Morbidity of Childhood Near-Drowning

Bradley Peterson, M.D.

From the Department of Anesthesia, Stanford University School of Medicine, Stanford, California

ABSTRACT. An assessment of morbidity in near-drowning was made from a review of emergency room and hospital records of 72 patients, ages 9 months to 20 years, who suffered near-drowning during the period January 1972 through June 1974. Fifteen patients (21%) evidenced severe anoxic encephalopathy; the remainder had no detectable neurologic deficits. Hypoxemia was demonstrated in 56 patients. Severe acidosis was not present unless respiratory failure occurred. Neither electrolytes, red blood cell hemolysis, nor cardiac arrhythmias presented a problem. Respiratory complications included pulmonary edema, aspiration pneumonia, atelectasis, shock lung, pneumothorax, and pneumomediastinum.

All children requiring cardiopulmonary resuscitation in the emergency room suffered anoxic encephalopathy. The occurrence of seizures, fixed and dilated pupils, flaccid extremities, and lack of response to deep pain in the emergency room had almost universal correlation with resultant severe anoxic encephalopathy, as did a submersion period of six or more minutes.

The morbidity of near-drowning is significant with regard to the number of children affected and the severity of the central nervous system insult received. The statement by the American Heart Association that resuscitative efforts in children should be continued for periods longer than ten minutes needs reevaluation, since neurologic recovery did not occur in any child requiring cardiopulmonary resuscitation (CPR) in the emergency room. More importantly, new methods of cerebral resuscitation need to be developed and established. In short, medical personnel need to think in terms of cardiopulmonary cerebral resuscitation (CPCR) rather than in terms of CPR. Pediatrics, 59:364-370, 1977, NEAR-DROWNING, HYPOXIA, ISCHEMIA, ANOXIC ENCEPHALOPATHY, CARDIOPULMONARY CEREBRAL RESUSCITATION (CPCR).

In May 1973, the American Heart Association and the National Academy of Sciences recommended "Standards for Cardiopulmonary Resuscitation (CPR) and Emergency Cardiac Care." Included is the recommendation, "In children, or in unusual circumstances, e.g., when the arrest is associated with hypothermia, resuscitation efforts should be continued for longer periods since recovery has been seen even after prolonged unconsciousness." An extension of this philosophy is aggressive resuscitation of children in emergency facilities. This includes near-drowning victims in whom the primary goal of treatment is the correction of hypoxemia.

However, with sophisticated life-support systems the major clinical problem in near-drowning has become neurologic prognosis. Although experimental studies of immersion and anoxic encephalopathy exist, there are no large clinical studies correlating neurologic outcome with clin-

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ADDRESS FOR REPRINTS: (B.P.) Department of Anesthesia, Stanford University Medical Center, Stanford, California 94305.
TABLE I
COMPARISON OF NEAR-DROWNING AND DROWNING
STATISTICS FOR SAN DIEGO COUNTY, JANUARY 1972
THROUGH JUNE 1974

<table>
<thead>
<tr>
<th>Age</th>
<th>No. of Near-Drowning Accidents</th>
<th>No. of Deaths by Drowning</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-21</td>
<td>72</td>
<td>65</td>
</tr>
<tr>
<td>1-3</td>
<td>38</td>
<td>20</td>
</tr>
</tbody>
</table>

TABLE II
NEAR-DROWNING AND DROWNING ACCIDENTS DIVIDED
ACCORDING TO BODY OF WATER, SAN DIEGO COUNTY,
JANUARY 1972 THROUGH JUNE 1974

<table>
<thead>
<tr>
<th>Body of Water</th>
<th>No. of Near-Drownings</th>
<th>No. of Drownings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pool (&lt; 4 yr)</td>
<td>42 (30)</td>
<td>30 (18)</td>
</tr>
<tr>
<td>Seawater</td>
<td>22</td>
<td>20</td>
</tr>
<tr>
<td>Bathtub</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Other*</td>
<td>3</td>
<td>12</td>
</tr>
</tbody>
</table>

*Includes pond, irrigation ditch, whirlpool bath, wading pool.

ical course. Therefore, physicians continue to work with a guarded optimism based on hearsay
knowledge and isolated case reports1-9 of neurologic recovery following prolonged immersion.
In childhood near-drownings, aggressive resuscitation based on this optimism proves tragic
when cardiopulmonary function but not central nervous system (CNS) function is retrieved.
The purposes of this study were (1) to quantitate the morbidity of near-drowning in children
and (2) to determine if current practice in management might be improved.

