Cardiovascular Collapse in Acute Poliomyelitis

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Cardiovascular collapse accounted for approximately one-third of the deaths from acute poliomyelitis in two epidemics. This syndrome occurred early in the disease and only in cases of bulbar involvement. In all cases there was morphologic involvement of the medulla and in 75 per cent of the cases there was a varying degree of myocarditis. There was a much smaller incidence of less severe myocarditis in the cases dying of other causes. It is suggested that the combination of sustained vasoconstriction arising as a result of the medullary lesion and the presence of interstitial myocarditis leads to circulatory failure and pulmonary edema.

Although cardiovascular collapse had previously been noted in patients with acute poliomyelitis, Baker and his colleagues first indicated that this lethal symptom complex is a relatively common feature of bulbar poliomyelitis. They examined in detail the medulla of 80 cases dying of poliomyelitis and concluded that this syndrome was due to involvement of the large cells in the ventromedial reticular substance of the medulla.

There are also reports in the literature noting the occurrence of an interstitial myocarditis in poliomyelitis. Electrocardiographic changes in a variable proportion of patients with acute poliomyelitis have also been reported. These reports do not record whether the histologic changes described by Baker and his associates occurred in their cases. On the other hand, the reports by Baker and his colleagues deal mainly with the central nervous system findings.

Maloney and Whittenberger suggested from their experiments on dogs that a high negative intratank pressure may play an important role in cardiovascular collapse in respirator patients by interfering with the return of blood to the heart, particularly if the circulation is failing.

In view of the uncertain role played by different factors in the etiology of this syndrome, it seemed worthwhile to review our cases of cardiovascular collapse.

Case Material

During 1952 and 1953 a total of 1359 cases were admitted to the acute poliomyelitis wards of the Winnipeg Municipal Hospitals. Of these, 523 had bulbular involvement and there were 82 deaths in the whole series. The immediate cause of death in 22 cases was cardiovascular collapse (table 1). Autopsies were carried out on 20 of these 22 cases and on 47 of the remaining patients who died. The deaths of the latter were due to a variety of causes such as secondary bacterial infection, asphyxia and gastrointestinal ulceration. Six patients, showing clinical evidence of this syndrome, survived (table 2).

All patients with any evidence of bulbar involvement were carefully followed clinically and a running record of blood pressure, pulse, temperature, respirations and state of consciousness was maintained. Cases coming to autopsy were examined completely in order to determine as accurately as possible the factors leading to the lethal outcome.

Clinical Syndrome

The syndrome of cardiovascular collapse is a complication of the early acute phase of bulbular poliomyelitis. All these patients had clinical evidence of pharyngeal involvement. This syndrome was not encountered in any of 640 patients with only spinal paralysis. Sixty-eight of these were respirator cases; there were five deaths and three postmortem examinations in this spinal group.

In the 22 cases shown in table 1, the average time from onset of illness to death was five days and the signs of cardiovascular collapse
TABLE 1.—Fatal Cases

