Norepinephrine in Shock Following Myocardial Infarction

Influence upon Survival Rate and Renal Function

By John J. Sampson, M.D., and Albert Zipser, M.D.

A clinical study is presented of the influence of continuous intravenous norepinephrine therapy upon shock accompanying myocardial infarction. The significant reduction in immediate and eventual mortality rates usually associated with this condition are discussed. Complications are defined, and a possible first reported instance is presented of ventricular tachycardia developing in the presence of complete heart block during the administration of large doses of norepinephrine.

SEVERE and sustained hypotension complicating myocardial infarction is correlated with a fatality rate\(^1\) between 80 per cent and 90 per cent. It may be assumed that hypotension and the accompanying inadequate coronary circulation are detrimental to the functional integrity of the myocardium and therefore warrant prompt correction. The various therapeutic measures employed until recently have not provided relief of marked sustained hypotension, nor have they substantially influenced the high fatality rate. This report presents an evaluation of the influence of continuous norepinephrine therapy upon immediate and eventual survival from shock occurring in patients with myocardial infarction.

METHODS AND MATERIALS

Criteria for Therapy. The plan for treating patients in shock was to administer phenylephrine hydrochloride (Neo-Synephrine) or other pressor amines by the intramuscular or intravenous route intermittently as indicated. If the systolic blood pressure was elevated to above 100 mm. Hg and satisfactorily maintained, further therapy was withheld. On recurrence of shock similar medication was employed through one to three episodes. Norepinephrine by continuous drip was instituted if (1) the patient was known to have been in shock longer than one hour, (2) the initial episode of shock was severe with systolic pressure below 80 mm. Hg for at least 15 minutes, (3) more than three transient episodes of shock had occurred within 12 hours, (4) or if Neo-Synephrine proved ineffective in relieving any episode of shock.

Pharmacology. L-Norepinephrine has the following pharmacologic effects:

1. Constriction of peripheral arteries, capillaries and veins,\(^2\) an effect which is probably not mediated through neural pathways or via the adrenal cortical hormones.
2. Slowing of the heart rate when the vagi are intact and increasing the rate when the vagi are blocked.\(^3\)
3. Increasing the coronary artery blood flow, resulting in elevation of oxygen content of coronary sinus blood and of infarcted myocardium.\(^4\)
4. No significant change in cardiac output.\(^1\)
5. Decreasing cerebral blood flow.\(^6\)
6. Induction of ectopic ventricular arrhythmias in complete heart block and other conditions which are largely experimental.\(^7\)
7. Alteration of renal function.\(^8\)
   a. Depression of sodium and potassium excretion.
   b. Constriction of efferent renal arterioles with resultant reduction of the total renal blood flow but increased glomerular filtration fraction.
   c. Increased urine formation.

The pharmacologic actions most likely to cause concern in the prolonged use of this drug, especially in high concentration, were increased cardiac work precipitating heart failure, gross impairment of renal function, and induction of ventricular fibrillation or tachycardia. One possible example of the latter was observed in this series.

Administration. Norepinephrine was administered intravenously after diluting 4 mg. of the bitartrate monohydrate (Levophed\(^*\)) in 1000 ml. of 5 per cent aqueous solution of dextrose. The use of solutions containing sodium chloride was avoided because of

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* Levophed supplied through the courtesy of Winthrop-Stearns, Inc. New York, N. Y.
the renal retention of sodium in patients with myocardial infarction. The flask was connected to one arm of an “Y” tube, the other arm being clamped off. A pressure clamp controlled the rate of flow which was observed in the filter chamber. When prolonged administration was necessary a polyethylene tube was threaded into the vein in place of a needle. Care was used to avoid and limit extravasation of the solution into the subcutaneous tissues.

The flow was started at approximately 10 drops or 2.5 micrograms of norepinephrine per minute. Blood pressures were determined at intervals of 30 to 60 seconds initially, and after each change in rate of flow until the response was apparent and stabilized. Rise of the blood pressure was often abrupt and pronounced necessitating immediate readjustment of the flow.

The rate of administration was regulated to maintain the systolic blood pressure at about 100 mm. Hg which was usually adequate to abolish the manifestations of shock. In previously hypertensive patients a systolic pressure of 110 to 125 mm. Hg was sought. When therapy was prolonged, the output of an adequate volume of urine was an added criterion of appropriate response.

