Kenneth Tardiff,[1] *M.D., M.Ph.; Elliot Gross,*[2] *M.D.; Jeffrey Wu,*[3] *M.D.; Marina Stajic,*[4] *Ph.D.; and Robert Millman,*[1] *M.D.*

Analysis of Cocaine-Positive Fatalities

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ABSTRACT: A review of all autopsy and toxicology reports for persons dying in New York City in an 11-month period found 935 persons dying with cocaine in their bodies. Cocaine-positive fatalities were more likely in the young black and Hispanic and male population. In addition to cocaine and its metabolites, heroin and other opiates were found in 39% of persons and ethanol in 33% and barbituates and minor tranquilizers in only 2% of the deceased. Cocaine overdose was responsible for 4% of the deaths and overdose with heroin and cocaine for 12% of the deaths. Violence was often the cause of death. Thirty-eight percent died of homicide, seven percent of suicide, and eight percent from accidents. Of particular interest were 6 persons who died of acute cardiac events directly related to cocaine as well as 4 cases of ruptured dissections of the ascending aorta, and 9 cases of cerebral hemorrhage. Autopsy findings for these individuals are described, and possible mechanisms of death are discussed.

KEYWORDS: toxicology, pathology and biology, cocaine, death

In the 1980s, the perception of cocaine has changed from its being viewed as a benign "recreational" drug to that of a scourge with profound psychological, social, and medical consequences. This has come about largely because of increased availability of cocaine, especially in the free-base form. A report has reviewed many of the medical complications of cocaine use [*1*]. Most case reports have focused on acute cardiac events, including arrhythmias and acute myocardial infarctions [*2-12*]. Autopsies in three cases of acute myocardial infarction following cocaine use have been described [*13-15*]. One case of acute rupture of the ascending aorta in a man smoking free-base cocaine has been reported [*16*]. Six cases of cerebrovascular accidents, five with cerebral hemorrhage and one with occlusion of a cerebral artery, have been reported [*2,17-20*]. A recent review of cases brought to the Dade County Medical Examiner's Office found fifteen instances of cocaine and death attributed to coronary artereosclerosis and five attributed to intracranial hemorrhage [*21*].

This paper presents the results of a systematic study of all cases brought to the Office of the Chief Medical Examiner in New York City where cocaine was found in the deceased on routine toxicological tests. It describes the patterns of death and focuses on cases where cocaine use was directly associated with cardiovascular and neurological deaths.

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[1]Associate professor and professor, respectively, of psychiatry and of public health, Cornell University Medical College, New York, NY.

[2]Associate professor of forensic medicine, New York University School of Medicine, New York, NY.

[3]Resident, Department of Internal Medicine, St. Elizabeth's Hospital, Boston, MA.

[4]Director of toxicology, Office of the Chief Medical Examiner, New York, NY.

Methods

All autopsy and toxicology reports of persons dying in New York City from 1 Jan. 1986 to 1 Nov. 1986 and reported to the Office of the Chief Medical Examiner were reviewed. Reported deaths include those occurring by violence (accident, homicide, suicide) or when medically unattended in an unusual or suspicious manner or in a correctional institution. Radioimmunoassay (RIA) was used to detect cocaine or benzolyecgonine and opiates or both; gas chromatography (GC) for ethanol, lidocaine, amitriptyline, and methadone; thin-layer chromatography (TLC) for quinine; and high-pressure liquid chromatography for acetaminophen. There were 935 cases in which cocaine was detected in routine toxicological analysis of a broad spectrum of drugs and alcohol. A structured instrument was used to record data on demographic characteristics, cause of death, and the presence of alcohol or other substances of abuse in the deceased.

Results

The demographic characteristics of persons with cocaine in their bodies are presented in Table 1. Approximately two thirds were younger than 34 years. Of the 16 cases under 10 years, only 1 child was older than 4 months; 8 cases involved stillbirths and 2 infants died a few hours after birth from seizures. The rest died within a few months of birth, with most found dead in bed and one found on a bench.

