AGONAL PHYSIOLOGY AND BLOOD GAS TENSIONS
WITH APPLICATION TO THE SUDDEN INFANT DEATH SYNDROME

A thesis submitted in partial satisfaction of the requirements for the degree of
Master of Science in Health Science

by

James O. Aldrich

January, 1972
"Beautiful is what can be seen, more beautiful what can be understood, by far the most beautiful is that which we don't know."

____________  Neils Stensen
1638-1686
The thesis of James O. Aldrich is approved:

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ABSTRACT

AGONAL PHYSIOLOGY AND BLOOD GAS TENSIONS
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The partial pressures of oxygen (pO₂), carbon
dioxide (pCO₂), and pH were determined in the blood of
young adult mice sacrificed either by cardiac arrest, or
by asphyxiation. Cardiac arrest was produced by electric
shock, and asphyxiation by sudden occlusion of the
trachea. A single blood sample, drawn from the left
ventricle three minutes after death was analyzed. Death
by asphyxiation resulted in a significantly lower pO₂ than
death due to cardiac arrest. The type of death was not
associated with pCO₂ or pH. Possible application of these
procedures to the study of sudden infant deaths (SID
Syndrome) in humans was discussed.
CHAPTER I
INTRODUCTION

A reliable method for determining whether the heart or lungs fail first during an agonal episode should be applicable in determining cause of death in sudden infant deaths (SID Syndrome) (8). Lung failure with a continued heart beat should result in a smaller amount of oxygen in the blood stream. Mitoefer found that respiratory obstruction in dogs resulted in lower blood oxygen when compared with cardiac arrested dogs (28:657). In the case of dogs, blood circulation continued for some seven to nine minutes following airway occlusion (28:655). An hypoxic condition exists in both modes of death because of failure of the "respiratory cycle" where blood loses carbon dioxide (CO₂) and takes up oxygen (O₂) in the lungs and loses O₂ and takes up CO₂ in the tissues (27:286). Mitoefer's studies indicate that occlusion of the airway does not cause circulation to cease immediately (28:655). With this continued circulation and the hypoxic cell condition it is possible that more blood O₂ is removed by the cells in respiratory obstruction. Conversely, in cardiac arrest oxyhemoglobin does not reach the hypoxic cells, resulting in a higher pO₂.
Suwa et al., have shown that after three minutes of circulatory arrest, dogs have a higher pCO₂ than control animals (36:40). In asphyxiated mice it was found that pCO₂ increased sharply when the respirator was shut off for three minutes (24:436). The removal of bicarbonate (HCO₃⁻), including carbamino-bound CO₂, and hydrated carbonic acid (H₂CO₃) from the blood occurs in the lungs where it is converted to gaseous CO₂ (17:54). In both types of death, cardiac arrest and occlusion of the airway, CO₂ cannot be eliminated via the lungs, therefore it seems reasonable that differences would not exist except for some CO₂ liberated by the tissues.

Another important consideration in studies attempting to diagnose cause of death through the use of blood gas analysis is the effect of time on the pO₂ and pCO₂ of the blood sample. Earlier work indicated that the distinction between modes of death could be made up to six hours following the agonal episode (28:657). This distinction was in part a result of the relationship between time and blood temperature. The internal organs have a temperature drop of approximately 0.5°C for six hours following the agonal episode (28:657). This relatively small temperature decrease does not greatly effect the pO₂. Astrup, et al., have shown that a blood sample of known O₂ saturation (10%) at 38°C yields a pO₂ of 11.0 mmHg (41). A sample at 30°C indicates a
pO₂ of 6.0 mmHg (4). This small difference of 4 mmHg for a 8°C temperature difference indicates that a six hour period following death (temperature decrease of approximately 3°C) would not appreciably affect the pO₂ readings. Even at higher pO₂ values of 30 mmHg the difference for an 8°C change was 7.3 mmHg. Therefore the variation of sampling time in this present work of 0 to 3 minutes would not affect the pO₂ values with regards to temperature decrease.

Following death, whether by cardiac or respiratory obstruction, there is a lowering of pH (24:445) (25:179). Therefore, pH is not a valid indicator as to type of death.

Statement of the Problem

The purpose of this study is to extend Mitheofer's work by measuring pO₂ values post mortem. It is hypothesized that mice sacrificed via cardiac arrest or respiratory obstruction will have statistically significant differences in blood pO₂.