If the pressor response was inadequate or could be maintained only with a rate of flow exceeding 40 drops per minute, a higher concentration of norepinephrine was administered from a second flask connected to the other arm of the “Y” tube. Concentrations varying from 2 to 32 mg. per liter and doses of 1.5 to 150 micrograms per minute were used depending upon the necessities of maintaining the blood pressure, avoiding excessive fluid intake and preventing clot formation in the needle or catheter resulting from very slow rate of flow.

Blood pressures and flow rates were determined every 15 minutes for at least three hours and every 30 minutes for the remaining period of this therapy. When discontinuance of the drug was contemplated, and after trial on a lowered dosage, an infusion of 5 per cent dextrose solution was maintained for several hours to permit rapid resumption of the pressor therapy if shock returned. Although norepinephrine deteriorates rapidly in an alkaline medium, Ringer’s solution and citrated blood were occasionally administered simultaneously through the “Y” tube from the accompanying flask without loss of the pressor effect.

Records were kept of the concentration of the drug, rate of flow in drops per minute, blood pressure, pulse rate and rhythm, fluid intake and urine output, and untoward responses. Renal function studies included the measurement of water balance and, in some cases, determination of renal blood flow via para-aminobenzene acid clearances and glomerular filtration rate by means of endogenous creatinine clearance tests.

**Results**

Prolonged intravenous norepinephrine therapy was given to 30 patients, ages 43 to 84 (table 1). All had clinical and electrocardiographic evidence of recent myocardial infarction, 19 with involvement of the anterior wall and 11 with involvement of the posterior wall. There were 19 males and 11 females. Five of the patients were previously hypertensive. Infarction occurred in three cases during or shortly after major surgical operations. In one patient shock followed the intravenous administration of procaine amide for ventricular tachycardia.

Fourteen patients experienced recurrent episodes of hypotension with transient recovery in three following intravenous blood transfusions and in eight following Neo-Synephrine injections. Sixteen had been in persistent shock for longer than one hour or were in very severe shock. All patients treated exhibited systolic blood pressures of 80 mm. Hg or below except for four previously hypertensive individuals who manifested shock with systolic blood pressures between 88 and 100 mm. Hg.

Initial therapy consisted of 500 ml. intravenous blood transfusions in five cases, three with transient benefit and two without apparent effect. All five of these responded subsequently to norepinephrine. Intermittent doses of Neo-Synephrine in 16 cases provided transient elevation of the blood pressure in all but four. Three of the latter and all of the former responded to sustained norepinephrine infusions.

Twenty (67 per cent) of the 30 patients survived the episode of shock under norepinephrine therapy, and 16 (53 per cent) recovered clinically and were later discharged from the hospital. Four patients died subsequent to successful treatment of the shock, one from cardiac rupture eight hours later, two others abruptly 4 and 18 days later, and one from Stokes-Adams attacks (ventricular fibrillation) 19 days later. Ten patients (33 per cent) died during norepinephrine therapy (table 2). Seven of these manifested pressor responses, and the other three exhibited hypo-
tension resistant to doses as high as 60 micrograms per minute.

Certain observations of the norepinephrine therapy are summarized in table 3.

1. With one exception the duration of shock prior to treatment was over twice as great in the patients who died as in those who survived.

2. Seven of the 10 patients who died during therapy did so within six hours of the start of treatment. The exceptions were one who relapsed into shock when the flow stopped after 70 hours of successful therapy and failed to respond to reinstituted treatment, a second who died abruptly after 181 hours of continuous therapy, and a third who died of ventricular fibrillation. The survivors required from three hours to five and one-half days of continuous treatment.

3. The average dose needed to elevate the blood pressure initially was more than two times as great in those that died as in the survivors.

4. The average maximum dose needed in attempts to maintain the blood pressure in the fatal cases was over five times as large as in the survivors.

Examples of some characteristic findings are demonstrated by the following five cases.

Case 8 (fig. 1) illustrates the transient effect of Neo-Synephrine and the prompt response to 3 to 4 micrograms per minute of norepinephrine. The pressure fell rapidly when the flow was obstructed, but responded promptly again when therapy was restored. In general a stable level of blood pressure was maintained by minor adjustments in dosage to correct the tendency of the pressure to fluctuate. The blood pressure was sustained at a safe level without norepinephrine after a two-hour trial of very low dosage. The pressure did not rise with a con-
constant dose to indicate that the drug was no longer required.

