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Drowning : importance, pathophysiology and resuscitation

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DROWNING: IMPORTANCE, PATHOPHYSIOLOGY and RESUSCITATION

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INTRODUCTION

Incidence of Drowning

In the United States, 6,900 dorwnings occurred during 1961. Of this figure, 4,400 happened as public accidents while the victims were either swimming, playing or falling in the water. Transportation on the water involved 1,100 of which 3/4 involved boats with a capacity of less than ten. The home was the site of 500 with victims found in bathtubs, pools, wells cisterns, cesspools and open bodies of water. Drowning represents the fourth cause of accidental death and the first cause, motor vehicle accidents, result in five to six times as many deaths. The death rate, deaths per 100,000 population, was four which represents a major decrease from 1905 when the death rate was eleven. In a state like Nebraska with a population of about 1,500,000, approximately sixty deaths would occur every year.

Drowning is a distressing cause of death since it frequently occurs in the young child at play and adults enjoying holidays and recreation. Almost 2/3 of the victims are between the ages of five and

forty-five with 1/3 in the school age population of five to nineteen years old. Drowning represented 17% of all accidental deaths in the last group. It is important to emphasize that in young children under five years, 95% of those drowned were around the home. Accidents result in more deaths than the next four causes in the one to fourteen age group and all other causes in the fifteen to twenty-four age group. In both the last two groups, five out of six accidental deaths are males. In May, June, July and August an average of 1,000 drownings occur per month. (1a) (2) (3), see tables 1 and 2.

Investigation of the Drowning Accident

Drowning has received little attention and has been neglected considering the rate of death. All too frequently little or no inquiry is made into: 1) personal history of victim, 2) predisposing factors, 3) details of drowning and rescue, and 4) treatment received. (1) (4). Each case should be investigated as fully as the circumstances permit and the findings sent as soon as possible to whatever local authority has accepted the responsibility for the investigation. See table 3. The information

should be collected from: 1) the victim if he survives, 2) companions and relatives if he dies, 3) rescuers and witnesses, 4) any one who gave first aid, 5) ambulance personal who transported the patient, 6) any attending medical practitioner, and 7) the pathologists who conducted any post mortem examination. (1a).

Urgency for Understanding Drowning

Neglect of this subject by physiologists and medical men has occurred inspite of the fact that it remains a vital and growing problem. Progress in the understanding of drowning has been almost negligible and over the last 150 years eminent persons have made statements concerning the neglect of this subject. (1) (4) (5) (6) (7). However, various lay organizations, i.e. Boy Scouts, Red Cross, YMCA and etc., have taken great interest in drowning with emphasis on prevention through safety, swimming, and life saving instruction.

PATHOPHYSIOLOGY OF DROWNING

Predisposing Factors

These include bravado, carelessness, ignorance, ill health, overindulgence in drink or food, sheer misfortune or foul play. Sometimes cramps, often attributed to cold and exposure to cold prior to entering water, occur and cause near drowning or drowning in good swimmers.

Narratives of Drowning

Much can be learned from cases of near drowning with regard to events beforehand and sensation during. Narratives often sound convincing and others seem influenced by vivid imagination.

One considered reliable is that of Lowson (8), a medical man, who when about to drown, "breathed in" when apnoea became intolerable. He immediately swallowed the water and this occurred about ten times. This gave him some relief which he suggested might have been due to the sedative effects of CO_2 increase. Then he became unconscious, but recovered on the surface and after a few breaths swam to shore, where he vomited copious amounts of water. Later there were no clinical signs or symptoms that he had inhaled water into his lungs. What is difficult to understand is that, once having lost consciousness in the water, he should recover on the surface.

He no doubt owes his life to a brisk laryngeal reflex. It would appear likely that if such a victim remained submerged the reflex closure of the glottis would cease and water would be aspirated.

A convincingly written account occurs in the medical literature (9) about a youth who nearly drowned. He described a calm feeling of tranquility after exertion ceased, and no longer thought of drowning as evil or of being rescued nor did he feel pain. The outstanding events of his life were presented to him as a "panoramic view." This published report led to reports from many more rescued victims of having "been to heaven." All reported that when struggling ceased their experience was pleasurable and painless. By speculation, the fortunate near-drowned are prevented from inhaling water by a glottic spasm and by having an increase in CO_2. The associated anoxia perhaps leads to flashbacks of memory. Glottic spasm conceivably may account for the bulk of survivals in cases of prolonged submersion.

Miles (1a) in 1962 reports on an article by Dr. Cullen in 1894 who attended a lady that nearly drowned. This report was lacking in any colorful imaginery. The lady went with her husband and sister

to the sea during a fog. She was swimming near the shore and put her feet down to stand up, but was sucked under the surface by a strong current and sank into a hole. She swallowed water but got her mouth above the surface of the water with difficulty. Not being able to overcome the strong swirling of water she shouted for help before going down. She experienced panic and fear. As she sank she gasped involuntarily and immediately experienced a scorching pain associated with the salt water entering her lungs. She felt burning suffocation, suffering, and had an intense desire that someone had noticed her difficulty. No fear of death, desire to be saved, thought of children, or dreams were experienced by her. Not more than three minutes elapsed from when she first sank, to when her husband, who had heard her cry, rescued her and used vigorous means to revive her.

Drowning Without Inhalation

Function of Epiglottis

Any water which enters the victims mouth while struggling during submersion initiates a reflex that causes the epiglottis to close over the

glottis to prevent aspiration of water. Many sensitive areas in the back of the pharynx and upper trachea when irritated initiate this reflex. The afferent limb of the reflex passes mainly through the nineth nerve into the medulla oblongata and the efferent limb passes mainly through the vagus to the muscle of the epiglottis. Therefore, the victim either coughs the water out or swallows it into the stomach without aspiration into the lungs (10a).

Control of Respiration

Without inhalation of oxygen due to apnoea the CO_2 content increases, acidity of plasma occurs, and O_2 content decreases. The victim will experience intolerable apnoea and have violent inspiratory efforts. This is due to the action of:

1) CO_2 and acidity directly on the respiratory center,
2) O_2 stimulating the carotid and aortic chemoreceptors with afferent impulses passing to the medulla via the vagi,
3) the emotion factor, i.e. fear, causes neurogenic stimulation (10b) (11a).

Obstructive Asphyxia

This occurs when free breathing is prevented

and therefore no pulmonary exchange of gases occurs as in fulminating (12a), a cute (12b), progressive (12c) or carbon monoxide poisoning (12d) anoxia. It is simulated by strangulation or drowning without aspiration of water into the lungs. It isn't similar to suffocation as occurs in a fire.

Swann (12e) in 1949 made the following observations in experiments on dogs which were not anesthetised. Survival lasts for about eight minutes due to absence of "blowing off" of O_2 and its retention in an inflated lung. The pH becomes exceedingly low, below the isoelectric point for body proteins. This is due to a change in the acid-base balance with first an acidosis of CO_2 excess and then a metabolic acidosis of increased lactic acid. The CO_2 content and blood bicarbonate first increases due to lack of "blow off" and then decreases as increased lactic acid displaces the weaker acid from its base. The CO_2 is probably driven from the blood into the tissues. The heart begins an early strong bradycardia before very much anoxia occurs and tachycardia does not occur until terminally with circulatory failure.

Percent of Victims

The reflex closure of the glottis is more likely to occur and last longer in victims without excessive exhaustion as for example a non-swimmer falling into deep water. If the victims falls into the water after being stunned or while intoxicated with a respiratory center depressed, violent respiratory efforts are less likely to take place (1a).

Glaister (20) in 1942 and Smith and Fiddles (21) in 1949 described asphyxial death due to submersion in water. Otter (22) in 1960 felt laryngospasm in human drowning occurred for "some short time," while Redding (23) in 1961 concluded this must occur for a "prolonged time." The reflex probably doesn't prevent final aspiration of water into the lungs in most victims because, with severe hypoxia and hypercapnia, this reflex is lost and the victim takes a "dying gasp" with flooding of the lungs.

Drowning with Aspiration

General For Both Fresh and Salt Water

Percent of Victims

As discussed under drowning without aspiration this probably accounts for approximately 85-90% of

all victims.

Inhalation of Water into Lungs

Miles (1a) in 1962 described the sensation of inhalation of water as possibly being an agonizing scorching pain similar to the pipe smoker who normally does not inhale his smoke and inadvertently does so. With first aspirations of water, glottic spasm may prevent immediate flooding of the lungs as discussed under drowning without aspiration. After submersion, apnoea soon becomes intolerable and animals swallow water violently with high negative intrathoracic pressures occurring. Often the initial swallowing of water into the stomach during glottis spasm is immediately followed by vomiting. Then in the absence of glottis spasm, inhalation of water into the lungs occurs in a series of gasps. These involuntary inspirations usually draw large quantities of water into the lungs. The time at which this occurs depends partly on how long the animal can hold its breath. Swann (24) in 1951 found this occurring from zero to four minutes after submersion with two minutes the average. Donald (25) in 1955 with a review of the literature concluded this occurred in one to two minutes. Redding (26) in 1961 gave no definite time for preceding events, but

stated all happened in rapid sequence.

Lung

Pathology

Pulmonary Edema

Cot (14) in 1931 believed the high protein content of the fluid in the lungs suggested a fulminating pulmonary edema in victims who aspirated water into their lungs and that victim drowned in own fluid. However, Swann (24) in 1951 believed dog experiments revealed this a secondary phenomena, also occurring simultaneously with absorption of water or diffusion of salt. With aspiration of small quantities of water instead of large volumes as above, Halmagyi (26) in 1961 concluded that severe pulmonary damage could occur without pulmonary edema. Findings by Pattle (27) in 1958 suggested the presence of protein contained in the froth of the airways could not be regarded as good evidence of pulmonary edema.

Changes in Compliance and Results

The immediate gross decrease in compliance will occur if there is wide spread distribution of a small amount of water. This is due to the resulting widespread closure of terminal airways with a

consequence of decreasing the number of distensible units participating in volume exchange of gases. The loss of compliance occurred without a change in the lung volume or in the end expiratory transpulmonary pressure. The final result is an increasing venous admixture even if the lungs can be ventilated with 100% O_2. In salt water drowning, compliance continues to decrease as more fluid accumulates in the lungs (26) and is unchanged with fresh water.

The above were the conclusions of Colebatch and Halmagyi (28) in 1961, and the following is an elaboration. It is important to remember that this concerns aspiration of a small amount of water and not a large amount which is associated with obstructive asphyxia of the trachea.

The stability of the terminal airspaces depends on a low surface tension material lining them (27), but a film of water with a high surface tension causes their immediate collapse. If this is widespread, a gross fall in compliance occurs with a small amount of water. Persistent closure of the airspaces after absorption of the water was found to be due in part to a vagal reflex from abundant

local receptors in the epithelium lining the airspaces found by Gaylor (29) in 1934. Bronchospasm as opposed to airspace closure did not play an important part.

The results of the airspace collapse are:

1) A gross increase in the elastic work of breathing with associated dyspnea of fluid aspiration described by Mendelson (30) in 1946.
2) Increase venous admixture due to blood flow through collapsed areas rather than uneven ventilation or grossly impaired diffusion. Any compensation by redistribution of blood flow away from the hypoxic area of the lungs found by Rahn and others (27) in 1953 is limited and arteriol oxygen saturation decreases at an accelerated rate.

The low surface tension in usual pulmonary edema, which causes foam formation with exudate, is in marked contrast to fresh water aspiration, though it may occur secondarily with salt water.

Pulmonary Hypertension

This concerns aspiration of a small amount of water and is reported by Halmagyi and Colebatch (31)

in 1961. Significant degree of this was produced by arteriolar constriction in the lungs due to penetration of the fluid into the terminal airways and caused by a mechanism unknown. Hypoxia was eliminated as a cause because no relation was found between vaso-constriction and changes in arterial or venous O_2 saturation.