Extent of This Study

Different methods of determining pO₂, pCO₂ and pH of a blood sample will be reported in the review of the literature section of this paper. This study utilized the glass electrode technique for pH measurement. The determinations of the pO₂ and pCO₂ values in blood (in Swiss strain mice) were based on the principle of
selective absorption of gases.

**Definition of Terms**

**Acid:** Hydrogen ion donor. Example: carbonic acid may give off a hydrogen ion forming a bicarbonate ion.

**Base:** Hydrogen ion acceptor. Example: bicarbonate ions may accept a hydrogen ion forming carbonic acid.

**Actual pH:** Refers to the pH of anaerobically drawn blood.

**Actual pCO₂ mmHg:** The partial pressure of carbon dioxide in anaerobically drawn blood. The sum of the carbonic acid concentration and the concentration of dissolved carbon dioxide is derived as 0.03 x pCO₂.

**Actual Bicarbonate Concentration:** Bicarbonate (HCO₃⁻) concentration in plasma at the pCO₂ actually present. Expressed in mMol/L.

**Total CO₂ of Plasma:** The CO₂ derived from carbonic acid and bicarbonate in plasma from anaerobically drawn blood.

**CO₂ - Combining Power of Plasma:** The total CO₂ of plasma, separated at the actual pCO₂ from the cell and equilibrated with CO₂ at pCO₂ of 40 mmHg.
Standard Bicarbonate: The bicarbonate concentration in the plasma of blood equilibrated at a pCO₂ of 40 mmHg and with oxygen for full saturation of the hemoglobin.

Buffer Base (BB): The sum of buffer anions, i.e., mainly bicarbonate and proteinate ions. Whole blood BB refers to fully oxygenated blood and gives the value in meq/L blood. Plasma BB gives the value in meq/L plasma. BB without specification refers to whole blood.

Base Excess (BE): It is defined as zero for blood with pH of 7.40 at pCO₂ of 40 mmHg. Positive values indicate an excess of base (or deficit of fixed acid); negative values indicate a deficit of base (or excess of fixed acid). Whole blood BE gives the value in meq/L blood referring to fully oxygenated blood. Plasma BE gives the value in meq/L plasma. Without specification BE refers to whole blood.

Meq/L: A system of equivalents or milliequivalents (meq), which expresses concentrations in terms of the number of (+) and (-) chargers per unit volume.
CHAPTER II

REVIEW OF THE LITERATURE

SID Syndrome - Epidemiologic

The ability to determine cause of death is often a matter of observation. However, in the case of sudden and unexplained infant deaths (SID Syndrome), cause of death has so far eluded investigators. Since the results of the experiment described in this thesis are directly applicable to SID Syndrome victims a review of this literature will be given (8). Basically there are two categories of studies that are examined: 1) Those dealing with contributions of epidemiology in studying the SID Syndrome and 2) physiologic or pathologic findings in the study of the SID Syndrome.

A review of the literature on the SID Syndrome invariably reveals the excellent article by Valdes-Dapena (38:123). This particular paper surveys the literature from 1954 to 1966. It was written to educate practitioners in pediatrics and pathology, as well as researchers in this area, as to what research has taken place throughout the world from 1954 to 1966. In dealing with the epidemiologic factors it is pointed out that there is a serious problem in getting accurate death rates since
pathologists do not enter "cause unknown" on a death certificate. It is stated that Landing has set the figure to be 25,000 to 30,000 per year in the United States (44:1). Age is also an important epidemiologic factor with a peak incidence of between two and four months (39:630) (20:291) (45:291) 22:134). The consensus of reviewed articles indicates a preponderance of male deaths in the SID Syndrome (12:31). Race was found to be an additional factor with a disproportionate number of victims being Negro (19:53). Other factors reported in this review article deal with season (higher incidence during cold months), geographic location (generally accepted that it is most prevalent in urban areas), time factor (great majority die between midnight and 6 A.M. (21:592)). Recurrences within the family was also found to be mentioned in six reports, but the number of recurrences were very small (1:633) (32:914).

Cooke points out that cause of death in most cases remains obscure although many reasons are often put forth (15:1550). In this study 122 cases of sudden infant death were studied for epidemiologic and pathologic patterns in the syndrome. Those factors having to do with the epidemiology of the syndrome will be discussed here. The incidence rate reported in this study was nine sudden and unexpected deaths per 100,000 population. A definite seasonal variation was also shown in this study.
with a higher prevalence in colder months. Other factors such as social grade of parents, birth weight of the infant and size of family were looked at and found to compare with other studies (38:125-27) (18:458). In their conclusions the authors state that, "no one theory fits all the facts, and the problem remains open." The only real difference in observations in this study was the striking feature of the number of children who were members of a twin pair, there were sixteen such children. No real explanation or theory for this was presented. It should be noted that even though the authors consider the problem open, they believe the most tenable theory is that the infants die from anoxia due to laryngospasm mediated through the autonomic nervous system.

