Drowning Hemoglobinuria

By Charles E. Rath, M.D.

It has been amply demonstrated in the experimental animal\(^1\) and in drowned individuals\(^2\) that fresh water may gain access to the circulation through the lungs. Moritz\(^3\) has summarized the blood chemical changes that occur during drowning, and Swann\(^4\) has clarified the terminal physiologic and biochemical events in the dog during fresh water and sea water drowning. These studies have shown that in fresh water drowning the various blood constituents are more dilute in the left heart than on the right, whereas the reverse is true in salt water drowning.

Pathologists\(^5\) have long recognized that marked hemolysis of the blood in the left heart is usually observed in cases of drowning in fresh water. Swann\(^6\) has noted marked hemodilution and severe hemolysis of the blood in all of the experimental dogs drowned in fresh water, and Yamakami\(^7\) has demonstrated hemoglobinuria in rabbits following experimental drowning. Jaundice following submersion in fresh water has been reported in the European literature but most of these cases were apparently due to Weil's disease.\(^8\)

The author has been unable to find a report of hemoglobinemia and hemoglobinuria in a patient who lived following submersion. Such a case is the substance of this report.

Case Report

PBBH 2B426 J. H. A 15 year old white school boy was admitted to the Peter Bent Brigham Hospital on May 19, 1948 in coma. About thirty minutes before admission he was found at the bottom of his high school swimming pool. He had been submerged for an unknown period of time, probably not more than five minutes. Immediately upon discovery he received artificial respiration and voluntary respiration returned within a few seconds. Shortly thereafter an artificial resuscitator with oxygen was applied and he received oxygen continuously during his transport to the hospital in a police ambulance.

Physical Examination on Admission

When first seen in the emergency room, the patient was found to be confused and disoriented. He was moaning incoherently and thrashing about in bed. Bloody froth was noted in the mouth. Respiration were irregular. The lips and nailbeds were cyanotic and the pupils constricted. The eyes roamed aimlessly. There was no neck rigidity. The temperature was 103.0 F. The radial pulse was 120 and the B.P. was 180/90. Examination of the lungs revealed coarse inspiratory and expiratory rales throughout both lungs. There was no evidence of consolidation. The heart was normal except for sinus tachycardia. The abdominal examination was negative. The deep tendon reflexes were equal and active and the Babinski responses were plantar in type.

Laboratory Data

In the emergency room the patient passed dark urine which gave a 4+ protein reaction with nitric acid. A few red blood cells and many white blood cells were found in the sediment.

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DROWNING HEMOGLOBINURIA

and the supernatant urine gave a 4+ benzidine and guaiac reaction. Blood studies done shortly after admission revealed the following: hemoglobin 15.9 Gm./100 cc., hematocrit 45.7 per cent, red cell count 3,030,000 per cu. mm., reticulocyte count 1 per cent, white blood cell count 17,850 per cu. mm. The peripheral smear showed a moderate increase in segmented neutrophils. No abnormal cells were seen. The platelets appeared normal. A few red cells showed punctate basophilia but no spherocytes were found. The BUN was 19 mg. per cent; this subsequently rose to a maximum of 24 mg. per cent on the second hospital day. Total protein 6.2 Gm./100 cc., blood sugar 150 mg. per cent, CO₂ 23.2 mM./L., Cl 107 mEq./L. The serum hemochromogen (about ninety minutes after submersion) was 98 mg./100 cc. and 4.8 mg./100 cc. sixteen hours later. Total blood methemoglobin 0.047 mg./100 cc. and total blood sulfhemoglobin zero. Serum bilirubin: direct .56 mg., indirect 1.57, total 2.13 mg. Subsequent urines after the admission urine were benzidine negative. One week later the serum bilirubin was normal. Saline fragility normal. Presumptive tests for warm hemolysis, cold hemolysis and acid hemolysis were negative. Cold agglutinins were negative. The spinal fluid was normal. On the fourth hospital day the PSP test revealed 35 per cent excretion in fifteen minutes (65 per cent in two hours). On the tenth hospital day the urea clearance was 85 per cent. X-rays: initial films of the chest suggested pulmonary edema but were clear four days later. Skull films normal. Two electroencephalograms demonstrated a slowed EEG with spikes. A psychiatric examination revealed no abnormality.

Hospital Course

Immediately following admission, the patient was given .2 Gm. sodium amytal, intravenously, to facilitate the performance of a lumbar puncture. He promptly became areflexic and anoxic. Positive pressure oxygen was instituted and as the effect of the amytal subsided the neurologic consultant demonstrated a decerebrate type of response with a Magnus and de Kleijn tonic neck reflex, hyperactive deep tendon reflexes, and hyperactive mass response to all external stimuli. Twenty-four hours after admission, however, the patient responded to direct questioning and was able to recall incidents immediately prior to the drowning episode. Evidence of hemolysis rapidly disappeared from the serum and the urine cleared. The patient rapidly recovered and when discharged on the eleventh hospital day was apparently completely normal.