Cardiovascular Changes

Hypoxia (31 as above)

This wasn't observed if fluid was present in one lobe only. However, with fluid throughout the lung a precepitous fall and prolonged depression was not observed to be associated with the amount of water or speed of recovery as determined by the rate of absorption of fluid. Recovery did not vary markedly between fresh or salt water. The cause was the collapsed airspaces discussed earlier under cahnges in compliance. The hypoxia produced was near or beyond the limit of tolerance for conscious state demonstrated by Lassen (32) in 1959 to be a cerebral venous O_2 pressure of 15 to 25 mm Hg.

Myocardial Impairment (31 as above)

The right ventricular work as especially marked following inhalation of large amounts of fluid

by increased: 1) mean pulmonary arterial pressure, 2) pulmonary blood flow, 3) cardiac output, and 4) hypoxemia. The pulmonary arterial constriction may offer a satisfactory explanation as to why the right ventricle is dilated in victims of drowning as opposed to the left ventricle dilation characteristic of suffocation. Martin (33) in 1932 felt that aspiration of water mechanically blocked pulmonary circulation and increased the work of an anoxic myocardium.

Drowning with Inhalation of Fresh Water

Summary

The sequence of events are as follows:

1) Aspiration of water into the lungs due to gasping from apnoea after about one minute of obstructive asphyxia from glottis spasm and/or breath holding.
2) Hemodilution of the pulmonary venous blood within about two minutes by diffusion of hypotonic water through the alveolar tissue and capillary wall.
3) Simultaneous damage to the alveolar tissue and capillaries with RBC into the alveoli and pulmonary arteriolar contraction.

4) Diffusion of proteins and electrolytes from the blood into the hypotonic water remaining in the alveoli at same time as hemodilution. Therefore, the specific gravity of the blood is decreased in association with 2) to 3).

5) Water shifts between the intravascular, extracellular and intracellular compartments locally and peripherally.

6) Hemolysis of RBC osmotically with release of Hb and K^+ into the plasma in association with hemodilution.

7) Anoxia due to lack of O_2 from obstructive asphyxia and/or collapse of the airspaces from aspirated water.

8) Acidosis due first to respiratory acidity from increased CO_2 and then to metabolic acidity from increased lactic acid.

9) An abrupt slowing of the pulse with a wide pulse pressure followed by sudden onset of ventricular fibrillation within three to four minutes of submersion.

This is the result of:

1) the relative decreased Na^+ and increased K^+,

2) the anoxic myocardium,

3) the increased circulatory load on the heart and,

4) the acidosis.

Summaries found in the literature contributing to the above are: Swann and others (13) in 1947, Swann and Brucer (12) in 1949, Swann and Spafford (24) in 1951, Donald (4) in 1955, Redding (52) in 1960, Redding (23) in 1961, Otter (22) in 1961, and Miles (1a) in 1962. See figures 1 and 2 for experimental physiologic changes.

Research

Pulmonary

Aspiration of Chemical Tracers: dyes, bismuth salts, ferricyanide were used in drowning animals and found to penetrate deep into the lungs, even to complete inundation of all alveoli by Martin (33) in 1932. Swann (24) in 1951 used D_2O in water and found it appeared rapidly in the dogs blood.

Hemodilution: Colin (35) in 1873 demonstrated that the pulmonary epithelium had an enormous capacity to absorb water. Brouardel and Vibert (36) in 1880 experimented with the esophagus tied off to prevent water entering the stomach with absorption.

They found absorption of water occurred primarily into the lungs and caused dilution of the blood. Karpovitch (37) in 1933 demonstrated the same fact. Hemodilution was revealed to be a function of hypotonic water diffusing into isotonic blood because of a difference in osmotic pressure by Moritz (17b) in 1944. At this time also, he found the blood in the left heart more dilute than right heart blood and that the Hb, Cl^-, K^+ and Na^+ were diluted. Swann (14) in 1947 proved the blood volume enormously increases with a fall in specific gravity. He determined the average amount of water absorbed in dogs by D_2O dilution of blood to be 50 ml of water for each 100 ml of circulating blood within three to four minutes after submersion in 1951; this may reach 50% (24). At the same time a total of 1,000 ml was absorbed, but a large amount entered the intersitial spaces.

Pulmonary Edema: Swann (24) in 1951 found lung fluids to contain essentially dilute serum with a considerable quantity of protein showing that pulmonary edema occurs. However, Halmagyi (26) in 1961 felt it could safely be concluded that severe functional or structural damage of alveolar

walls and pulmonary capillaries could occur by a quantity of water insufficient to be associated with pulmonary edema. There can be a rapid fall in plasma proteins due to hemodilution, and diffusion into the alveoli if aspiration of large volumes of water occur according to work of Spitz (38) in 1961.

Blood

Electrolyte Changes: Swann (24) in 1951 found electrolyte changes in dogs corresponded to Moritz's findings of electrolyte changes in humans (17b) in 1944. Swann also found changes uniform due to hemodilution except for K^+ which will be explained later. See pages 19, 20 and 21. The post mortem blood levels of serum salts was nothing short of disastrous with 1/3 normal concentration present. Even with relatively dry lungs, the decrease in salts was in the lethal range of 77-85 mg./L for Na^+. A decrease in NaCl with associated increase in the Mg^{++} and K^+ was found in humans to be greater on the left side of the heart than on the right side by Bowden (39) in 1957. They were not consistent, however, and could represent post mortem change to be discussed later. See pages 31 and 32.

Hemolysis and Fatal Electrolyte Change: Swann (14) in 1947 reported hemolysis lead to a plasma Hb of 2.8% and that this caused a terminal increase in plasma proteins after the initial decrease due to hemodilution and diffusion into alveoli. Spitz (38) in 1961 confirmed this terminal rise as due to Hb. Swann (19) in 1956 stated Hb in plasma may reach 6mg% in dogs. Bowden (39) in 1957 found increased Hb but described it as happening very soon in contrast to Swann who described it as a terminal event. Rath (40) in 1954 reported a case of hemoglobinemia and hemoglobinuria in a 15 year old boy during recovery from aspiration of fresh water into the lungs.

Swann (24) in 1951 from dog experiments, hypothesized that hemolysis was due to anoxia and hemodilution. Gordon (41) in 1951 caused hemolysis by injection of water into anoxic animals. Swann (24) in 1951 also hypothesized that K^+ release from RBC was most important electrolyte change leading to ventricular fibrillation. Bernstein (42) in 1954, however, demonstrated that in the dog the main intracellular cation was Na^+ and not K^+ as in man. Swann (43) in 1951 had already begun

to change his mind and decided that in dogs the drastic decrease in Na^+ due to hemodilution was the most important fatal electrolyte change. Potassium increase in that occurred was then thought by Swann (19) to be slight and the result of anoxia alone as Fenn (44) in 1940 had found. Swann (19) in 1956 described the drastic decrease of Na^+ in dogs as the main fatal electrolyte change and not hemolysis releasing K^+. Bowden (39) in 1957 noticed the increased K^+ was negligible and believed this was due to the short time his animals were exposed to anoxia. Otter (22) in 1960 found that the electrolyte change was not necessarily fatal if not associated with anoxia.

This author believes evidence shows that in the dog the drastic Na^+ reduction is the main electrolyte change contributing to death. In humans, however, since K^+ is the main intracellular cation, hemolysis is more important because K^+ is released in large quantities. The other salts, especially Na^+, are mainly decreased in humans by hemodilution. This would result in a marked increase in the ratio of K^+/Na^+ in man. The evidence shows that in dogs Na^+ is drastically reduced without corresponding

drastic reduction in K^+ even though with hemolysis Na^+ is released instead of K^+. This either means the quantity of Na^+ released is not great or K^+ does not diffuse into the alveoli as well as Na^+. Since the current literature read by the author in 1960 and 1961 discusses K^+ from hemolysis in man as the primary factor, this author accepts the quantity of K^+ released by hemolysis as the fatal electrolyte imbalance.

Ventricular Fibrillation

Incidence and time of Onset: Garry (45) in 1924 hypothesized this occurred in dogs due to ionic unbalance of unknown cause. Banting (15) in 1938 found it occurred in his dog experiments "sometimes" and Lougheed (16) in 1939 reported the same. Swann (13) in 1947 hypothesized that it might not occur in dogs with small amounts of aspiration and hemolysis. Swann (24) in 1951 had ventricular fibrillation occur 30 seconds after cardiac standstill occurred at 1½ minutes. Gordon (50) in 1954 had it occur in one to three minutes after submersion of horses, cows and pigs and concluded man's risk of this are just as great. Donald's (4) review in 1955 stated fibrillation

may occur three to seven minutes after submersion and time of onset not closely related to degree of hemodilution. Spitz (38) in 1961 had fibrillation occur at 5 and 20 minutes in only two of eight dogs. This author notes a difference in procedure of this investigation as cause for variance of incidence and time of onset. The variables seem mainly to be the amount of hemodilution and anoxia with higher incidence and more rapid onset occurring when variables are greater.

Contributory Factors: Refer to, "Hemolysis and Fatal Electrolyte Change" p. 20 in this thesis for discussion of change in K^+/Na^+ ratio. The research concludes it is the change in this ratio that is important with the decrease in Na^+ the probable cause in dogs and the increased K^+ the probable cause in man. This became of importance when Swann (24) in 1951 found his EKG tracings on dogs were like Nahium and Hoff's (46) in 1939 caused by K^+ poisoning. EKG like K^+ poisoning was produced by Wener (47) in 1949 by saponin injection with resulting hemolysis. Winkler (48) (49) in 1938 and 1939 found fibrillation occurred with a K^+/Na^+ ratio of 0.10 by injection of 15 meg. KCl into blood with 145 meg.

Na^+. The normal K^+/Na^+ ratio in dogs was .035.

Otter (22) in 1960, confirmed evidence that progressive myocardial aschemia associated with large volumes of water causing obstructive asphyxia increases the possibility of fibrillation and with small aspiration the onset and frequency is decreased. Spitz (38) in 1961 had the same results with addition of an overloading of the circulation being a factor.

Respiratory Failure

Swann (19) in 1956 stated animals continued to make gasping movements for as long as two minutes after fibrillation which in man would deceive a would be rescurer as victims for practical purposes are dead from circulatory failure.

Cerebral Death

Swann (51) in 1953 stated cerebral tissues sensitivity to O_2 lack is such that an attempt at rescue after more than four minutes of anoxia has persisted is senseless.

Drowning With Inhalation of Salt Water

Summary

Refer to p. 15 for summary of fresh water drowning. The sequence of events are as follows:

1) Aspiration of water into the lungs due to gasping from apnoea after about two minutes of obstructive asphyxia from glottis spasm and/or breath holding. There then occurs a transient apnoea before more inhalation occurs due to vagal stimulation. Relatively, this is the same as fresh water, but transient apnoea is less.

2) Water from the hypotonic blood diffuses across the pulmonary membrane into the intrapulmonic spaces filled with hypertonic sea water due to the osmoloaity gradient. The blood is therefore concentrated with an increased specific gravity. This is opposite of fresh water with associated hemodilution where water leaves alveoli and enters the blood instead of hemoconcentration.

3) There is no diffusion or loss of RBC into the sea water in the alveoli as with fresh water.

4) Diffusion of proteins into the sea water independently from movement of water and salts.

The proteins diffuse in the same direction as in fresh water but to a greater extent.

5) Salts i.e., Na^+, Cl^- and Mg^{++}, leave the alveoli and enter the blood. Therefore, hypernatremia occurs due to this and 2).
6) Water shifts between intracellular, extra cellular and intravascular compartments locally and peripherally. This is in the opposite direction from fresh water where overhydration occurs instead of dehydration.
7) Hemolysis of RBC osmotically with release of Hb and K^+ into the plasma doesn't occur as in fresh water, since there is no hemodilution.
8) Anoxia due to lack of O_2 from obstructive asphysia and/or collapse of the airspaces from aspirated water occurs as in fresh water.
9) Acidosis due first to respiratory acidity from increased CO_2 and the metabolic acidity from increased lactic acid occurs as with fresh water. However, the fixed base, i.e. Na^+ is greatly increased here instead of

decreased as in fresh water.

