	0.28	58
0.11	0.11	0.04	0.29	61
0.12	0.12	0.05	0.27	71
0.12	0.12	0.05	0.25	69
-		0.07	0.21	81
-	-	0.05	0.20	78
0.11	0.11	0.04	0.23	67
0.09	0.09	0.04	0.24	65
0.07	0.07	0.04	0.23	72
0.05	0.05	0.05	0.23	60
0.04	0.04	0.04	0.21	70
0.05	0.06	0.05	0.26	60
0.07	0.07	0.05	0.25	60
0.07	0.07	0.05	0.26	54
0.07	0.07	0.05	0.24	51
0.07	0.08	0.05	0.29	44

Table	11.	Continued

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heart rate per minute	respirations per minute	P amplitude mV	T amplitude mV
150	24	0.15	0.10
257	28	0	0.13
25	25	-	-

pig #3 - Na pentobarbital prior to sample 1. Atropine sulfate prior to sample 1. Infusion preceded samples 2-9.

138	15	0.15	0.10
150	15	0.15	0.15
150	15	0.15	0.20
167	17	0.17	0.20
180	19	0.15	0.10
164	17	0.15	0.10
137	22	0.15	0.22
133	14	0.02	0.30
124	14	0.05	0.30
132	21	0	0.35
134	22	0.02	0.40
131	18	0.02	0.20
124	34	0.02	0.42
138	34	0.10	0.32
157	22	0.12	0.18

pig #4 - Na pentobarbital prior to sample 1. Atropine sulfate
prior to sample 1. Infusion preceded samples 2-18.180140.150.15164190.15-.05167260.20-.08

24

0.20

-.05

P duration second	P-Q interval second	QRS duration second	Q-T interval second	T-wave angle degrees
0.07	0.07	0.05	0.25	68
-	-	0.04	0.21	80
-	-	-	-	-
0.05	0.07	0.04	0.28	27
0.05	0.08	0.04	0.28	36
0.05	0.08	0.04	0.27	40
0.05	0.08	0.04	0.23	44
0.04	0.08	0.04	0.24	54
0.06	0.08	0.04	0.24	50
0.07	0.08	0.04	0.24	53
0.05	0.05	0.04	0.25	51
0.07	0.07	0.05	0.25	54
-	-	0.07	0.24	60
0.03	0.03	0.04	0.27	68
0.01	0.08	0.04	0.26	48
0.06	0.08	0.04	0.28	69
0.06	0.07	0.04	0.27	56
0.05	0.08	0.04	0.23	45
	0.07	0.00	0.03	27
0.04	0.07	0.03	0.21	57
0.05	0.08	0.03	0.20	52
0.05	0.07	0.03	0.20	27
0.05	0.07	0.03	0.20	35

heart rate per minute	respirations per minute	P amplitude mV	T amplitude mV
141	15	0.20	05
150	25	0.20	05
157	24	0.18	05
150	26	0.20	0.10
157	26	0.20	0.15
171	27	0.15	10
176	28	0.15	0.10
144	27	0.10	15
167	21	0.10	10
150	29	0.05	12
153	42	0.08	15
150	23	0.10	0.10
150	22	0.05	0.20
114	34	0	0.30
pig #5 - Na pe prio	entobarbital prior t r to sample 1. Infu	o sample l. Atro sion preceded sam	pine sulfate ples 2-14.
160	56	0.15	0.45
157	44	0.15	0.60
164	54	0.20	0.40
185	24	0.15	0.40
167	43	0.15	0.50
200	25	0.15	0.50
185	28	0.10	0.50
200	21	0.10	0.50
140	21	0.15	0.55
136	22	0.15	0.55
138	24	0.10	0.55

P duration second	P-Q interval second	QRS duration second	Q-T interval second	T-wave angle degrees
0.05	0.08	0.03	0.21	22
0.05	0.07	0.03	0.20	42
0.05	0.07	0.03	0.19	45
0.05	0.07	0.03	0.20	65
0.05	0.07	0.03	0.20	65
0.06	0.07	0.03	0.18	65
0.06	0.07	0.03	0.18	64
0.05	0.08	0.03	0.18	65
0.07	0.07	0.03	0.17	62
0.05	0.07	0.03	0.17	70
0.04	0.07	0.03	0.17	60
0.03	0.06	0.03	0.18	61
0.05	0.07	0.03	0.19	64
-	-	0.08	0.22	78
0.04	0.08	0.04	0.22	64
0.04	0.07	0.04	0.22	68
0.05	0.08	0.04	0.21	69
0.05	0.08	0.04	0.20	73
0.05	0.08	0.04	0.20	70
0.05	0.08	0.04	0.20	71
0.05	0.08	0.04	0.20	71
0.05	0.08	0.04	0.21	69
0.05	0.07	0.04	0.23	68
0.05	0.07	0.04	0.24	67
0.06	0.09	0.04	0.24	72