Follow-up

The patient was seen and examined two weeks, one year, and two years after discharge from the hospital. He remained completely well, showed no evidence of renal impairment, and had no recurrence of hemoglobinuria. When last heard from, four years after admission, he was still asymptomatic.

Discussion

A consideration of the features of this case presentation raises several questions: (1) Was hemoglobinuria produced as a result of the passage of fresh water through the alveolar walls into the circulation or was it the result of some other hemolytic mechanism? (2) What was the approximate amount of water that entered the circulation? (3) What was the relationship between the submersion and the central nervous system changes? (4) Was there any renal damage associated with this episode?

There is no evidence in this case that the hemoglobinemia was caused by a mechanism other than intravascular hemolysis resulting from the effect of fresh water entering the general circulation. There had been no previous history of hemoglobinuria and a four year follow-up has recorded no recurrence of this phenomenon. Presumptive tests revealed no evidence of increased red cell fragility to hypotonic saline and no evidence of warm, cold, or acid hemolysins.
The cold agglutination titer was not increased and no abnormal antibody was found. The Coombs' test was not done nor were more sensitive technics employed for the demonstration of hemolysins.

Much evidence exists to show that hemolysis may be produced by the passage of fresh water through the lungs into the general circulation. In 1880 Brouardel made reference to the presence of hemoglobin in the plasma of blood taken from the left heart at autopsies of individuals drowned in fresh water. Moritz stated that marked hemolysis of the blood taken from the left heart may help distinguish fresh water from salt water drowning. Peiper, in 1884, demonstrated the rapid absorption, through the lungs, of various solutions instilled in the trachea of dogs. Strychnine, warm hemoglobin solution, egg albumin, and fresh bile were tested. Following the instillation of 60 cc. of a half and half mixture of hemoglobin and water, albumin was demonstrated in the urine in 40 minutes and red urine giving a positive test for hemoglobin was collected in 48 minutes.

Carrara, in 1901, demonstrated a lowering of the freezing point in the left heart following salt water drowning. Yamakami consistently demonstrated hemoglobinuria in rabbits following repeated submersion in fresh water to the point of semiconsciousness. In 1921, Gettler called attention to the changes in the concentration of blood electrolytes after drowning and Moritz has reviewed these changes. The changes in electrolytes are presumably too great to be due to postmortem diffusion between the intravascular fluid and fluid in the lung alveoli. The electrolyte disturbances and concentration of plasma hemoglobin are most pronounced in the left ventricle. This is probably because the last cardiac efforts move pulmonary blood into the left auricle but are not vigorous enough to mix it thoroughly with the rest of the blood.

Swann, et al., have more recently reported extensive physiologic and biochemical studies of fresh and sea water drowning in dogs. They noted "violent hemolysis" in the blood of all dogs during fresh water drowning. This was found within three to six minutes and was associated with a marked drop in hemoglobin, blood density, plasma protein, and chloride. In two dogs, the plasma at the fifth or sixth minute contained 3.6 and 2.8 Gm. per cent of hemoglobin respectively. By using a water solution of 10 per cent D2O as the drowning fluid, Swann and Spafford demonstrated that a large volume of the drowning fluid enters the dog's body fluids. Admittedly crude computations indicated that the volume of water entering the circulation was somewhere between 504 ml. and 3700 ml.

In an attempt to localize the point of entrance of water into the circulation, the present author performed three brief experiments. In the first experiment (table 1) a 15 kilo female dog was anesthetized with nembutal and 620 cc. of distilled water was injected into a tracheal catheter over a period of 95 minutes. The plasma hemochromogen level rose from an initial level of 7.0 mg./100 cc. to a maximum of 22.0 mg./100 cc. at 65 minutes. There was no hemoglobinuria.

In a second experiment on the same dog (table 2) 1800 cc. of distilled water was injected into a tracheal catheter during a period of 45 minutes. In this instance the plasma hemochromogen level rose to a maximum of 44.0 mg./100 at 35 minutes. There was no hemoglobinuria.

A third experiment (table 3) was done to exclude the possibility that some of
DROWNING HEMOGLOBINURIA

the water might be coughed up around the tracheal catheter and swallowed. This time the tracheal catheter was tied in place. The same dog was again used and 1000 cc. of distilled water labeled with 100 mg. sodium salicylate/1000 cc. was injected into the tracheal tube over a period of 65 minutes and salicylic acid levels were determined on the plasma. Rough calculations, based on an estimated plasma volume of 100 cc. at 20 minutes and 800 cc. at 65 minutes, indicate that approximately 110 cc. and 128 cc. of distilled water had entered the circulation through the lungs at 20 minutes and 65 minutes respectively. The degree of hemolysis per 1000 cc. of distilled water injected was somewhat less than that observed by Landsteiner and Finch in humans.

