Provocation of coronary spasm by dopamine in patients with active variant angina pectoris


ABSTRACT The effects of dopamine on arteries are different depending on the dose, route of administration, and receptor population. Its administration can cause vasodilation by stimulation of dopaminergic receptors, vasoconstriction by stimulation of $\alpha$-adrenergic and serotoninergic receptors, and even spasm of coronary arteries when given intracranially in dogs. The ability of dopamine to provoke coronary spasm was assessed in 18 patients with active vasospastic angina in whom this amine was infused at rates of 5, 10, and 15 $\mu$g/kg/min for periods of 5 min each. The 12-lead electrocardiogram and blood pressure (cuff) were monitored throughout the whole test. In nine patients dopamine caused angina and ischemic electrocardiographic changes suggestive of coronary spasm: ST segment elevation in six patients and ST segment depression in the absence of important coronary stenoses in the remaining three. Infusion of dopamine was repeated during coronary angiography in three patients with positive test results; this provoked occlusive coronary spasm with ST segment elevation in two patients and nonocclusive spasm with ST segment depression in the remainder. In conclusion, infusion of dopamine provokes coronary spasm in a sizeable proportion of patients with active vasospastic angina. Its administration may be detrimental in patients susceptible to coronary spasm, such as those with acute myocardial infarction.


IN THE VAST MAJORITY of patients with active vasospastic angina, coronary spasm is consistently provoked by ergonovine maleate.1–3 This test is useful for the diagnosis of variant angina but does not provide information about the mechanism of spontaneous spasm, since ergonovine is not a biogenic substance. A number of more “physiologic” vasoconstrictor stimuli have been tested to this end. In an early study, Yasue et al.4 reported that administration of epinephrine consistently resulted in coronary spasm; however, their data were not subsequently confirmed. Furthermore, Cierchia et al.5 failed to induce coronary spasm with phenylephrine, a “pure” $\alpha$-receptor agonist, in 12 consecutive patients with active variant angina. More recently, Ginsburg et al.6 induced coronary spasm in four patients with variant angina by infusion of histamine after blockade of cycloheximide H$_2$ receptors. This finding was confirmed at our institution in about 50% of patients with variant angina.

Dopamine is a biogenic amine known to produce spasm of cerebral arteries when given intracranially in dogs.7 To investigate its potential role in precipitating coronary spasm, we administered dopamine to 18 patients with active variant angina.

Patients and methods

Patients (table 1). Eighteen patients (14 men, four women; mean age 49 ± 10 years) with variant angina, admitted to our institution from January 1982 to December 1984, were selected for study. They all reported angina predominantly at rest (>7 episodes/week) with frequent attacks in the early morning hours. Holter monitoring showed episodes of transient ST segment elevation in nine patients; the remaining nine had both ST segment depression and ST segment elevation alternating in the same leads during different episodes.

Two patients had previous myocardial infarctions, occurring 12 and 18 months before the study, respectively. All patients were normotensive and had no evidence of cardiac failure. Sixteen patients were in sinus rhythm and two were in atrial fibrillation. No patients had conduction disturbances or left ventricular hypertrophy on the electrocardiogram (ECG) that could prevent the interpretation of ST segment changes. Coronary angiography was performed in 17 patients; one patient declined this investigation. Each patient gave informed consent for participating in the study.
TABLE 1
Clinical, electrocardiographic, and angiographic findings