heart rate per minute	respirations per minute	P amplitude mV	T amplitude mV
133	23	0.08	0.55
129	20	0.02	0.50
133	20	- 05	0.50
133	24	0.10	0.50
pig #6 - Na pe prior	entobarbital prior t r to sample l. Infu	o sample l. Atro sion preceded sa	pine sulfate mples 2-15.
180	42	0.15	0.25
237	31	0.20	0.12
232	36	0.20	0.20
237	43	0.25	0.30
231	48	0.15	0.30
225	54	0.20	0.35
240	42	0.20	0.30
232	48	0.20	0.40
200	38	0.15	0.35
240	27	0.10	0.25
240	30	0.10	0.20
240	17	0.10	0.25
248	15	0.10	0.10
218	17	0.05	10
240	16	0	0.80
267	20	0	15
180	29	02	0.20
pig #7 - Na pe prio:	entobarbital prior t r to sample l. Infu	to sample l. Atro sion preceded sa	pine sulfate mples 2-12.
180	26	0.02	02
206	30	0.08	02

P duration second	P-Q interval second	QRS duration second	Q-T interval second	T-wave angle degrees
0.06	0.10	0.04	0.24	72
0.07	0.12	0.04	0.24	73
0.05	0.12	0.04	0.24	68
0.08	0.08	0.04	0.24	66
0.04	0.07	0.04	0.20	53
0.04	0.06	0.04	0.19	51
0.04	0.06	0.04	0.18	55
0.04	0.06	0.04	0.18	57
0.04	0.07	0.04	0.19	62
0.04	0.06	0.04	0.18	64
0.04	0.06	0.04	0.18	62
0.05	0.07	0.04	0.17	59
0.05	0.07	0.04	0.18	65
0.05	0.07	0.04	0.17	64
0.07	0.07	0.04	0.20	65
0.05	0.07	0.04	0.16	65
0.06	0.06	0.04	0.18	65
0.08	0.08	0.04	0.15	65
-	-	0.11	0.24	86
-	-	0.05	0.14	71
0.03	0.08	0.04	0.17	60
0.03	0.07	0.03	0.19	29
0.03	0.06	0.03	0.15	17
		-		

heart rate per minute	respirations per minute	P amplitude mV	T amplitude mV
171	32	0.05	05
180	28	0.10	05
164	34	0.05	10
185	33	0.05	05
180	23	0.05	02
167	29	0.10	05
189	28	0.05	02
176	38	0.02	01
185	24	0.05	02
110	12	0	20
206	16	0.05	22
195	21	0.05	10
189	39	0.10	05
212	23	0.03	05
pig #8 - Na p pric	entobarbital prior t or to sample 1. Infu	o sample l. Atro sion preceded sa	pine sulfate mples 2-10.
169	25	0.30	02
155	34	0.25	0.10
170	44	0.30	02
180	48	0.30	0.05
180	54	0.30	0.10
195	50	0.20	0.08
176	52	0.30	0.10
171	44	0.30	0.17
180	50	0.20	0.10
189	50	0	0.32
10)			

P duration second	P-Q interval second	QRS duration second	Q-T interval second	T-wave angle degrees
0.03	0.06	0.03	0.15	25
0.03	0.06	0.03	0.15	30
0.02	0.06	0.04	0.17	.38
0.03	0.06	0.03	0.15	22
0.03	0.06	0.03	0.15	17
0.03	0.06	0.03	0.15	15
0.02	0.06	0.03	0.13	20
0.03	0.07	0.03	0.15	31
0.04	0.07	0.03	0.14	41
0	0	12	0.30	42
0.02	0.06	0.03	0.15	70
0.03	0.06	0.04	0.14	61
0.03	0.06	0.04	0.17	50
0.03	0.07	0.04	0.14	57
0 03	0.07	0.04	0 20	42
0.03	0.07	0.04	0.20	48
0.05	0.07	0.04	0.20	56
0.05	0.07	0.04	0.20	55
0.05	0.07	0.04	0.21	47
0.05	0.07	0.04	0.19	58
0.05	0.07	0.04	0.20	65
0.06	0.08	0.04	0.19	62
0.07	0.09	0.04	0.18	65
-	-	0.05	0.20	74
-	-	0.04	0.17	78

heart rate per minute	respirations per minute	P amplitude mV	T amplitude mV
167	60	0.10	0.20
124	66	0.10	0.18
124	66	0.08	0.12
115	76	0.10	0.15
128	68	0.20	0.15
pig #9 - Na pe prio:	entobarbital prior t r to sample l. Infu	co sample l. Atro sion proceded sa	pine sulfate mples 2-9.
208	35	0.10	0.45
212	30	0.12	0.45
218	36	0.12	0.55
209	46	0.20	0.60
191	31	0.15	0.55
188	33	0.10	0.70
180	25	0.10	0.80
167	24	0.10	0.70
131	16	0	0.40
180	27	0.15	0.70
180	20	0.20	0.55
165	33	0.20	0.55
111	24	0.10	0.30
103	44	0.10	0.15
155	54	0.15	0.10
229	39	0.10	0.20

	brior co sampre	r. musion precede	\sim samples 2 iv.
144	30	0.12	0.12
137	42	0.10	0.12

