Original Articles

Asphyxial Death: The Roles of Acute Anoxia, Hypercarbia and Acidosis

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Respiratory and circulatory responses to acute, severe hypoxia, hypoxia combined with moderate hypercarbia, and severe hypercarbia were measured in 22 dogs under conditions of apnea and spontaneous breathing. Survival times before circulatory collapse and physiological death indicated that depletion of oxygen stores progresses ten times more rapidly than accumulation of lethal quantities of hydrogen ion. Consequently, the responses to acute asphyxia differed little from those to breathing an oxygen-free atmosphere. Depletion of oxygen arrested respiratory drive before causing cardiac arrest in systole. During CO_2 retention, acidosis progressively paralyzed the myocardium; respiratory drive persisted until the circulation failed. Arterial P_{O_2} at the time of death was 5 to 10 mm. of mercury during anoxia with and without moderate CO_2 retention. Death occurred during extreme CO_2 retention over a narrow range of *p*H of arterial blood, from 6.50 to 6.45, whereas Pa_{CO_2} varied from 149 to 400 mm. of mercury, suggesting that acidosis causes death, rather than hypercarbia. Hypoxia and acidosis became additive in producing circulatory collapse at a Pa_{O_2} below 25 mm. of mercury and at a *p*Ha below 6.80. The rate of depletion of oxygen or accumulation of hydrogen ion did not alter the lethal values of either.

ACUTE severe interference with ventilation of the lungs causes changes in respiratory gas tensions, acid-base balance, electrolytes, and neurohormones eventually leading to cardiac arrest. Resuscitation from cardiac arrest involves an attempt to treat simultaneously all of these derangements. Because time is of the essence in successful resuscitation, more precise information on the most lethal disorder might lead to a more efficient order of treatment. The present study is concerned with identifying those factors which correlate most consistently with arrest of the circulation resulting from various combinations of hypoxia and hypercarbia.

We subjected dogs to anoxia and hypercarbia, singly and in combination with and without the work of breathing, until respiration and circulation ceased and until electrocardiographic activity disappeared. Our observations are in general agreement with those of others.[1–6] However, we have been able, by disregarding the differences in time-course to death caused by the different modes of respiratory failure, to establish more pointed correlations between extreme degrees of hypoxemia and acidemia and the occurrence of respiratory and circulatory arrest.

Methods

Twenty-two apparently healthy pound dogs weighing 12 to 18 kg. were anesthetized with single, minimal doses of thiamylal sodium. A cuffed endotracheal tube was inserted and a light plane of methoxyflurane anesthesia was maintained. Polyethylene catheters were inserted into the abdominal aorta, the inferior vena cava, and into the right ventricle for continuous recording of arterial, central venous, and right ventricular blood pressures using Statham strain gauges. A precordial electrocardiogram was obtained with needle electrodes. Airway pressure was measured at the endotracheal tube with a Statham strain gauge. A pneumotachograph (original Fleisch) was connected to the endotracheal tube.

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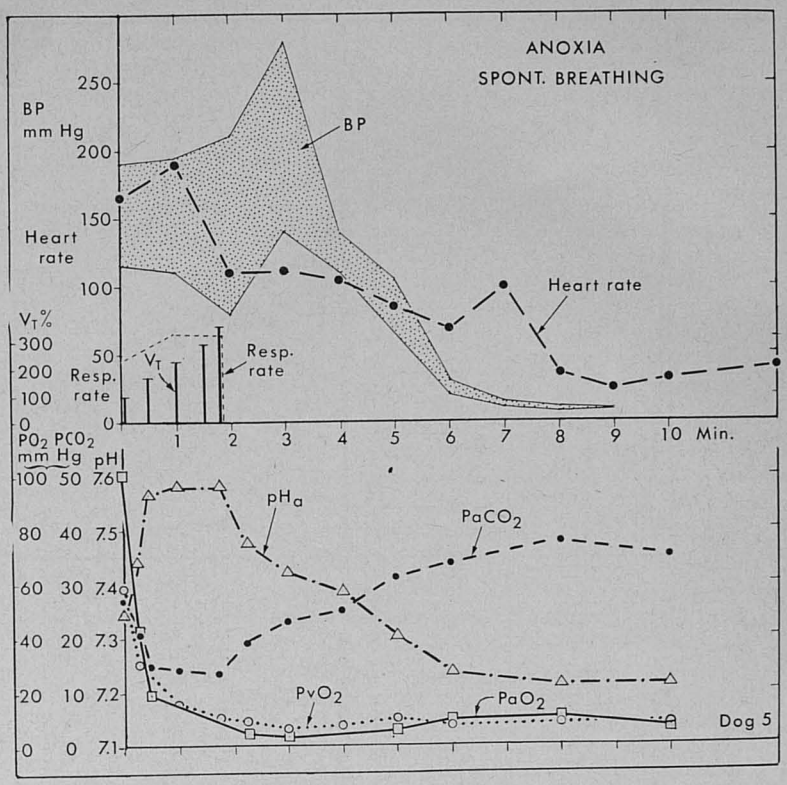

FIG. 1. A representative course of a spontaneously breathing dog, exposed to anoxia, illustrating the relation between cardiorespiratory events and altered blood gas tensions. BP is arterial blood pressure; V_T is tidal volume expressed as percentage increase over the initial tidal volume. See text for details.