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr/sex)</th>
<th>Angina Onset (mo)</th>
<th>No. of attacks/wk</th>
<th>Basal ECG</th>
<th>Diameter reduction of coronary arteries (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>LAD</td>
</tr>
<tr>
<td>1</td>
<td>36/M</td>
<td>4</td>
<td>14</td>
<td>N</td>
<td>70</td>
</tr>
<tr>
<td>2</td>
<td>62/M</td>
<td>3</td>
<td>15</td>
<td>N</td>
<td>70</td>
</tr>
<tr>
<td>3</td>
<td>56/F</td>
<td>6</td>
<td>10–15</td>
<td>N</td>
<td>70</td>
</tr>
<tr>
<td>4</td>
<td>50/M</td>
<td>2</td>
<td>20</td>
<td>N</td>
<td>70</td>
</tr>
<tr>
<td>5</td>
<td>42/M</td>
<td>7</td>
<td>15</td>
<td>OIMI</td>
<td>70</td>
</tr>
<tr>
<td>6</td>
<td>50/M</td>
<td>3</td>
<td>10–15</td>
<td>AF</td>
<td>70</td>
</tr>
<tr>
<td>7</td>
<td>40/M</td>
<td>2</td>
<td>10</td>
<td>N</td>
<td>50</td>
</tr>
<tr>
<td>8</td>
<td>53/M</td>
<td>2.5</td>
<td>10–15</td>
<td>AF</td>
<td>50</td>
</tr>
<tr>
<td>9</td>
<td>56/M</td>
<td>24</td>
<td>7–10</td>
<td>N</td>
<td>50</td>
</tr>
<tr>
<td>10</td>
<td>55/M</td>
<td>2</td>
<td>14</td>
<td>N</td>
<td>50</td>
</tr>
<tr>
<td>11</td>
<td>66/M</td>
<td>3</td>
<td>7–15</td>
<td>N</td>
<td>50</td>
</tr>
<tr>
<td>12</td>
<td>64/F</td>
<td>1</td>
<td>14</td>
<td>N</td>
<td>50</td>
</tr>
<tr>
<td>13</td>
<td>53/M</td>
<td>12</td>
<td>10</td>
<td>N</td>
<td>50</td>
</tr>
<tr>
<td>14</td>
<td>46/M</td>
<td>2</td>
<td>15</td>
<td>N</td>
<td>50</td>
</tr>
<tr>
<td>15</td>
<td>37/M</td>
<td>3</td>
<td>10</td>
<td>N</td>
<td>50</td>
</tr>
<tr>
<td>16</td>
<td>32/M</td>
<td>1</td>
<td>10</td>
<td>N</td>
<td>50</td>
</tr>
<tr>
<td>17</td>
<td>50/F</td>
<td>5</td>
<td>10</td>
<td>N</td>
<td>50</td>
</tr>
<tr>
<td>18</td>
<td>35/F</td>
<td>2</td>
<td>14</td>
<td>OIMI</td>
<td>50</td>
</tr>
</tbody>
</table>

*= not done; AF = atrial fibrillation; LAD = left anterior descending coronary artery; LC = left circumflex coronary artery; N = normal; OIMI = old inferior myocardial infarction.

Provocative test protocol. All patients underwent testing with dopamine and ergonovine in random order. Both tests were performed in the morning (between 9 A.M. and 1 P.M.) on different days within 1 week. All provocative tests were performed in the exercise laboratory, which was adjacent to the catheterization laboratory. None of the patients were on β-blockers or digoxin at the time of the study. Calcium-antagonists and long-acting nitrates were withdrawn at least 2 days before the tests; only sublingual nitroglycerin was used during this period, but never less than 2 hr before the tests. Smoking and alcohol consumption were prohibited on the test days. Blood pressure (cuff sphygmomanometer) and 12-lead ECGs were obtained during the control period, at 1 min intervals during the tests and for 15 min during recovery. Three ECG leads were monitored continuously throughout the whole session. Myocardial ischemia was relieved by amyl nitrite and subsequent intravenous injection of isosorbide dinitrate.

Dopamine test. With the patient in the supine position, dopamine was infused intravenously, through an antecubital vein, at incremental rates of 5, 10, and 15 μg/kg/min for periods of 5 min each. In the last 10 patients the test was repeated within a few days (2 to 4) during coronary angiography, according to the same protocol.

Ergonovine test. Ergonovine maleate was administered intravenously in incremental doses of 0.025, 0.05, 0.1, 0.2, and 0.3 mg at intervals of 5 min, with the patient in the supine position. In 15 patients the test was repeated within a few days during coronary angiography, according to the same protocol. In those patients in whom both dopamine and ergonovine tests were performed at angiography, dopamine was always given first.

Data analysis. Electrocardiographic changes were considered diagnostic of myocardial ischemia when either ST segment elevation greater than 0.1 mV or rectilinear or downsloping ST segment depression greater than 0.1 mV occurred in at least two different leads. ST segment elevation was considered only if observed in leads without pathologic Q waves.

Coronary stenoses were considered significant for reduction of internal lumen diameter of 70% or greater.

Continuous data were expressed as mean ± 1 SD.

Results

Dopamine caused ST segment elevation in six patients, ST segment depression in four, and no ischemic changes in eight. Five of the 10 patients with a positive response to dopamine and three of the eight patients with a negative response had coronary arteries without significant stenoses (figure 1).

Positive response to dopamine with ST segment elevation (table 2)

ECG data. In six patients, dopamine precipitated ST segment elevation and angina (figure 2). In all six, ergonovine (mean cumulative dose 0.208 ± 0.124 mg) also precipitated angina and ST segment elevation similar to that observed after dopamine.