P duration second	P-Q interval second	QRS duration second	Q-T interval second	T-wave angle degrees
0.05	0.09	0.04	0.21	68
0.05	0.08	0.04	0.23	65
0.05	0.08	0.04	0.24	65
0.05	0.08	0.03	0.26	70
0.05	0.07	0.03	0.24	68
0.03	0.08	0.04	0.20	72
0.03	0.07	0.04	0.20	66
0.03	0.07	0.04	0.20	68
0.03	0.07	0.04	0.20	66
0.05	0.07	0.04	0.23	69
0.06	0.08	0.04	0.23	64
0.07	0.09	0.04	0.25	69
0.04	0.10	0.09	0.24	67
-	_	0.07	0.29	52
0.06	0.07	0.04	0.24	68
0.04	0.08	0.04	0.25	70
0.08	0.08	0.04	0.28	70
. 0.08	0.10	0.04	0.32	65
0.08	0.11	0.04	0.34	55
0.05	0.09	0.04	0.20	75
0.03	0.07	0.04	0.17	63
• • •				20
0.04	0.07	0.04	0.24	28
0.05	0.07	0.04	0.22	45

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heart rate per minute	respirations per minute	P amplitude mV	T amplitude mV
120	21	0.10	0.40
125	22	0.08	0.20
132	18	0.10	0.20
136	15	0	0.20
138	18	0	0.25
145	17	0	0.35
120	18	0	0.20
138	18	0.03	0.22
133	21	0.08	0.15
133	18	0.08	0.20
138	20	0.05	0.20
pig #11 - Na p:	a pentobarbital prior receded samples 3-9.	to sample 2. I	nfusion
154	50	0.15	0.35
190	56	0.10	0.30
190	52	0.10	0.30
195	50	0.10	0.25
151	50	0.10	0.50
151	52	0.10	0.52
157	52	0.10	0.60
149	48	0.10	0.60
137	34	0.05	0.70
157	48	0	0.60
151	50	0	0.55
162	52	0	0.52
116	52	0	0.35
131	52	0	0.50

P duration	P-Q interval	QRS duration	Q-T interval	T-wave angle
second	second	second	second	degrees
0.04	0.07	0.04	0.22	46
0.04	0.07	0.04	0.23	59
0.04	0.07	0.04	0.21	52
0.03	0.06	0.04	0.20	58
-	-	0.04	0.20	52
-	-	0.04	0.20	55
-	-	0.04	0.19	65
-	-	0.05	0.20	56
0.02	0.05	0.04	0.19	60
0.03	0.06	0.04	0.19	45
0.04	0.06	0.04	0.19	49
0.03	0.07	0.04	0.20	47
0.03	0.07	0.04	0.23	64
0.04	0.07	0.04	0.20	58
0.04	0.07	0.04	0.20	60
0.04	0.07	0.04	0.19	55
0.03	0.07	0.04	0.22	71
0.04	. 0.07	0.04	0.20	74
0.04	0.07	0.04	0.20	78
0.04	0.07	0.04	0.20	68
0.05	0.07	0.04	0.20	75
-	_	0.04	0.19	78
-	-	0.04	0.20	73
-	-	0.04	0.19	72
-	-	0.05	0.22	71
_	-	0.04	0.20	71

heart rate per minute	respirations per minute	P amplitude mV	T amplitude mV
136	52	0.05	0.40
111	64	0.05	0.40
pig #12 - Na pre	pentobarbital prior ceded samples 2-9.	to sample 1.	Infusion
120	25	0.15	15
130	26	0.20	07
133	20	0.10	0.20
119	20	0.10	0.30
109	16	0.20	0.30
108	18	0.05	0.20
109	20	0	0.20
20	30	0	0.10
144	30	05	0.22
120	34	0.20	0.20
pig #13 - Na j pre	pentobarbital prior ceded samples 3-7.	to sample 2.	Infusion
160	60	0.10	0.20
185	24	0.30	0.25
190	24	0.20	0.30
162	20	0.20	0.20
152	20	0.15	0.35
150	20	0.15	0.40
130	10	0	20
144	16	0.15	0.52