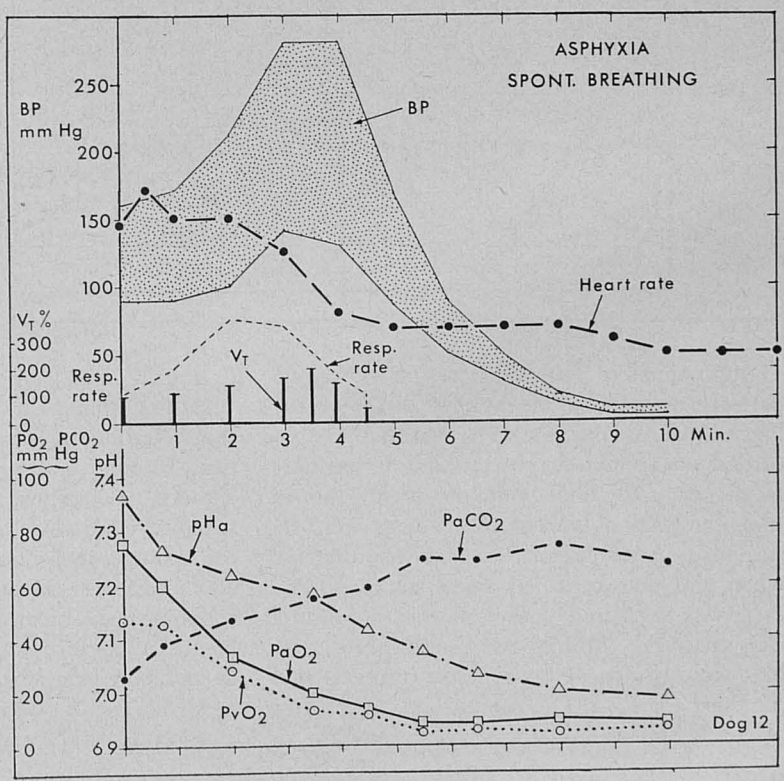

FIG. 2. A representative course of a spontaneously breathing dog exposed to asphyxia. Pa_{O_2} declines more slowly and the time course of the cardiorespiratory events is slightly more protracted than during anoxia.