Froggatt also mentions many factors identified by Valdez-Dapena (18:457-68). This study took place in Belfast, Northern Ireland (population 1.4 million) where 162 cases were observed from August 1, 1965 to July 31, 1967. The definition of sudden infant death used by these investigators is as follows: "The death of a child who was thought to be in good health or whose terminal illness appeared to be so mild that the possibility of a fatal outcome was not anticipated." They extended this to include the contingency that the autopsy lesions were not such as are generally conceded to show a "cause" for death.
This study examined the clinical profile of the infant and considered it an important factor. It was found that the infants were either symptom free when last alive or else had minor symptoms, usually in the upper respiratory tract. Death was found to be usually quiet in the sense that the mother was not disturbed—even when in the same room with the child. Froggatt pointed out that in Ireland the SID Syndrome is not a certifiable cause of death, therefore limiting the incidence rates available. In Froggatt's study an incidence of 2.3 deaths per 1,000 live births was found which compared favorable with American rates (1:664) (31:478) (10:5). It was pointed out that there is a real immunity enjoyed in the first months of life and a sharp decline of incidence after four months. The usual seasonal pattern was verified. Cases of SID Syndrome were found to occur in colder months with the highest prevalence of deaths occurring between midnight and breakfast. The author warns however that this must be interpreted with care because the natural regimen of households bias the estimated time of death. Socio-economic level indicates that families of infants who died were at a disadvantage compared to families where no deaths occurred. Maternal factors dealing with pregnancy (obstetric history no different for mothers of dead infants and mothers of control) and illegitimacy (sudden death found to
be more common in illegitimate than legitimate children) were additional factors examined by Froggatt. Infant factors indicated that the infants dying were on the average less mature than the chosen controls. Heritable mechanisms were looked for and on the basis of collected data it was concluded that they play at most a small part in sudden unexpected death in infants. The authors further state that "epidemiology alone cannot explain how or why infants die. Knowledge from other disciplines is required" (18:468).

Valdes-Dapena also studied the relationship between the incidence of sudden unexpected death in infancy and poverty (40:387-94). Of the 337 deaths occurring during a three year period it was shown that such deaths occurred more frequently among Negro infants (215) than among Caucasian ones, relative either to the total number of live births or to the total number of deaths of infants seven days to one year of age. This difference was in large part dependent upon the very high incidence of sudden death during infancy in Negro infants of low socio-economic level.

Peterson studied the SID Syndrome in King County, Washington (31:478). Peterson reports that one sudden infant death could be expected for every three hundred babies born alive. The age distribution is typical, as were
the data on sex, race and season of death. He also observed that there is a regular progression of susceptibility which is inversely related to birth weight. It was also pointed out that the higher risk associated with illegitimacy must be interpreted with caution because other factors may contribute to the increased death rate among illegitimate children. The authors did not report hour of death because they felt that these data were frequently a "matter of conjecture." Peterson concludes that "deaths occur in certain high risk infants under diurnal influences and are triggered by mild, unrecognized infection with microbiologic agents not found in previous studies, or perhaps by some seasonally influenced factor."

The important and different data presented in this paper deal with the inverse relationship of birth weight and the regular progression of susceptibility although it was not discussed in detail.

J. J. O'Reilly did a study of "cot death" or "crib death" for the State Health Department in Queensland, Australia (30:1084-87). In the five year period from 1962 to 1966, autopsies were performed on eighty infants who died suddenly and unexpectedly in the Brisbane metropolitan area (population 700,000). It was stated that this number represents almost thirty percent of all infant deaths between the ages of four weeks and twelve months in that area. The age of infants in this study was reported to be three to five months and apparently not ill. However,
they were found dead following a period of sleep. These observations agree with other studies (1:633) (18:458).

After reviewing these preceding articles it would seem important that a three-tiered approach be used in assessing the worth of the epidemiological approach to the problem of the SID Syndrome.

(1) Are there differences in the incidence of the condition between different countries or communities and can the etiology of the syndrome be attributed to some factor?