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex &amp; Age</th>
<th>Duration Bulbar Signs—Days</th>
<th>Max. Temp.</th>
<th>Respirator and/or Tracheotomy*</th>
<th>Blood Pressure Phases Duration in Hours</th>
<th>Duration of Nor-adrenaline—Hours</th>
<th>Clinical Pulmonary Edema</th>
<th>Autopsy Findings§</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Normal</td>
<td>Hypertensive</td>
<td>Hypotensive</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>F 21</td>
<td>1</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>10</td>
</tr>
<tr>
<td>2</td>
<td>F 19</td>
<td>2</td>
<td>101 F.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>M 33</td>
<td>2</td>
<td>100 F.</td>
<td>—</td>
<td>2</td>
<td>—</td>
<td>—</td>
<td>&lt;1</td>
</tr>
<tr>
<td>4</td>
<td>M 8</td>
<td>&lt;1</td>
<td>102 F.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>1</td>
</tr>
<tr>
<td>5</td>
<td>M 10</td>
<td>&lt;1</td>
<td>105 F.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>6</td>
<td>2½</td>
</tr>
<tr>
<td>6</td>
<td>F 27</td>
<td>2</td>
<td>106 F.</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td>M 12</td>
<td>3</td>
<td>104 F.</td>
<td>&lt;1</td>
<td>—</td>
<td>2</td>
<td>—</td>
<td>2</td>
</tr>
<tr>
<td>8</td>
<td>M 4</td>
<td>1</td>
<td>106 F. R &amp; T</td>
<td>10</td>
<td>—</td>
<td>1</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>9</td>
<td>F 15</td>
<td>2</td>
<td>103 F.</td>
<td>—</td>
<td>36</td>
<td>3</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>10</td>
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<td>T</td>
<td>12</td>
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<td>—</td>
<td>5</td>
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<tr>
<td>11</td>
<td>M 35</td>
<td>2</td>
<td>103 F. T</td>
<td>13</td>
<td>—</td>
<td>8</td>
<td>—</td>
<td>8</td>
</tr>
<tr>
<td>12</td>
<td>F 35</td>
<td>2</td>
<td>105 F. R</td>
<td>17</td>
<td>—</td>
<td>8</td>
<td>—</td>
<td>8</td>
</tr>
<tr>
<td>13</td>
<td>F 21</td>
<td>2</td>
<td>102 F.</td>
<td>29</td>
<td>—</td>
<td>7</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>14</td>
<td>M 15</td>
<td>4</td>
<td>104 F.</td>
<td>75</td>
<td>&lt;1</td>
<td>—</td>
<td>—</td>
<td>5</td>
</tr>
<tr>
<td>15</td>
<td>F 29</td>
<td>&lt;1</td>
<td>106 F. R</td>
<td>24</td>
<td>—</td>
<td>2½</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>16</td>
<td>M 27</td>
<td>&lt;1</td>
<td>102 F.</td>
<td>24</td>
<td>—</td>
<td>1 (190/150)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>17</td>
<td>M 29</td>
<td>2</td>
<td>106 F. R &amp; T</td>
<td>17</td>
<td>—</td>
<td>1 (154/124)</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>18</td>
<td>F 17</td>
<td>1</td>
<td>104 F. R &amp; T</td>
<td>25</td>
<td>4</td>
<td>2 (160/?)</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>19</td>
<td>F 11</td>
<td>1</td>
<td>106 F.</td>
<td>4</td>
<td>5</td>
<td>2 (210/130)</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>20</td>
<td>M 15</td>
<td>&lt;1</td>
<td>105 F.</td>
<td>6</td>
<td>3</td>
<td>3 (170/?)</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>21</td>
<td>M 40</td>
<td>&lt;1</td>
<td>100 F.</td>
<td>2</td>
<td>3</td>
<td>3 (170/?)</td>
<td>25</td>
<td>25</td>
</tr>
<tr>
<td>22</td>
<td>M 30</td>
<td>2</td>
<td>104 F.</td>
<td>28</td>
<td>2</td>
<td>165/5</td>
<td>25</td>
<td>25</td>
</tr>
</tbody>
</table>

* This refers to the use of the tank respirator and/or tracheotomy prior to onset of the cardiovascular collapse. Cases 1, 5, 6, 10, 13, 19, 20, 22 went into respirators some time after that and, similarly, cases 1, 5, 6, 12, 15, 22 had tracheotomies later.
† The dose of noradrenaline was adjusted to try and maintain systolic pressure above 100 mm. Hg.
‡ Lesser grades of pulmonary edema, as evidenced by basal crepitations, were often not detected since the chest was not always examined serially.
§ Graded as indicated in the text.

almost always occurred within 48 hours of the first evidence of bulbar paralysis which, however, was not always extensive or severe. The patients were usually alert, exceedingly apprehensive and agitated. Body temperature was recorded at 104 F. or over in approximately half the cases. The pulse rate was fast during the stage of shock and often difficult to feel at the wrist, but always regular.

In table 1, the cases have been arranged according to the blood pressure findings. In cases 1 to 6, systolic pressure was below 80 mm. Hg when first recorded. Cases 16 and 17 were hypertensive on admission. The remaining cases were normotensive on admission; cases 7 to 15 remained so until their blood pressure fell, but the last four cases in the table developed transient hypertension before the stage of shock. The fall in blood pressure was sometimes precipitous but in others it fell over a period of hours. In the shock stage, the extremities were cold and clammy and mottled with peripheral grey cyanosis.

