Editorial
Mechanism of Circulatory Failure in Fresh and Sea Water Drowning

OLDER concepts as to the cause of death from drowning postulated that spasm of the glottis prevents entry of water into the lungs, that impairment or cessation of the heart beat is due to asphyxia and that use of artificial respiration offers the greatest chance for revival. Recent experimental studies have revealed that this mechanism is dominant in a minority of cases only: 20 per cent in our series and about 40 per cent in the series reported by Fainer, Martin and Ivy. In the majority of animals submitted to experimental drowning in fresh water, death is due to ventricular fibrillation within a few minutes after submersion. While the mechanisms which incite fibrillation are still not fully understood, considerable experimental information has become available about events leading up to such a state. It is the purpose of this editorial to review briefly the known facts and the working hypothesis to which they have given rise.

When dogs are submerged in fresh water, sooner or later, they make a strong inspiratory movement and aspirate a large volume of water. This floods the bronchial tree and the water then enters the blood stream rapidly. The course of the inundation was followed using deuterium oxide in the water as a tracer; some 50 ml. of water are added to each 100 ml. of circulating blood in the course of only 3 or 4 minutes of submersion. In one extreme case, the “blood” was found to be 51 per cent of the “drowning water” at 2 minutes after submersion and 72 per cent at 3 minutes.

The absorption of water dilutes correspondingly all of the blood constituents: Na, Cl, Ca, proteins, hemoglobin, etc. An exception is the behavior of K: this rises to about 8 mEq./L. To the dilution of salts may be ascribed the considerable hemolysis that may take place, e.g., plasma hemoglobin of 6 Gm. per cent. Soon ventricular fibrillation supervenes which, of course, arrests at once the function of the heart as a pump. This reaction was discovered by Banting, Hall, Janes, Leibel and Lougheed. During the submersion a fulminating pulmonary edema also occurs, probably due to irritation of the pharyngo-bronchial regions. This results in the movement of many of the blood constituents into the intrapulmonic spaces; for example, just after death, the Na of the lung fluids is about 25 mEq./L. and the plasma proteins about 2 Gm. per cent. The latter accounts for the frothy fluid, sometimes tinged with hemoglobin, that often oozes from the nares and mouth of the victim of drowning.

The conditions under which hemodilution of electrolytes induces ventricular fibrillation have been studied experimentally. In the first place we have found that fibrillation cannot be produced experimentally by injecting large volumes of water intravenously. The precedent condition is anoxia: the animal must be anoxic in order for an experimental hydremic plethora to cause fibrillation. We originally thought that the fibrillation was a K fibrillation, due primarily to the imbalance of Na and K ions. The hemolysis during drowning would release K, and this, coupled with the decrease in Na,
would cause the fibrillation. Attempts to verify the hypothesis have, however, failed: when one injects, during anoxia, large volumes of 0.3 per cent NaCl, thus minimizing hemolysis and presumably diluting the plasma K, fibrillation still develops. Furthermore, the original hypothesis overlooked the fact that in the dog, the main intracellular cation of the erythrocytes is not K, as in man, but Na. Hemolysis, then, in this species, would not release K ions; the slight increase observed in the plasma K during drowning may be a consequence of anoxia alone.

The fibrillation is due, we now suspect, to the lowering of the plasma Na. It can be induced, as discussed above, by injecting dilute saline solution under anoxia. Furthermore, an imminent attack of fibrillation during experimental hydremic plethora with anoxia, may not only be aborted by injecting intravenously a concentrated solution of NaCl, but it temporarily restores the contractility of the weakened heart as well. Once established, the fibrillation cannot be arrested by intracardiac injection of Na or K or Ca salts. Such injections, to be successful, would have to be accompanied with cardiac massage in order to perfuse the given drug throughout the myocardium.