Angiographic data. Baseline coronary angiography showed insignificant narrowings in two patients, one-vessel disease in one, and two-vessel disease in two and was not performed in the remaining patient. In patients 11, 14, and 15, both dopamine and ergonovine tests were repeated during angiography. In patients 11 (figure 3) and 15, both drugs provoked coronary spasm followed by ST segment elevation and angina; spasm occurred in an angiographically normal artery in one patient and was superimposed on a significant stenosis in the other. In patient 14, ergonovine precipitated coronary spasm, whereas dopamine failed to cause obvious changes in coronary diameter or the electrocardiographic changes observed during the first test.

![FIGURE 1: Results of dopamine testing. The crossed portion of the histograms represents patients without significant coronary stenoses (<70%).](image-url)
performed a few days before. In patients 3 and 4, no provocative tests were performed at angiography; the latter had spontaneous spasm.

**Positive response to dopamine with ST segment depression (table 2)**

**ECG data.** Dopamine precipitated ST segment depression and angina in four patients. Ergonovine (mean cumulative dose 0.325 ± 0.264 mg) precipitated ST segment elevation and angina in three patients and ST segment depression and angina in the remainder.

**Angiographic data.** Baseline coronary angiography showed insignificant narrowing in three patients and one-vessel disease in one. In patients 13 and 17, both dopamine and ergonovine tests were repeated during angiography. In patient 13, both tests provoked nonocclusive coronary spasm of an apparently normal artery followed by ST segment depression and angina (figure 4). Patient 17 had spontaneous nonocclusive spasm superimposed on a noncritical coronary narrowing; this was followed by ST segment depression and angina. In this patient, dopamine, which had provoked ST segment depression a few days earlier, failed to cause obvious angiographic or electrocardiographic changes. After return to baseline, administration of ergonovine resulted in occlusive spasm of the same artery, followed by angina and ST segment elevation. In patients 6 and 8, only the ergonovine test was performed during angiography and precipitated coronary spasm in both.

**Negative response to dopamine (table 2)**

**ECG data.** In eight patients dopamine given at the maximal infusion rate did not precipitate diagnostic ST segment changes; two of these patients, however, complained of typical chest pain during the test. With dopamine, all patients exhibited nonspecific T wave peaking and prominent U waves in the precordial leads. Ergonovine (mean cumulative dose 0.262 ± 0.203 mg) precipitated ST segment elevation and angina in all.

**Angiographic data.** Baseline coronary angiography showed nondiagnostic narrowing in three patients, one-vessel disease in four, and two-vessel disease in one. In patients 9, 10, 12, 16, and 18, both dopamine and ergonovine tests were repeated during angiography. Dopamine did not cause spasm or ischemic electrocardiographic changes in any of the patients, whereas ergonovine did so in all five. In patients 1, 5, and 7 only the ergonovine test was performed during angiography and provoked coronary spasm in all.

**Hemodynamic findings and adverse effects.** At peak infusion, dopamine caused an increase in both heart rate (16 ± 8 beats/min) and systolic blood pressure (23 ± 9 mm Hg). The hemodynamic response was similar in patients with a positive or negative response to the dopamine test. None of the patients had major adverse effects during infusion of dopamine; 11 patients complained of palpitation and eight of mild headache. The ECG showed occasional isolated premature ventricular beats in five patients, without signs of ischemia. In two patients frequent premature ventricular beats and couplets were seen during dopamine-induced ST segment elevation.

Ergonovine did not cause an appreciable increase in heart rate; systolic blood pressure never increased by more than 10 mm Hg.

**Discussion**

**Dopamine-induced coronary spasm.** Dopamine, at the doses given in this study, precipitated myocardial ischemia in 10 of 18 patients with variant angina. Although dopamine increases myocardial oxygen demand and direct angiographic evidence for spasm was obtained in only three patients, our data provide indi-
rect evidence that in at least six others myocardial ischemia was caused by coronary spasm. Indeed, in four of these patients dopamine precipitated ST segment elevation, an accepted electrocardiographic marker of coronary spasm.\textsuperscript{8–12} In the remaining two, dopamine caused ST segment depression and angina, but neither of them had significant coronary narrowings (only 50% diameter reduction in one vessel). Therefore it is likely that myocardial ischemia was predominantly caused by nonocclusive coronary