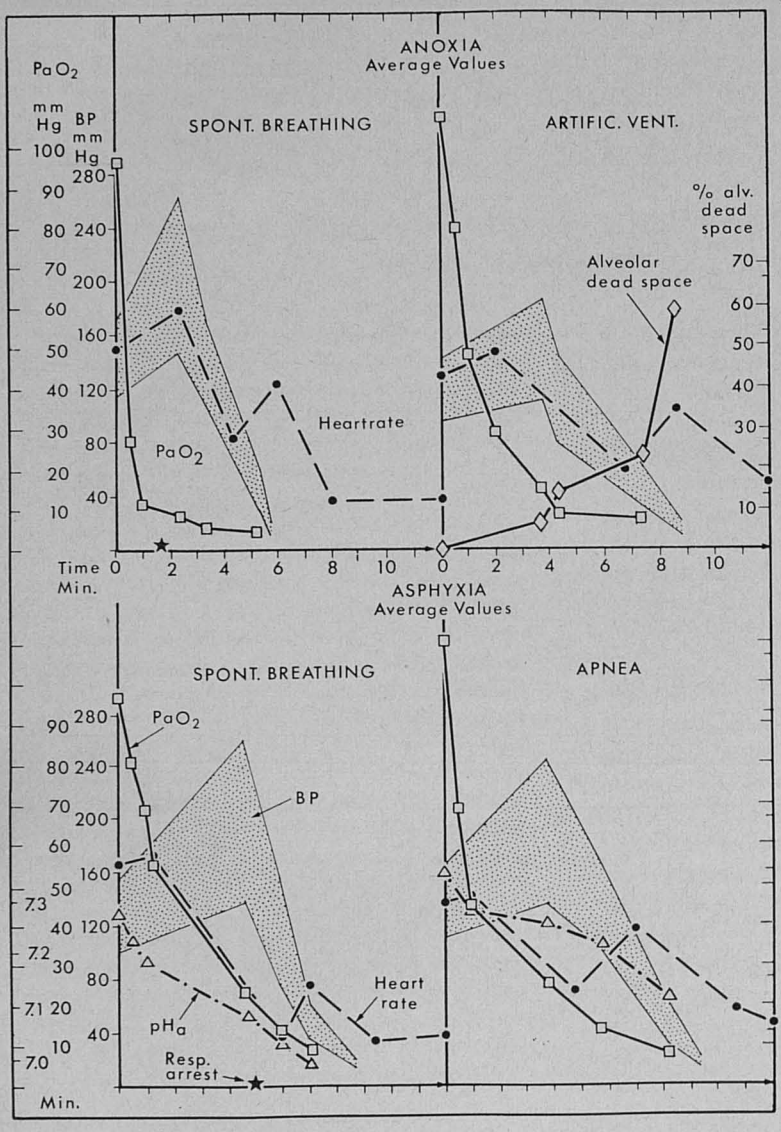

FIG. 3. The pertinent average circulatory values for all dogs exposed to anoxia and asphyxia. A star indicates the time of respiratory arrest in the spontaneously breathing dogs. Alveolar dead space, expressed as per cent of total alveolar ventilation, is shown for the artificially ventilated dogs which were exposed to anoxia.

Approximately one hour was used to prepare the animals before the start of the experiment. All parameters were continuously recorded on an Offner polygraph. Respiratory frequency and tidal volumes were derived from the pneumotachogram. Values were recorded as percentage of the control obtained when the dog breathed room air. Expired P_{CO_2} was measured with a Beckman infrared CO_2 analyzer. Arterial and venous blood samples were drawn at appropriate intervals and analyzed for P_{CO_2}, P_{O_2}, and *p*H using a Clark electrode, and a Radiometer potentiometer.

Anoxia was produced in spontaneously breathing animals by passing high flows of nitrogen (containing less than 0.5 per cent oxygen) through a non-rebreathing circuit (Ayres T-piece). Asphyxia was produced in spontaneously breathing animals by attaching to the endotracheal tube a sealed rebreathing bag containing air. Hypercarbia was produced in spontaneously breathing dogs using a similar breathing circuit into which oxygen was added to maintain inflation of the bag.

Apnea was induced with intermittent doses of succinylcholine chloride. In the paralyzed

TABLE 1. Summary of Pertinent Respiratory and Circulatory Values During Apneic Oxygenation

	Dog	16	17	18	19	Average*
Starting time	Time (min.)	0	0	0	0	0
	Pa_{O_2} (mm. Hg)	290	520	570	530	540
	Pv_{O_2} (mm. Hg)	45	57	92	57	69
	*p*Ha	7.41	7.50	7.25	7.36	7.34
	Pa_{CO_2} (mm. Hg)	31	27	44	25	32
Time of maximum B. P.	Time (min.)	41	30	33	33	32
	Pa_{C_2} (mm. Hg)	31	220	346	246	271
	Pv_{O_2} (mm. Hg)	20	100	81	94	92
	*p*Ha	6.80	6.76	6.71	6.66	6.71
	Pa_{CO_2} (mm. Hg)	205	215	192	214	207
Time of B. P. = initial value	Time (min.)	45	59	47	77	61
	Pa_{O_2} (mm. Hg)	17	124	224	122	157
	Pv_{C_2} (mm. Hg)	5	122	84	73	93
	*p*Ha	6.77	6.66	6.59	6.46	6.57
	Pa_{CO_2} (mm. Hg)	210	215	253	286	251
Time of B. P. = 60 mm. Hg	Time (min.)	46	81	84	90	85
	Pa_{O_2} (mm. Hg)	14	65	98	96	86
	Pv_{O_2} (mm. Hg)	6	43	45	38	42
	*p*Ha	6.76	6.53	6.64	6.44	6.48
	Pa_{CO_2} (mm. Hg)	210	215	396	400	337
Time of last ECG activity	Time (min.)	85	134	96	100	110

The times are given in minutes after start of CO_2 retention.

* Average values are from the last three experiments. Dog 16 was omitted from the averages because his course was influenced by hypoxia.

animals, anoxia was produced by artificially ventilating the dogs with a time-cycled, preset-volume, constant-flow automatic lung ventilator operating into a non-rebreathing system through which a high flow of nitrogen was passed. Asphyxia was produced in paralyzed dogs by obstructing the endotracheal tube. Hypercarbia was induced in paralyzed dogs (apneic oxygenation) by passing a high flow of oxygen through an Ayres T-piece, following thorough denitrogenation.