The question arises: does ventricular fibrillation occur in man during accidental submergence in fresh water? (If so, resuscitation is, of course, at present impossible since ventricular fibrillation is irremediable for practical purposes, except in the operating room.) The presumption is strong, in our opinion, that the accident occurs, but proof for it is exceedingly tenuous. The only suggestive evidence is to be found in data on the composition of the heart blood of victims of drowning. This, it was once proposed by Gettler, might furnish evidence in forensic medicine as to whether or not drowning was the cause of death. Since, after ordinary asphyctic death, the heart blood’s composition changes anyway, e. g., the serum Cl decreases to about 80 mEq./L. at 24 hours after death from suffocation, it is generally agreed that blood analysis is of dubious value unless an autopsy is performed promptly after death. Let us take 12 hours or less post mortem as the time within which blood samples must be drawn for reliable analysis. Moritz reviewed two such cases of drowning in fresh water in which the blood Cl was some 20 mEq./L. less than the normal value of 100. This suggests hemodilution. On the other hand, if the same limitation on the use of data is made, a recent report by Durlacher, Freimuth and Swan presents three cases of fresh water drowning in which the plasma Cl was found to be essentially normal. They also report data on the hematocrit, specific gravity of plasma, plasma proteins and plasma K of drowning victims; none of them differed clearly from the values obtained on victims of death due to causes other than drowning. Furthermore, the claim that hemolysis is frequent in victims of drowning is not supported by the study of Durlacher and associates for they found hemolysis to be equally severe in victims of drowning and the controls. One case of semi-drowning was recently described by Rath, in which mild hemoglobinuria and hemoglobinemia were observed (but not apparent hemodilution).

These data, in summary, meager as they are, do not support at all the hypothesis that hemodilution occurs in man during fresh water drowning. We feel, however, that the presumption is strong that it does indeed take place, because it is so obvious and so severe in the dog and because it has been reported in many other species (horses, cows and pigs). The hemodilution would then cause ventricular fibrillation. The total pattern of response is seen in most dogs and in all horses, cows and pigs. It appears sometimes in sheep and goats but not at all (except transiently) in cats, rabbits and monkeys. This follows the general rule that the larger an animal’s heart, the more prone it is to permanent ventricular fibrillation. Man, presumably, is subject to the same rule.

Submergence of dogs in sea water also causes an electrolyte imbalance but it is opposite in sign, sea water being about three and one-half per cent salts in contrast with a 0.9 per cent concentration of salts in plasma. With aspira-
tion, the salts of sea water diffuse into the blood stream, again with explosive speed. The plasma Na rises to about 220, Cl to 250, Mg to 30 mEq./L., etc., all within 4 to 6 minutes after submersion. Hemoconcentration, as shown by increases in hemoglobin and plasma proteins, also occurs. A fulminating pulmonary edema likewise develops. Ventricular fibrillation is not observed in any animals (i.e., dogs, horses, cows, pigs): the plasma Na is high, not low as in fresh water drowning, and hence the conditions initiating fibrillation are absent. The heart, in spite of the adverse electrolyte environment, continues to function with almost the efficiency that it shows during uncomplicated asphyxia.7

With respect to resuscitation of dogs from semi-drowning, the animals recover rapidly, providing ventricular fibrillation does not supervene and providing adequate resuscitating measures are taken before circulatory failure.8 The animals do not appear damaged at all by the experience. One characteristic of the process of drowning, which is of practical interest in resuscitation, is that the animals often continue making gasping movements after fibrillation has occurred. They are observed as long as two minutes after the animal has been killed by the fibrillation. Again, we suspect that man reacts in the same way: such behavior would deceive the would-be resuscitator cruelly, for the victim, although breathing, is, for practical purposes, dead.

As knowledge of drowning accidents is accumulated, it is suggested that adequate measurement of the blood composition be made, for this seems to us the clue to the whole situation. In particular, measurements obtained on victims of semi-drowning, soon after the accident, are bound to expedite our understanding of the process of drowning in man. With this, better therapy of the semi-drowned may well be initiated. If the disturbance in blood electrolytes is as severe in man as it is in the dog and many other infra-human mammals,9 the situation imperatively demands rectification. Replacement transfusion is one of several logical therapies.1 The possibility of kidney damage from hemoglobinemia should not be overlooked. The procurement of electrocardiographic evidence of ventricular fibrillation in victims of drowning would also be valuable. Complexes characteristic of fibrillation persist for fairly long periods after death: in dogs they are obvious and clear at about 20 minutes after submersion, faint at about 24 minutes, but gone at 26 minutes. In a properly prepared emergency center, valuable records on drowning victims might be obtained. Attempts at defibrillation appear to us impractical at present; drugs, intracardiac injections, slapping the chest, would be ineffectual. Furthermore, in man, the process of irreversible insult to the brain from anoxia is so rapid that any circulatory resuscitation that might be accomplished10 is probably futile. Also, we have long been puzzled by the deaths which follow, by one or two days, an episode of semi-drowning; they are sometimes called “deaths from exposure.” Are they perhaps due to a salt imbalance or to an irreversible cerebral insult from the anoxic experience? At present, it is impossible to say, but more knowledge of the blood changes during drowning will help furnish an answer.

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