MATERIALS AND METHODS

Seventy-two cases of near-drowning were obtained retrospectively from hospital emergency
department logs for the period from January 1972 through June 1974. Drowning was defined as
submersion resulting in asphyxia and death while submerged or within one day. Near-drowning was
defined as submersion of sufficient gravity to result in the victim being transported to a hospital
emergency department but not severe enough to result in death within the first day.
Hospitals in the study included Children's Hospital, University of California Hospital-San
Diego, U.S. Naval Hospital-Balboa Park, Mercy Hospital, Sharp Memorial Hospital, Doctors
Hospital, Coronado Hospital, Bay General Hospital, and Paradise Valley Hospital. These hospitals
provide 50% of the pediatric beds in San Diego County.

RESULTS

Incidence and Scene of Accident

Young children and home swimming pools were involved in most of the near-drowning
accidents (Tables I and II). For the 1- to 3-year-olds, the swimming pool accounted for 80% of
near-drowning accidents and 90% of drowning accidents. For these young children, near-drowning
was usually not observed. Rather they were discovered to be “missing” and were subsequently
found in the pool. The parent’s estimation of the duration of time that the child was missing and/or
immersed surprisingly correlated with the child’s neurologic prognosis (Table III). The position
of the child in the water and the time estimated to transfer the patient to the emergency room did
not correlate with neurologic outcome.

Evaluation in Emergency Room

Upon arrival at the emergency department, the patients were divided into two groups: those with
and those without cardiopulmonary arrest in the emergency facility. The nonarrested group (58
patients) was tachycardic, tachypneic, cyanotic, normotensive, and normothermic. Bilateral rales
were usually present. Seven patients had depressed levels of consciousness; two patients
had seizures. Arterial blood gases demonstrated abnormally large alveolar arterial oxygen
gradients in 70% of the 58 nonarrested patients. There was not always a correlation between
hypoxemia and cyanosis or between hypoxemia and level of consciousness.

The arrested group (14 patients) was resuscitated with return of cardiac function within
minutes. Thirteen of these patients were 3 years of age or younger and one was 6 years of age.
Severe metabolic acidosis (HCO₃ < 15) occurred only in those patients who suffered an arrest. Electrolyte values were generally normal with the exception of hypokalemia (Table IV). All
patients had normal hematocrit levels. No patient developed renal complications, including the
patient with a plasma hemoglobin level of 151 mg/100 ml. Chest roentgenographs obtained in
the emergency facility were abnormal in 60% of patients (Table V).
TABLE III

CLINICAL DATA CORRELATING WITH NEUROLOGIC MORBIDITY

<table>
<thead>
<tr>
<th></th>
<th>Estimated Immersion Time &gt; 5 min</th>
<th>Cardiac Arrest in Emergency Room</th>
<th>Neurologic Signs* of Injury or Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1 (57 patients; no detectable neurologic sequelae)</td>
<td>0</td>
<td>0</td>
<td>0†</td>
</tr>
<tr>
<td>Group 2 (14 patients; severe anoxic encephalopathy)</td>
<td>14</td>
<td>13</td>
<td>14</td>
</tr>
</tbody>
</table>

*Neurologic signs include fixed and dilated pupils on arrival at emergency department, abnormal motor function, coma on day 2, and seizures.
†Two patients in group 1 suffered isolated seizures.

Electrocardiograms (ECGs) were obtained on 12 patients at the time of arrival in the emergency facility. Only supraventricular arrhythmias were noted, all converting to sinus rhythm with correction of hypoxia and acidosis.

**Morbidity**

The morbidity of near-drowning is pulmonary and neurologic. The most common respiratory complications were aspiration pneumonia and pulmonary edema (Table V). In the absence of neurologic damage, all pulmonary complications cleared within days.