Results

The time course of typical responses to anoxia and hypercarbia is illustrated in figures 1, 2, and 4. Average values of pertinent respiratory and cardiovascular parameters, obtained from each group at times of significant events, are presented in figure 3 and tables 1 and. 2. The points in time considered to be significant were: time of maximum pulse rate, time of maximum blood pressure which we assume marked the onset of circulatory failure, time during circulatory failure when the systolic blood pressure equaled the initial blood pressure, and the time when the systolic blood pressure fell to 60 mm. of mercury. This last value was arbitrarily designated as marking the cessation of effective blood flow; it always occurred when the blood pressure was falling rapidly and within a minute or two of the time when the pulse pressure approximated zero. Also included is the time of respiratory arrest in spontaneously breathing animals. These events were selected to establish correlations between progressive steps in circulatory and

TABLE 2. Summary of Pertinent Respiratory and Circulatory Values During Hypercarbia with Spontaneous Breathing

	Dog	20	21	22	Average
Starting time	Time (min.)	0	0	0	0
	Pa_{O_2} (mm. Hg)	480	510	117	369
	Pv_{O_2} (mm. Hg)	73	66	54	64
	*p*Ha	7.25	7.17	7.23	7.22
	Pa_{CO_2} (mm. Hg)	44	44	24	37
Time of maximum B. P.	Time (min.)	46	41	55	47⅓
	Pa_{O_2} (mm. Hg)	34	100	42	59
	Pv_{O_2} (mm. Hg)	30	68	28	42
	*p*Ha	6.71	6.65	6.52	6.63
	Pa_{CO_2} (mm. Hg)	199	134	264	199
Time of B.P. = initial value	Time (min.)	47½	53	56½	52⅓
	Pa_{O_2} (mm. Hg)	12	40	31	28
	Pv_{O_2} (mm. Hg)	8	22	13	14
	*p*Ha	6.68	6.66	6.52	6.62
	Pa_{CO_2} (mm. Hg)	197	144	264	202
Time of B. P. = 60 mm. Hg	Time (min.)	48	54½	57	53⅓
	Pa_{O_2} (mm. Hg)	11	11	28	17
	Pv_{O_2} (mm. Hg)	7	9	9	8
	*p*Ha	6.68	6.66	6.52	6.62
	Pa_{CO_2} (mm. Hg)	197	149	264	203
Time of respiratory arrest	Time (min.)	47	54½	56½	52⅔
	Pa_{O_2} (mm. Hg)	13	11	31	18
	Pv_{O_2} (mm. Hg)	9	9	13	10
	*p*Ha	6.68	6.66	6.52	6.62
	Pa_{CO_2} (mm. Hg)	197	149	264	203
Time of last ECG activity	Time (min.)	112	123	75	103¼

The times are given in minutes after start of CO_2 retention.

respiratory failure and changes in arterial blood P_{O_2}, P_{CO_2}, and *p*H.

Anoxia. During anoxia, respiratory rate and tidal volume increased in the spontaneously breathing dogs, the minute ventilation becoming 4.7 times the control ventilation. Oxygen was washed from the lungs and pulmonary capillary blood during the first minute, as shown by the rapid decline of Pa_{O_2}, to an average tension of 10 mm. of mercury at the end of the first minute, and by the consistently higher values of P_{O_2} in venous blood as compared with the arterial after the first few seconds of exposure to nitrogen. There was a sudden concurrent reduction of P_{CO_2} and increase of *p*H which was sustained until respiratory activity ceased suddenly shortly before the end of the second minute of exposure. Following the second minute, P_{CO_2} increased gradually, *p*H declined at a comparable rate, and Pa_{O_2} and Pv_{O_2} remained constant at a very low level approximating 5 mm. of mercury.

Heart rate increased an average of 20 per cent during the first minute, following which pronounced bradycardia developed. Arterial systolic, diastolic, and pulse pressures increased and reached maximum values approximately 30 seconds after cessation of respiration. As the Pa_{O_2} fell below 10 mm. of mercury, the circulation failed rapidly. The electrocardiogram, on the other hand, persisted for more than 30 minutes, presenting at times a normal pattern (fig. 6). A progressively increasing alveolar dead space, indicated by an expanding arterial-end tidal difference in CO_2 tension, appeared as arterial blood pressure commenced to fall and increased sharply as systolic blood pressure fell below 60 mm. of mercury (fig. 3). No significant differences could be detected in the courses of the artificially ventilated, paralyzed dogs as compared with the spontaneously breathing anoxic dogs, with the exception that the events progressed somewhat more slowly in the paralyzed dogs, in which arterial *p*H and P_{CO_2} were sustained without significant change—until failure of the circulation was well developed.