P duration second	P-Q interval second	QRS duration second	Q-T interval second	T-wave angle degrees
0.04	0.07	0.04	0.21	74
0.04	0.07	0.04	0.23	73
0 04	0.09	0.04	0.26	58
0.04	0.09	0.04	0.25	62
0.05	0.09	0.04	0.24	57
0.05	0.08	0.04	0.26	45
0.05	0.09	0.04	0.26	55
0.05	0.08	0.04	0.27	59
0.03	0.09	0.04	0.27	66
-	-	0.05	0.27	68
-	-	0.17	0.37	80
0.02	0.08	0.04	0.28	62
0.05	0.09	0.04	0.30	66
0.04	0.08	0.04	0.23	51
0.04	0.07	0.04	0.22	46
0.03	0.06	0.04	0.22	66
0.04	0.07	0.04	0.22	51
0.04	0.07	0.04	0.23	65
0.04	0.06	0.04	0.23	70
-	-	0.08	0.27	72
0.04	0.06	0.04	0.23	72

heart rate per minute	respirations per minute	P amplitude mV	T amplitude mV
pig #14 - Na pr	pentobarbital prior fior to sample 3. Inf	to sample 1. Atro	opine sulfate amples 2-8.
164	38	0.10	0.40
171	34	0.10	0.45
209	24	0.30	0.40
212	26	0.20	0.40
200	36	0.15	0.40
206	32	0.20	0.40
198	35	0.15	0.45
195	35	0.12	0.50
212	34	0	0.40
211	46	0.10	0.55
200	46	0.20	0.50
pig #15 - Na pr	pentobarbital prior tior to sample 2. Inf	to sample 2. Atro	opine sulfate amples 3-7.
145	31	0.10	0.22
195	31	0.12	0.45
200	34	0.10	0.50
200	34	0.10	0.35
206	37	0.10	0.50
267	40	0	0.60
141	38	0	0.60
83	. 44	0	0.60
190	46	0.05	0.60
pig #16 - Na pr	pentobarbital prior fior to sample 2. Inf	to sample 2. Atro	opine sulfate amples 3-9.
178	48	0.20	0.40
178	37	0.25	0.30

duration second	P-Q interval second	QRS duration second	Q-T interval second	T-wave angle degrees
0.05	0.07	0.04	0.22	59
0.05	0.07	0.04	0.21	66
0.05	0.08	0.04	0.18	60
0.04	0.08	0.04	0.18	70
0.05	0.08	0.04	0.19	68
0.05	0.08	0.04	0.19	69
0.05	0.08	0.04	0.19	68
0.07	0.08	0.04	0.19	72
-	-	0.05	0.19	72
0.07	0.08	0.04	0.18	73
0.07	0.08	0.04	0.18	70
0.04	0.09	0.04	0.19	38
0.04	0.08	0.04	0.18	53
0.04	0.08	0.04	0.18	66
0.04	0.08	0.04	0.18	57
0.04	0.08	0.04	0.18	59
-	-	0.03	0.19	45
-	-	0.04	0.21	68
-	-	0.04	0.19	74
0.04	0.08	0.04	0.17	68
0.03	0.07	0.04	0.19	75
0.03	0.07	0.04	0.21	55

heart rate respirations per per ampl minute minute m	P T itude amplitude
	V mV
167 36 0.	20 0.35
167 40 0.	20 0.35
162 33 0.	20 0.40
138 34 0.	20 0.40
133 34 0.	15 0.50
138 33	02 0.50
141 38	0 0.50
150 22 0.	05 0.45

P duration second	P-Q interval second	QRS duration second	Q-T interval second	T-wave angle degrees
0.03	0.07	0.04	0.21	62
0.03	0.07	0.04	0.20	62
0.03	0.07	0.04	0.21	65
0.03	0.07	0.04	0.23	64
0.04	0.07	0.04	0.24	71
0.02	0.09	0.05	0.23	72
-	-	0.05	0.23	72
0.07	0.08	0.05	0.21	77

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pig #	ŧ pento	no Na obarbital	initial sample after Na pentobarbital	increase in the T-wave amplitude	đ	S-T epression
1	no	sample	3.9	8.8	no	depression
2	no	sample	3.9	no increase	no	depression
3	no	sample	4.3	8.2	no	depression
4	no	sample	3.8	no increase		7.0
5	no	sample	3.9	no increase	no	depression
6	no	sample	3.7	9.8	no	depression
7	no	sample	4.0	10.5	no	depression
8	no	sample	4.0	7.5		6.6
9	no	sample	3.8	7.7	no	depression
10	no	sample	4.0	6.2		8.2
11		3.7	3.7	5.4	no	depression
12	no	sample	3.6	6.4		5.5
13		3.5	3.5	7.3		9.9
14	no	sample	3.6	7.3		9.0
15		4.0	4.0	6.2		7.2
16		3.3	3.5	7.9	no	depression
mean		- 3.6	3.8	7.6		7.6
±sd		0.3	0.2	1.5		1.5
#		4	16	13		7