Neurologic morbidity was substantial (Table VI). On arrival in the emergency department, 21 patients had impairment of consciousness; 24 hours later, 15 remained unconscious. These were the same 15 who had suffered a cardiac arrest. They had fixed, dilated pupils on arrival in the emergency facility; their pupils were reactive one to two hours after resuscitation. They usually had flaccid extremities on day 1, confusing voluntary movements by day 2, and sometime thereafter decerebrate and decorticate posturing. There was generally an initial lack of response to deep pain; this returned within hours. Assessment of brain stem function was done in the 15 children sustaining a cardiac arrest: the oculovestibular reflex was noted to be present in four children and absent in three; caloric stimulation was not effective in inducing labyrinthine nystagmus in four patients; and vertical eye bobbing was noted in two patients. Fourteen of these 15 patients suffered a seizure during the initial three days.

Electroencephalograms (EEG) were recorded on 11 of the 15 arrested patients. In all cases except one, they were grossly abnormal at 48 hours after arrest.

*Every patient requiring CPR in the emergency facility developed permanent brain damage* (Fig. 1). Only one patient arriving with a heartbeat suffered irreversible brain injury. Found pulseless, he had been resuscitated by a rescue squad, using mask oxygen and closed chest massage.

In terms of neurologic morbidity, it was possible to divide the patients into two very distinct groups: group 1, those with no neurologic sequelae, and group 2, those with severe anoxic encephalopathy (Table III). There was one

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**TABLE IV**

SERUM ELECTROLYTES DRAWN IN EMERGENCY ROOM

<table>
<thead>
<tr>
<th></th>
<th>Freshwater (50)*</th>
<th></th>
<th>Seawater (22)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No Cardiac Problems (37)</td>
<td>Cardiac Arrest (13)</td>
<td>No Cardiac Problems (21)</td>
<td>Cardiac Arrest (1)</td>
</tr>
<tr>
<td>Sodium</td>
<td>136 ± 5.9†</td>
<td>140 ± 4.7</td>
<td>142 ± 4.5</td>
<td>150</td>
</tr>
<tr>
<td>Potassium</td>
<td>3.5 ± 0.5</td>
<td>2.8 ± 0.55</td>
<td>4.1 ± 0.8</td>
<td>3.3</td>
</tr>
<tr>
<td>Chloride</td>
<td>100 ± 6.4</td>
<td>93 ± 5.0</td>
<td>105 ± 7.4</td>
<td>...</td>
</tr>
</tbody>
</table>

*Number of cases in parentheses.
†± SD.
FIG. 1. Relationship of resuscitation to anoxic encephalopathy after near-drowning.

patient who did not fall into these two groups. Now age 2, two years after the accident, she remains with marked truncal ataxia, strabismus, and some optic atrophy; however, her verbal skills are appropriate for her age. She was estimated to have been immersed for two to three minutes, was found breathing shallowly at poolside, was given resuscitation by a person trained in modern techniques, but on arriving in the emergency room with fixed, dilated pupils required CPR. She remained in a coma for several days. She did not have a seizure. Her EEG, abnormal at 48 hours after the accident, was normal after seven days.

DISCUSSION

Drowning ranks as one of three leading causes of accidental death in the United States. It is ranked as the second commonest cause of death in people under 24 in South Carolina. In California, drowning ranks as the second commonest cause of death in the age group from 1 to 4 years. Delayed deaths from near-drowning approach the number of deaths from drowning. In this study, delayed deaths were significant (Table VI). It could be assumed by combining delayed deaths with drowning deaths that drowning and near-drowning may rank as the leading

### TABLE V

**Respiratory Complications of Near-Drowning**

<table>
<thead>
<tr>
<th>Complication</th>
<th>No. (%) of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspiration pneumonia</td>
<td>29 (40)</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>16 (29)</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>14 (20)</td>
</tr>
<tr>
<td>Shock lung; adult respiratory distress syndrome</td>
<td>4-6 (5-10)</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>7 (10)</td>
</tr>
<tr>
<td>Pneumomediastinum</td>
<td>3 (5)</td>
</tr>
</tbody>
</table>