**FIGURE 3.** Left, Baseline coronary angiogram of patient 11, showing an apparently normal left anterior descending coronary artery (LAD) indicated by the arrows. Right, Angiogram showing occlusive spasm of the LAD after infusion of dopamine (15 \( \mu \)g/kg/min); ST segment elevation in \( V_4 \) was seen on the ECG. Bottom, Spasm of the LAD and ST segment elevation were relieved by administration of amyl nitrite.
spasm resulting in subendocardial ischemia\textsuperscript{12} rather than by an increase in myocardial oxygen demand. Our interpretation is supported by the observation that in these two patients, a maximal exercise test performed in the same week did not cause ST segment changes in spite of an increase in heart rate–blood pressure product similar to that observed during infusion of dopamine. Furthermore, one of the these two patients developed spontaneous nonocclusive spasm during angiography accompanied by ST segment depression similar to that observed a few days earlier during the dopamine test. Although in this same patient administration of dopamine during angiography did not produce either angina or ST segment changes, the hemodynamic response was similar to that seen during the first test. Finally, other patients who had a similar hemodynamic response to dopamine did not develop electrocardiographic signs of myocardial ischemia even in the presence of significant coronary stenoses. The role played by coronary vasoconstriction in precipitating myocardial ischemia is difficult to establish in one patient with significant coronary atherosclerosis in whom dopamine provoked ST segment depression; in this patient the test was not repeated at angiography.

**Receptors involved.** Dopamine can cause vasodilation by stimulation of two subtypes of peripheral dopaminergic receptors: DA\textsubscript{1}, located postsynaptically, and DA\textsubscript{2}, located on postganglionic sympathetic nerves whose activation inhibits catecholamine release.\textsuperscript{13} In this study, however, patients who did not develop coronary spasm after dopamine did not show any obvious dilation of epicardial arteries. What turns the potential coronary dilator stimulus of dopamine into coronary spasm is unknown. It might be postulated that in variant angina the stimulation of dopaminergic receptors could result in a paradoxical constrictive response. Yet pretreatment with domperidone, an antagonist of peripheral dopaminergic receptors,\textsuperscript{14} failed to prevent dopamine-induced angina and ST segment elevation in three of our patients (Nos. 2, 3, and 4). This finding, however, does not rule out the possibility that in patients with variant angina, a decreased sensitivity of peripheral dopaminergic receptors could have unmasked the vasoconstrictor effects of dopamine.

---

**TABLE 2**

Electrocardiographic and angiographic results of dopamine and ergonovine tests

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Maximal infusion rate (µg/kg/min)</th>
<th>ECG changes</th>
<th>Angina</th>
<th>Cumulative dose (mg)</th>
<th>ECG changes</th>
<th>Angina</th>
<th>Vessel undergoing spasm</th>
<th>Stimulus</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>15</td>
<td>II-III-aVF↑</td>
<td>+</td>
<td>0.175</td>
<td>V1-V5↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>2</td>
<td>15</td>
<td>II-III-aVF↑</td>
<td>+</td>
<td>0.675</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>3</td>
<td>10</td>
<td>V2-V3↑</td>
<td>+</td>
<td>0.675</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>4</td>
<td>15</td>
<td>II-III-aVF</td>
<td>+</td>
<td>0.175</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>5</td>
<td>15</td>
<td>V2-V3↑</td>
<td>+</td>
<td>0.175</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>6</td>
<td>15</td>
<td>II-III-aVF</td>
<td>+</td>
<td>0.175</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>7</td>
<td>15</td>
<td>0</td>
<td>0</td>
<td>0.175</td>
<td>V2-V4↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>8</td>
<td>15</td>
<td>II-III-aVF</td>
<td>+</td>
<td>0.175</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>9</td>
<td>15</td>
<td>V1-V4↑</td>
<td>+</td>
<td>0.175</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>10</td>
<td>15</td>
<td>0</td>
<td>0</td>
<td>0.175</td>
<td>V2-V3↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>11</td>
<td>15</td>
<td>V1-V4↑</td>
<td>+</td>
<td>0.175</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>12</td>
<td>15</td>
<td>0</td>
<td>0</td>
<td>0.175</td>
<td>V2-V3↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>13</td>
<td>10</td>
<td>V2-V3↑</td>
<td>+</td>
<td>0.175</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>14</td>
<td>15</td>
<td>V1-V2↑</td>
<td>+</td>
<td>0.175</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>15</td>
<td>15</td>
<td>V1-V2↑</td>
<td>+</td>
<td>0.175</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>16</td>
<td>15</td>
<td>V3-V4↑</td>
<td>+</td>
<td>0.175</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>17</td>
<td>15</td>
<td>V3-V4↑</td>
<td>+</td>
<td>0.175</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
<tr>
<td>18</td>
<td>15</td>
<td>V3-V4↑</td>
<td>+</td>
<td>0.175</td>
<td>V5-V6↑</td>
<td>+</td>
<td>LAD</td>
<td>E</td>
</tr>
</tbody>
</table>