Gross clinical pulmonary edema was a notable feature in more than a third of the cases. In two of these (cases 18 and 22), who survived for several hours after the onset of pulmonary edema, the autopsy findings suggested some regression of the pulmonary edema before death. A rapidly fatal termination within a few hours was more the rule. Of the 22 cases, 5 were in respirators and five had tracheotomies at the time of the onset of the cardiovascular collapse. It can be seen from table 1 that 13 of the 22 cases were given continuous infusions of noradrenaline with variable results. It seemed of definite but temporary
Table 2.—Survivors

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex &amp; Age</th>
<th>Duration Bulbar Signs—Days</th>
<th>Max. Temp.</th>
<th>Respirator and/or Tracheotomy*</th>
<th>Blood Pressure Phases Duration in Hours</th>
<th>Duration of Noradrenaline—Hours*</th>
<th>Clinical Pulmonary Edema*</th>
<th>Final Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>23</td>
<td>F, 28</td>
<td>2</td>
<td>103 F</td>
<td>R &amp; T</td>
<td>48 Normal—20 Hypertensive—40 Hypotensive—0</td>
<td>24 24 6 6 0</td>
<td>++</td>
<td>Died 6 months later. Cause undetermined.</td>
</tr>
<tr>
<td>24</td>
<td>F, 26</td>
<td>1</td>
<td>104 F</td>
<td>R</td>
<td>20 Normal—24 Hypertensive—32 Hypotensive—0</td>
<td>24 24 6 6 0</td>
<td>+ + +</td>
<td>Died 3 months later. Secondary infection.</td>
</tr>
<tr>
<td>25</td>
<td>M, 26</td>
<td>4</td>
<td>106 F</td>
<td>R &amp; T</td>
<td>96 (170/? Normal—6 Hypertensive—6 Hypotensive—0</td>
<td>6 6 0</td>
<td></td>
<td>Remains with severe spinal paralysis.</td>
</tr>
<tr>
<td>26</td>
<td>M, 19</td>
<td>3</td>
<td>103 F</td>
<td>R &amp; T</td>
<td>24 (195/? Normal—46 Hypertensive—46 Hypotensive—0</td>
<td>?48 46 0</td>
<td></td>
<td>Remains with severe spinal and respirator paralysis.</td>
</tr>
</tbody>
</table>

* See footnotes to table 1.

![FATAL CARDIOVASCULAR COLLAPSE](image1)

![TRANIENT CARDIOVASCULAR COLLAPSE](image2)

Fig. 1. Record of rectal temperature, heart rate, systolic blood pressure and dose of noradrenaline throughout the hospital course of case 20 (table 1).

Fig. 2. Record of pulse rate, systolic blood pressure and dose of intravenous noradrenaline during the episodes of hypotension in case 23 (table 2).

benefit in six of these cases and in five of the survivors shown in table 2.

A typical example of the fulminating course is illustrated in figure 1.

There were a few cases amongst the remaining patients who died from bulbar poliomyelitis which showed some features of this syndrome, both clinically and pathologically; for example, a sharp drop in blood pressure and post-mortem evidence of pulmonary edema. These may have been examples of the same syndrome but at autopsy other causes of death were found and they have been excluded from this series.

Table 2 shows six patients who recovered from an attack of hypotension. The duration of the low blood pressure phase was sometimes difficult to assess when the patient had an infusion of noradrenaline. If the blood pressure was maintained when the infusion was temporarily stopped, the drug was discontinued. Such a test is illustrated in figure 1.

The clinical course of case 23 is illustrated in figure 2. She developed ascending spinal paralysis requiring respirator treatment. The next day a
tracheotomy was necessary for bulbar paralysis. On the day following the tracheotomy she became extremely apprehensive and had the episodes of hypotension treated with noradrenaline as shown in the figure. Following this, she remained well but severely paralyzed. She could neither swallow nor breathe. Six months later she suddenly convulsed, became unconscious and died a few days later. The central nervous system at autopsy showed marked loss of lower motor neurones in all areas as well as severe involvement of the medullary reticular substance. The heart was grossly and microscopically normal. Terminal partial atelectasis of a few hours' duration involved both lungs. The cause of death was not apparent.