Swine plasma K levels (mEq/L) at which certain electrocardiographic (Lead II) changes appear

Table 12.

no P wave	increase in the T-wave angle	heart block	return of the P wave	
8.8	8.8	9.0	7.3	
8.0	7.8	11.2	-	
8.4	6.1	8.4	7.7	
8.3	6.1	8.3	-	
didn't disappear	no increase	8.8	didn't disappear	
9.8	9.8	9.8	9.1	
10.5	. 8.1	10.5	7.5	
8.7	6.6	8.7	7.7	
9.3	no increase	9.3	8.5	
7.6	6.2	8.2	7.1	
8.5	5.4	8.0	7.3	
8.2	7.9	9.9	6.4	
9.9	7.3	9.9	7.6	
9.0	5.3	9.0	7.7	
7.2	4.9	8.3	6.5	
9.2	7.9	9.2	8.5	
8.8	7.0	′ 9.2	7.6	
0.9	1.4	0.9	0.8	
15	14	16	13	

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	DELWEE	en porcions for	sambiru	y,		
PCV [*]	PCV %	RBC's 10 ⁶ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %
pig #16 -	Na pent precede	cobarbital given ed samples 2-10.	prior	to sample	l. Infu	usion
22.5	22.0	3.5	6.5	63	19	30
21.9	21.4	3.5	6.5	61	19	30
21.8	21.4	3.5	6.5	61	18	29
21.8	21.3	3.4	6.3	63	19	30
22.3	21.8	3.5	6.3	62	19	30
22.4	21.8	3.5	6.5	62	19	30
22.5	21.9	3.5	6.5	63	19	30
23.2	22.6	3.6	6.8	63	19	30
23.1	22.4	3.5	6.5	64	19	29
22.6	21.9	3.5	6.4	63	18	29

Table 13. Hematologic data of a pig infused intravenously with an isotonic NaCl solution (Infused in 100 ml portions at 7.64 ml/minute. Five minutes allowed between portions for sampling)

* includes leukocytes.

Table 14. Blood electrolyte data of a pig infused intravenous: with an isotonic NaCl solution (Infused in 100 ml portions at 7.64 ml/minute. Five minutes allowed between portions for sampling)									
plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%			
pig #16	- Na p ent o preceded	barbital (samples	given pric 2-10.	or to sam	ple l. I	nfusion			
140	4.0	26	101	10.8	7.0	74			
140	3.9	24	102	10.5	6.7	75			
141	3.8	24	102	10.4	6.7	70			
141	3.9	24	103	10.3	6.3	71			
141	3.8	24	103	10.8	6.5	60			
140	3.7	24	104	10.7	6.3	62			
140	3.8	24	103	10.7	6.3	66			
140	4.1	24	104	10.6	6.3	76			
138	4.0	23	104	10.4	6.4	74			
137	4.1	23	105	10.4	6.4	80			

^{*}1 part blood and 19 parts H_2^0 .

**1 part blood and 1 part H_2O .
diluted [*] whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L
			L		
5.3	1.5	43	-9	118	35
5.4	1.5	43	-9	121	29
5.5	1.5	44	0	121	33
5.5	1.5q	43	0	122	20
5.5	1.5	43	0	128	26
5.4	1.5	44	0	128	32
5.4	1.5	43	0	128	28
5.4	1.6	43	0	124	27
5.4	1.6	43	9	125	22
5.4	1.5	43	9	119	22

heart rate per minute	respirations per minute	P amplitude mV	T amplitude mV
pig #16 - Na p prec	entobarbital give ceded samples 2-10	n prior to sample 1.	Infusion
170	30	0.10	0.35
178	32	0.05	0.35
176	32	0.05	0.30
164	35	0.10	0.20
171	32	0.05	0.20
164	26	0.05	0.20
164	24	0.10	0.30
164	26	0.10	0.20
171	26	0.10	0.20
167	28	0.10	0.20

Table 15. Electrocardiographic data (Lead II) of a pig infused intravenously with an isotonic NaCl solution (Infused in 100 ml portions at 7.64 ml/minute. Five minutes allowed between portions for recording)

