Urinary Catecholamine Excretion in Myocardial Infarction

By K. DAVID HAYASHI, M.D., ARTHUR J. MOSS, M.D., AND PAUL N. YU, M.D.

SUMMARY

The catecholamine excretion rates in urine were studied in three groups of patients with acute myocardial infarction. Group I patients had no complications. Group II patients had minor complications. In groups I and II urine was obtained during the first night of the patient’s admission. Group III patients had major complications, and urine was collected during one episode of complications. Catecholamines were measured by spectrophotofluorometric assay using the trihydroxyindole method.

A lack of elevation in basal catecholamine excretion in patients without complications and an elevated catecholamine excretion in those with complications suggest that myocardial necrosis or ischemia per se is not responsible for increased catecholamine excretion and that the increase in catecholamine excretion may reflect the presence of complicating factors.

Recurrence or persisting chest pain, pulmonary congestion or edema, and ventricular arrhythmias were almost always associated with elevated catecholamine excretion. Although in some cases a causal relationship of these complications with observed increase in catecholamine excretion is suggested, further study is required for a definitive conclusion.

Additional Indexing Words: Diurnal variation Chest pain Pulmonary congestion Ventricular arrhythmias

INCREASED urinary catecholamine excretion has been reported in many patients with acute myocardial infarction.1-5 Patterns of 24-hour excretion of catecholamines during the hospital course of such patients have been described, and elevated catecholamine levels were usually accompanied by complicating clinical events.4,5

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Experimental studies in dogs after coronary artery ligation demonstrated that the infarcted myocardium lost 75% of its catecholamine content within the first 24 hours.6 Thus, it is possible that increased catecholamine excretion in acute myocardial infarction is due to the release of catecholamine from the infarcted tissue. Gazes and co-workers,7 however, reported elevation in plasma catecholamine levels following acute myocardial infarction as well as in angina pectoris. This finding suggests that an elevation in catecholamine levels following acute myocardial infarction reflects the body’s reaction to the pathophysiologic state rather than catecholamine release from the necrotic, ischemic myocardium.

Since increased urinary excretion of free norepinephrine has been described in patients with congestive heart failure,8 this condition is another possible cause of increased catecholamine excretion when infarction is complicated by left ventricular failure.
The association of catecholamine administration and serious ventricular arrhythmias is widely recognized in both clinical and experimental studies. A close relation between increased catecholamine excretion and development of cardiac arrhythmias has been suggested in patients with acute myocardial infarction.\(^4\)\(^,\)\(^5\)

It is the purpose of this paper to examine the urinary excretion rate of catecholamines in patients with acute myocardial infarction and to analyze the factors which may increase urinary catecholamine excretion. Special attention is directed to the incidence of heart failure and cardiac arrhythmias in relation to catecholamine excretion rates.

Methods

Twenty-eight patients with acute myocardial infarction admitted to the Coronary Care Unit of the University of Rochester Medical Center were studied. The diagnosis was based upon any two or all three of the following criteria: (a) typical clinical history, (b) classical serial electrocardiographic changes, and (c) significant alterations in serum glutamic-oxaloacetic transaminase (SGOT) level. Those who had received catecholamines or sympathomimetic drugs prior to, or during, the collection of urine were excluded.

Patients were included in the appropriate groups according to the following features:

**Group I**

This group of six patients had all the following features: (1) no recurrent or persisting chest pain; (2) no pulmonary congestion on chest film; (3) no prior diuretic therapy; and (4) minor or no cardiac arrhythmias. In all but one patient 6-foot chest films were obtained for evaluation of the pulmonary congestion. An anteroposterior roentgenogram was taken while the subject was supine. Minor cardiac arrhythmias if present, consisted of sinus bradycardia (less than 60 beats/min), wandering atrial pacemaker, or rare ventricular premature contractions. Arrhythmia was monitored by continuous visual inspection and by direct recording of the arrhythmic episode when observed.