Considering all ages, half were black, 30% were Hispanic, and 80% were male. There were 39 (4.2%) individuals whose deaths were attributed to cocaine intoxication manifested by circulatory and respiratory failure. An additional 112 (12.0%) died from combined heroin and cocaine intoxication. In addition to those attributable to intoxication, a large proportion died of trauma classified as (37.5%) homicide, a smaller but significant proportion from physical or chemical injuries, classified as suicide (6.6%) and accident (7.5%).

Other substances detected in addition to cocaine included opiates in 362 cases (38.7%) and ethanol in 305 cases (32.6%). Only a few had barbituates or minor tranquilizers in addition to cocaine.

Of particular interest, 45 persons (4.8%) died of occlusive coronary artery disease, 19 (2.0%) of cerebral hemorrhage, and 6 (0.6%) hemorrhages as a result of aortic rupture. Based on reports from those investigating the scene of death and interviewing persons with knowledge of the death, it was determined that cocaine was responsible for 6 who died from acute cardiac events, 4 of ruptured dissections of the ascending aorta, and 9 as a result of cerebral hemorrhage. The case reports for the individuals follow.

Case 1

A 40-year-old black man collapsed on the street and was unresponsive to emergency care at a nearby hospital. Cocaine was detected in blood (<0.1 mg/L) and in bile, ethanol (0.03 g/dL) in blood. Autopsy findings included: coronary atherosclerotic disease with an occlusive thrombus in the left anterior descending coronary artery; atherosclerotic occlusion of greater than 75% in the left main, right main, and left circumflex coronary arteries; old and recent left ventricular myocardial infarcts; and cardiac hypertrophy.

Case 2

A 37-year-old Hispanic man who was a known intravenous (IV) drug abuser was found dead in his apartment. Cocaine was detected in blood (8.0 mg/L) and in bile ethanol (0.03 g/dL) in blood. Autopsy demonstrated occlusive coronary thombosis (the artery was not specified), an old infarct in the lower third of the ventricular septum, an acute anterolateral wall infarct, and cardiac hypertrophy.

TABLE 1—*Characteristics of persons with cocaine in their bodies at time of autopsy.*

	Persons	Percentage
AGE (YEARS)		
0-10	16	1.7
11-24	160	17.1
25-34	416	44.5
35-44	237	25.3
45-64	88	9.4
65+	18	1.9
Total	935	100.0
RACE		
Black	473	51.6
Hispanic	271	29.6
White/Asian	172	18.8
Total	916	100.0
SEX		
Male	744	80.2
Female	184	19.8
Total	928	100.0
PRESENCE OF OTHER SUBSTANCES		
Ethanol	305	32.6
Heroin/opiates	362	38.7
Barbituates/minor tranquilizer	19	2.0
CAUSE OF DEATH		
Cocaine overdose	39	4.2
Acute narcotism	112	12.0
Chronic narcotism	14	1.5
Acute alcoholism	2	0.2
Chronic alcoholism	31	3.3
Occlusive coronary artery disease	45	4.8
Cerebral hemorrhage	19	2.0
Ruptured aorta	6	0.6
Other natural causes	104	11.1
Homicide	350	37.5
Suicide	62	6.6
Motor vehicle accident	33	3.5
Other accident	37	4.0
Pending or unknown	81	8.7
Total	935	100.0

Case 3

A 35-year-old black man with a history of "heart disease" was found dead in his apartment. Cocaine was detected in blood (1.0 mg/L), urine and bile, ethanol (0.02 g/dL) in blood. Autopsy findings included atherosclerosis of the left anterior descending artery (50% occlusion) with complete occlusion by superimposed thrombus 1.5 cm distal to the ostium, and atherosclerosis of the aorta, with occlusion just proximal to the bifurcation by brownish yellow thrombus.

Case 4

A 27-year-old Hispanic man complained of epigastric pain while playing basketball with friends, who took him to a nearby hospital, where he died several hours later. Cocaine was detected in blood (< 0.1 mg/L) and bile, ethanol in blood (0.04 g/dL) and brain. There was no history or skin manifestations of chronic intravenous drug abuse. Autopsy findings included an acute thrombus of the left anterior descending coronary artery, with pulmonary congestion.