10) A gradual slowing of the pulse, a severe atrial hypertension with little increase in the pulse pressure, and a gradual decrease in both systolic and diastolic blood pressures slowly fades away till asystoli occurs from asyphxia and acidosis in about eight minutes after submersion. There is no wide pulse pressure and no abrupt loss of blood pressure as would occur with ventricular fibrillation in fresh water drowning.

11) The respiratory movements usually stop sometime before the pulse is lost. This is in contrast to fresh water where gasping may continue for two minutes after death.

Summaries found in the literature contributing to the above are: Swann (13) in 1947, Donald (4) in 1955, Redding (52) in 1960, Redding (23) in 1961 and Miles (1a) in 1962. See figures 3 and 4 for physiology of salt water drowning.

Research

Pulmonary

Hemoconcentration: Moritz (17b) in 1944 found right heart blood to be more dilute than left heart blood and concluded isotonic blood lost its water into the alveolar spaces filled with hypertonic sea water. Swann (13) in 1947 found this to be rapid and violent occuring in two to three minutes after submersion. He gave the figure 0.26 of the total volume of water in the victims blood as removed into the sea water in the lungs. This was confirmed again by Swann (24) in 1951 with the use of D_2O injected into the blood before aspiration and finding it in the sea water of the lungs afterwards. Another good estimate was provided by $SO_4^{=}$ ion which the pulmonary membrane is relatively impermiable to. Swann (24) in 1951 calculated from this that 150ml or 15ml/kg body weight of water leaves the blood. This would correspond to approximately 1 liter in a 70 Kg man or 20 to 25% of his blood volume.

Pulmonary Edema: Cot(14) in 1931 felt fulminating pulmonary edema occurred because lung fluid was rich in protein. Swann (24) in 1951 found 2 to 4 mg% of serum proteins in lung fluid substantiating Cot's impression and described this as due to

osmotic dilution of sea water which is four times as concentrated as blood. Because of this a rapid edema develops by the same mechanism which creates hemoconcentration. Therefore, water is added to that already in the lungs (14) and (24). Redding (23) and Halymagyi (26) in 1961 mention the same pulmonary edema. Swann (24) in 1951 stated edema does not account for all the lung fluid since it is always to dilute with respect to one or the other components.

Electrolyte Changes In the Blood

Jetter (53) in 1943 concluded salts of sea water passed into the blood since large quantities of Mg (15.3mg% or 19 times normal) was found in the plasma. Swann (13) in 1947 found with dogs that Cl^- increased in the left heart and in 1951 (24) he found salts in the blood very high with N^+ in excess of 200 meg/L. Fulton (54) in 1946 had concluded that Na^+ of this concentration was incompatible with life. Swann (24) in 1951 states in dogs that all salts of the sea water passed into blood equally well except $SO_4^=$ which the alveolar capillary membrane was relatively impermeable to. Where he excepted 12 meg./L he gound only 1.5meg/L. This wasn't attributed to

precipitation in blood as $CaSO_4$, since the amount of $SO_4^{=}$ in the sea water could precipitate all the Ca^{++} in the blood and still be greatly increased. The impermeability of the pulmonary membrane to $SO_4^{=}$ is unexpected because most capillary walls are quite permeable to this ion and it has been used by Lavietes (55) in 1936 to study interstitial water. Swann (24) in 1951 also used D_2O in the blood before aspiration to show water of two fluids on each side of pulmonary membrane are evenly distributed. The Na^+, K^+, and Ca^{++} were near equal on each side with the blood's increased HCO_3^- due to CO_2 buildup being neutralized by excess $MgSO_4$ and Cl^- in the lung fluid. Bowden (39) in 1957 also with studies on 5 human victims commented on excess of Na^+, Cl^-, Mg^{++}, and K^+ being due to both hemoconcentration and also passage of these electrolytes from sea water in lungs. He noted that the level of 6 to 14 meg/L of Mg^{++} is considered incompatible with life and that this was reached in four of five cases.

Cardiac Failure

Swann (13) in 1947 found no fibrillation, but that the pulse slowed, the systolic and diastolic

pressure rose with some increase in pulse pressure, and then slowly fell without a decrease in pulse pressure till terminally when it gradually disappeared. The venous pressure was well sustained until cardiac failure and at death it was zero. He confirmed this again in 1951 (24) and stated effective contractions ceased six to eight minutes after submersion. Revival by artifical respiration was certain with systolic blood above 115 mm Hg, but below 50 mm Hg, failure was irrevisible. The time between this was as short as 20 seconds in dogs. Donald's (4) review in 1955 stated heart failure was to sever prolonged myocardial anoxia. Spitz (3) in 1961 postulated successful resuscitation prior to circulatory failure involved reversal of anoxic myocardium rather than peripheral vasodilation.

Respiratory Failure

Swann (24) in 1951 had results in dogs showing this occurs in a high proportion of cases before irreversible circulatory failure. The figures were: 1/3 before, 1/3 with onset, and 1/3 after circulatory failure. Bowden (39) in 1957 had EKG studies showing hearts mascular activity is extremely weak even when it exists for a comparatively long

time after respiratory failure and artifical respiration alone not thought enough to revive.

Autopsy

Biochemical Tests for Drowning

Examination of blood for biochemical changes may be difficult to interpret unless collected very soon after death due to post mortem changes of death. The following is a discussion of different tests that have been tried in the past to prove a person drowned and does not necessarily include tests used in research alone which are scattered throughout the rest of the thesis.

Attempts to distinguish fresh and salt water victims in the past included the use of:

1) Hb content by Brouardel (36) in 1880,
2) RBC by Pattauf (56) in 1892,
3) Hb and RBC concentration compared on right and left side of heart by Brouardel (36) in 1889 and again by Pattauf (56) in 1902,
4) Specific gravity on blood of right and left heart recommended by Placzek (59) in 1903,

5) Cl^- determination on blood in right and left heart recommended by Getter (60) in 1921.

Peripheral lung and stomach content examination for water algae may be extremely useful in cases where cause of drowning is uncertain was suggested by Donald (4) in 1955.

Moritz (17) in 1944 mentioned that concentration of Mg^{++} better than Cl^- in cases of salt water drowning to determine death due to drowning. However, within 6 to 12 hours after ordinary death a progressive fall in the Cl^- and a rise in the Mg^{++} occurs. Therefore, this test must be done early. Getter (60) in 1921 found in salt water drowning the Cl^- level in left heart blood was elevated and higher than right side. In fresh water the Cl^- level in the left heart blood was decreased and lower than the right side. He states a difference of more than 25 mg% between Cl^- levels on left and right sides indicates death due to drowning. However, Durlacher (56) in 1953 found Cl^- levels changed in unpredictable fashion in both drownings and controls and Bowden (39) in 1957 confirmed this. He stated that any cause of natural death may cause change of more than 25 mg%.

Durlacher (56) in 1953 found considerable overlapping of values with no significant difference between groups when he attempted to use: 1) ratios of plasma K^+/Na^+, 2) individual K^+ and Na^+ and, 3) true plasma Hb. They suggested the specific gravity of plasma more reliable than Cl^-. The specific gravity was found less in the left heart blood than in the right heart blood in all types of drowning irrespective of the salinity of the water. In nondrowning deaths vise versa is true.

Appearance

Curry (15) in 1815 observed that water did not enter the lungs and that the drowning patient appeared to have no injury to the structure of any of the essential parts of his body. This is true in this type of drowning where water does not enter the lungs and therefore, this is the type where victim has best chance of recovery. French investigators reviewed by Cot (17) in 1931 were in general agreement that when victims were white or pale little or no water had been aspirated and little or no froth was present in the respiratory passages; death was attributed to "syncope." However, they generally agreed that, if much water

has been aspirated into the lungs, death is due to obstructive asphyxia and the victim is blue or cyanosed.

Reason for Atuopsy and Procedure

Miles (1a) in 1962 wrote that one should makes sure that death was due to drowning if circumstances leave doubt of accident, a post mortem examination should remove it. This is especially true in a diving accident as drowning might only be sequel to another condition. Special attention should be paid to the lungs for signs of: 1) drowning, 2) inflammatory process, 3) obstruction of bronchi and bronchioles, and 4) emphysematous bullae. The heart and great vessels should be examined and special note made of any injury which could have caused unconsciousness.

Lungs

Distention

It is recommended by Miles (1a) in 1962 that the lungs be removed with care to avoid damage and examined. If they pit or show rib compressions, they are distended and have lost their elasticity due to fresh or especially salt water entering the

lungs. Otter (22) in 1960 found when fresh water aspiration occurred in dogs: 1) the whole lung swells markedly, 2) the color changes to pale grey, 3) it is firm to touch, 4) some pink water oozes through the pleura, and 5) the lung sinks in water. The amount of distention of the lung tends to increase when large volumes aspirated and usually is more prominent in sea water but values overlap.

Drainage of Water

The amount of water that could be drained from the lungs by gravity or pressure was not thought by many pathologists to be significant in clearing the airway when Donald (4) reviewed the literature in 1955. The drainage was from the trachea and bronchi only, i.e., the dead air space. Otter (22) in 1960 with dogs drowned in fresh water found that the amount of pinkish watery fluid siphoned off plus pink foam produced by pressure equaled the dead air space. Swann (24) in 1951 with dogs found water removed by gravity usually in excess of amount given in salt water, but some valves for fresh water overlapped. Redding (52) in 1960 with dogs found that a volume equal to or in excess of the salt water administered was

recovered from the lungs by passive drainage depending on length of time dog survived.

Presence of Froth

Swann (24) in 1951 noted a frothy fluid in lungs of dogs which was pink due to hemolysis with fresh water and colorless with salt water. Donald (4) in 1955 with his review stated small airways were obstructed by fine persistent froth and that while the trachea and large bronchi also contained froth they were not obstructed. Bowden (39) in 1957 described froth as typical of drowning which was whitish with salt and reddish with fresh water drowning, but was absent if death occurred before pulmonary edema or without aspiration. He noted this was a formidable barrier to entry of air into circulation and thought drainage or suction should be used. In the past, he also stated that froth was attributed to churning up of water, air, and mucus in air passages during artifical respiration. The new concept presented was that the mucouc exudate from the tissue was due to pulmonary edema and that very fine stable froth was produced without artifical respiration.

This orginated when Swann (24) in 1951 noted serum proteins in lung fluid of salt water especially, but also in lung fluid of fresh water which revealed pulmonary edema.

Miscroscopic

Otter (22) in 1960 did a microscopic exam of lungs of dogs drowned in fresh water. He found: 1) marked exudative edema with protein containing fluid in the alveoles, 2) stasis in dilated capillaries, 3) many erythocytes disintegrated, 4) marked intravascular hemolysis, 5) lixivated RBC in septa and alveoli, and 6) complete disintegration of the alveolar lining in many places. This occurred with both small and large amounts of water and was more marked in the latter. Donald (4) in 1955 found reference to large amounts of fluid within the parenchyma which was greater in salt than fresh water.

Heart

Donald (4) in 1955 noted in his review that with fresh water the right ventricle was dilated and the left ventricle was contracted. Otter (22) in 1960 with a large amount of fresh water found a flabby dilated heart on the right side, but

with a small amount, no dilation was present.

Stomach and Contents

Donald (4) in 1955 noted large amounts of water are found in the stomach due to reflex swallowing. Also, vomitus may be found in the air passages due to agonal vomiting or transfer during artifical respiration. Miles (1a) in 1962 suggested the same and that the presence of large quantities of food and alcohol may be significant, especially if there is also evidence of vomitus in the air passages.