In general the data reported in this report seem to indicate that the incidence of the SID Syndrome is fairly uniform which agrees with investigators who have reviewed other literature. These data seem to indicate a higher incidence in urban areas which is left unexplained. This could be an artifact resulting from better reporting of cases in urban areas. At present the etiology cannot be attributed to a single or number of factors.

(2) Having identified a factor or factors, we can establish the reality of cause and effect, and not just correlation, by seeing whether the disease can be prevented by changing the prevalence of the suspected agent.

This point in the classical epidemiological approach has not been reached in this problem and, therefore, the technical and professional problems cannot be considered at this time.

(3) Can we convincingly test formulated hypothesis which relate the occurrence of the condition to a personal characteristic of the victim in relation to the degree of exposure to an environmental agent?
However any relationship established may be a secondary one and the real cause or causes may be some unknown common factor. Quoting Dr. J. Berkson (11:99):

If an essential biologic association is to be established as a definitive scientific conclusion, that is to say, if it is to be considered "proved," the population must not be anything else than an experimental population. An association found is purely statistical investigation made on an existent population, by which I mean an investigation which is retrospective as regards either of the variables concerned, however strongly it may suggest association as a presumptive conclusion, is tentative until it is corroborated fully by means of experiment.

If however, we study the SID Syndrome in the human population, where experimentation and production of controlled populations are impossible, then Berkson's dictum must be relaxed.

The data resulting from the epidemiological studies suggest that it would be very difficult to pinpoint casual factors. It would seem that epidemiology has made its contribution, and further studies in this area are not indicated.

**SID Syndrome - Pathologic**

Dr. Beckwith states that it is essential that we make a distinction between sudden unexplained death and sudden unexpected death (9:44). Deaths which are definitely unexplained and show no a priori lesion upon pathologic investigation can be classified as the SID Syndrome. He believes that petechial hemorrhages are
the most distinctive feature of the syndrome. Other fairly constant pathologic characteristics are, pulmonary edema, fluid blood in the heart and empty urinary bladder. Beckwith states that "the mechanism of sudden death may be precipitated by a number of interrelated factors."
It is their belief that these factors terminate in laryngospasm via a final common pathway. This type of hypothesis would argue for death due to respiratory obstruction (lung failure). Some of the factors believed to cause the spasm include sleep, constitutional autonomic reactivity and upper respiratory infection. The authors stress the paucity of research in agonal physiology and ask why the pathogenesis of the SID Syndrome has not been explored. In conclusion it is stated that, "a prime goal of such research would be to develop an appropriate animal model."

Immunoglobulin levels in infants dying of SID Syndrome have been examined by Balduzzi (5:689-92). Balduzzi believes that a host factor rather than specific external cause may be responsible for death. The basis of this hypothesis evolves from the studies showing the peak incidence at ages two to four months. In certain children in this age group conditions could exist which might result in increased susceptibility to factors ultimately responsible for death. At two and three months of age the immune defenses are at a minimum as a consequence of
decreased amount of certain gamma globulins. Balduzzi therefore investigated the levels of the immunoglobulins in a group of infants who died suddenly and unexpectedly and in a control group of infants. The results from thirty-five infants indicated that there was no observable difference between the SID Syndrome victims and the control infants.

James explains the role of the heart in sudden infant death (23:479-504). One hundred and fifty hearts from infants of which sixty percent of their deaths were unexplained were studied. It was his contention that in any sudden death the mechanics of a lethal cardiac arrhythmias or conduction disturbance is of utmost importance. In his study the conduction systems of SID Syndrome victims were examined. It was found that there was a presence of focal resorptive degeneration in all the post natal hearts, both of "controls" and of sudden unexplained deaths, which suggests that it was an ubiquitous post natal process. James also felt that conduction disturbances were consequences of developmental histologic changes in the critical regions which may be the final common pathway in sudden infant deaths.

Morgan studied heart rates of infants (29:658). She indicated that it has been theorized that since the etiology of arrhythmias has not been documented it would be reasonable to suspect immaturity of the sympathetic
and parasympathetic cardiac regulation as causal factors in the initiation of such rhythms (2:179) (13:545). It was pointed out by Morgan that Stowens had suggested that a generalized neural spasm, mediated through the autonomic nervous system, may be responsible for the SID Syndrome (35:674). Morgan found marked sinus arrhythmia and sinus bradycardia in infants. This evidence would suggest that since these cardiac rhythms are present in normal infants then perhaps the role they have (if any) in SID Syndrome should be investigated. Once again, a method of determining if heart disfunction was cause of death would prove valuable in the initiation of such a study.