Case 9 (fig. 2) demonstrates the pattern of a patient who had been in shock intermittently for 14 hours with four significant but transient pressor responses to Neo-Synephrine. The instability of the natural mechanisms controlling blood pressure necessitated continual readjustment of dosage through five and one-half days of therapy. This case represents the unusual example in which a rising pressure is observed on a fixed or decreasing dose indicating that need for the drug was diminishing. In the majority of the patients, continued need for the drug could only be determined by cautiously lowering the dose and observing whether the blood pressure fell.

Case 25 (fig. 3) illustrates the requirement of a large dose (25 micrograms per minute) to maintain a satisfactory and stable blood pressure level. The initial episode of shock followed the intravenous administration of 200 mg. procaine amide for ventricular tachycardia and did not respond to Neo-Synephrine. To prevent recurrence of the paroxysmal ventricular tachycardia, a slow infusion of the procaine amide was then maintained. In spite of this therapy a second episode of the arrhythmia developed, and a precipitous fall in pressure followed the supplementary injection of 250 mg. procaine amide. Both times the blood pressure rose from undetectable levels to satisfactory levels with the use of norepinephrine. Death was due to abrupt ventricular tachycardia and fibrillation. In view of the occurrence of the initial episode prior to therapy, this instance of arrhythmia should not properly be attributed to norepinephrine toxicity.

Case 14 (fig. 4) illustrates extreme instability and wide fluctuation of the blood pressure despite a constant and moderately large dose. The possible stabilizing effect of 150 mg. cortisone orally is demonstrated as is the reduction in dose of norepinephrine required. Cessation of pressor therapy resulted in gradual return of shock but re instituted therapy re-

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Table 1.—Continued

<table>
<thead>
<tr>
<th>Case</th>
<th>Maximum Dose/min. (Microgram)</th>
<th>Duration Therapy (Hours)</th>
<th>Total Dose mg.</th>
<th>Results*</th>
<th>Remarks</th>
</tr>
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<tbody>
<tr>
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<td>11</td>
<td>28½</td>
<td>13</td>
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</tr>
<tr>
<td>2</td>
<td>14</td>
<td>69</td>
<td>22</td>
<td>A</td>
<td>Onset infarct during surgery</td>
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<tr>
<td>3</td>
<td>16</td>
<td>40</td>
<td>19</td>
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<td>Onset postoperatively</td>
</tr>
<tr>
<td>4</td>
<td>9</td>
<td>46</td>
<td>18</td>
<td>A</td>
<td>Phlebitis</td>
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<td>5</td>
<td>15</td>
<td>69</td>
<td>36</td>
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<td>5</td>
<td>11½</td>
<td>4</td>
<td>A</td>
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</tr>
<tr>
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<td>4</td>
<td>3½</td>
<td>1.6</td>
<td>A</td>
<td>None</td>
</tr>
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<td>8</td>
<td>4</td>
<td>66½</td>
<td>12</td>
<td>A</td>
<td>None</td>
</tr>
<tr>
<td>9</td>
<td>5</td>
<td>136</td>
<td>26</td>
<td>A</td>
<td>Phlebitis</td>
</tr>
<tr>
<td>10</td>
<td>10</td>
<td>119</td>
<td>64</td>
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<td>Onset postoperatively</td>
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<td>12</td>
<td>8</td>
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<td>4</td>
<td>A</td>
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<td>24</td>
<td>87</td>
<td>67</td>
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<td>14</td>
<td>15</td>
<td>49</td>
<td>39</td>
<td>A</td>
<td>Cortisone potentiation</td>
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<tr>
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<td>8</td>
<td>7</td>
<td>4</td>
<td>A</td>
<td>None</td>
</tr>
<tr>
<td>16</td>
<td>12</td>
<td>15½</td>
<td>10</td>
<td>A</td>
<td>Phlebitis</td>
</tr>
<tr>
<td>17</td>
<td>6</td>
<td>30</td>
<td>7</td>
<td>B</td>
<td>Died 18 days later</td>
</tr>
<tr>
<td>18</td>
<td>6</td>
<td>34</td>
<td>10</td>
<td>B</td>
<td>Died 19 days later</td>
</tr>
<tr>
<td>19</td>
<td>4</td>
<td>16</td>
<td>4</td>
<td>B</td>
<td>Died 4 days later</td>
</tr>
<tr>
<td>20</td>
<td>12</td>
<td>4</td>
<td>4</td>
<td>B</td>
<td>Cardiac rupture 8 hrs. later</td>
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<tr>
<td>21</td>
<td>150</td>
<td>181</td>
<td>420</td>
<td>C</td>
<td>Ventricular tachycardia</td>
</tr>
<tr>
<td>22</td>
<td>20</td>
<td>1</td>
<td>0.8</td>
<td>C</td>
<td>None</td>
</tr>
<tr>
<td>23</td>
<td>5</td>
<td>1½</td>
<td>0.5</td>
<td>C</td>
<td>Recurrent Stokes-Adams</td>
</tr>
<tr>
<td>24</td>
<td>12</td>
<td>6</td>
<td>4</td>
<td>C</td>
<td>Abrupt death</td>
</tr>
<tr>
<td>25</td>
<td>25</td>
<td>11½</td>
<td>12</td>
<td>C</td>
<td>Procaine amide caused shock</td>
</tr>
<tr>
<td>26</td>
<td>32</td>
<td>70</td>
<td>92</td>
<td>C</td>
<td>Died when flow ceased</td>
</tr>
<tr>
<td>27</td>
<td>16</td>
<td>5</td>
<td>4</td>
<td>C</td>
<td>B.P. 120/118 with therapy</td>
</tr>
<tr>
<td>28</td>
<td>24</td>
<td>1½</td>
<td>8</td>
<td>D</td>
<td>No pressor effect</td>
</tr>
<tr>
<td>29</td>
<td>60</td>
<td>5</td>
<td>8</td>
<td>D</td>
<td>No pressor effect</td>
</tr>
<tr>
<td>30</td>
<td>35</td>
<td>4½</td>
<td>7</td>
<td>D</td>
<td>No pressor effect</td>
</tr>
</tbody>
</table>