CNS

Donald (4) in 1955 noted that as in other asphyxial deaths small local hemorrhages occur throughtout the body, especially in the CNS. They are less marked in drowning andif they are significant violence should be considered.

Peripheral Interstitial Edema

Otter (22) in 1960 found with large amounts of fresh water in dogs that pinkish fluid was found in: 1) peritoneal cavity, 2) liver, 3) spleen, 4) kidney, 5) retroperitoneal tissue and 6) the mesenterial tissue.

RESUSCITATION

Methods

See table 6 for outline of methods.

Push Pull (active inspiratory and expiratory phase)

In a December Symposium Issue of the J. Appl. Physiol. (61a to j) in 1951, various authors in studies on artifical respiration present evaluations of:

1) the use of barbiturates and curare in normal adults to produce apnoea
2) air flow patterns and pulmonary ventilation of different manual negative pressure methods revealing that all push pull techniques are superior to the Schafer method, i.e. prone pressure, and should be done at the rate of 10 to 12 cycles/minute
3) circulatory system revealing the arterial O_2 saturation is best in push pull techniques, no deleterious cardio dynamic effects result from use of them, and one should be picked to replace the Schafer method
4) energy expended by operators revealing Schafer method as the least taxing and the Silvester, i.e. arm-left chest pressure, or Holger Neilsen, i.e. arm lift back pressure methods as the least taxing of the push pull group.

5) the teachability, i.e. pedogogy, and performance revealing that the Holger Neilson method surpasses all others as the easiest to learn, physically perform, correct and accurately perform, and also is the best all around considering ventilation, fatigue, teachability, and accuracy of performance

6) the pulmonary ventilation revealing that the Schafer method gave 1/2 the ventilation of the push pull methods of which the Holgar Neilsen type is the best

7) energy expended in a second study revealing the Silvester method the least taxing

8) the teachability and fatigue a second time revealing the Holger Neilsen type of the push pull methods is the easiest to learn and do physically

9) the mechanics of breathing revealing push pull methods give better ventilation than Schafer method

10) a comparison of methods revealing the Holgar Neilsen the most practical for long periods of time, but the hip-raising procedures more effective and emphasizing

all push pull methods should be known since one not ideal for all situations.

Mechanical

Both manual and mechanical methods were equally effacious for Fainer (62) in 1951 for resuscitation of dogs drown in fresh water and this was probably due to the frequency of ventricular fibrillation. Handford (63) in 1951 found that alternating positive (+13mm. Hg) and intermittent positive and negative (+13 to -9mm. Hg) pressures were equally effacious in resuscitation of curare apneic dogs, providing heart failure had not begun. Lucas (64) in 1958 studied manual (Holger Neilson) and IPPB by mouth to mouth apparatus (face mask with piece and tube connecting to oronasal mask) or resuscitator (bellows). He found manual methods have the advantage of requiring no apparatus, but require space and are ineffective in bronchoconstriction. The IPPB required apparatus, but was superior with bellows the best apparatus.

Mouth-to-Mouth

General

Johns (66) in 1951 conceived the idea of a mask-to-mask resuscitator suitable for use on nerve gas casualties in a contaminated atmosphere.

Elam (67) in 1954 was an early investigator with mouth-to-mask artifical respiration. Greane (68) in 1957 did research on the expired air resuscitation.

Dill (65a) in 1958 advocated disseminating information about expired air breathing. Gordon (65b) in 1958 after comparative studies of mouth-to-mouth versus manual artifical respiration had results indicating the unequivocal superiority of mouth-to-mouth method. He found it the only technique that assures adequate ventilation in all cases. With manual methods complete obstruction occurred in a significant number of cases and partial obstruction in all cases. The most important factor in assuring ventilation without obstruction was proper extension of the neck and elevation of the jaw.

Elam (65c) in 1957 found reoxygenation of the patients lungs was possible in four inflations, and within a circulation time the arterial O_2 saturation could be restored to normal with concomitant reduction of the CO_2 level. His data refuted the prejudice that exhaled air contains too little O_2 and too much CO_2. A suitable resuscitating gas was obtained by mild hyperventilation and the method could be maintained for prolonged periods

without undue fatigue or hypocopneic symptoms. The advantages of this technique were concluded to be:

1) resistance is sensed if airway becomes obstructed,
2) gurging is heard and felt if secretions accumulate,
3) expansion is visible during inflation,
4) spontaneous breathing can be felt,
5) both hands are available to support jaw and clean airway.

Safar (65d) in 1957 found with 164 untrained rescuers that 90% performed the technique satisfactorily after one demonstration and that disportion in size of rescuer and victim did not hinder the proficient application of the method.

Techniques

Early

Early techniques of: 1) pulling on the jaw with the thumb in the mouth for mouth-to-mouth or 2) pushing upward at the angles of the jaw for mouth-to-nose were recommended by Gordon (65a) in 1958. These presented problems in performance and teaching.

A summary of the difficulties of performance

from Elam (69) in 1960 is as follows:

1) Leakage around the rescuers thumb in the victims mouth is often excessive.
2) Inflation of the relaxed victims stomach during both methods had been a frequent complication, aggravating passive regurgitation of stomach contents.
3) Trismus had complicated or prevented inflation through the victims mouth.
4) Both methods were fatiguing for the rescuer's hands.
5) Insertion of the thumb in the victim's mouth through sharp teeth was hazardous, especially in near conscious victims.

A summary of the difficulties of teaching is as follows: (69)

1) Pulling on the jaw with the thumb or pushing upward at the angles of the jaw involves numerous fine details in technique which cannot be learned by some layman.
2) The modifications for infants and children are different from procedures used on adults.
3) Insertion of the thumb in the mouth during

practice is frequently distasteful.

4) Both methods are impractical and often impossible to demonstrate and practice effectively on conscious volunteers.

5) The instructor cannot evaluate whether trainees have learned these procedures.

Head Tilt

The advantages especially with mouth-to-nose method are as follows: (69) and Buchanan in 1960 (70)

1) Gastric inflation is negligible or absent.

2) Leakage is no problem because with use of nose the mouth is held closed and with use of the mouth the rescuer presses his cheek against the victim's nostrils.

3) Despite trismus, the head can be tilted which opens the nasopharyngeal and glottic air passages.

4) The method makes it easy to evaluate the performance.

5) The method is easy to teach and practice in a few minutes with a minimum of discussion of the anatomy of the air passages.

6) It is easy to remember the simple steps of tilting the head back as far as possible and inflating the lungs through to mouth or nose.

The instructions for the head-tilt oral resuscitation are as follows: (69) See figure 3

1) Lift the neck.
2) Tilt the head as far back as possible by holding the crown of the head with one hand, which when sufficient usually opens the mouth. The reader can try this on himself and note the result.
3) Pull the chin upward with the other hand. X-ray studies reveal this action produces a wider open air passage than that of normal conscious subjects. Note the similarity of procedures 2) and 3) to that used by the anesthesiologists in the administration of gas anesthesia.
4) Inflate the lungs through the nose or mouth or, in an infant, through both.
5) Remove your mouth to let the victim exhale passively and if necessary, let him exhale through his mouth by momentarily separating his lips.

Additional information is as follows: (69) (70)

1) The mouth is used by separating the lips of the victim by using your thumb.

This is done when the nasal canals are blocked.

2) In babies, be careful not to encroach on the air passages when pulling the chin upward.

3) The semilateral position with the victim's shoulder resting continuously on the rescuer's knee can be used to tilt the head to provide gravity drainage of fluid from the pharynx in drowning.

4) Small victims should be inverted and sharp blows applied between the shoulder blades to help dislodge any foreign body.

Oral pharyngeal airways (69) (70)

Specialized equipment, such as artifical airway, should be used only by persons trained in a) the management of the airway passages and b) artifical ventilation of the unconcious patient. The physician should remember that the casual rescuer, housewifes or passers-by initiates 2/3 of all resuscitations according to records of 1957-1958. They cannot diagnose with certainty the level of reflex response in an unconscious patient and may attempt procedure on any unconscious patient

which would be traumatic. The hazards of instrumentation of the throat in a semiconscious victim with an active gag reflex are laryngospasm or vomiting with aspiration. Unlike the anesthetized patient a asphyxia victim is not likely to have 1) an empty stomach, 2) belladonna to diminish secretions and vagal reflex, 3) depression from anesthetic drugs, and 4) curare to be paralyzed sufficiently to prevent retching and vomiting.

Dangers (60)

As long as the rib cage of the lung remains intact, the lung cannot be burst, but a vigorous adult could produce a localized tear in a lung congested by water inhalation with some bleeding or surgical emphysema. This is more likely in a child and care needs to be taken. Infection to the rescuer would be no greater danger than kissing, since he doesn't inhale the victims air. The victim would certainly rather risk the danger of an infection than the certainty of death and this is the most efficient method of resuscitation. However, practising this method on a large scale presents a danger of infection and possibly death. Therefore, practice masks have been developed

which allow no entrance of air into the lung, but simulate actual conditions of victim for pupils learning. Also, life size models have been developed for instruction purposes,but are much more expensive. Films are more successful method of teaching adjunct in this method than in the more complicated push-pull methods of artifical respiration.

Urgency of Administration (4) (70)

Donald (4) in 1955 and Buchanan (70) in 1960 emphasized the urgency of not wasting a second while clearing the airway, loosening clothes, examining the patient, draining the lungs, or selecting a slope which would result in a delay which could very likely be fatal. Therefore, this also applies while towing a patient ashore. Herein, lies another advantage of the mouth-to-mouth method, since it can be given in the water. This is a time when three or four breaths, could be much more important in providing survival than later on shore when more suitable circumstances are available for artifical respiration. Buchanan (70) discusses the method to use in chest high water and while towing the victim to shore. It is not the purpose of this paper to discuss rescue techniques and therefore,

no detail will be presented. However, the author feels this can definitely be done by life saving personnel since he is a former competitive swimmer, life guard and water safety instructor.

Research On Additional Methods Of Treatment

See table 7

Redding (52) and (71) in 1960 and (23) in 1961 did a series of experiments with dogs on intermittent positive pressure breathing with air and O_2. This was done with or without associated administration of external cardiac massage, plasma and defibrillation where needed in the three different types of drowning for successful resuscitation. The conclusions for treatment were gained through well organized experiments and are summarized in table 5. Colebatch (28) in 1961 used sheep to study IPPB with air and O_2 as a method of resuscitation for aspiration of small quantities of fresh and salt water. This proved highly effective. This results complemented Reddings and revealed more strenuous methods of resuscitation as CCCM, i.e. closed chest cardiac massage, defibrillation and plasma in appropriate condition may not be needed if victim does not inhale a large quantity of water.

External Cardiac Massage (59)

In the literature, the reader will find another term, closed chest cardiac massage (CCCM) which refers to the same method.

Jude (72) in 1961 published the following material on CCCM involving 138 episodes in 118 patients at John Hopkins Hospital over a 2½ year period. The author also attended a lecture given by him at Omaha Mutual in the fall of 1962.

Anatomy and Physiology

See figure 4

The effectiveness of CCCM is based on the fact that the heart fills most of the space between the lower sternum and the thoracic spine. The heart is restricted from lateral motion by the pericardium. It is compressed during rhythmic downward thrusts of the lower sternum. With exact placement of pressure on the lower sternum the relaxed thorax gives at the costochondral and costosternal junctions. Some motion occurs in the ribs themselves and in the costovertebral ligaments. With 60-80 intermittent compressions per minute, adequate circulation can be maintained to sustain the viability of the brain and other vital organs.

The negative intrathoracic pressure created between compression by the resilent expansion of the intact thorax enhances the cardiac venous filling phrase. Although pressure is exerted on venous system with each compression, the resultant pressure difference is forward: because of the heart valves.