Case 24 survived a severe episode of pulmonary edema but also died a few months later. On admission she had rapidly ascending spinal paralysis and went into a tank respirator. Within two hours she suddenly developed gross pulmonary edema and cyanosis. She remained alert and very apprehensive. A bronchoscope was immediately passed to facilitate removal of the frothy edema fluid and to deliver a high concentration of oxygen deep into the air passages. She was given 0.2 mg strophanthin and also a small dose of sodium pentothal to allay her apprehension. The pulmonary edema subsided in half an hour and the bronchoscope was removed after tracheotomy had been performed. Following this episode her systolic blood pressure was 100 and her pulse rate 140. The following day her systolic blood pressure fell to 80 and was returned to between 90 and 100 with an infusion of noradrenaline which was continued for 24 hours. Three days later tracheal obstruction produced a sudden fall in systolic blood pressure to 60 but this rose again a few minutes after the obstruction was relieved. During the following weeks she remained in a state of chronic ill health and she succumbed three months later to bronchopneumonia and pyopneumothorax. Autopsy examination was not allowed.

The other four survivors had not so severe a clinical course.

Cases 25 and 26 were also in respirators at the onset of cardiovascular collapse. The former was given 0.25 mg strophanthin intravenously when his systolic blood pressure fell to 90. Following this the blood pressure remained at 110 for several hours but then fell to 75 and his pulse rate rose to 170. An intravenous infusion of noradrenaline raised the blood pressure to 105 and the pulse rate slowed to 140. His course thereafter was uneventful but he has remained severely paralyzed. When case 26 developed a rapid pulse, falling blood pressure and sweating collapse, he was given intravenous strophanthin, intravenous Digoxin and oxygen without apparent effect. However, his blood pressure was elevated to 100 systolic by intravenous noradrenaline. This was continued for two days, after which blood pressure remained normal.

Cases 27 and 28 developed this shock syndrome without being in respirators although the former patient developed respiratory insufficiency shortly afterwards. His blood pressure recovered spontaneously. The blood pressure in case 28 was maintained for a day with noradrenaline. When it was discontinued his systolic blood pressure remained between 80 and 90 mm. Hg. He seemed so well and was without the cold, clammy peripheral cyanosis or the apprehension seen in the fatal cases that the drug was discontinued. He made steady improvement; his blood pressure finally came back to normal and at the time of discharge he was left with very little residual paralysis.

The occurrence of hypertension in these patients is of some interest. Six of the fatal cases (table 1) and four of those who survived (table 2) had hypertension prior to the onset of hypotension. There were other patients, not shown in the tables, who were probably examples of the same syndrome without the development of the hypotensive phase. One example will serve to illustrate this.

A 16-year old boy was admitted with a five-day history of illness and with pharyngeal and palatal paralysis of one day's duration. Temperature was 102 F, pulse 120 and respirations 25. Systolic blood pressure was 130. During the next two days his bulbar paralysis worsened but he developed practically no spinal paralysis. His blood pressure rose to 190/120. He became extremely agitated but remained quite alert. Temperature and pulse rate began slowly to subside to normal and by the eighth hospital day swallowing function began to recover and the patient felt quite well. However, his blood pressure, which had reached a height of 220/130, remained elevated above normal for 10 days, during this time it gradually subsided. At the time of discharge, three weeks after admission, his blood pressure was 125/70 and there was moderate residual weakness of the face and palate.

Pathology

The postmortem findings of the 20 cases autopsied are summarized in table 1. The main abnormal findings were limited to the central nervous system, the heart and the lungs. A few of these patients had clinically insignificant lesions of the upper gastrointestinal tract. There was no pathologic evidence of airway obstruction, bacterial infection or hemorrhage. The lungs showed a varying degree of acute pulmonary edema in all but one case.
Involvement of the Myocardium: The gross examination of the heart was usually normal although in two cases the heart muscle was considered abnormally flabby. The average heart weight was approximately 10 per cent greater than normal. This has been previously described.  