* A = Survived shock, clinical recovery; B = Survived shock, died later of other causes; C = Died during therapy despite pressor response; D = Died during therapy, no pressor response.
stored the pressure appropriately. In previous experiences with cortisone alone, doses of 300 mg. daily were ineffective in correcting hypotension following myocardial infarction. However, in one patient who had been refractory to Neo-Synephrine, a pressor response to this drug was exhibited four hours after receiving 150 mg. cortisone orally. It has been shown experimentally that cortisone sensitizes the contractile elements of the blood vessels to the pressor drugs, and potentiates their effect.

Case 21. This patient required exceptionally large doses of norepinephrine to sustain the blood pressure. The electrocardiogram demonstrated the pattern of a posterior myocardial infarction and complete A-V block with a ventricular rate of 48. The complete heart block persisted throughout the patient's illness. During the fourth day of continuous norepinephrine therapy ventricular tachycardia supervened while the drug was being administered at the unusually high but necessary rate of 150 micrograms per minute. Quinidine controlled the paroxysm which did not return despite similar high doses of norepinephrine during the succeeding three days. This represents the only instance in our series of a ventricular arrhythmia which might be attributed to norepinephrine toxicity, though such have been demonstrated in the experimental animal.

Renal Function

Urine Volume. In no instance did urine volume exceed 20 ml. per hour during the observed period of shock. Of the patients that responded to norepinephrine, only one exhibited oliguria (21 ml. per hour) during the therapy. The remainder showed adequate urine output, averaging 70 to 110 ml. per hour. There was generally a 20 per cent to 50 per cent positive water balance.

Although oliguria, possibly under hormonal influence, is a common complication of myocardial infarction, our patients showed a striking improvement in urine output during norepinephrine therapy, presumably due to elevation of effective filtration pressure. Under similar conditions of hydration, the urine flow in three cases during norepinephrine therapy and several days afterward was approximately identical, namely 1.64 and 1.78 ml. per minute, 1.42 and 1.25 ml. per minute, and 4.1 and 4.4 ml. per minute, respectively.

Clearances. The para-aminobenzoic acid clearances were lower during norepinephrine therapy than subsequent to the infusions despite similar levels of blood pressure and urine minute volumes. In three patients the renal plasma flow measured by this test rose from 120.5 to 157 ml. per minute (31 per cent), from 300 to 384 ml. per minute (28 per cent), and from 355 to 509 ml. per minute (42 per cent), respectively, after stopping the therapy. Glomerular filtration rates were approximately the same during and after therapy, namely 30 and 36 ml. per minute, 68 and 63 ml. per minute, and 54 and 58 ml. per minute. This conforms with previous observations that while renal plasma flow is reduced, glomerular filtration rate is essentially unchanged. A higher filtration fraction results from the apparent efferent arteriolar constriction. No clinical evidence of renal functional defects were observed nor was azotemia present even after five and one-half days of continuous norepinephrine therapy in another patient. The low creatinine clearances in all three of the above patients suggests that primary renal disease, probably nephrosclerosis, existed.