P duration second	P-Q interval second	QRS duration second	Q-T interval second	T-wave angle degrees
0.04	0.08	0.04	0.22	65
0.06	0.08	0.04	0.22	60
0.07	0.09	0.04	0.22	55
0.05	0.09	0.04	0.22	42
0.06	0.09	0.04	0.22	53
0.06	0.09	0.04	0.23	50
0.07	0.08	0.04	0.22	56
0.05	0.08	0.04	0.22	52
0.05	0.09	0.04	0.21	53
0.06	0.08	0.04	0.20	56

	arrowe	a permeen porci		sampring		
PCV*	PCV %	RBC's 10 ⁵ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %
pig #17 -	Na pento precede	obarbital giver d samples 2-12	n prior	to sample	l. Infu	ision
33.7	33.1	5.4	10.7	62	20	32
32.3	31.5	5.2	11.4	61	22	36
33.8	33.2	5.3	10.7	63	20	32
32.3	31.6	5.2	10.4	61	20	33
32.8	32.2	5.2	10.4	62	20	32
32.4	31.7	5.3	10.7	60	20	34
32.0	31.4	5.3	10.1	59	19	32
32.3	31.8	5.3	10.1	60	19	32
32.6	32.0	5.2	10.4	62	20	33
37.4	36.8	6.0	11.4	61	19	31
37.4	37.0	5.9	11.3	63	19	31

Table 16. Hematologic data of a pig infused intravenously with an isotonic solution of glucose (Infused in 50 ml portions at 3.82 ml/minute. Five minutes allowed between portions for sampling)

*includes leukocytes.

Table 17. Blood electrolyte data of a pig infused intravenously with an isotonic solution of glucose (Infused in 50 m1 portions at 3.82 ml/minute. Five minutes allowed between portions for sampling)

plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%
pig #17 -	- Na pento preceded	barbital samples	given pric 2-12.	or to samp	ple l. 1	Infusion
142	4.2	24	101	10.2	9.6	118
142	4.1	24	101	10.1	9.5	206
140	4.0	24	100	10.7	9.5	242
139	4.0	24	99	10.0	9.4	282
138	4.0	23	98	10.0	9.3	312
137	3.8	23	95	10.0	9.4	342
136	3.8	23	94	10.4	9.5	362
135	3.8	23	95	10.1	9.4	388
135	4.0	23	94	10.1	9.4	386
135	4.2	22	94	10.3	10.0	390
136	3.8	21	94	9.8	10.4	394
135	4.0	22	92	9.8	10.7	406

^{*}l part blood and 19 parts H_2O .

**1 part blood and 1 part H_2O .

diluted [*] whole blood Na mEq/L	diluted [*] whole blood K mEq/L	diluted ^{**} whole blood Cl mEq/L	RBC Na mEq/L	RBC K mEq/L	RBC Cl mEq/L
4.7	2.1	39	0	121	39
4.6	2.0	38	0	121	24
4.5	2.0	38	0	114	29
4.6	2.0	38	0	120	28
4.6	2.0	38	0	118	27
4.5	2.0	38	0	120	33
4.6	2.0	38	0	121	34
4.6	2.0	38	0	119	33
4.5	2.0	37	0	119	29
4.1	2.3	36	0	115	35
4.3	2.3	36	0	120	35
4.1	2.3	36	0	119	39

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Table 18. Electrocardiographic data (Lead II) of a pig infused intravenously with an isotonic solution of glucose (Infused in 50 ml portions at 3.82 ml/minute. Five minutes allowed between portions for recording)

heart rate per minute	e respirations per minute	P amplitude mV	T amplitude mV
pig #17 -	Na pentobarbital given preceded samples 2-12.	prior to sample 1.	Infusion
200	18	0.20	0.45
171	14	0.12	0.70
158	11	0.12	0.80
170	14	0.12	0.52
150	10	0.15	0.60
153	10	0.15	0.60
147	12	0.15	0.70
145	20	0.15	0.65
171	20	0.15	0.45
188	14	0.20	0.45
188	10	0.15	0.50
185	12	0.15	0.55

P duration second	P-Q interval second	QRS duration second	Q-T interval second	T-wave angle degrees
0.04	0.05	0.04	0.20	68
0.05	0.07	0.04	0.24	63
0.05	0.09	0.05	0.26	67
0.05	0.08	0.04	0.24	65
0.06	0.08	0.04	0.26	65
0.06	0.08	0.04	0.28	62
0.06	0.09	0.04	0.27	65
0.07	0.08	0.05	0.28	65
0.04	0.07	0.04	0.25	60
0.04	0.07	0.04	0.23	71
0.04	0.07	0.04	0.23	65
0.04	0.07	0.04	0.24	71

		,				
PCV*	PCV १	RBC's 10 ⁶ /cmm	Hb gm%	MCV u ³	MCH uug	MCHC %
pig #12 -	Na pento sulfate	obarbital giver given prior to	n prior sample	to sample 8.	l. Atro	opine
32.0	31.2	5.0	9.8	62	20	31
32.2	31.6	5.0	9.8	63	20	31
32.6	31.7	5.0	9.8	63	20	31
33.0	32.4	5.1	9.8	64	19	30
33.5	32.8	5.2	10.1	63	19	31
34.8	34.0	5.3	10.4	66	20	30
34.7	33.9	5.4	10.4	64	19	30
33.5	33.0	5.4	10.4	61	19	32
33.2	32.5	5.2	10.1	63	19	31
33.5	32.7	5.2	9.8	63	19	30
33.9	33.0	5.2	9.8	64	19	30
34.3	33.5	5.1	9.8	64	19	29

Table 19. Effect of time and sampling on the hematologic data of a pig (blood samples (15 ml) drawn at 20 minute intervals)

*includes leukocytes.