Asphyxia. Dogs subjected to asphyxia exhibited the same sequence of failure of respiration and circulation as the anoxic dogs except for a slightly more protracted course for all events and a slower decline of Pa_{O_2} (fig. 6). Pa_{CO_2} approximated 80 mm. of mercury terminally, and *p*Ha never declined below 7.0. Again, except for slightly accelerated deterioration during spontaneous breathing, no differences existed between the responses of spontaneously breathing and paralyzed dogs subjected to asphyxia.

Hypercarbia. Apneic Oxygenation: When circulatory failure and clinical death occurred in ten minutes or less during anoxia and asphyxia, blood pressure began to decline from maximum values 30 to 33 minutes after onset of CO_2 retention and declined gradually to reach 60 mm. of mercury at an average of 85 minutes (Table 1). At no time was arterial or venous oxygen tension within an hypoxic range. Pa_{CO_2}, on the other hand, had risen to about 207 mm. of mercury and *p*H averaged 6.71 when the blood pressure began to fall and 398 mm. of mercury and 6.48, respectively, at the time of circulatory collapse.

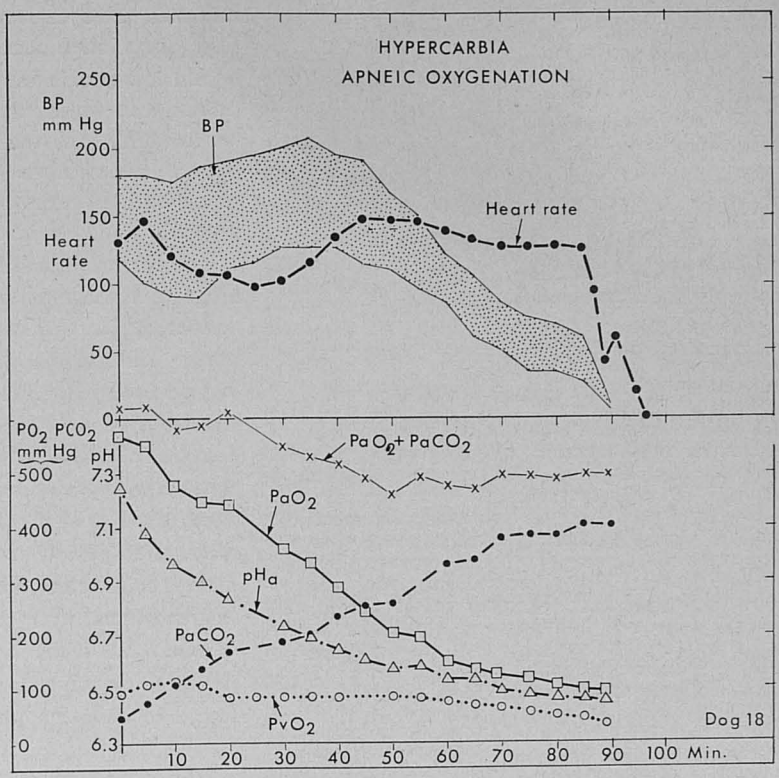

FIG. 4. A representative pattern of responses of a dog subjected to apneic oxygenation when hypoxia was not superimposed. The gradual decline of the line constructed by adding Pa_{O_2} and Pa_{CO_2} suggests the development of shunting in the lungs.

Hypercarbia with Spontaneous Respiration: The Pa_{O_2} of two animals in this group approximated 500 mm. of mercury at the start of the experimental period. The third inadvertently was not denitrogenated and started with an initial Pa_{O_2} of 117 mm. of mercury (Table 2). The courses of all three were, however, similar. At 47 minutes, blood pressure achieved a maximum value, the Pa_{CO_2} approximated 200 mm. of mercury, and *p*H was 6.6. Progression of events up to this time was similar to the group subjected to apneic oxygenation except that the systolic blood pressure had continued to rise. In the course of the next five minutes, however, the Pa_{O_2} and Pv_{O_2} reached severely hypoxic levels, and circulatory collapse occurred quickly. Spontaneous respiration persisted after the Pa_{CO_2} exceeded 200 mm. of mercury and the *p*H had fallen to 6.62; respiratory arrest occurred when the average Pa_{O_2} reached 18.3 mm. of mercury.