**Table 1.**—Intratracheal Instillation of 620 cc. Distilled Water in a Normal Dog in 95 minutes

<table>
<thead>
<tr>
<th>Time</th>
<th>Hematocrit (%)</th>
<th>Hemochromogen (mg./100 cc.)</th>
<th>Urine Hb.</th>
<th>Total protein serum (Gm./100 cc.)</th>
<th>Chloride (mEq./L.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>40</td>
<td>7.0 mg.</td>
<td>0</td>
<td>6.5</td>
<td>115</td>
</tr>
<tr>
<td>20 min.</td>
<td>30</td>
<td>5.9</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>65 min.</td>
<td></td>
<td></td>
<td>0</td>
<td>6.4</td>
<td>115</td>
</tr>
<tr>
<td>95 min.</td>
<td>37</td>
<td>20.0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>24 hrs.</td>
<td>42</td>
<td>5.0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 2.**—Intratracheal Instillation of 1800 cc. Distilled Water in the Same Dog in 45 minutes

<table>
<thead>
<tr>
<th>Time (min.)</th>
<th>Hematocrit (%)</th>
<th>Hemochromogen (mg./100 cc.)</th>
<th>Urine hemoglobin</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>38.4</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>27</td>
<td></td>
<td>34</td>
<td>0</td>
</tr>
<tr>
<td>35</td>
<td>38</td>
<td>44</td>
<td>0</td>
</tr>
<tr>
<td>80</td>
<td>37</td>
<td>10</td>
<td>0</td>
</tr>
</tbody>
</table>

Although, in the case here presented, it is possible that the hemoglobinemia and hemoglobinuria resulted from absorption of hemolyzed blood through alveolar capillary walls traumatized by asphyxia and the effects of the resuscitator, the experimental evidence supports the view that the hemoglobinemia was produced by intravascular hemolysis produced by absorption of hypotonic water into the circulation.

The approximate amount of water that entered the circulation of the patient

* These calculations were made by multiplying an estimated total blood volume of 1500 cc. by the hematocrit at 20 minutes and 65 minutes respectively to obtain the red cell mass. The plasma volumes were obtained by subtracting the red cell mass from 1500 cc. The volume of distilled water entering the circulation was estimated by multiplying the salicylate concentration in the plasma by the estimated plasma volume.
can be only roughly estimated. Since the hematocrit and serum chloride drawn within two hours of submersion showed no evidence of dilution, it is fairly certain that the amount of water absorbed was not great. Landsteiner and Finch injected distilled water intravenously in several patients. An injection of 300 cc. produced a rise in serum hemochromogen to 47 mg./100 cc. without hemoglobinuria. Injections of 600 cc. and 900 cc. of water resulted in serum hemochromogen levels of 201 mg./100 cc. and 380 mg./100 cc. respectively with hemoglobinuria. Krumbhaar in experiments on dogs, found that the intravenous injection of distilled water in amounts equivalent to 3 per cent of the body weight produced hemoglobinemia and hemoglobinuria.

The patient in this report weighed 60 Kg. Reference to Krumbhaar’s data suggests that a minimum of 180 cc. of distilled water must have entered the circulation to produce hemoglobinuria. However, if the calculation is based on the patient’s serum hemochromogen level of 98 mg./100 cc. and reference to the human data of Landsteiner and Finch, one arrives at a figure of about 500 cc. of absorbed water.

The relationship between the central nervous system changes and the submersion is not clear. Gellhorn reviewed the effect of water intoxication on the electroencephalogram. Slow waves and convulsive potentials were increased in rats intoxicated by water. The minor electroencephalogram changes noted in this patient cannot be adequately explained. It is possible that the patient had inherited cerebral dysrhythmia and that a convulsive seizure was responsible for his falling into the swimming pool. However the patient had no previous or subsequent convulsive seizures and there was no definite family history of convulsive seizures. It seems likely that the tonic neck reflexes and the mass reaction indicating decortication were the result of cerebral anoxia incident to submersion rather than the effect of water intoxication.

There is little evidence of renal damage other than the albuminuria and transient elevation of the blood urea nitrogen. Proteinuria constantly ac-
companies hemoglobinuria and is not surprising. The transient elevation of the blood urea nitrogen may represent a mild ischemic episode similar to that described by Oliver.

**SUMMARY**

1. A case of hemoglobinemia and hemoglobinuria with recovery following incomplete drowning is presented.
2. The pathogenesis of the intravascular hemolysis is discussed.

**REFERENCES**

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