**Group II**

One or more of the following features were present in this group of 14 patients: (1) physical and mental agitation manifested by no sleep, increased physical activity in bed, and verbal expressions; (2) recurrent or persisting chest pain; or (3) prior diuretic therapy for pulmonary congestion. In some patients minor arrhythmias similar to those observed in group I were present.

**Group III**

In this group of nine patients, one or more of the following features were present: (1) pulmonary edema; (2) ventricular tachycardia; (3) pulmonary embolism; (4) multiple ventricular premature contractions (more than 5/min); (5) marked sinus tachycardia (more than 110/min); or (6) supraventricular tachycardia. The grouping of the patients was made according to the aforementioned criteria independently by two authors and agreement was achieved in every patient. Patient U.P. was admitted twice because of acute myocardial infarction. The first admission was uneventful, while the second admission was complicated by recurrent chest pain. She was included, therefore, under group I on the first, and under group II on the second admission.

**Collection of Urine**

Samples of urine were obtained during the first hospital night from patients in groups I and II. Daytime samples of urine were also collected on the following day from four group I and seven group II patients. To correlate the effect of major clinical events and the rate of catecholamine excretion, urine samples were collected at the time of complications in group III patients.

For the purpose of comparison, urinary catecholamine excretion rates were also determined in (1) a group of five patients (group IV) with coronary insufficiency but no complications and (2) another group of five patients (group V) having other types of cardiac disease with major complications similar to those seen in group III patients with acute myocardial infarction. Urine was collected on the first hospital night from group IV patients and during the course of the complication, from group V patients.

Because of diurnal variations and effect of physical activity on catecholamine excretion, the data in three control groups (A, B, and C) were included. Group A consisted of nine patients on the ophthalmology service from whom overnight urine specimens were collected. Group B consisted of the same nine patients as group A from whom samples of urine were obtained during rest in bed in the daytime. Group C included seven normal laboratory workers from whom urine was collected during their working hours.

None of the patients had clinical manifestations of obstruction to urinary flow. Urine was voided spontaneously in all but a few patients. It was assumed that complete emptying of the urinary bladder was accomplished in each patient. The amount of urine was measured immediately, and an aliquot of 20 ml was put in a
Urinary Catecholamine Excretion

graduated test tube with added sodium metabisulfite in an amount of 0.5 mg/ml of urine. The exact time of urination was recorded to the minute with each sampling. Samples were frozen until the time of assay.

Catecholamine Assay

Assays of catecholamines were performed by the trihydroxyindole method described by Anton and Sayre with some modifications. Aluminum adsorption and elution of catecholamines were performed in test tubes rather than in beakers. Measurements of fluorescence were performed on an Amino-Bowman spectrophotofluorometer and slit arrangement no. 4 using an IP28 photomultiplier tube. Double-distilled de-ionated water was used throughout. Differential analysis of norepinephrine and epinephrine was made by fluorescence measurement at two different sets of excitation and emission wavelengths. Recovery rates were checked with each run of the assay procedure. The average recovery rate of 34 consecutive specimens of urine for norepinephrine was 38.7%. On the same sample of urine, the standard deviation of the recovery rate was 3.6%. The differences between six duplicate determinations for norepinephrine varied from 0.5 to 3.8 with a mean of 2.05 ng/min. The corresponding values for epinephrine were 0.3, 1.9, and 1.02 ng/min. Since recovery rates for epinephrine did not differ from those for norepinephrine, the value for norepinephrine was used for the calculation of both norepinephrine and epinephrine. Final values were corrected for recovery rate and expressed as excretion rate in nanograms (ng) per minute.

The t-test was employed for statistical analysis.

Results

Values from three control groups are shown in figure 1. The mean norepinephrine level varied from 10.8 ng/min during the night to 31.8 ng/min during the day, whereas the corresponding levels for epinephrine were 3.8 ng/min and 9.2 ng/min. In figure 2, the values for group I and II patients are compared with those for control group A. The values of group III patients are shown in figure 3. Data from individual patients in groups I, II, and III are summarized in tables 1 to 3, respectively.

