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 "drowning water" 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 pre-
sumably diluting the plasma K, fibrillation
still develops. Furthermore, the original hy-
pothesis overlooked the fact that in the dog,
the main intracellular cation of the erythro-
cytes 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 in-
duced, as discussed above, by injecting dilute
saline solution under anoxia. Furthermore, an
imminent attack of fibrillation during exper-
imental hydremic plethora with anoxia, may
not only be aborted by injecting intravenously
a concentrated solution of NaCl, but it tem-
porarily restores the contractility of the weak-
ened heart as well. Once established, the fibril-
lation cannot be arrested by intracardiac
injection of Na or K or Ca salts. Such injec-
tions, to be successful, would have to be
accompanied with cardiac massage in order to
perfuse the given drug throughout the myo-
cardium.

The question arises: does ventricular fibril-
lation occur in man during accidental sub-
mergence in fresh water? (If so, resuscitation
is, of course, at present impossible since
ventricular fibrillation is irremediable for prac-
tical purposes, except in the operating room.)

The presumption is strong, in our opinion, that
the accident occurs, but proof for it is exceed-
ingly 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 Dur-
lacher, 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 hemoly-
sis is frequent in victims of drowning is not
supported by the study of Durlacher and asso-
ciates for they found hemolysis to be equally
severe in victims of drowning and the controls.

One case of semi-drowning was recently de-
scribed by Rath, in which mild hemoglobinur-
ria and hemoglobinemia were observed (but
not apparent hemodilution).

These data, in summary, meager as they are,
do not support at all the hypothesis that hemo-
dilution occurs in man during fresh water
drowning. We feel, however, that the presump-
tion 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, rab-
bits and monkeys. This follows the general
rule that the larger an animal's heart, the more
prone it is to permanent ventricular fibrilla-
tion. 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 aspara-
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.19 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,17 the situation imperatively demands rectification. Replacement transfusion is one of several logical therapies. 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 accomplished 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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