<table>
<thead>
<tr>
<th>Table 2.—Results of Norepinephrine Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical Recovery</strong>—16 (63%)</td>
</tr>
<tr>
<td><strong>Subsequent Death</strong>—4 (13%)</td>
</tr>
<tr>
<td><strong>Death During Therapy</strong>—10 (33%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 3.—Comparative Observations in Surviving and Fatal Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Norepinephrine in Myocardial Infarction</strong></td>
</tr>
<tr>
<td><strong>Prior Duration of Shock</strong></td>
</tr>
<tr>
<td>(53%)</td>
</tr>
<tr>
<td><strong>Duration of Therapy</strong>—3–136 Hrs. (Ave. 59.5)</td>
</tr>
<tr>
<td><strong>Initial Effective Dose</strong>—2½–20 µg./Min. (Ave. 7)</td>
</tr>
<tr>
<td><strong>Max. Dose Needed</strong>—1½–24 µg./Min. (Ave. 7.4)</td>
</tr>
</tbody>
</table>
Fig. 1. Case 8. Therapy and blood pressure

Fig. 2. Case 9. Therapy and blood pressure
NOREPINEPHRINE IN SHOCK FOLLOWING MYOCARDIAL INFARCTION

Fig. 3. Case 25. Therapy and blood pressure

Fig. 4. Case 14. Therapy and blood pressure
Complications

Congestive heart failure was not observed to increase in any of our cases during the therapy. Two instances of arrhythmia were recorded. Norepinephrine probably bore no causal relationship to one (case 25) as noted above. In the other patient (case 21) high doses of norepinephrine may be incriminated in the precipitation of ventricular tachycardia in the presence of complete heart block. Six patients developed mild phlebitis localized above the site of venous infusion. One patient had a large slough of skin and subcutaneous tissue where the solution leaked around a polyethylene tube in a vein of the lower leg. A skin graft was necessary in this case.

Discussion

The mechanism of the shock-like state complicating myocardial infarction has not been completely explained. There are probably both cardiac and peripheral vascular components involved.\(^\text{14, 15, 16}\) Systolic ballooning of the ventricular myocardium seems to be related to the shock and is corrected by its relief.\(^\text{17}\) Hypotension may follow immediately after the onset of the infarct, in which case about 50 per cent of the patients recover spontaneously within one hour, or it may be delayed for several hours to several days. The delayed gradual onset of shock following myocardial infarction has been described\(^\text{14}\) as the clinical manifestation of progressive failure of the infarcted left ventricle and as almost invariably fatal. In our series, 16 of the 20 patients who recovered from episodes of shock with norepinephrine therapy developed the severe hypotension five hours or more after the infarction, and nine of these were delayed for more than 24 hours.

The division of patients with shock into two categories, those with low venous pressure and those with elevated venous pressure,\(^\text{16}\) for purposes of different therapy is not borne out in this study. The former was attributed to peripheral vascular or neurogenic reflex mechanisms and was treated with pressor agents, whereas the latter was presumed to reflect profound heart failure and was treated by rapid digitalization, pressor agents being deemed contraindicated here. In our series, several patients in shock with elevated venous pressures responded satisfactorily to norepinephrine pressor therapy alone.

The necessity for promptly correcting this shock state is becoming increasingly apparent, but it is difficult to evaluate the effect of therapy upon immediate and eventual recovery, because patients may exhibit early spontaneous improvement. Withholding specific treatment may permit the exhibition of this spontaneous response. However, if hypotension is severe, such delay may result in irreparable damage to other poorly irrigated areas of myocardium or in "irreversible" shock. It is apparent, therefore, that severe shock should be treated promptly.

The therapeutic measures employed until the past two years included rapid intravenous or intra-arterial infusions of blood or plasma, and individual or intermittent doses of pressor amines: ephedrine, Paredrine, Propadrine, desoxyephedrine, Neo-Synephrine and Mephentermine. Mephentermine, in addition, has been employed in a continuous slow intravenous drip for one-half to one and one-half hours.\(^\text{1}\) None of these measures appeared to induce arrhythmias or aggravate congestive heart failure. However, their influences were necessarily transient, and their chief value was in bridging the critical period of one or more episodes of severe hypotension.