No significant elevation in the excretion rates of either norepinephrine or epinephrine was noted in group I patients when compared with those observed in control group A. However, group II patients had elevated mean

![Figure 1](image1)

**Figure 1**
Control values, $N$ = norepinephrine; $E$ = epinephrine; various symbols and horizontal bars $=$ mean and standard error of the mean respectively. Numbers in parentheses indicate the number of cases. Note the diurnal change in catecholamine excretion (compare control A and control B).

![Figure 2](image2)

**Figure 2**
Basal catecholamine excretion rates in patients without complications and those with minor complications. While there is no significant increase in catecholamine excretion in uncomplicated patients, a significant increase in $N$ excretion rate is observed in patients with minor complications compared with the controls. Symbols and abbreviations are the same as in figure 1.
Table 1

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>N</th>
<th>E</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>G.B.</td>
<td>45</td>
<td>M</td>
<td>20.3</td>
<td>3.2</td>
<td>Lidocaine drip for rare PVCs primarily because of history of myocardial infarction complicated by ventricular tachycardia</td>
</tr>
<tr>
<td>J.C.</td>
<td>57</td>
<td>M</td>
<td>17.5</td>
<td>1.1</td>
<td>Atropine given for wandering atrial pacemaker. Hypertension in past</td>
</tr>
<tr>
<td>L.C.</td>
<td>55</td>
<td>M</td>
<td>5.6</td>
<td>4.8</td>
<td>Atropine given for bradycardia. History of MI</td>
</tr>
<tr>
<td>F.F.</td>
<td>72</td>
<td>F</td>
<td>15.9</td>
<td>3.7</td>
<td>No complications. History of MI</td>
</tr>
<tr>
<td>I.M.</td>
<td>71</td>
<td>M</td>
<td>24.2</td>
<td>2.3</td>
<td>Lidocaine given on admission for PVCs with prompt suppression. No complications when studied. Essential hypertension</td>
</tr>
<tr>
<td>U.P.</td>
<td>57</td>
<td>F</td>
<td>21.1</td>
<td>9.6</td>
<td>No complications</td>
</tr>
</tbody>
</table>

**Abbreviations:**
- N = norepinephrine
- E = epinephrine
- PVCs = premature ventricular contractions
- SEM = standard error of the mean
- MI = myocardial infarction

Catecholamine excretion rates in patients with acute myocardial infarction and major complications. A marked increase in both norepinephrine and epinephrine excretion rates is found in the group III patients compared with the controls. Note also the scale is different from that in figures 1 and 2. Symbols and abbreviations are the same as in figure 1.

Discussion

Group I patients were clinically uncomplicated except for minor cardiac arrhythmias in a few, in whom the chest pain had subsided.
and required no further analgesics at least several hours prior to the urine collection period. In contrast, group II patients had complicating factors including recurrent or persisting chest pain at the time of study.

The amount of catecholamines found in the samples of night urine collected from group I patients was considered to represent the basal level of excretion. The excretion rates of both norepinephrine and epinephrine were not increased on the first day in this group of patients. Furthermore, in three additional uncomplicated patients, normal catecholamine excretion rates were observed on the third hospital day. These findings suggest that uncomplicated acute myocardial infarction per se does not cause a significant increase in catecholamine excretion. Although psychologic reaction to acute myocardial infarction may result in increase in 24-hour urinary catecholamine excretion, the relatively normal levels of both norepinephrine and epinephrine in group I and group IV patients in our present series indicate that this factor probably plays a minor role in catecholamine excretion during sleep in uncomplicated cases.