An attempt to isolate viral agents responsible for sudden unexpected death in apparently well infants was conducted by Valdes-Dapena (41:398). In this early study it was found that as far as present techniques could demonstrate viral infections did not play a role in the cause of sudden deaths in infants.

Studies by Ray some years later again failed to yield any significant data pertaining to viruses among the SID Syndrome cases (10:145). However, in additional studies using a different protocol, in which the SID Syndrome cases and controls were studied as soon as possible after death, non-polivivirus isolates were reported. In the SID group 37.5% showed the viruses and in the control group
only 16.2% had the isolate (10:155). It was Ray's conclusion that viruses may have a significant role in the pathogenesis of the SID Syndrome, and that some stereotypes may have relatively greater importance than others. Ray went on to say "until the pathogenesis of this syndrome is more clearly defined, the relative significance of any single factor must remain speculative."

A recent theory is that death is caused by severe imbalance of electrolytes (26:1440). McGaffey believes that this imbalance may result in acidosis leading to rapid death from cardiac arrest or anoxia. Acidosis may result from a metabolic deficiency and it was theorized that perhaps the adrenal glands may be hypoactive since they were found to be lighter than the "control" glands. It was also reported that cases showed evidence of pulmonary congestion and petechial hemorrhage. Since there is a normal acidosis of sleep, then subacute or chronic acidosis caused by chronic hypoadrenalism might cause sudden death. McGaffey states "a borderline case of acidosis aggravated by mild additional anoxia and carbon dioxide retention, such as with an acute respiratory infection or aspiration, plus the acidosis of sleep, might be sufficient to cause death."

Present data, both experimental and epidemiological, indicate that we are still ignorant with respect to the etiology of the SID Syndrome. Basic research is needed to
promote theories about what happens to the infant's vital functions during the agonal episode. The remainder of this report is concerned with basic research performed to establish whether the heart or lungs failed first in the SID Syndrome animal model.

**Gas Transport in Blood**

This study is limited to the analysis of three parameters of the blood, pH, pCO$_2$, and pO$_2$. Blood pH, pCO$_2$, and pO$_2$, indicate the acid-base status of the blood. A review of how O$_2$ and CO$_2$ are transported in the blood is now provided.

When O$_2$ diffuses into the blood in external respiration, most of it enters the red blood cells or erythrocytes and unites with the hemoglobin in these cells forming a compound called oxyhemoglobin. The complex protein hemoglobin contains iron and has a great affinity for O$_2$. As the blood passes through the alveolar capillaries, the hemoglobin becomes saturated with O$_2$. The dissolved oxygen and hemoglobin react to form the oxyhemoglobin given by equation 1 (16:9).

\[ \text{O}_2 + \text{Hb} \rightarrow \text{HbO}_2 \]

Oxyhemoglobin is a very unstable compound and when the blood reaches the capillaries in the tissues throughout the body where pO$_2$ is low, the compound breaks down into hemoglobin and the O$_2$ diffuses into the cells. The
reaction that occurs in the tissue capillaries is provided in equation 2.

\[(2) \quad \text{HbO}_2 \rightleftharpoons \text{Hb} + \text{O}_2\]

When CO\textsubscript{2} enters the blood from the tissues, it first combines with water forming carbonic acid, H\textsubscript{2}CO\textsubscript{3} according to equation 3 (17:53).

\[(3) \quad \text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3\]

Sodium and Potassium ions then react with H\textsubscript{2}CO\textsubscript{3} to form bicarbonates. Most of the CO\textsubscript{2} is transported in the plasma in the form of sodium bicarbonate (NaHCO\textsubscript{3}).

Since the concentration of gases in the blood is proportional to the partial pressure of the gas, the actual amount of CO\textsubscript{2} and O\textsubscript{2} can be calculated by measuring these pressures. This partial pressure is a useful index in the development of a post mortem method of diagnosing cause of death (28:657).

**Blood Gas Analysis**

One method of measuring the partial pressure of a gas in blood is to extract that gas from the blood sample with a micromanometric apparatus (42:523). The gas present per unit volume of the sample is then calculated using formula 4.

\[(4) \quad \text{Gas} = P \times f_t \times R_v\]

Where: Gas is in millimoles per liter of blood

- \(P\) = pressure in mmHg
- \(f_t\) = temperature factor (43:29)
- \(R_v\) = instrument volume ratio a/b
where: \( a = \) volume of gas in the chamber when \\
P is measured \\
b = volume of sample blood

This method of measuring the partial pressures of 
blood gases is effective and was used in this study with 
certain modifications. This new method was of sufficient 
sensitivity for the present study. A brief description 
of the methods follows.