Table 20	Fable 20. Effect of time and sampling on the blood electrolytedata of a pig (blood samples (15 ml) drawn at 20minute intervals)						
plasma Na mEq/L	plasma K mEq/L	plasma CO ₂ mEq/L	plasma Cl mEq/L	plasma Ca mg%	plasma P mg%	plasma glucose mg%	
pig #12	- Na pento sulfate	barbital g given pric	given pric or to samp	or to samp ple 8.	ole l. A	tropine	
141	3.7	21	101	11.3	7.5	73	
140	3.7	22	100	11.4	7.6	76	
141	3.8	22	101	11.4	8.3	70	
141	3.9	23	101	11.4	8.7	70	
140	3.8	24	100	11.2	9.3	68	
140	3.7	22	100	10.7	9.1	81	
141	3.9	23	99	10.6	9.6	59	
140	4.0	23	99	10.4	9.4	54	
141	4.3	22	99	10.3	9.5	49	
140	4.3	23	99	9.9	9.3	53	
140	4.3	24	99	9.8	9.5	52	
140	4.3	23	99	9.5	9.5	53	

^{*}l part blood and 19 parts H_2^0 .

** 1 part blood and 1 part H_2^0 .

diluted [*] whole blood Na mEg/L	diluted [*] whole blood K mEg/L	diluted ^{**} whole blood Cl mEg/L	RBC Na mEg/L	RBC K mEg/L	RBC C1 mEg/L
4 8	2 0	40	0	192	36
4.9	2.1	40 20	12	127	30
4.9	2.1	39		126	31
4.9	2.1	39	12	123	32
5.0	2.2	38	28	128	28
4.7	2.3	38	6	129	27
4.8	2.2	38	12	124	28
4.7	2.2	38	0	127	28
4.7	2.1	37	0	123	24
4.9	2.2	37	12	128	25
4.9	2.2	37	18	127	25
4.8	2.2	38	12	125	29

heart rate per minute	respirations per minute	P amplitude mV	T amplitude mV
pig #12 - Na p sul:	pentobarbital given fate given prior to	prior to sample 1. sample 8.	Atropine
240	46	0.15	0.30
254	38	0.15	0.35
245	29	0.07	0.30
248	30	0.10	0.30
263	28	0.05	0.25
232	16	0.05	0.25
216	36	0.15	0.30
222	34	0.20	0.35
174	36	0.20	0.25
185	30	0.20	0.20
178	38	0.20	0.15
169	25	0.15	0.15

Table 21. Effect of time and sampling on the electrocardiographic data (Lead II) of a pig (blood samples (15 ml) drawn at 20 minute intervals)

P duration second	P-Q interval second	QRS duration second	Q-T interval second	T-wave angle degrees
0.03	0.07	0.04	0.17	65
0.03	0.07	0.04	0.17	64
0.03	0.07	0.04	0.17	56
0.03	0.07	0.04	0.17	61
0.03	0.07	0.04	0.16	62
0.03	0.07	0.04	0.18	61
0.04	0.07	0.04	0.19	67
0.04	0.07	0.04	0.19	66
0.05	0.08	0.04	0.24	58
0.04	0.07	0.04	0.24	57
0.04	0.08	0.04	0.24	65
0.05	0.08	0.04	0.24	55

and the second			
data from Table 3	Coldman and Good (1967)	Ullrey <u>et al</u> . (1967) (2 months old)	
plasma	plasma	plasma	
<u>Na mEq/L</u> 145±2 (60)*	133±11 (7)	150±1 (30)	
<u>K mEq/L</u> 5.4±0.5 (60)	7.7±2.7 (7)	5.5±0.2 (30)	
<u>Cl mEq/L</u> 100±2 (43)	- -	-	
$\frac{\text{Co}_2 \text{ mEq/L}}{22\pm3 (43)}$	-	_	
<u>Ca mg/100 ml</u> 10.8±1.1 (60)	-	11.8±0.2 (30)	
<u>P mg/100 ml</u> 9.8±1.0 (60)	-	8.1±0.2 (30)	
<u>glucose mg/100 ml</u> 109±10 (60)	95±17 (7)	-	

Table 22. Plasma electrolytes (mean and standard deviations) from a group of representative pigs 9-10 weeks of age compared to values in the literature

* number of pigs in parenthesis.