### TABLE VI

**Neurologic Sequelae of Near-Drowning**

<table>
<thead>
<tr>
<th>Sequelea</th>
<th>No. (%) of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>No sequelae</td>
<td>57 (80)</td>
</tr>
<tr>
<td>Anoxic encephalopathy</td>
<td>15 (20)</td>
</tr>
<tr>
<td>Severe organic brain disease</td>
<td>14</td>
</tr>
<tr>
<td>Mild to moderate organic brain disease</td>
<td>1</td>
</tr>
<tr>
<td>Death within 6 mo</td>
<td>7</td>
</tr>
</tbody>
</table>
cause of death in the age group from 1 to 4 in California.\textsuperscript{11} (With the continuing increase in the number of swimming pools, these figures will probably increase.) In this study, as in others, swimming pool accidents usually involved very young children, while seawater accidents involved older children and were often associated with boating and surfing accidents and occasionally alcohol and drugs.\textsuperscript{2,12}

A very neglected statistic is the number of children who suffer anoxic encephalopathy as a result of near-drowning (Table VI). This is a recent phenomenon: a conversion of deaths by drowning to near-drowning anoxic encephalopathy as a consequence of sophisticated CPR. Fuller predicted this in 1962.\textsuperscript{13}

Hypoxia has recently been identified as the primary problem in near-drowning.\textsuperscript{2} Known causes of hypoxemia in near-drowning include apnea, aspiration of fluids\textsuperscript{2,6} and debris,\textsuperscript{1,13} bacterial pneumonitis,\textsuperscript{2} decreased lung compliance,\textsuperscript{14,15} bronchospasm, laryngospasm,\textsuperscript{14,15} pulmonary edema,\textsuperscript{2} alveolar hemorrhage,\textsuperscript{2,15} loss of surfactant\textsuperscript{17} or shock lung,\textsuperscript{18} pneumothorax, and pneumomediastinum. All were noted in this study except aspirated debris. Pneumothorax and pneumomediastinum occurred only in those patients requiring mechanical ventilation and are probably complications of the therapy. Surfactant levels were not measured. However, several cases of shock lung were noted; all were freshwater accidents. Although not expected,\textsuperscript{2} pulmonary edema occurred with equal frequency in seawater and pool-water near-drownings. Other possible causes of hypoxemia include neurogenic pulmonary edema\textsuperscript{19} and hypoxemic-induced respiratory distress syndrome.\textsuperscript{20}

Electrolyte problems were minimal, in agreement with earlier reports.\textsuperscript{2,21} Experimental data demonstrating rapid intrabody fluid shifts\textsuperscript{16} and data showing that only small amounts of seawater and freshwater enter the circulation in partially immersed nonanoxic dogs\textsuperscript{22} best explain the laboratory values in human near-drownings, i.e., mild hyponatremia, hypochloremia, hypokalemia, and limited hemolysis occurring in freshwater near-drownings and mild hypernatremia, hyperchloremia, and absence of hemolysis in seawater near-drownings.

As previously reported, humans, in contrast to dogs, showed only supraventricular arrhythmias. Oliguria, though reported in the literature,\textsuperscript{6,23} did not occur. In the cases reported, acute tubular necrosis and/or myoglobinemia were probably the causes of renal shutdown. High plasma hemoglobin levels in near-drownings have not been associated with renal damage.\textsuperscript{2,24}

**Anoxic Encephalopathy**

Anoxic brain damage in long-term survivors of cardiac resuscitation has been rarely reported. Likewise, there are only a few reported cases of CNS damage occurring in near-drowning victims.\textsuperscript{2,25} To offset these reports are cases in which the patient recovered after a severe cerebral insult.\textsuperscript{5-9} There also exists a hearsay knowledge of significant incidence of neurologic recovery for resuscitated near-drowning victims. All this leads to the misconception that irreversible brain damage is an uncommon complication in near-drowning.