**pO₂ Measurement**

In 1956, Clark developed the first practical direct-
reading pO₂ electrode (14:42). In the Clark electrode, 
a platinum cathode and a silver reference electrode (anode) 
are separated from the sample by a thin membrane. A 
thin layer of electrolyte comprising a phosphate buffer 
and potassium chloride is trapped between the membrane 
and the tip of the electrode. O₂ molecules pass from the 
sample through the membrane and into the electrolyte until 
an equilibrium is established and the pO₂ in the electro-
lyte is the same as that of the sample. O₂ reaching the 
cathode is reduced to water and as a result of electron 
loss from the cathode, a current will flow between the 
cathode and the anode. This current is proportional to the 
pO₂ of the sample on the other side of the membrane.
pCO₂ Measurement

An electrode which would directly measure pCO₂ was originally devised by Stow and Randall (34:646). This electrode was modified and put into a more usable form by Severinghaus and Bradley (33:515). This electrode is basically a pH electrode-measuring the pH of a very small volume of sodium bicarbonate trapped between the sensitive glass surface of the electrode, and a thin membrane of teflon. The teflon is permeable to CO₂ but not to the bicarbonate ion of the blood sample on the other side of the teflon barrier. The CO₂ from the sample enters the bicarbonate solution and changes its pH (CO₂ combines with water to form carbonic acid, H₂CO₃, thus lowering the pH, and this change is detected by the glass electrode. The pH measured by the glass electrode depends upon the ratio between CO₂ and bicarbonate in the layer between the electrode and the membrane; therefore, the pH measured is a function of the pCO₂ of the sample outside the membrane.

pH Measurement

Electrometric and colormetric techniques are used to measure pH. Many people are familiar with the test where litmus paper turns pink when immersed in an acid and blue when immersed in a base. This is a colormetric pH measurement in its simplest form. The utility of the
colormetric test is very limited and generally this method is not very accurate.

Electrometric pH measurements are based on the fact that when immersed in a liquid, certain electrodes (such as hydrogen, quinhydrone, etc.) develop voltages that depend on the hydrogen-ion concentration of the liquid. Originally only this type of electrode was available for electrometric pH measurements and this was not highly accurate. Bates described the theory and practice of electrometric methods of pH determinations which utilize highly accurate glass electrodes (6:19) (7:126). The active element of a glass electrode is a membrane of a special glass. If the membrane forms a partition between two liquids of differing hydrogen-ion concentrations, a potential is produced between the two sides of the membrane. This potential is proportional to the difference in pH between the liquids. In this experiment a radiometer, thermostatted electrode originally described by Siggaard Anderson et al was used for electrometric pH determinations (3:49).

Summary

A review of the literature reveals a paucity of research in blood gas analysis following death. Beckwith suggested that one important step in solving the problem of the SID Syndrome would be to develop a method of determining whether the heart or lungs failed first (8). Such
knowledge would help considerably in eventually getting at a cause of death.

Initial work by Mithoefer indicated that a post-mortem diagnosis to differentiate between death caused by cardiac or respiratory failure could be made via blood gas determinations (28:654). It was found that the most applicable method of blood determinations was through the use of glass and diffusion type electrodes.
CHAPTER III

PROCEDURE

Three experiments were performed. Male mice of the Swiss strain weighing 14 to 24 grams, and aged four to five weeks were used. Animals were anesthetized with a sodium pentobarbital injection (65 mg/kg) and treated with the anticoagulant heparin (0.05ml/mouse, I.P.).

Once anesthetized, a ventral sagittal incision was made from the mentum to approximately 1 mm above the genitalia, and the skin was pulled back. In those mice receiving the respiratory obstruction, the trachea was exposed and occluded with a hemostatic clamp. After three minutes elapsed, and without removing the clamp, the sternum was split and the left rib cage removed exposing the heart. In all cases, the heart had ceased to beat by the time the thorax was opened. Blood was then removed from the left ventricle via 150 microliter glass capillary tube. Care was taken to insure that the sample was obtained under strictly anaerobic conditions. In most animals, capillary action was sufficient to acquire the 130 microliter sample, but in some cases a gentle kneeding of the animal was required. Blood samples were immediately analyzed using a Radiometer BMS-3 which
utilizes a glass electrode for pH, Severinghaus electrode for pCO₂ and a Clark electrode for pO₂.