+ = present; 0 = absent; ↑ = ST segment elevation; ↓ = ST segment depression; D = dopamine; E = ergonovine; S = spontaneous; other abbreviations as in table 1.

\textsuperscript{a}Patients in whom dopamine test was repeated at angiography.

\textsuperscript{b}Nonocclusive spasm after both dopamine and ergonovine.

\textsuperscript{c}Nonocclusive spontaneous spasm and occlusive spasm after ergonovine.
**FIGURE 4.** Left, Baseline coronary angiogram of patient 13, showing an apparently normal left anterior descending coronary artery (LAD) indicated by the arrows. Right, Angiogram showing nonocclusive spasm of the LAD after infusion of dopamine (10 μg/kg/min); ST segment depression in V4 was seen on the ECG. Bottom, Spasm of the LAD and ST segment depression were relieved by administration of amyl nitrite.
mediated by other receptors. Studies in vitro have shown that the vasoconstrictor effects of dopamine are mediated by both \(\alpha\)-adrenergic and serotonin receptor.

\[\text{Vasoconstriction, in fact, is only attenuated by pretreatment with either phenolamine or cyproheptadine and entirely prevented when both antagonists are used.}\] It is worth noting that the same classes of receptors seem to be responsible for the vasoconstrictor effects of ergonovine maleate, the most potent stimulus known to trigger coronary spasm in patients with variant angina.\(^1\)\(^-\)\(^3\)

In the central nervous system, endogenous dopamine is an important neurotransmitter. Exogenous dopamine, however, does not cross the blood-brain barrier\(^17\); consequently, it is unlikely that central dopaminergic receptors played any role in mediating the spasmodenic effects of the drug.

**Pathogenetic implications.** Coronary spasm may be provoked in the same patient by a number of apparently unrelated vasoconstrictor stimuli.\(^18\) This seems to suggest that a local supersensitivity of coronary smooth muscle to constrictor stimuli plays a key role in the pathogenesis of coronary spasm.\(^19\)-\(^21\) Yet powerful constrictors such as phenylephrine do not cause coronary spasm\(^5\); therefore, the interaction of adequate stimuli with hyperreactive smooth muscle is probably needed to cause it.

Our study shows that, like other constrictor agents, dopamine also has the ability to provoke coronary spasm. This observation is of interest because dopamine is a biogenic amine released by peripheral sympathetic nerves.\(^22\)\(^,\)\(^23\) Although it would be tempting to speculate that dopamine could be one of the possible mediators of coronary spasm, Robertson et al.\(^24\) found that spontaneous episodes of coronary spasm were not preceded by a detectable increase in coronary sinus dopamine levels. However, this technique is probably unable to pick up a highly targeted release of dopamine. The ability of various combinations of \(\alpha\)-adrenergic and serotonin antagonists with dopaminergic agonists to prevent coronary spasm could help in identifying the pathogenetic role of these receptor classes in individual patients.

**Clinical implications.** At the present time, the routine use of dopamine as an alternative test for the detection of coronary spasm does not appear to be warranted in view of the lower sensitivity of this agent compared with ergonovine.

Dopamine is frequently used as an inotropic agent in patients with acute myocardial infarction complicated by heart failure. Since coronary spasm may play a role in the early stages of myocardial infarction,\(^25\)-\(^29\) the potential spasmodenic effects of dopamine might be detrimental in this setting.

We thank Miss Bharti Kava for her careful secretarial help in the preparation of the manuscript and Miss Jean Powell for her assistance in the preparation of figures.

**References**


CIRCULATION
23. Thorner MO: Dopamine is an important neurotransmitter in the autonomic nervous system. Lancet 1: 662, 1975
Provocation of coronary spasm by dopamine in patients with active variant angina pectoris.
F Crea, S Chierchia, J C Kaski, G J Davies, A Margonato, D O Miran and A Maseri

Circulation. 1986;74:262-269
doi: 10.1161/01.CIR.74.2.262

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1986 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/74/2/262

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/