Steps See figure 5

1) Rapid Diagnosis

There is no need of 100 % proof that arrest is present as this method, if properly applied, is not dangerous. In 15-20 seconds the diagnosis can be made by checking the major pulses, percordial heart beat, pupillary constriction, and respiration.

2) Artifical Ventilation

Use mouth-to-mouth by stopping CCCM 12 times a minute.

3) Artifical Circulation

Place the heal of one hand on the lower 1/3 of the sternum parallel to it and push the sternum down 1½ to 2 inches 60-80 times per minute. Do this till a O_2 plateau has been reached to protect the CNS. Then apply IPPB with 100% O_2 and complete rest of treatment.

4) Drug Therapy

Epinephrine 0.5 mg IV or I^C is given as soon as possible. CaCl or Ca gluconate can be given, if weak cardiac action returns again, in dose of ½ or 1gm of 10% solution IV or IC. Na HCO_3 44 meg. (3.75gm) IV every 5-10 minutes in adults (½ dose in children) maintains blood pH, improves cardiac action, and response to vasopressors is better. Continuous infusion of aramine or levophed may be necessary. Quinidine gluconate or procaine amidi can be used in cases difficult to defibrillate or maintain.

5) EKG

Obtained to confirm type of arrest.

6) Defibrillation

Use 440 volts of A.C. applied along the long axis of the heart for 0.25 seconds. This is not detrimental to the normal beating heart and therefore, try defibrillation without positive EKG diagnosis if can not get it quickly.

7) Post Resuscitation Care

EKG is monitored in intensive care with continuous cardiovascular and respiratory support as necessary. If CNS damage evident, then give hypothermia of 32-24°C. and maintain 72 hours.

Criteria of Effectiveness of CCCM

Color improves, pupils contract, spontaneous gasping, swallowing, movements of the extremities occur and BP is in the range of 90-150 mm Hg systolic.

Criteria for Cessation of Efforts of CCCM

Success occurs when strong pulse spontaneously begins and BP is 70 mm Hg or greater. Artifical respiration is continued till patient does not tire.

Failure occurs when absence of EKG activity continues for one hour, dilated fixed pupils remain, and gasping respirations are not present.

Summary of Rescue (1a)

1) Immediately start artifical respiration. This can be done in the water by mouth-to-mouth method. Do not waste time choosing a site on shore. Rapidly scoop out any obvious obstruction in the mouth with a finger or handkerchief. Continue artifical respiration (AR) uninterrupted for 15 minutes, when a short pause may be taken to assess position and rearrange the victim. In absence of medical opinion, continue AR for one hour or more, even though the outcome usually is established by 15 minutes. There may be no other reason for this than presence of relatives who refuse to give up hope.

2) While AR is being administered, other bystanders can be employed to summon professional assistance, gently remove clothes, and cover the victim to keep him warm.

3) If signs of respiration return, adjust AR so that its rhythm is in phase with respiration of patient and continue until natural breathing is fully established.

4) Handle patient at all times with care.

5) If heart fails, no radial or carotid pulse and no heart beat, then due CCCM.

6) Transfer with a minimum of delay to hospital for chest X-ray, O_2, IV plasma, hypothermia, and defibrillation if necessary.

Summary

Reason for Topic

This thesis topic was chosen because of: 1) the author's past history of competitive swimming, life guarding, instructor of swimming and life saving; and 2) his future role of a physician with a desire to understand a subject medically that he has had experience with as a lay person.

Introduction

Incidence

In the United States 6,900 drownings occurred in 1961, which represents the fourth cause of accidental death. It is a distressing cause of death because it frequently occurs in the young child and adults at work and play. See tables 1 and 2.

Investigation of Accidents

With advanced methods of treatment, records become increasing important to determine the effectiveness, applicability, and means of improving resuscitation. See table 3.

Pathophysiology

Types of drowning discussed were:

a. Obstructive asphyxia due to reflex closure of epiglottis without aspiration of water.

b. Drowning with aspiration of fresh water

c. Drowning with aspiration of salt water

Aspiration Drowning

This was discussed generally as to the lung pathology resulting in pulmonary edema, decrease in compliance, pulmonary hypertension, hypoxia and myocardial impairment.

Fresh Water Drowning

See summary on page 15 for sequence of events which are mainly:

1) Hemodilution of blood by aspirated hypotonic water in alveoli.

2) Diffusion of proteins and electrolytes from blood into water in the alveoli.

3) Hemolysis of RBC with release of K^+

4) Death due to sudden ventricular fibrillation within 3-4 minutes due to increase in K^+/Na^+ ratio, anoxia, increase circulatory load, and acidosis.

A review of the literature follows which established this sequence of events. See figure 1 for physiology at the alveolus and table 4 for classic changes in the cardio-respiratory system and blood.

Salt Water Drowning

See summary on page 24 for sequence of events which are mainly:

1) Hemoconcentration of the blood due to diffusion of water into the hypertonic salt water in alveoli.

2) Diffusion of proteins independently into the sea water with pulmonary edema in the alveoli.

3) Diffusion of salts from sea water into the blood.

4) Death due to gradual development of asystoli in 8 minutes from asphyxia and acidosis. A review of the research then follows which established this sequence of events. See figure 2 for the physiology at the alveolus and table 5 for the classic changes

in the cardiopulmonary system and blood.

Autopsy Examination

a. Biochemical tests were reviewed which have been used in attempts to prove death by drowning and none found to be confirmatory, absolutely.

b. Findings of the autopsy were:

1) Distention of lungs, especially in fresh water drowning.

2) Drainage of water from lungs includes primarily that in dead air space and the rest is trapped in the alveoli.

3) Froth due primarily to pulmonary edema is pink in fresh water due to hemolysis of RBC.

4) Microscopic damage to alveolar walls and pulmonary capillaries.

5) Comments made also on heart, stomach, CNS, and peripheral edema.

Resuscitation

Research presented concerning following changes in methods artifical respiration:

1) Schafer to Holger-Neilson (61) in 1951.

2) Mask, bellows, and other means IPPB in mid 1950's.

3) Adoption of mouth-to-mouth method. See page 42 for summary of Head-Tilt technique and figure 3 for procedure.

Research presented concerning resuscitation of dogs as follows:

1) without inhalation of water--nothing or IPPB with air

2) Inhalation of sea water--IPPB with O_2, plasma and CCCM as needed.

3) Inhalation of fresh water--IPPB with O_2, CCCM and defibrillation. See table 7.

Use of CCCM (72)

Steps are summarized as follows:

1) rapid diagnosis, 2) artifical respiration, 3) CCCM till CNS protected and then, 4) drug therapy (especially epinephrine IC), 5) EKG, 6) defibrillation if needed, and 7) post resuscitation treatment.

See figure 4 and 5 for anatomy, physiology, position of heel of hand, steps and complications.

Important References

1. Miles (1) 1962 wrote a book with two chapters on drowning and treatment.
2. Guyton (10) 1959 contains the normal physiology.
3. Swann (24) 1951 presents the classic physiology in different types of drowning in dogs which subsequent researchers tried to verify, correct and expand.

4. Redding (23) 1961 presents the research by use of dogs on methods of resuscitation.

5. Symposium on manual methods of negative pressure artificial respiration (61) 1951 where Holger-Neilsen method recommended to replace Schafer.

6. Symposium on mouth-to-mouth Resuscitation (65) 1958 where this recommended to replace Holger-Neilsen method.

7. Elam (69) 1960 presents the Head Tilt technique of mouth-to-mouth resuscitation.

8. Buchanan (70) in 1960 serves as a good review of drowning, resuscitation and application of mouth-to-mouth method during actual water rescue.

9. Jude (72) 1961 presents the method of CCCM.

Conclusions

The work of Swann (24) in 1951 on drowning provided the classic physiology which later researchers sought to confirm and expand the details. See tables 4 and 5. Improved methods of resuscitation were found in the push-pull techniques of which the Holger-Neilsen was recommended to be taught nationally in place of Schafer in 1951 (61). However, even as late as 1955, ventricular fibrillation was considered fatal by Donald (4).

In 1958 (65) mouth-to-mouth resuscitation became the recommended method to replace the Holger-Neilsen. See figure 3. This had several advantages: 1) less tiring, 2) better control of airway, 3) easy to learn, 4) could be done in the water, and 5) later adaptable to external cardiac massage. Numbers 2), 4), and 5) are especially significant, since older methods always were associated with airway obstruction, a few breaths early are much more valuable than resuscitation under more suitable conditions, a few seconds or minutes later, and an attempt can be made to sustain life without spontaneous effective cardiac action. Closed chest cardiac massage, CCCM, technique was perfected and received attention (72) by Jude in 1958-1960 as a possible method of sustaining life till the heart could be defibrillated or the CNS protected till effects of anoxia on myocardium could be overcome and spontaneous beats start. See figures 4 and 5.

In 1961 Redding (23) used the full course of resuscitation, i.e. IPPB, O_2 plasma, CCCM, and defibrillation where indicated, on dogs with effective results. See table 7.

This author believes the methods of administration

of mouth-to-mouth resuscitation in the water, as presented by Buchanan (70) in 1960, can be taught to pupils in all life saving courses. He would also recommend that all life saving and first aid courses include instruction in CCCM. For life saving personnel and rescue squads, simple explanations of the pathophysiology of drowning and its treatment by mouth-to-mouth breathing with or without CCCM, as indicated, should be included as part of instruction. Hospital emergency rooms should be prepared to handle drowning victims as indicated in the thesis.

Skeptism as to the applicability and effectiveness of these methods is warranted, but with the cooperation of lay organizations actively interested in this subject, i.e. the Boy Scouts, Fire Department Rescue Squads, Red Cross and YMCA, a large enough series of cases could be collected to obtain more significant data than is available or ever will be made available by research on animals.

Respectfully submitted,

Dwight F. Rickard M_4

TABLE 1

Drowning and Accidents 1961 (2)

	Totals	Death Rate of Drowning	Ranking of Accidents in Leading Causes of Accidental Death	Death Rate Of Accidents
All Ages	6,900	3.6%	4th	52
Under 1yr.			6th	102
0-4	750	4	1st	31
5-14	1,400	4	1st	19
15-24	1,450	6	1st	56
25-44	1,400	3	2nd	42
45-64	1,100	3	4th	53
65-74	300	3	5th	94
75 & over	150	3	6th	289

Males	5,500	= 85%
Females	900	= 15%

TABLE 2

Causes of Death 1961 (2)

Cause	Death Rate
Heart Disease	363
Cancer	147
Vascular Lesions	108
Accidents	52
Motor Vehicle	21
Falls	11
Fires-burns	4
Drowning	4
Others	12

TABLE 3

Form For Reporting Drowning (1a)

PART I - Incidence Report should consist of:

1. Personal history should include: name, address, sex, age, race, occupation, and relevant medical history, i.e., emphysema, old myocardial infarction, etc.
2. Relevant activities of victim before incidence should include: last meal, activities immediately before going in, how long and activities in water, cause of distress and how noticed.
3. Environmental history should include: time, location, weather, and water conditions, i.e., temperature, fresh or salt water.
4. Details of rescue should include: time lapse, method, condition during and obvious injury.
5. Details of resuscitation should include notes on: general appearance, color, consciousness, breathing, vomitus, frothy secretions, type, duration, and difficulties of resuscitation and victims response.

PART II - Hospital and Medical Report (1a)

1. Chest X-ray if victim lives.

2. Clinic condition of lungs and upper respiratory tract.
3. Clinic condition heart and vascular system.
4. Blood sample for HCT, electrolytes, pCO_2, and pO_2; obtain first two even if patient dies.
5. Post mortem examination if victim dies should be summarized with note on lungs, heart, stomach, evidence of injury, and preceding illness.