In experiments where cardiac arrest was the mode of death, animals were anesthetized and treated with heparin as were the occluded airway subjects. Cardiac arrest was induced by passing a 120 volt alternating current across the chest for four to five seconds via electrodes clipped to each side of the thorax. Surgical techniques were the same, except that the trachea was not clamped. Following electric shock the animal's sternum was split, and blood removed from the left ventricle. Blood samples were analyzed as in occluded airway animals.

Experimental Design

Experiment 1. The agonal episode was induced in nine animals in each of the two groups—cardiac arrest and occluded trachea. Blood samples were taken immediately from animals sacrificed by cardiac arrest. Samples were obtained from subjects sacrificed by airway occlusion after a three minute interval. This time interval was necessary for the cessation of all respiration movements. All samples were analyzed immediately to determine the pCO₂, pO₂ and pH values.

Experiment 2. Three groups were sacrificed, each containing six mice. Group A was sacrificed by occlusion of the airway with an elapse of three minutes before blood
was removed. Group B was sacrificed by cardiac arrest with a three minute elapse before blood was removed. Group C was also sacrificed by cardiac arrest but the blood was removed immediately. A three minute time period in Group B was employed in order to detect any effect of the sampling time differential on pO$_2$, pCO$_2$ or pH values.

**Experiment 3.** Ten animals were sacrificed via cardiac arrest, and ten animals by occlusion of the airway. Subjects in the occluded airway group were treated as in previous experiments. In the cardiac arrested subjects, three minutes elapsed before the sternum was split and the left ventricle punctured as in group two of Experiment 3. Bloods were analyzed as in the other studies.
CHAPTER IV

RESULTS

The blood gas changes after death were such that the discrimination between each mode of death could be made on the pO₂ values but not on pCO₂ or pH values. In all three experiments, earlier work was confirmed (28:654) (37:520).

Experiment 1. As indicated in Table I, statistical differences were obtained on pCO₂ between occluded airway and cardiac arrested subjects (see Figure 1). The reasons for this are not clear, since a similar response was not observed in other experiments. It was found that pO₂ differences were highly significant in each experiment and therefore consistent with the hypothesis that pO₂ should be lower in the occluded airway group (see Figure 2).

Experiment 2. Cardiac arrest was studied in two ways; Group B had a three minute waiting period before blood samples were taken, while in Group C blood samples were taken immediately. Results indicated that a three minute interval did not cause significant differences in pO₂, pCO₂ or pH values; however, significant differences in pO₂ were obtained when blood samples from
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p < .05*

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p > .05

*Considered statistically significant; evaluated at 16 degrees of freedom. (Student's t Test was used as the principle statistical procedure to obtain p values)
FIGURE 1

EXPERIMENT I
SACRIFICE BY RESPIRATORY OR CARDIAC MEANS
pCO₂ VALUES

(1) Cardiac
(2) Respiratory
FIGURE 2

EXPERIMENT 1
SACRIFICE BY RESPIRATORY OR CARDIAC MEANS
pO₂ VALUES

CUMULATIVE PERCENTAGE

mmHg

(1) Respiratory
(2) Cardiac
the occluded airway group were compared to either cardiac 
arrest group. Once again, pCO₂ values were not signifi-
cantly different (see Table II and Figures 3 and 4).

Experiment 3. As illustrated in Table III, Figure 5
and Figure 6, results support and confirm the previous 
experiment. Statistically significant differences for 
pO₂ were obtained.

Apparently pH is not associated with mode of death 
(see Table I, Table II, Table III, Figure 7, Figure 8 and 
Figure 9).
<table>
<thead>
<tr>
<th></th>
<th>Group A Occluded Airway</th>
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<th>Group C Cardiac Arrest Only</th>
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Group A vs. B: \( p > 0.05 \)
Group A vs. C: \( p > 0.05 \)
Group B vs. C: \( p > 0.05 \)

Group A vs. B: \( p < 0.05 \)*
Group A vs. C: \( p < 0.05 \)*
Group B vs. C: \( p < 0.05 \)*

*Considered statistically significant; evaluated at 10 degrees of freedom
FIGURE 3

EXPERIMENT 2
SACRIFICE BY RESPIRATORY OR CARDIAC MEANS
pO2 VALUES

(1) Respiratory
(2) Cardiac
(3) Cardiac + 3 minutes
FIGURE 4

EXPERIMENT 2
SACRIFICE BY RESPIRATORY OR CARDIAC MEANS
pCO₂ VALUES

(1) Cardiac
(2) Cardiac + 3 minutes
(3) Respiratory
### TABLE III

MEANS, STANDARD DEVIATION, AND P VALUES
FOR CARDIAC ARRESTED AND OCCLUDED
AIRWAY ANIMALS - 3

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* Considered statistically significant; evaluated at 18 degrees of freedom
FIGURE 5