**Table 2**

**Group II Patients**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Excretion (ng/min)</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>D.A.</td>
<td>86</td>
<td>M</td>
<td>73.7</td>
<td>10.3</td>
</tr>
<tr>
<td>F.B.</td>
<td>59</td>
<td>M</td>
<td>32.6</td>
<td>0.7</td>
</tr>
<tr>
<td>H.B.</td>
<td>60</td>
<td>M</td>
<td>18.9</td>
<td>16.6</td>
</tr>
<tr>
<td>H.C.</td>
<td>56</td>
<td>F</td>
<td>44.7</td>
<td>12.3</td>
</tr>
<tr>
<td>E.D.</td>
<td>77</td>
<td>F</td>
<td>11.4</td>
<td>1.1</td>
</tr>
<tr>
<td>G.H.</td>
<td>39</td>
<td>F</td>
<td>25.2</td>
<td>4.1</td>
</tr>
<tr>
<td>N.H.</td>
<td>50</td>
<td>F</td>
<td>49.7</td>
<td>2.7</td>
</tr>
<tr>
<td>B.I.</td>
<td>53</td>
<td>F</td>
<td>71.3</td>
<td>11.9</td>
</tr>
<tr>
<td>J.K.</td>
<td>57</td>
<td>M</td>
<td>36.6</td>
<td>9.2</td>
</tr>
<tr>
<td>A.N.</td>
<td>62</td>
<td>M</td>
<td>33.3</td>
<td>6.7</td>
</tr>
<tr>
<td>U.P.</td>
<td>57</td>
<td>F</td>
<td>36.6</td>
<td>9.2</td>
</tr>
<tr>
<td>M.R.</td>
<td>74</td>
<td>M</td>
<td>54.7</td>
<td>13.3</td>
</tr>
<tr>
<td>W.S.</td>
<td>54</td>
<td>M</td>
<td>33.5</td>
<td>6.2</td>
</tr>
<tr>
<td>P.V.</td>
<td>48</td>
<td>M</td>
<td>47.3</td>
<td>8.7</td>
</tr>
</tbody>
</table>

| Mean    |        |      | 39.8               | 8.7     |
| SEM     |        |      | 5.6                | 1.3     |

**Abbreviations:** SVT = supraventricular tachycardia; CHF = congestive heart failure. Other abbreviations and symbols are the same as those used in table 1.
Group III Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>N</th>
<th>E</th>
<th>Excretion (ng/min)</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>F.B.</td>
<td>51</td>
<td>M</td>
<td>166.9</td>
<td>71.1</td>
<td>Sinus tachycardia 110–115/min, temp. 38.2°C; history of MI. Mild chronic emphysema</td>
<td></td>
</tr>
<tr>
<td>G.D.</td>
<td>60</td>
<td>M</td>
<td>51.3</td>
<td>23.9</td>
<td>Severe chest pain; morphine sulphate given; pulmonary rales 1/2 way up chest. CVP rising from 4 → 11 → 14 cm H2O; BP 140/90 → 90/70; HR 100. Substernal pain persisting; morphine sulphate given. VT and then VF developed; patient could not be resuscitated. Hypertension noted in 1963. History of MI</td>
<td></td>
</tr>
<tr>
<td>M.B.</td>
<td>79</td>
<td>F</td>
<td>52.1</td>
<td>14.5</td>
<td>HR 100; R 44, breath sounds decreased; dullness to percussion at posterior left base. Bilateral effusion on chest x-rays; Po2 60; high-pitched expiratory wheezes and rales; ethacrynic acid given</td>
<td></td>
</tr>
<tr>
<td>C.F.</td>
<td>78</td>
<td>M</td>
<td>99.3</td>
<td>35.5</td>
<td>AF with frequent PVCs; HR around 100</td>
<td></td>
</tr>
<tr>
<td>R.H.</td>
<td>67</td>
<td>M</td>
<td>59.8</td>
<td>76.7</td>
<td>Lidocaine i.v. infusion started for VT which responded, leaving some PVCs</td>
<td></td>
</tr>
<tr>
<td>F.M.</td>
<td>52</td>
<td>M</td>
<td>93.6</td>
<td>147.8</td>
<td>Pulmonary edema developed. Chronic prostatitis; good urinary stream with no recent dysuria; prostate felt flat, firm, and nontender on rectal examination</td>
<td></td>
</tr>
<tr>
<td>J.S.</td>
<td>46</td>
<td>M</td>
<td>225.6</td>
<td>35.9</td>
<td>Persistent sinus tachycardia at 118–120/min, occasional PVCs (0–8/min)</td>
<td></td>
</tr>
<tr>
<td>P.S.</td>
<td>41</td>
<td>M</td>
<td>61.6</td>
<td>58.3</td>
<td>Swelling and pain at site of CVP catheter in arm; catheter was removed; sharp, pleuritic and substernal chest pain and rise in temp. to 38.6°C followed; HR increased to 110. No evidence of pulmonary infarction on chest x-rays. History of chronic cough</td>
<td></td>
</tr>
<tr>
<td>J.T.</td>
<td>53</td>
<td>M</td>
<td>85.7</td>
<td>22.9</td>
<td>Meperidine for recurrent chest pain. ST 138/min, bilateral basilar rales and S3; digoxin and furosemide started; ST persisting when studied. History of angina pectoris and chronic bronchitis</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: CVP = central venous pressure; BP = blood pressure (mm Hg); HR = heart rate (beats/min); R = respiratory rate (/min); VT = ventricular tachycardia; VF = ventricular fibrillation; Po2 = arterial oxygen tension (mm Hg); AF = atrial fibrillation; ST = sinus tachycardia; S3 = third heart sound. Other abbreviations and symbols are the same as those used in table 1. For the calculation of mean and SEM, the values of first determinations were used when more than one determination was obtained in a given subject.