TABLE 4

Course of Fresh Water Drowning in Dog (12a)

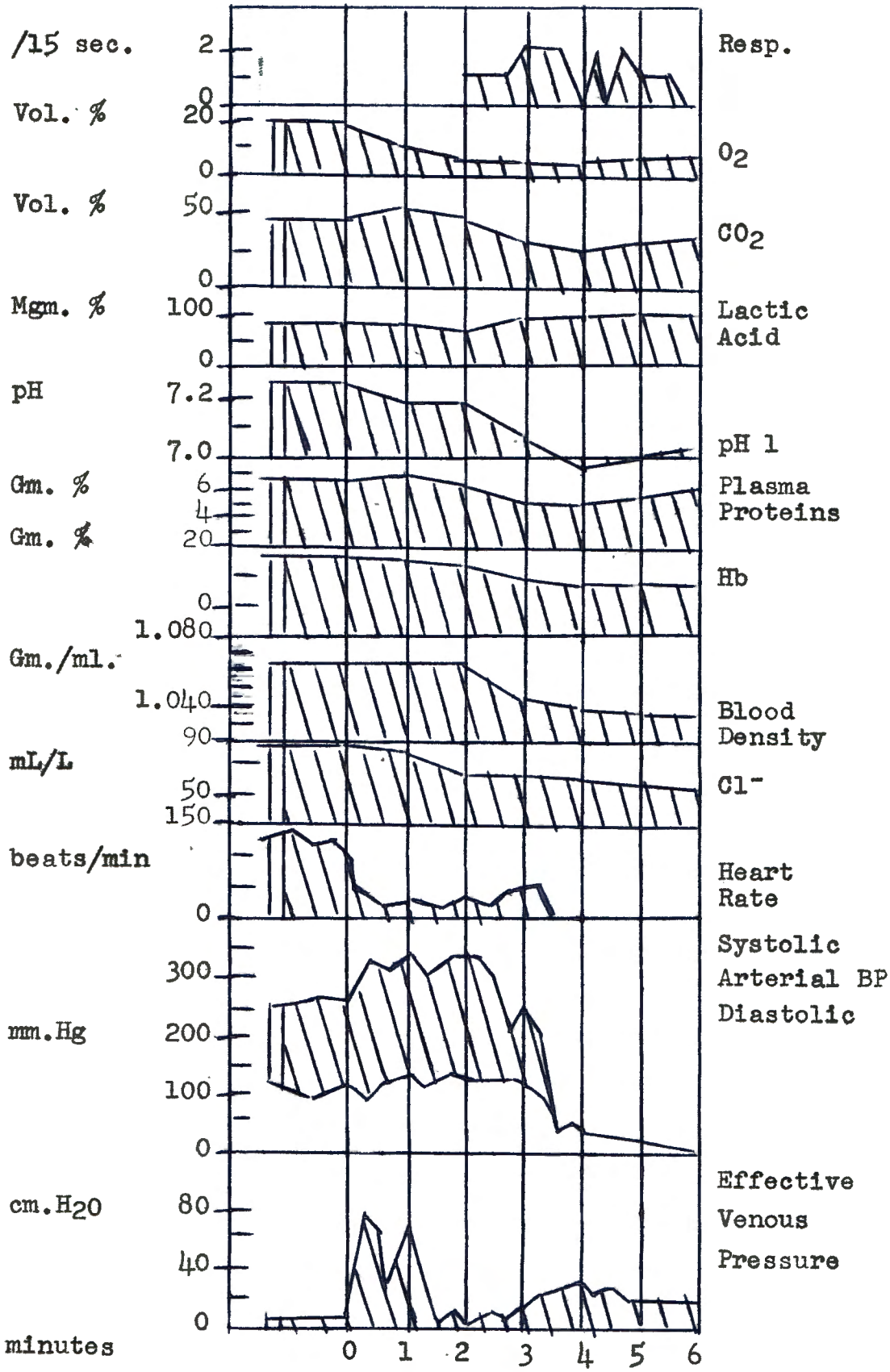


TABLE 5
Course of Sea Water Drowning (12a)

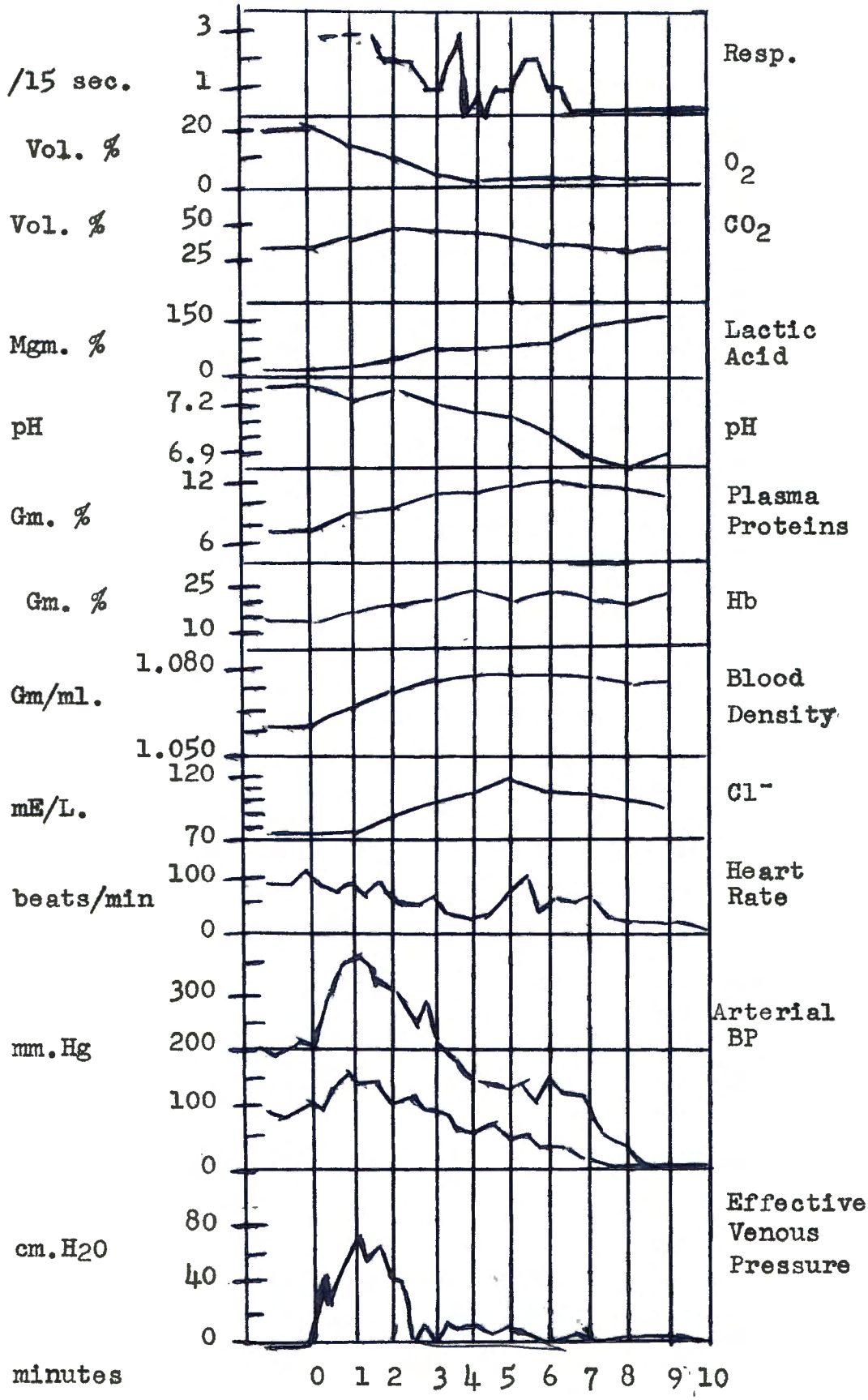


TABLE 6

Methods of Resuscitation (1b) (61b) (10a)

A. Negative Pressure

1. Mechanical

   a. iron lung

   b. chest respirator

2. Rocking

3. Manual

   a. prone pressure, i.e., Schafer or PP.

   b. arm-left back-pressure, i.e., Holger Neilsen, HN or ALBP.
   c. arm-left chest-pressure, i.e., Sylvester or ALCP
   d. hip-lift back-pressure, i.e., HLBP

   e. hip-roll back pressure, i.e., HRBP

B. Positive Pressure

1. Expired Air

   a. mouth to nose

   b. mouth to mouth

   c. mouth to apparatus

2. Bellows and Bags

3. Mechanical - intermittent positive pressure or IPPB

TABLE 7

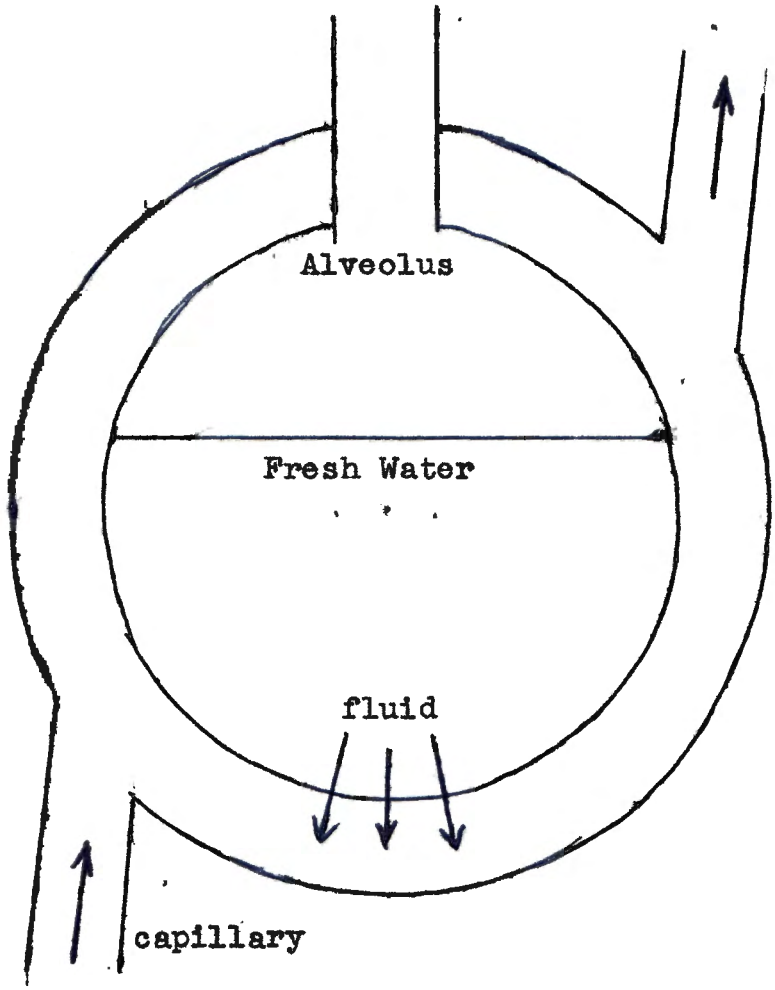
Effectiveness of Resuscitation Techniques in Experimental Drowning. (23)

Injury	Treatment	Result
1. Spontaneous breathing with fresh water in lungs	none	Spontaneous survival possible
2. Spontaneous breathing with sea water in lungs	none	Spontaneous survival possible
3. Obstructive asphyxia until apneic	IPPB/air	Survival
4. Obstructive asphyxia with sea water in lungs	IPPB/air	Partial reoxygenation, delayed pulmonary edema and death
5. Same as 4.	Prolonged IPPB/O_2 with plasma infusion	Complete reoxygenation and survival
6. Obstructive Asphyxia with fresh water in lungs.	IPPB/air	Sudden ventricular fibrillation
7. Same as 6.	IPPB/O_2	Occasional prevention of ventricular fibrillation
8. Same as 6 and 7	IPPB/O_2, CCCM and 400 volt shock	Defibrillation and resumption of spontaneous circulation