Widdowson and McCance (1956) (adult pigs)	Cummings and Kaiser (1959) (sows)	Birkeland (1968) a&b (4-5 months old)
serum	plasma	plasma
144±5 (13)	145 (6)	132 (16)
6.0±0.6 (13)	4.3 (6)	3.5 (16)
106 [±] 4 (13)	101 (6)	-
-	27 (6)	-
-	-	10.9 (16)
-	-	-
-	-	121 (18)

APPENDIX B. FIGURES

Figure 1. The erythrocyte K concentration of 8 sows and their fetal pigs selected at different stages of gestation. One of the 8 sows was selected at 67, one at 77, one at 86, one at 91, one at 109, one at 110, and one at 111 days of gestation



Figure 2. The plasma Na concentration of 8 sows and their fetal pigs selected at different stages of gestation. One of the 8 sows was selected at 67, one at 77, one at 86, one at 87, one at 91, one at 109, one at 110, and one at 111 days of gestation.

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Figure 3. The plasma K concentration of 8 sows and their fetal pigs selected at different stages of gestation. One of the 8 sows was selected at 67, one at 77, one at 86, one at 87, one at 91, one at 109, one at 110, and one at 111 days of gestation



Figure 4. The plasma Cl concentration of 8 sows and their fetal pigs selected at different stages of gestation. One of the 8 sows was selected at 67, one at 77, one at 86, one at 87, one at 91, one at 109, one at 110, and one at 111 days of gestation

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Figure 5. The mean corpuscular volume of 8 sows and their fetal pigs selected at different stages of gestation. One of the 8 sows was selected at 67, one at 77, one at 86, one at 87, one at 91, one at 109, one at 110, and one at 111 days of gestation



Figure 6. Nonrespiratory sinus arrhythmia (shown twice) and the effect of respiration on lead I of an unanesthetized pig

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Figure 7. Nonrespiratory sinus arrhythmia (shown once) of an unanesthetized pig





Figure 8. Electrocardiogram of an unanesthetized pig





Figure 9. Change in the configuration of the T wave in lead II due to excitement (Fifteen minutes prior to this electrocardiogram the T wave was monophasic (+) in lead II)

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Figure 10. Electrocardiogram of an anesthetized pig

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Na pentobarbital
Figure 11. The effect of a rising plasma K concentration on the electrocardiogram of an anesthetized pig (sodium pentobarbital). Pig number 12 in Table 12





Eq/L plasma K 5.5 mEq/L

plasma K 5.9 mEq/L plasma K 6.4 mEq/L plasma K 7.2 mEq/L





Figure 12. The effect of a rising plasma K concentration on the electrocardiogram of an anesthetized pig. Pig number 16 in Table 12







Figure 13. The effect of a rising plasma K concentration on a negative QRS complex in the electrocardiogram of an anesthetized pig (sodium pentobarbital). Pig number 9 in Table 12



plasma K 8.2 mEq/L

plasma K 3.8 mEq/L

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plasma K 8.5 mEq/L

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Figure 14. The effect of a rising plasma K concentration on the T-wave angle of lead II in the electrocardiogram of an anesthetized pig

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Figure 14. Continued

Figure 15. The effect of a rising plasma K concentration on the electrocardiogram of an anesthetized pig. Pig number 15 in Table 12

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Figure 15. Continued

Figure 16. The effect of a rising plasma K concentration on the electrocardiogram of an anesthetized pig. Pig number 13 in Table 12

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Figure 16. Continued

Figure 17. Heart block in the electrocardiogram of an anesthetized pig (sodium pentobarbital) as the result of an elevated plasma K concentration





LEAD I

Figure 18. Peaked T waves in lead II with a normal plasma K concentration followed by a slight increase in T-wave amplitude at a higher plasma K concentration in an anesthetized pig (sodium pentobarbital)







plasma K 8.4 mEq/L

Figure 19. Depressed S-T segment (leads II and III) as a result of an elevated plasma K concentration and the effect of additional sodium pentobarbital on the heart rate



injection of additional Na pentobarbital Figure 20. The effect of infusing intravenously 850 ml of isotonic NaCl solution over a 3 hour period on the electrocardiogram of an anesthetized pig (sodium pentobarbital). A is prior to infusion and B is after infusion. Note Table 15





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plasma K 4.0 mEq/L plasma Na 140 mEq/L plasma Ca 5.4 mEq/L



plasma K 4.1 mEq/L plasma Na 137 mEq/L plasma Ca 5.0 mEq/L

Figure 21. The effect of infusing intravenously 500 ml of isotonic glucose solution over a 3 hour period on the electrocardiogram of an anesthetized pig. Note Table 18



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Figure 22. The effect of the initial injection of sodium pentobarbital on heart rate and the electrocardiogram and the subsequent effect on the electrocardiogram of the pig after 3 1/2 hours of drawing blood samples (no infusion). Note Table 21