With the exception of one patient, a significant increase in catecholamine excretion was observed in all group II and III patients who had recurrent or persistent chest pain. The catecholamine excretion rate of the patient E.D. in the daytime on the third day was 14.1 ng/min for norepinephrine and 2.2 ng/min for epinephrine, possibly indicating low basal levels. Any elevated level of catecholamine excretion that might have been present at the time of chest pain could have been lowered by the large amount of urine collected over a period of 9 hours in this patient.

Our finding of increased catecholamine excretion associated with chest pain supports the suggested sympatho-adrenal response to...
URINARY CATECHOLAMINE EXCRETION

Table 4

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Excretion (ng/min)</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.C.</td>
<td>82</td>
<td>M</td>
<td>68.0* 47.3*</td>
<td>Was found dyspneic and unresponsive in bed. ECG showed atrial flutter with 2:1 block and pulmonary edema on x-rays. Rhythm changed to atrial flutter-fibrillation after lidocaine, but patient was still in failure when studied</td>
</tr>
<tr>
<td>A.D.</td>
<td>76</td>
<td>M</td>
<td>137.9* 159.9*</td>
<td>Admitted with subxyphoid pain and vomiting. Diagnosis: rheumatic heart disease, mitral regurgitation, and AF. Runs of VT and multiple PVCs when studied on day 1. Developed abdominal swelling on day 2; intestinal infarction due to mesenteric occlusion found at laparotomy</td>
</tr>
<tr>
<td>R.D.</td>
<td>67</td>
<td>F</td>
<td>78.9 734.7</td>
<td>Admitted in pulmonary edema; frequent PVCs. Two hours later, improving but still in failure; no PVCs when studied</td>
</tr>
<tr>
<td>M.F.</td>
<td>57</td>
<td>F</td>
<td>134.2 95.6</td>
<td>Admitted in respiratory distress with paroxysmal atrial tachycardia which was persisting when studied. Angina pectoris; diabetes mellitus, receiving chlorpropanide</td>
</tr>
<tr>
<td>M.K.</td>
<td>77</td>
<td>F</td>
<td>135.7 92.3</td>
<td>Admitted in pulmonary edema with multiple PVCs. Diagnosis of hypertensive cardiovascular disease</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td></td>
<td>110.9 226.0</td>
<td>Still in moderate failure when studied</td>
</tr>
<tr>
<td>SEM</td>
<td></td>
<td></td>
<td>15.4 128.4</td>
<td></td>
</tr>
</tbody>
</table>

* Catheterized urine.