In 14 of the 20 cases autopsied, there was histologic evidence of myocarditis. Blocks for microscopic examination were taken from the anterior and posterior wall of the left ventricle, the septum and the pulmonary conus. The lesions are graded in table 1 as mild, moderate or severe. We considered lesions, mainly limited to perivascular spaces, to be mild. Those which also showed scattered focal lesions in the myocardium were considered to be moderately severe. Diffuse involvement was considered to be a severe grade. The exudate consisted of lymphocytes, plasma cells, macrophages and polymorphonuclear cells, the latter predominating in the more severe cases (fig. 3). Although the majority of the muscle fibers were usually spared, there was often involvement of a few fibers adjacent to the lesion. These fibers were swollen, fragmented, had lost their striations and showed a granular cytoplasm. Some of the fibers showed cloudy swelling and a tendency to coalesce with adjacent fibers.

Among the other 47 postmortem examinations for poliomyelitis, where the cause of death was asphyxia, gastrointestinal hemorrhage or secondary infection, 12 hearts showed a mild degree of inflammatory reaction and five had septic abscesses in the myocardium as well as in the other organs. Four of the 12 were in cases dying of septicemia and it is difficult to distinguish between a mild septic reaction and a virus inflammation in these cases. In none of these cases was the inflammatory reaction severe or moderately severe as it was in nine of the 14 cases showing myocarditis and who died with the syndrome of cardiovascular collapse.

Discussion
Several mechanisms could be responsible for inducing cardiovascular collapse in polio-
myelitis. From the data presented, some of these can be excluded as primary factors in our cases.

Destruction of the vasmotor center was considered responsible by Baker and co-workers.\textsuperscript{4} Such a lesion should lead to a general absence of sympathetic activity with postural hypotension. Although measurements of blood flow were not made, it was clinically obvious that our patients had intense vasoconstriction of the skin at least. Symptoms were not alleviated by tilting the head down. Histologically, the area of the reticular substance in the medulla was involved in all or our cases and no case of this syndrome was found in patients with spinal involvement only. The medullary area was also involved in cases dying of other causes. But death in such cases occurred at a somewhat longer interval after the onset of paralysis than did cardiovascular collapse.

Myocarditis was more common and also more severe in the cases dying of cardiovascular collapse compared with other bulbar cases coming to autopsy. It is difficult to envision a virus myocarditis limited only to cases with bulbar poliomyelitis. Indeed, electrocardiographic evidence indicates that spinal and nonparalytic cases may also have transient myocardial changes.\textsuperscript{6, 9} From these considerations and also from the fact that one-fourth of the cases did not show myocarditis, it seems unlikely that the heart lesion is the basis for this syndrome although its presence would probably aggravate it.

It has been suggested\textsuperscript{13} that all deaths from acute poliomyelitis can be attributed to hyperventilation. It is difficult to categorically deny this on our evidence. We have seen patients, other than those reported here, in terminal shock from airway obstruction. However, the majority of the present patients were not in respiratory distress at the onset of the collapse; nor were there any indications at post-mortem examination of airway obstruction or asphyxia from other causes.

Unfortunately, arterial puncture for blood gas analysis was carried out only on two of these patients during the phase of shock. Case 27 (table 2) did have a moderate respiratory acidosis (carbon dioxide partial pressure 77 mm.; pH 7.28); case 12 (table 1) had a normal arterial carbon dioxide partial pressure (45 mm.) a few hours before death although the blood pH was 7.32. Many other cases with airway obstruction were seen with severe acidosis which promptly recovered when ventilation was returned to normal.\textsuperscript{14}

The undesirable effects on the circulation of prolonged positive pressure breathing are well known.\textsuperscript{11, 15, 16} However, as only five of our patients were in respirators at the onset of cardiovascular collapse, this is unlikely to be a factor of prime importance in our cases.

Severe hyperthermia may be lethal. Approximately half of the fatal cases had fever with rectal temperatures of 104 F. or over. It is not considered likely that such a mortality rate would result from comparable body temperatures in other infectious diseases.

Although there is no reason to suspect acute adrenal insufficiency in poliomyelitis, the occurrence of lethal shock made us consider this possibility. The adrenal glands at autopsy in some cases showed a varying degree of histologic change characteristic of stress.\textsuperscript{17, 18} There was no difference in the appearance of the glands between the cases dying of cardiovascular collapse and the remaining 47 cases autopsied.