IPPB = intermittent positive pressure breathing

CCCM = closed chest cardiac massage

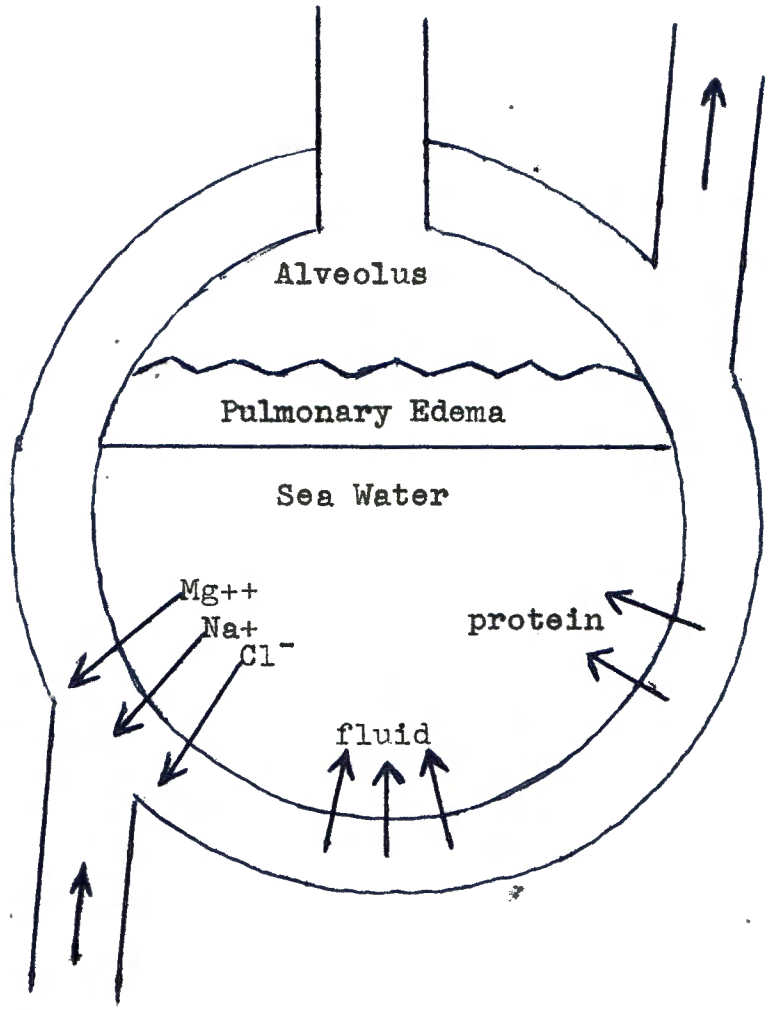
Figure 1
Changes Produced by Aspiration of Fresh Water. (23)



A. Result
1. Ventricular fibrillation
2. Hemodilution
3. Hypervolemia
4. Hemolysis
5. Hyponatremia

B. Treatment
1. IPPB/O_2
2. Closed chest cardiac massage
3. External Defibrillation

Figure 2
Changes Produced by Aspiration of Sea Water. (23)



A. Results
1. Hemoconcentration
2. Hypovolemia
3. Hypoproteimemia
4. Hypernatremia

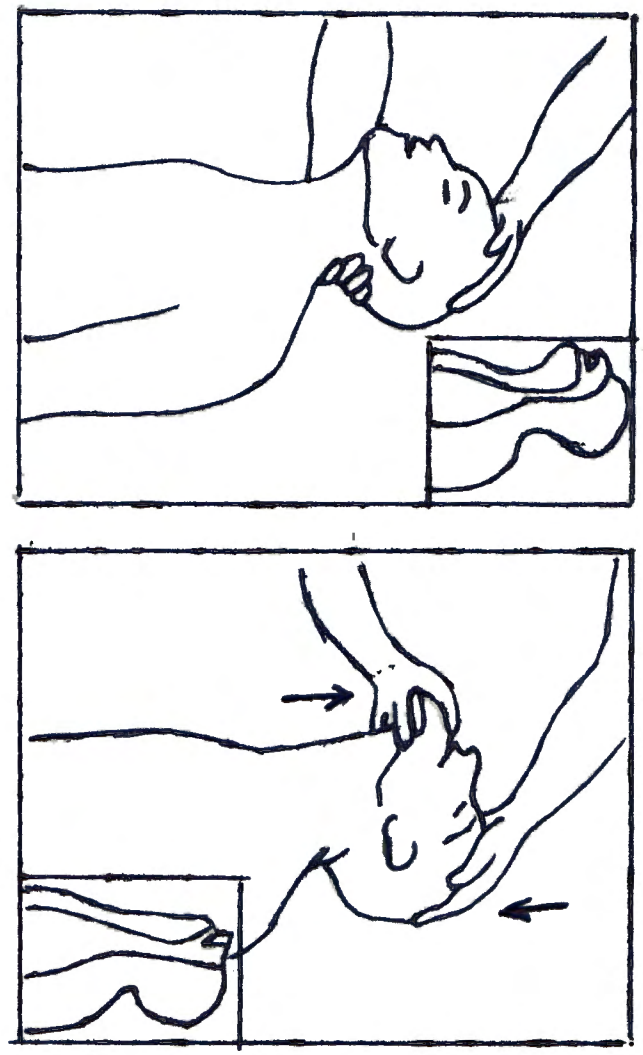
B. Treatment
1. IPPB/O_2
2. Plasma Infusion

Figure 3

Head-tilt oral method of resuscitation (69)

A.

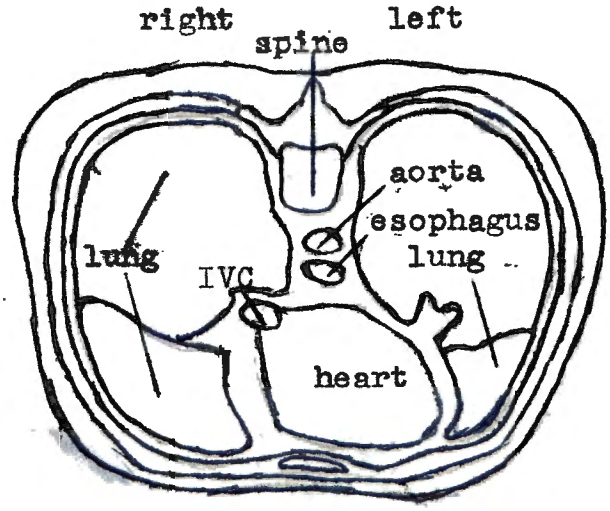
B.



A. neck is lifted; B, head is fully tilted back. The position allows a larger airway than the conscious person has, as noted in the insert of B. This position of the head is similar to that used by an anesthesiologist.

Figure 4

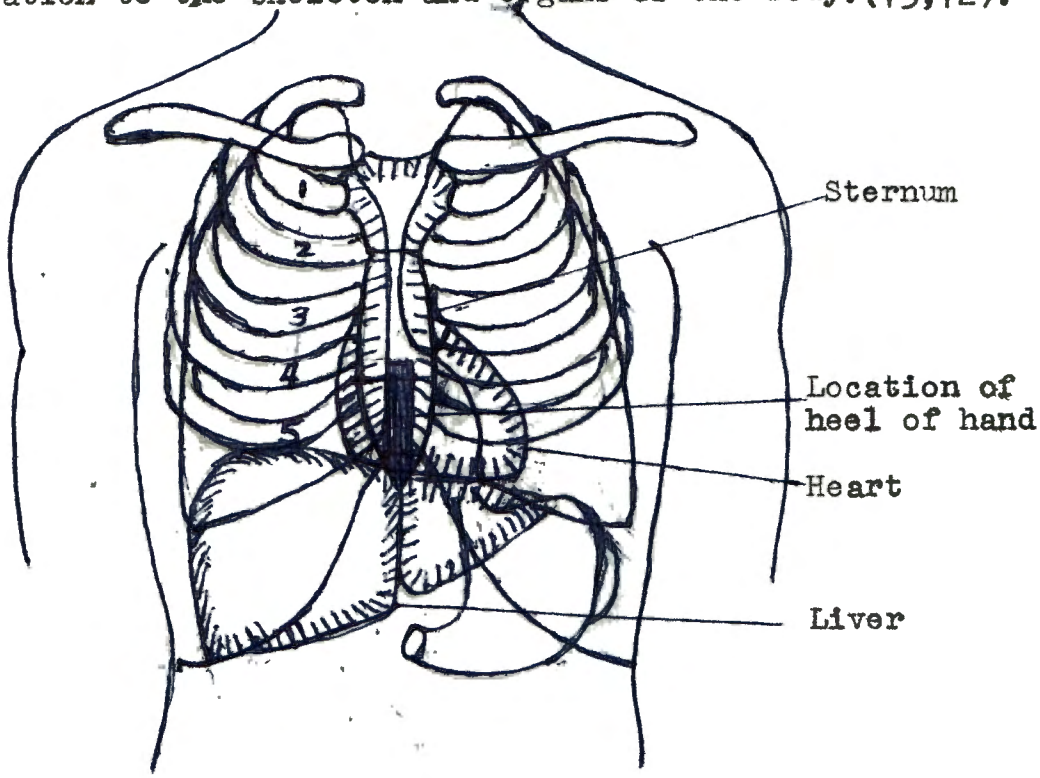
Anatomy and Physiology of CCCM



Since the heart is restricted by the pericardium and fills the space between the sternum and the spine, a downward thrust on the sternum causes compression of the atria and ventricles. Although pressure is exerted on the venous system with each compression, the resultant pressure difference is forward because of the heart valves.

Figure 5

Position of the heel of the hand in CCCM in relation to the skeleton and organs of the body. (73,72).



A. Steps in CCCM

1. Rapid Diagnosis
2. Artifical Ventilation
3. Artifical Circulation till CNS protected
4. Drug Therapy (0.5mg epinephrine IC)
5. EKG
6. Defibrillation if necessary
7. Continued CV and pulmonary support
8. Postresuscitation Therapy

B. Complications

1. Fracture or cracked ribs most common.
2. Pneumothorax; B/ with chest catheter drainage.
3. Subcapsular liver hematoma or rupture from hand to low down.

4. Tears of major BV's possible.
5. Fracture of sternum due to hand to high up.
6. Intercostal hematoma not unusual.
7. Bone morrow emboli to pulmonary arteries found in 50% of the patients.
8. Hemopericardium not seen.
9. Rupture of the heart not seen.

BIBLIOGRAPHY

1. Miles, Stanley, M.D., Underwater Medicine, Philadelphia, J.B. Lippincott Company, 1962,
   a. Chapter XIII, Drowning, p. 200-212,
   b. Chapter XIV, The Treatment of the Apparently Drowned, p. 215-233.

2. Accident Facts, National Safety Council, Chicago, Illinois, 1962.

3. Iskrant, A.P., Accident Mortality Data As Epidemiologic Indicators, Amer. J. Public Health. 50:161-172 (Feb.) 1960.

4. Donald, K.W., Drowning, Brit, Med. J., 2:155-160 (July-Sept.) 1955.

5. Curlewis, Judge, Pres. of Life Saving Association of Australia, Opening of International Conference on Life Saving, 1960. Cited by: (1a).

6. Editorial, "Drowning", Brit. Med. J. 2:373 (Aug. 18) 1894.

7. Curry, J., Observations on Apparent Death, London, E. Cox and Son, 1815. Cited by: (1a).

8. Lowson, J.A., Sensations in Drowning, Edin, Med. J. 13:41, 1903. Cited by: (1a).

9. Editorial, What Drowning Feels Like, Brit. Med. J. 2:823, 1894. Cited by: (1a).

10. Guyton, A.C., M.D., Textbook of Medical Physiology, Philadelphia and London, W.B. Saunders Co., 1959,
    a. Chapter 36, Mechanisms of Respiration, p.434-447.
    b. Chapter 39, The Control of Respiration, p472-482.
    c. Chapter 37, Physical Principles of Gaseous Exchange, p. 448-459.
    d. Chapter 18, Pulmonary Circulatory System, p. 204-212.
    e. Chapter 40, Physiology of Respiratory Disorders and Their Treatment, p. 483-497.
    f. Chapter 20, Blood Flow Through Special Areas of the Body, p. 222.