Critical Limits of Hypoxia and Acidemia. The responses of the anoxic, the asphyxiated, and the spontaneously breathing hypercarbic animals suggest that circulatory collapse occurs when oxygen is reduced below a certain minimum. The response of the apneic oxygenation group indicates that circulatory collapse occurs when a certain degree of acidemia is reached. These relationships are summarized in figure 5. Circulatory failure began, as indicated by the onset of fall of blood pressure from the maximum and the decline to the initial value, at somewhat lower values for Pa_{O_2} in the anoxic group than the asphyxiated group, and failure was complete (blood pressure equaled 60 mm. of mercury) in both groups at about the same low Pa_{O_2}. The spontaneously breathing hypercarbic dogs, on the other hand, failed at appreciably lower values for Pa_{O_2} and slightly higher *p*Ha values than the apneic oxygenation group. In summary, in the absence of severe acidosis, circulatory failure occurred at an arterial P_{O_2} between 5 and 10 mm. of mercury. In the absence of hypoxia, circulatory failure occurred when arterial *p*H reached 6.5. When extreme hypoxia and respiratory acidosis coexisted, circulatory failure occurred at values

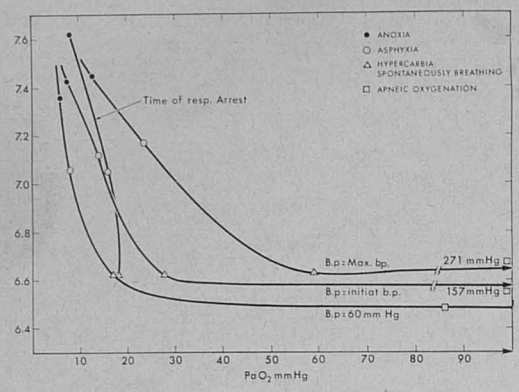

FIG. 5. The average values of Pa_{O_2} and *p*Ha are plotted for each group of dogs at the times of maximum blood pressure, when the blood pressure had declined to the initial value, and when it reached 60 mm. of mercury. The average Pa_{O_2} and *p*Ha of each group of spontaneously breathing dogs is plotted at the time of respiratory arrest. The solid lines join the points representing the various groups. The paralyzed and spontaneously breathing dogs have been averaged within the groups subjected to hypoxia and asphyxia. Hypoxia and acidosis appear to act synergistically to initiate reduction of blood pressure. Final circulatory collapse, on the other hand, occurred either at a Pa_{O_2} below 10 mm. of mercury or at a *p*H of 6.5, with an additive effect being evident only in the average of the spontaneously breathing hypercarbic group (the triangle).

of P_{O_2} and *p*H only slightly higher than either of these extremes, indicating a slight additive action.

The Electrocardiogram. The most consistent changes in the electrocardiogram during anoxia and asphyxia were ST segment and T-wave changes, and A-V dissociation. ST segments were elevated and T waves were of high voltage and peaked. After circulatory collapse, the T wave frequently became fused with the QRS complex, which either was of low voltage or exhibited a single negative wave. Transient A-V dissociation appeared almost uniformly during anoxia and asphyxia but usually reverted to a sinus rhythm before circulatory collapse (fig. 6).

Premature supraventricular and ventricular complexes and bigeminy were seen during hypoxia and during the first thirty minutes of hypercarbia. During hypercarbia without superimposed hypoxia, however, A-V dissociation, bigeminy, and peaked T-waves never occurred before circulatory arrest. Within 17 minutes after circulatory arrest, a permanent A-V dissociation occurred in all groups of dogs.

During anoxia and asphyxia, the ECG terminated in ventricular fibrillation as frequently as in stand-still, whereas 6 dogs subjected to severe hypercarbia terminated in stand-still. A seventh hypercarbic dog was also severely hypoxic and terminated in fibrillation.

Ventricular fibrillation occurred in hypoxic dogs an average of 17 minutes after circulatory failure, the range being from two to 32 minutes. The range appeared not to be influenced by terminal *p*H. Electrocardiographic activity persisted an average of 18 minutes after ventricular fibrillation started, or a total of 35 minutes after circulatory collapse.

At post mortem examination, the hearts of all dogs subjected to anoxia and asphyxia were firmly contracted, in contrast to the dilated hearts found after exposure to severe hypercarbia. In cases where anoxia was superimposed on severe acidosis, the hearts were found to have an intermediate rigor.

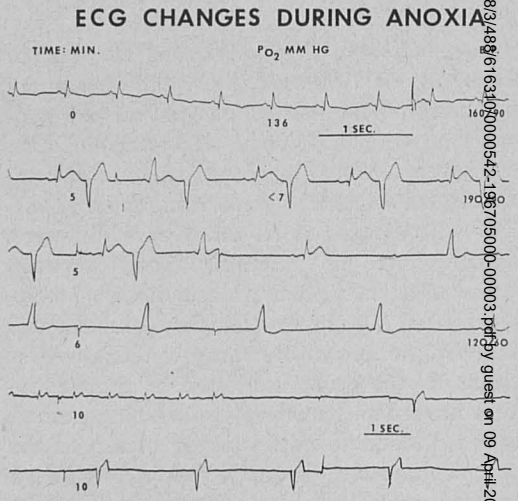

FIG. 6. Electrocardiographic changes occurring during exposure to anoxia. The third line continues directly from the second, as does the sixth from the fifth. The paper speed was reduced to one-half for the tracing at ten minutes. Note the peaked T waves, the elevated ST segments, and the transient A-V dissociation at five minutes. There is reversion to a sinus rhythm at six minutes, before circulatory collapse. A persistent block develops after circulatory stasis, during the tenth minute.