EXPERIMENT 3
SACRIFICE BY RESPIRATORY OR CARDIAC MEANS
pO$_2$ VALUES

CUMULATIVE PERCENTAGE

mmHg

(1) Respiratory
(2) Cardiac
FIGURE 6

EXPERIMENT 3
Sacrifice by Respiratory or Cardiac Means
pCO₂ Values

CUMULATIVE PERCENTAGE

(1) Cardiac
(2) Respiratory
FIGURE 7

EXPERIMENT 1
SACRIFICE BY RESPIRATORY OR CARDIAC MEANS
pH VALUES

(1) Respiratory
(2) Cardiac
FIGURE 8

EXPERIMENT 2
SACRIFICE BY RESPIRATORY OR CARDIAC MEANS
pH VALUES

(1) Cardiac + 3 minutes
(2) Respiratory
(3) Cardiac
FIGURE 9

EXPERIMENT 3
SACRIFICE BY RESPIRATORY OR CARDIAC MEANS
pH VALUES

CUMULATIVE PERCENTAGE

pH


(1) Respiratory
(2) Cardiac
CHAPTER V

DISCUSSION

The purpose of these experiments was to study blood gas tensions following death. It was hypothesized that by measuring pO\textsubscript{2} in mice, it would be possible to predict whether the heart or the lungs failed first during the agonal event. In addition there should be no significant difference in pCO\textsubscript{2} of pH. Previous workers have reported that when dogs were sacrificed by cardiac arrest or respiratory obstruction, and pO\textsubscript{2} measured following death, the mode of death could be determined (28:657). It was found that the pO\textsubscript{2} remained higher following cardiac arrest than when death resulted from respiratory obstruction. The present experiments with mice indicate that mode of death can also be determined in this species by similar methods. Mithoefer concluded that a pO\textsubscript{2} value of more than 25 mmHg should indicate cardiac arrest (28:656). Values of Swiss strain mice are extremely close, 20 mmHg, (mean pO\textsubscript{2} of all cardiac arrested animals). The low pO\textsubscript{2} in both experiments is probably due to removal of O\textsubscript{2} from the blood by tissues as circulation continues following airway occlusion. In the present study, the heart was observed to continue beating up to two and one half
minutes following the clamping of the trachea. Earlier studies have shown that this tissue hypoxia does result in mice after three minutes of asphyxia (24:435).

No differences were noted in pCO₂ values in two of the three experiments. The obtained p value for pCO₂ (see Table II) in Experiment 1 was significant (p<.05). Two animals in the occluded airway group of this experiment hypoventilated excessively on receiving the anesthetic. These two subjects had much higher pCO₂ values than the group average which could account for this inconsistency. As shown in Tables II and III, significant differences were not obtained on pCO₂ in Experiments 2 and 3. This is consistent with the hypothesis.

Differences in pH were observed in Experiment 2 between both the occluded airway subjects and also the cardiac arrest subjects (see Table II). The results could not be replicated. It is, therefore, unlikely that hydrogen ion concentrations are associated with these modes of death.
CHAPTER VI

CONCLUSIONS

Results strongly suggest that by measuring pO₂ following death it is possible to predict whether the lungs or heart failed first. Tissue hypoxia exists in both modes of death, but in the occluded airway animals the circulation of blood continues, allowing oxyhemoglobin to reach the tissues. With cardiac arrest, blood is not forced through the tissues, and deoxygenation is retarded.
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8. Beckwith, J. Bruce, M.D., Children's Orthopedic Hospital and Medical Center, Seattle, Washington. Personal Communication, Oct. 5, 1970. This hypothesis is a result of the thinking of Dr. Beckwith as stated in this communication, "Some specific projects I would like to see carried out center around the area of agonal physiology. One of the key issues is whether S.I.D.S. is the last analysis cardiac arrest or respiratory obstruction."


22. Jacobsen, T., and J. Viogt. "Sudden and Unexpected


34. Stow, R. W., R. F. Baer, and G. F. Randall, "Rapid Measurement of the Tension of Carbon Dioxide in


APPENDIX A

Data Listings for Experiment 1
### Group A  Occluded Airway

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APPENDIX B

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