11. Sodeman, W.A., M.D., Pathologic Physiology, Philadelphia and London, W.B. Saunders Co., 1961
    a. Chapter 19, Pulmonary Ventilation and Respiration: Tests of Respiratory Functin by John H. Seabury, p. 591-628.
    b. Chapter 31, The Nervous System by B.J. Alpers and E.L. Mancall, P. 1125.

12. Swann, H.G. and M. Brucer, The Cardiorespiratory and Biochemical Events During Rapid Anoxic Death, Texas Rep. Biol. and Med. 7:1949
    a. Fulminating Anoxia p. 507-538.
    b. Acute Anoxia p. 539-552.
    c. Progressive Anoxia p. 553-568.
    d. Carbon Monoxide Poisioning: p. 569-592.
    e. Obstructive Asphyxia p. 592-603.

13. Swann, H.G. and others, Fresh Water and Sea Water Drowning: A Study of the Terminal Cardiac and Biochemical Events, Texas Rep. Biol. and Med., 5-6:423-437, 1947-48.

14. Cot, C., Les Asphyxies Accidentelles, N. Maloine, Ed., Paris, 1931. Cited by: (13).

15. Banting. F.G., and others, Physiological Studies in Experimental Drowning, Canad. Med. Assn. J., 39:226, 1938. Cited by: (13).

16. Lougheed, D.W., and others, Physiologic Studies in Experimental Asphyxia and Drowning, Canad. Med. Assn. J. 40:423, 1939. Cited by: (13).

17. Moritz, A.R., Chemical Methods for Determination of Death by Drowning, Physiol. Rev. 24:70-88, 1944. Cited by: (13), (23) and (56).

18. Fainer, D.C., and others, Resuscitation of Dog from Fresh Water Drowning, J. Applied Physiol. 3:417, 1951.

19. Swann, H.G., Mechanics of Circulatory Failure in Fresh and Sea Water Drowning, Circulation Research. 4:241-244, 1956.

20. Glaister, J., Medical Juresprudence and Toxicology London, E. and S. Livingston, 1942. Cited by: (1a).

21. Smith, H.G., and F.S. Fiddles, Forensic Medicine London, J. and A. Churchill, 1949. Cited by: (1a).

22. Otter, G. den, Investigation On Cause of Death in Fresh Water Drowning, Acta. Physiol. Pharmacol. Neerl. 9:415-430, 1960.

23. Redding, J.S., and others, Resuscitation From Drowning, J.A.M.A., 178:1136, 1961.

24. Swann, H.G. and Norma R. Spafford, Body Salt and Water Changes During Fresh and Sea Water Drowning, Texas Rep. Biol. Med. 9:356-382, 1951.

25. None

26. Halmagyi, D.F.J., Lung-Changes and Incidence of Respiratory Arrest in Rats after Aspiration of Salt and Fresh Water, J. Appl, Physiol. 16(1): 41-44. 1961.

27. Pattle, R.E. Proc. Roy. Soc. London. D. 148:217, 1958. Cited by: (26).

28. Colebatch, H.J.H., and D.R.J. Halmagyi, Lung Mechanics and Resuscitation After Fluid Aspiration, J. Appl. Physiol. 16(4):684-696, 1961.

29. Gaylor, J.B., Brain 57:143, 1934. Cited by: (28).

30. Mendelson, C.L., Am. J. Obstet. Gynecol., 52:191 1958. Cited by: (28).

31. Halmagyi, D.R.J., and H.J.H. Colebatch, Ventilation and Circulation After Fluid Aspiration, J. Appl. Physiol. 16(1):35-40, 1961.

32. Lassen, N.A., Physiol. Rev. 39:183, 1959. Cited by: (31).

33. Martin, E., Hepatic Lesions in Death from Drowning Annal. D. Me'd, Le'gale, 12:372, 1932. Cited by: (13).

34. Rahn, H., and H.T. Bahnson, J. Appl. Physiol. 6:105, 1953.

35. Colin, G., Traite' de Physiologie Compare'e des Animaux, Paris, Vol. II, 2nd ed., 1873. Cited by: (13).

36. Brouardel, P. and C. Vibert, Etude sur la Submersion, Annal, D'hygiene publique and de me'd. le'gale, 3rd series. 4:452, 1880.

37. Karpovich, P.V., Water in the Lungs of Drained Animals. Arch. Path., 15:828, 1933. Cited by: (13).

38. Spitz, Mechanism of Death in Fresh Water Drowning: An Experimental Approach to the Problem, Arch. Path. (Chicago) 71:661-668, June, 1961.

39. Bowden K., Drowning, M.J. Australia, 1:39-43, 1957.

40. Current Comment, Drowning Hemoglobinuria., M.J. Australia, 1:715, 1954.

41. Gordon, A.S., Conference on Artifical Respiration, Medical Division Special Report No. 5, Army Chemical Center, Md. April 1951, p. 33. Cited by: (19).

42. Bernstein, R.E., Potassium and Sodium Balance In Mammalian Red. Cells, Science, 120:459, 1954. Cited by: (19).

43. Swann, H.G., Studies in Resuscitation, A.F. Technical Report No. 6696, Wright Air Development Center, Wright Patterson Air Force Base, Ohio, 1951. Cited by: (19).

44. Fenn, W.O., Role of Potassium in Physiologic Processes, Physiol. Rev., 20:277, 1940. Cited by: (19).

45. Garry, W.E., Auricullar Fibrillation, Physiol. Rev., 4:215,1934. Cited by: (13).

46. Nahum, L.H. and Hoff, H.E: Observations on Potassium Fibrillation. J. Pharmacol. and Exper. Therap., 65;322-331, 1939. Cited by: (13).

47. Wener, J., Stansfield, H., and others, Potassium Autointoxication from Hemolysis of Red cells, Am. Heart J., 37:881-891, 1949, Cited by: (13).

48. Winkler, A.W., and others, Electro-cardiographic Changes and Concentration of Potassium in Serum Following Intravenous Injection of Potassium Chloride, Am. J. Physiol., 124:478-483, 1938.

49. Winkler, A.W. and others, Cardiovascular effects of Potassium, Calcium, Magnesium and Barium, Experimental Study of Toxicity and Rationale of Use in Therapeutics, Yale. J. Biol. and Med., 13:123-132, 1940.

50. Gordon, A.S. and others, Fed. Proc. 13:58, 1954, Cited by: (4).

51. Swann, H.G., Anesthesiology, 14:1261, 1953. Cited by: (22).

52. Redding, J.S., Drowning Treated With Intermittent Positive Pressure Breathing. J. Appl. Physiol., 15:849-854, 1960.

53. Jetter, W.W. and A.R. Moritz, Changes in the Magnesium and Chloride Contents from Drowning in Fresh and in Salt Water, Arch. Path., 35:601, 1943. Cited by: (13).

54. Futlon, J.R., Editor, Howell's Textbook of Physiology, 15th Ed., Philadelphia, W.B. Saunders Co., 1946, Cited by: (24).

55. Levietes, P.H. and others, Volume of Extra-cellular Fluids of Body, J. Clin. Invest., 15:261-268, 1936. Cited by: (24).

56. Durlacher, S.H., and others, Blood Changes In Man Following Death Due to Drowning, Arch. Path., 56:454-461, 1953.

57. Paltauf, A., Einige Bermerkungen über den Tod durch Ertrinken, Berl. klin. Wchnschr., 29;298-302, 1892. Cited by: (56).

58. Brouardel, P., La penetaison, la strangulation, la suffocation et la submersion, Paris, J.B. baillie're et fils, 1897, Cited by: (56).

59. Placzek: Du Blutidchte als Zeichen des Ertrinkeignstodes, Vrtlyschr. geaitchi. Med. 25:13, 1903, Cited by: (56).

60. Gettler, A.O., A Method for the Determination of Death by Drowning. J.A.M.A., 77:1650 1921. Cited by: (56) (39).

61. Symposium on Manual Methods of Negative Pressure Artifical Respiration, J. Appl. Physiol. 4:403-495, 1951.
   a. Sadove, M.S., and others, Barbiturate-Curare-Induced Apnea for Artifical Respiration Studies on Normal Adults. I, p. 403.
   b. Gordon, A.S., and others, Air Flow Pattern and Pulmonary Ventilation During Manual Artifical Respiration on Apneic Normal Adults.II, p. 408.
   c. _____, and others, Circulatory Studies During Artificial Respiration on Apneic normal Adults. III, p. 421.
   d. _____, and others, Energy Expenditure of Operators During Manual Artifical Respiration. IV. p. 439.
   e. _____, and others, Pedagogical and Performance Factors of Manual Artifical Respiration with Naval Personnel. V, p. 447.
   f. Karpovich, P.V., and others. Pulmonary Ventilation in Manual Artifical Respiration, p. 458.
   g. _____, and Creighton J. Hale, Energy Expended in Administering Artifical Respiration, p. 467.
   h. _____ and _____, Manual Artifical Respiration; Pedagogical and Fatigue Factors in Its Use, p. 472.
   i. Whittenberger and others, Mechanics of Breathing in Relation to Manual Methods of Artifical Respiration, p. 476.
   j. Nims, Robert G., Comparison of Methods for Performing Manual Artifical Respiration in Apneic Patients, P. 486.

62. Fainer, D.C., and others, Resuscitation of Dogs from Fresh Water Drowning, 3:417-426, 1951.

63. Handford, S.W., and others, Positive and Alternating Positive-Negative Pressure Resuscitation From Curare Poisoning, 3:535-553, 1950.

64. Lucas, B.C.B., and H.W. Whitcher, Artifical Respiration: Comparison Between Manual and Intermittent Positive-Pressure Methods, 2:887-889, 1958.

65. Symposium On Mouth-to-Mouth Resuscitation (Expired Air Inflation), J.A.M.A., 167:317-341, 1958.
a. Dill, D.B., Introduction, p. 317.
b. Gordon, A.S. and others, Mouth-to-Mouth Versus Manual Artifical Respiration For Children and Adults, p. 320.
c. Elam, J.O., and others, Oxygen and Carbon Dioxide Exchange and Energy Cost Of Expired Air Resuscitation, p. 328.
d. Safar, Peter, Ventilatory Efficacy of Mouth-to-Mouth Artifical Resuscitation, p. 335.

66. Johns, R.J. and D.Y. Cooper, New Field Resuscitator, Chemical Corps Medical Laboratories Research Report 76 (Aug.) 1951. Cited by: (65a).

67. Elam, J.O., and others, Artifical Respiration by Mouth-to-Mask Method: Study of Respiratory Gas Exchange of Paralyzed Patients Ventilated by Operator's Expired Air, New England J. Med. 250: 749-754 (May 6) 1954. Cited by: (65a).

68. Greene, D.G., and others, Expired Air Resuscitation in Paralyzed Human Subjects, J. Appl, Physiol. 11:313-318 (Sept.) 1957, Cited by: (65a).

69. Elam, J.O., and others, Head-Tilt Method of Oral Resuscitation, J.A.M.A., 172:812-815, 1960.

70. Buchanan, V.D. Rescue Breathing for the partly drowned, Med. J. Australia, 1:405-412, 1960.

71. Redding, J.S., and others, Treatment of Salt Water Drowning Aspiration, J. Appl. Physiol. 152:1113-1116, 1960.

72. Jude, J.R. and others, Cardiac Arrest, J.A.M.A, 178:1063-70, 1961.

73. Adams, F.D., Physical Diagnosis, 14th Ed., Baltimore, Williams and Wilkins Co., 1958, p. 205.