The lungs appeared grossly normal after death except for some congestion of dependent portions.

Discussion

The causes and the progression of failure of vital functions during acute, extreme anoxia and hypercarbia can be clearly discriminated. The rapid early fall of P_{O_2} during pure anoxia induces a tachycardia which is quickly replaced by reflex bradycardia and hypertension. Failure of the circulation begins at Pa_{O_2} below 15 mm. of mercury as the stores of oxygen available to the myocardium become exhausted. Failure proceeds rapidly as the Pa_{O_2} is depressed below 10 mm. of mercury, and confirms the results of Clowes,[7] Cater,[8] and Swann.[6] The coincidental appearance of increase in alveolar dead space, as the systolic blood pressure declines below the initial pressure, indicates ventilation in excess of perfusion and evidence of decreasing perfusion of the lungs.[9, 10] The accelerated expansion of alveolar dead space as the systolic blood pressure falls below 60 mm. of mercury marks the point of circulatory collapse (fig. 3). These relationships justify our arbitrary designation of these blood pressures as points of significant hemodynamic change.

Acute respiratory acidosis, uncomplicated by anoxia, acts over a narrrow range of *p*H to cause circulatory failure. Decline of blood pressure begins at *p*Ha 6.7 and circulatory arrest is essentially complete at *p*Ha 6.5.

When anoxia and respiratory acidosis coincide, cardiovascular instability occurred at considerably higher values of Pa_{O_2} as suggested by the position of the point of maximum blood pressure of the asphyxiated group of dogs, in figure 5. There is, however, little influence on the terminal Pa_{O_2}, indicated by the position of the point designating blood pressure equal to 60 mm. of mercury in the same group of dogs.

The differences in the survival times of the different groups of dogs can be explained entirely on the rate of depletion of oxygen and the rate of accumulation of carbon dioxide and hydrogen ion, as determined by the different experimental conditions. The spontaneously breathing, anoxic dogs died early as a consequence of rapid blow-off of oxygen stores and increased consumption of oxygen in performing the work of breathing. The paralyzed animals which were subjected to anoxia by being artificially ventilated with nitrogen exhibited a more gradually declining Pa_{O_2} and died later, presumably because they were not involved in a vigorous hyperpnea. The asphyxiated dogs also depleted their oxygen stores more slowly because they had larger volumes of oxygen available at the start of the experiment and because there was no wash-out of oxygen. Acidemia appeared to play no important role in causing death during the two forms of asphyxia used in these experiments. Arterial P_{O_2} declined much more slowly in the animals subjected to CO_2 retention. Circulatory failure began in most of the dogs when *p*Ha was in the range 6.6–6.7 (tables 1 and 2), and proceeded slowly toward arrest as the *p*H fell to 6.5, except in those whose oxygen tensions fell below 30 mm. of mercury. The later failed abruptly and were usually dead within five minutes at a *p*Ha of 6.6 and a Pa_{O_2} of 20 mm of mercury.

Respiratory arrest preceded circulatory collapse in most instances, but the difference in time was greatest when the Pa_{CO_2} was low and diminished as the Pa_{CO_2} was elevated. Although this suggests that extremes of both hypoxia and respiratory acidosis can paralyze the neurones of the respiratory center, as they apparently do to the muscles of the heart, a more likely explanation is that oxygen deprivation alone was responsible for respiratory arrest in our experiments. The tendency for the line depicting the point of respiratory arrest in figure 5 to cross from the line of maximum blood pressure at high *p*H to the line of blood pressure equal to 60 mm. of mercury at low *p*H supports the view that the respiratory center fails at a Pa_{O_2} somewhat higher than that causing heart failure and that, in the absence of extreme arterial hypoxemia, the medullary centers continue to function until circulation to the brain has stopped. Further evidence that the respiratory center can function at extremely high carbon dioxide tensions in the absence of severe arterial hypoxemia or circulatory failure comes from the work of Graham, Hill, and Nunn,[11] who gradually added carbon

dioxide to the inhaled gas mixture and observed respiratory arrest when the CO_2 concentration was close to 40 per cent, a value higher than was achieved by our spontaneously breathing hypercarbic dogs. Furthermore, Graham's dogs resumed spontaneous respiration and breathed concentrations of carbon dioxide as high as 60 and 70 per cent.