The CNS damage in near-drowning is caused primarily by hypoxemia and ischemia and commences at their onset. However, further CNS insult occurs after resuscitation. Here, factors of flow, pressure, and metabolism are involved; these include CNS perfusion pressures, autoregulation, pseudoautoregulation, no reflow phenomenon, intracerebral steal, intracerebral squeeze, intracranial pressure, cerebral edema, cerebral osmolality, temperature, and the biochemical milieu.\textsuperscript{24-32}

The reversibility of brain damage is dependent on the therapy available. With present clinical standards of practice,\textsuperscript{1} irreversible CNS damage begins four to ten minutes after submersion. This estimate assumes that after submersion, hypoxemia begins in seconds, that ineffective circulation develops in two to six minutes, and that microscopic CNS damage occurs in two to three minutes of total ischemia.\textsuperscript{2,34-35} This estimate is consistent with clinical data\textsuperscript{6} (Table III) and with EEG data, suggesting that a physiologic threshold is crossed over after five minutes of cerebral ischemia.\textsuperscript{33}

However, new methods have demonstrated that the damage incurred prior to and during resuscitation is reversible and that the functional part of the CNS insult progresses during the initial hours following resuscitation.\textsuperscript{28,30,32,37,38}
Promising therapeutic modes for cerebral resuscitation include hypothermia and barbiturates. Hypothermia decreases the cerebral metabolic rate and is a factor in cases of neurologic recovery following prolonged immersion. Barbiturates also protect the brain from hypoxia and ischemia. Specifically, large doses of thiopental given 5, 30, and 60 minutes after ischemic injury have prevented neurologic sequelae in monkeys. They may act by decreasing the cerebral metabolic rate, by decreasing cerebral edema, and by decreasing cerebral blood flow in proportion to the decrease in cerebral metabolic rate. They may also act in other obscure ways. Other techniques for cerebral resuscitation include hemodilution; hyperventilation; use of steroids, mannitol, and glycerol; and continuous intracranial pressure monitoring. Patients in this series were not treated with these modalities with the exception of the occasional use of steroids, mannitol, and hyperventilation.

Age also affects reversibility; but is probably only significant in the neonate who benefits from increased anaerobic metabolism and in the older patient who suffers because of arteriosclerosis. In this series, age had no correlation with neurologic outcome.

With the present standards of CPR, permanently brain-damaged children are salvaged because the heart, unlike the brain, does not incur irreversible damage at 37°C until it has suffered 30 minutes of total ischemia. This creates a significant time period during which only the heart can be retrieved. Presently, the results of CPR in the emergency department are very poor. Of 99 patients requiring CPR in the Stanford Emergency Department, 28 survived to be admitted to the intensive care unit. Twenty-six of these 28 later died, while one remains alive in a vegetative state. The one patient who recovered function was resuscitated at the scene by paramedics. His pupils were not fixed and dilated; however, his ECG was a flat line on arrival in the emergency department. Other supporting data shows that all near-drowning victims arriving in the emergency department with fixed and dilated pupils died. That fewer children die and more children exist with anoxic encephalopathy in contrast to adults is probably secondary to the relatively excellent state of their cardiovascular system.

There is an obvious need for reevaluation of CPR. The ideal answer is improvement in methods to the point where we are practicing cardiopulmonary cerebral resuscitation (CPCR). Until CPCR is developed and standardized, the physician must be aware of the limits of CPR. The question is which child will indubitably develop severe irreversible brain damage. Neurologic findings one hour after arrest have been correlated with prognosis. However, there are also factors a physician should consider at resuscitation (Table VII). These data and those of others stress the poor prognosis of the arrested patient with fixed, dilated pupils. This prognosis, however, is improved if immersion occurred in cold water, if the duration of immersion was less than six minutes, if the patient had taken an overdose of barbiturates, and, importantly, if there is prompt, on-the-scene, sophisticated resuscitation. This basic information can be gathered in the initial seconds after arrival in the emergency department. CPR begun on arrival can then be terminated if judged appropriate by the physician. If resuscitation is continued, methods of cerebral resuscitation must be considered. Conservative limited cardiopulmonary therapy may be worse than no therapy at all.

The major attack on the problem, however, must be prevention. Required fencing of pools; perhaps even required licensing of pool owners; necessitating basic knowledge of treatment of water accidents; education of the general public; and establishment of strong paramedic programs should all be part of prevention.

REFERENCES
42. Eliastam M: Personal communication.

ACKNOWLEDGMENT

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