Abbreviations and symbols are the same as those used in tables 1 and 3.

Mean and SEM were calculated as in table 3.

pain,12 as well as the report of Gazes and co-workers7 who demonstrated increased plasma catecholamine concentration in patients with acute myocardial infarction or angina pectoris.

Patient H.B. received glyceryl trinitrate 20 min before the urine collection period. His norepinephrine excretion was within normal limits. Kobayashi and associates13 reported the effect of glyceryl trinitrate on plasma and urinary excretion of 3H-norepinephrine and 3H-epinephrine after infusion of the same agents in man. An abrupt decrease of the radioactivity of 3H-catecholamine during infusion was noted after sublingual administration of glyceryl trinitrate. These authors postulated that the drug acted as an accelerator of catecholamine inactivation. It is possible that the normal urinary excretion of norepinephrine by patient H.B. may have been partly due to this acceleration of catecholamine inactivation.

Patient C.C. (table 4) had atrial flutter-fibrillation and signs of cardiac decompensation, while patient M.F. (table 4) had paroxysmal atrial tachycardia with respiratory distress. Both had elevated excretion rates of norepinephrine and epinephrine. Our preliminary observation on patients without acute myocardial infarction complicated by supraventricular tachycardia failed to demonstrate a direct correlation between the presence of the arrhythmia and magnitude of catecholamine excretion. Some patients had normal values, while others had high values. Thus, the elevation in the catecholamine excretion in these patients may not be directly related to supraventricular tachycardia, but rather a reflection of the borderline or impending cardiac decompensation associated with the arrhythmia.

High values of catecholamine excretion were observed in all patients with marked and persistent sinus tachycardia. Patient J.S. (table 3) had concomitant ventricular premature contractions, while patient J.T. (table 3) had chest pain and signs of cardiac decompensation. It is uncertain whether elevated catechol-
amine excretion in patients with acute myocardial infarction and sinus tachycardia was a specific finding.

All patients with ventricular tachycardia or frequent ventricular premature contractions had marked elevations in catecholamine excretion. A substantial decrease in catecholamine excretion was observed in patients C.F. (table 3), R.D., and M.K. (table 4) a few hours after the initial determination when the ventricular premature contractions were much less frequent or absent. Temporal correlation of ventricular arrhythmias and increased catecholamine excretion suggests a cause-effect relationship. The occurrence of ventricular arrhythmias during the administration of catecholamines is widely recognized in both clinical and experimental studies. Furthermore, some workers have suggested that an increased urinary catecholamine excretion may be associated with cardiac arrhythmias in acute myocardial infarction. Since only a small fraction of the injected norepinephrine appears unaltered in the urine, the level found in our patients with ventricular arrhythmias may reflect a sufficient amount of tissue catecholamines which were responsible for the production of arrhythmias. As a result of decreased cardiac output or other unknown factors, ventricular arrhythmias in turn may stimulate the sympa-tho-adrenal system and increase urinary free catecholamine excretion.

Elevated values of norepinephrine and often of epinephrine excretion were found in patients who were in pulmonary edema or manifested radiologic evidence of pulmonary congestion. This finding suggests a possible role of pulmonary congestion as a factor for the increased sympatho-adrenal activity. Valori and associates described a case in which pulmonary venous hypertension and congestion due to sudden onset of mitral regurgitation were associated with concomitant rise in urinary catecholamine excretion. Saito showed a strong positive correlation between magnitude of pulmonary artery pressure and urinary excretion of free norepinephrine in 60 patients with various types of either congenital or acquired cardiac lesions. Although the pre-

cise mechanism is not known at present, the available evidence seems to suggest a close relationship between sympatho-adrenal activity and altered pulmonary circulatory state.

Since urinary epinephrine is primarily derived from the adrenal glands, the increased epinephrine level suggests augmentation of the adrenal activities. It is uncertain whether the adrenal participation is related to poor perfusion of the adrenal glands, neurogenic influence on the adrenal activities, or some unknown chemical factors.

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