The mechanism of heart failure during anoxia is different from that during respiratory acidosis. Circulatory failure progresses rapidly during anoxia in contrast to the gradual decline during hypercarbia, in keeping with the difference in storage capacity of the blood and tissues for oxygen and carbon dioxide. Myocardial irritability is high in the anoxic heart and ventricular fibrillation is likely to occur, whereas ventricular fibrillation never occurred in dogs whose *p*H was below 6.70 despite the presence of severe hypoxia. After death, the hearts of the anoxic and mildly acidemic dogs were firmly contracted [12]; the hearts of severely hypercarbic dogs were dilated and relaxed. These differences can be explained by the high concentration of catecholamines released in response to both anoxia and hypercarbia and by the interference with the effect of catecholamines on the myocardium that occurs at decreasing *p*H.[3, 13, 14] Thus, during our experimental conditions called anoxia and asphyxia, heart failure was due to exhaustion of the aerobic enzyme systems which are responsible for repolarization and relaxation of myocardium [15] and which account for more than 90 per cent of the energy releasing systems in the heart.[16] During our conditions of hypercarbia, the myocardium was progressively paralyzed by acidosis to the effects of catecholamines, on which myocardial contractibility is dependent.

Summary and Conclusions

Acute respiratory failure results in death from cardiac arrest. The form of cardiac arrest depends upon whether hypoxia or respiratory acidosis predominates. Hypoxia arrests the heart in systole whereas respiratory acidosis stops the heart in diastole. Ventricular fibrillation tends to develop after cardiac arrest in hearts which have incurred hypoxia; concurrent, severe acidosis prevents ventricular fibrillation. Hypoxia causes cardiac arrest in lightly anesthetized dogs when the arterial P_{O_2} is 10 mm. of mercury or less. Hypercarbia becomes lethal when the arterial *p*H is depressed below 6.5. These limits of Pa_{O_2} and *p*Ha are independent of the time required to deplete body stores of oxygen or to accumulate hydrogen ion. Respiratory drive ceases at a Pa_{O_2} slightly higher than that at which cardiac arrest occurs.

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Circulation

SINGLE-UNIT TRANSFUSIONS The single-unit transfusion has great advantage over the two- or three-unit transfusion, if the single unit is used to avoid two or three units of blood. Risk of serum hepatitis to patients receiving single transfusions from transfusions derived from prison and Skid Row populations is at least ten times that from the use of volunteer donors. However conservative one is in the use of blood, the largest number of patients transfused will always be those receiving single transfusions—between 35 and 40 per cent of the total number of patients transfused in a civilian general hospital. The most practical method for reducing the hazard of serum hepatitis from blood is to stop using blood from prison and Skid Row donors. To limit the use of blood by giving one transfusion instead of two, two instead of three, etc., is helpful, but of far less benefit than would be measured to insure the use of blood from quality donors. If it is necessary that blood from undesirable donors be used to meet demands, such blood should be labeled as carrying a significantly increased hazard of transmitting serum hepatitis. (*Allen, J.: The Advantages of the Single Unit Transfusion, Ann. Surg. 164: 475 (Sept.) 1966.*)

Abstractor's Comment: Clinical operating room experience indicates that frequently the single-unit transfusion may spell the difference between impending shock and circulatory compensation, yet the anesthesiologist is often called upon to defend this action before the vigilant Transfusion Committees of some hospitals. The information in this article may be of help.

AUTOTRANSFUSION Transfusion with a patient's own blood has obvious advantages over transfusion with blood from others, be it fresh donor, bank, cadaver, or frozen blood. The hazards of incompatible blood, viral hepatitis, sensitivity reactions, and so forth are eliminated. Blood drawn from a patient prior to elective surgery and reinfused during surgery is limited in use to good risk patients in whom its need can be anticipated. Blood collected from the abdominal cavity during surgery has been reinfused on rare occasions, while blood from the chest cavity is routinely reinfused in open heart procedures. The major reason for the lack of enthusiasm for this technique has been the absence of suitable and readily available collecting and filtering apparatus. A simple intraoperative transfusion set utilizing silicone collecting bottles, siliconized steel wool, and a fine 200 micro screen for filtration is described and has been used successfully in dogs and in humans. (*Dyer, R. H., Jr.: Intraoperative Autotransfusion, Amer. J. Surg. 112: 879 (Dec.) 1966.*)

Abstractor's Comment: In the day of disposable blood oxygenators it is certainly not beyond the capabilities of industry to develop and make readily available a good inexpensive, disposable, collection and administration set for intraoperative autotransfusion.