In personal experiences with intravenous and intra-arterial infusions of blood and plasma,\(^\text{18}\) beneficial results rarely occurred if shock had existed for longer than four hours. In other series\(^\text{19}\) a significant percentage of favorable responses are reported to most forms of routine therapy for such cases of shock, provided treatment was instituted within three hours. Thus in shock accompanying myocardial infarction, as in surgical or hemorrhagic shock, an apparently irreversible state often develops after several hours. However, in spite of the frequency of this observation, such irreversibility after prolonged shock is not a constant finding. Patients in this series who had been in shock for three or more hours were often resistant even to con-
Continuous norepinephrine therapy. However, of those who responded and recovered, nearly 40 per cent had been in shock longer than three hours, and one for 25 hours before coming under our care. The mechanisms resulting in “irreversible” shock may be self correcting if sufficient time is given for recovery. Such recovery may be quite abrupt after days of virtually artificial maintenance of blood pressure.

Persistent shock could not be attacked effectively with previous methods of intermittent therapy. The introduction of norepinephrine in a sustained intravenous drip by Kappert presented a more rational means of treating shock from various causes. Subsequent studies demonstrated effective application of such therapy to shock accompanying myocardial infarction. Other pressor amines may prove equally satisfactory for sustained intravenous therapy.

Conclusions and Summary

1. A total of 20 (67 per cent) of the 30 patients with shock accompanying myocardial infarction recovered from the episodes of shock under continuous intravenous norepinephrine therapy. Sixteen (53 per cent) recovered completely and were later discharged, while four died of other causes subsequent to the therapy. This is apparently a significant reduction in the fatality rate from reported series of comparable cases in which various forms of intermittent therapy had been used.

2. The average dose of norepinephrine required to elevate and sustain the blood pressure was 7.5 micrograms per minute, although optimal response in different patients was generally obtained with doses varying from 1.5 to 25 micrograms per minute. Rarely do patients respond to higher doses if 25 micrograms per minute proves unavailing; however, in isolated instances doses as high as 150 micrograms per minute have been found necessary. Hence these larger doses should be tried before concluding that the patient is refractory to the treatment.

3. Complications were not unduly consequential, considering the serious nature of the condition under treatment. Varying degrees of tissue necrosis may result from extravasation of norepinephrine into the subcutaneous tissues, however this has not warranted interdicting the therapy. Phlebitis and venous spasm had no significant influence on recovery. No instance was observed suggesting the precipitation or aggravation of congestive heart failure by norepinephrine. In one patient with complete heart block, ventricular tachycardia developed during the administration of large doses (150 micrograms per minute). Further observations of the effects of such doses in comparable cases are necessary to determine the existence of a causal relationship.

4. Renal plasma flow was diminished and glomerular filtration rate essentially unchanged by infusion of norepinephrine. No sequel of renal damage was demonstrated following prolonged therapy. The resultant satisfactory level of blood pressure, as compared with that in the shock state, improves total renal function as evidenced by the prompt increase of urine flow.

5. The continuous administration of this pressor agent with the attendant high percentage of recovery suggests a modification of previous concepts of “irreversible” shock. The artificial maintenance of blood pressures by this means apparently permits the organism to survive a critical period until the natural mechanisms for maintaining the blood pressure are resumed. This restitution was more often abrupt than gradual and occurred from several hours to five and one-half days after starting therapy.

6. The damage to the heart and other vital organs from shock persistent longer than three hours as well as from recurrent episodes of severe hypotension is illustrated by the greater mortality of such cases even with norepinephrine therapy. It is suggested that the early use of intravenous norepinephrine drip therapy in shock accompanying myocardial infarction may demonstrate even further the life saving potential of this drug.

Sumario Español

Se presenta un estudio clínico de la influencia del uso continuo de norepinefrina intravenosa en la terapia del choque que acompaña el...
infarto del miocardio. La reducción significativa en la mortalidad inmediata y eventual usualmente asociada con esta condición se discute. Las complicaciones se definen, y un posible primer caso se informa de taquicardia ventricular desarrollándose en presencia de un bloqueo cardíaco completo durante la administración de grandes dosis de norepinefrina.

REFERENCES

Norepinephrine in Shock Following Myocardial Infarction: Influence upon Survival Rate and Renal Function
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