The transient hypertension of six of the fatal cases and four of the six surviving cases, as well as other cases without cardiovascular collapse, may indicate that severe vasoconstriction may be an important factor leading to heart failure and pulmonary edema.\textsuperscript{19, 20} Such a postulated mechanism would be greatly enhanced by an already weakened myocardium from viral myocarditis. Although we have no direct evidence to support this hypothesis, it is put forward as the suggested mechanism in our cases as it seems to best fit the clinical and pathologic data. Further investigation along these lines would be very helpful in confirming or refuting this suggestion.

On the basis of this tentative hypothesis about the mechanism of this syndrome, one would expect that vasoconstrictor drugs would be of no benefit in treatment. Indeed, animal experiments\textsuperscript{21} suggest that such therapy may actually be harmful in spite of temporary im-
provement in blood pressure and clinical appearance. Despite this, the use of noradrenaline seemed to be of definite benefit in some of our cases. In others, although it temporarily elevated the blood pressure, it appeared to have no beneficial influence on the course of the disease. Of the surviving cases shown in table 2, noradrenaline seemed an important factor in case 23. Case 24 was most gravely ill when she had pulmonary edema; she recovered from this before she was given noradrenaline. None of the other four survivors at any time seemed as critically ill as the fatal cases. We attribute their survival more to a mild form of the syndrome rather than to the therapy.

From the experimental work on animals, the use of hypertensive drugs in the early hypertension phase might be of value. However, we have no experience with this possible method of treatment.

**Summary**

1. A series of 22 fatal cases and 6 surviving cases of cardiovascular collapse in acute poliomyelitis is reported.

2. Clinically, the syndrome came on acutely within 1 or 2 days after the onset of bulbar paralysis. It was characterized by apprehension, hyperthermia, fast regular pulse and was sometimes preceded by a short period of hypertension. It then progressed to a state of shock with cold, sweating and cyanosed extremities. Pulmonary edema was a common terminal feature.

3. At autopsy, there was involvement of the medulla in all cases and pulmonary edema in all but one case. Interstitial myocarditis was found in 75 per cent of the cases.

4. Treatment with noradrenaline appeared to be of benefit in some cases but this was usually transient. Four of the six surviving cases had a mild form of the syndrome.

5. It is suggested that the mechanism for the syndrome is a combination of vasoconstriction resulting from a medullary lesion and a viral myocarditis. This leads to acute heart failure and pulmonary edema.

**Addendum**

Since these cases were studied we have only once encountered this syndrome. The blood pressure of an adult male, admitted with acute bulbar poliomyelitis, was 170/100 and pulse rate was 90. He rapidly deteriorated, became agitated and somewhat disoriented and 14 hours after admission was cold and clammy; the blood pressure could not be recorded; his heart rate had risen to 160. Rather than increasing his blood pressure, a hypertensive agent, Arfonad, was given by continuous intravenous infusion for 26 hours at a rate which maintained the systolic blood pressure between 70 and 80 and the diastolic between 60 and 65. His color and general condition improved markedly. When the drug was stopped his blood pressure remained normal although at the present time he is still in the acute stage of his disease.

This single case seems to corroborate the discussion above but further experience is obviously necessary before conclusions can be drawn.

**Acknowledgments**

The technical assistance of Miss K. Nagy and Mrs. E. Pullen is gratefully acknowledged.

**Summary in Interlingua**

Collapso cardiovascular esseva responsabile pro circa duotertios del mortos in duo epidemias de poliomyelitis acute. Iste syndrome occurriva testo in le curso del morbo e solmente in casos de involvensimento bulbar. In omne casos il habeva un involvimento morphologic del medulla, e in 75 pro cento del casos varie grados de myocarditis esseva observate. Le frequentia e le severitate de myocarditis esseva multo minus extense inter le casos de mortes per altere causas. Nos presenta le hypotheses que le combination de (1) vasoconstriction continuo resultante del lesion medullar e (2) le presentia de myocarditis interstital es le causa de disfallimento circulatori e edema pulmonar.

**References**


*Trimethaphan camphor sulphonate from Hoffmann-LaRoche.*


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