Case 5

A 38-year-old white man walking with his wife collapsed and died. There was no history of heart problems or hypertension. Cocaine was detected in blood (0.1 mg/L) and in bile, lidocaine in blood and liver, and methadone in blood, liver, and urine. Autopsy findings included old and recent myocardial infarcts, atherosclerosis of left anterior descending, left circumflex (60 to 70% occluded) and right main (90 to 95% occluded) coronary arteries, cardiac hypertrophy (580 g) with flabby myocardium, and aneurysmal formation of the anterior and posterior walls.

Case 6

A 33-year-old white man was seen in an emergency room complaining of chest pain radiating to his left arm. Sent home on Maalox, he was found dead in his apartment several hours later. Cocaine was detected in blood (0.1 mg/L), liver, and urine. No other drugs were detected. Autopsy findings were: atherosclerotic coronary artery disease in the left anterior descending artery (70 to 90% occluded), right main artery (70 to 80% occluded), and left circumflex artery (60% occluded), left ventricular myocardial ischemia cardiac hypertrophy, and pulmonary and hepatic congestion.

Case 7

A 36-year-old black man visiting a friend complained of chest pain, lay down to rest, and was found dead hours later. Cocaine was detected in blood (0.1 mg/L) and bile, ethanol (0.02 g/dL) in blood. Autopsy revealed dissection of the ascending aorta, with a 5.7-cm transverse tear, dissection of the medical layer, and a 1.3-cm calcified perforation in the descending aorta. There was no evidence of Marfan's syndrome, nor was there any suggestion of tertiary syphilis or trauma.

Case 8

A 28-year-old Hispanic man was hospitalized complaining of 3 to 5 h of chest pain, beginning after intranasal cocaine snorting. He died several hours later. Cocaine was detected in blood (0.9 mg/L) and bile. Autopsy revealed a ruptured dissecting 5.1-cm aneurysm of the ascending aorta. The dissection separated the media and adventitia, with intimal and medial separation as well, and extended to the right subclavian artery. There was a hemopericardium of 700 cc of clotted and liquid blood. There was no suggestion of Marfan's syndrome, tertiary syphilis, or trauma.

Case 9

A 45-year-old Hispanic woman was found dead at home after complaining of chest pain for several days. Cocaine was detected in blood (< 0.1 mg/L), bile, and liver. Autopsy revealed an aortic dissection extending from the ascending aorta to the bifurcation. There was

a posterior rupture with hemopericardium (250 cc) and hemothorax (1500 cc). There was no suggestion of Marfan's syndrome, tertiary syphilis, or trauma.

Case 10

A 42-year-old black man complained of chest pain. His wife took him to a hospital where he was given unknown medications and sent home. There his chest pain resumed, and he became unresponsive. Cocaine was detected in his blood (<0.1 mg/L) and bile. Autopsy revealed a dissection of the ascending aorta, beginning about 2 cm above the coronary ostia and reentering 4.5 cm distally and a hemopericardium (300 cc). There was no suggestion of Marfan's syndrome, tertiary syphilis, or trauma.

Case 11

After lifting an object at work, a 44-year-old black man complained of headache, stiff neck, and photophobia. He was taken to a hospital where he was diagnosed with a ruptured cerebral aneurysm and died one week later. Cocaine or metabolites or both were detected in his blood, bile, and liver on admission. At autopsy quinine was detected in bile and liver; ethanol in bile. Autopsy revealed a ruptured aneurysm of the anterior communicating artery with intracerebral hemorrhage, pneumonia, anoxic encephalopathy, and fatty liver.

Case 12

A 27-year-old Hispanic man "took some drugs," according to his wife, and later that day complained of headaches. His wife found him dead on the floor later that evening. There was a family history of cerebral aneurysms. Cocaine was detected in blood (1.8 mg/L) and bile; opiates in blood and bile; and acetominophen in blood. Autopsy revealed a ruptured 2.0-cm aneurysm of the anterior cerebral communicating artery, with acute massive subarachnoid and intraventricular hemorrhage; cardiomegaly and acute visceral congestion; and needle tracks.

Case 13

A 29-year-old black woman complained of headache at work and collapsed. A computed tomography (CT) scan at a nearby hospital suggested a subarachnoid hemorrhage. She went into cardiopulmonary arrest and died. Cocaine was detected in blood (0.2 mg/L) and bile, lidocaine in blood. Autopsy demonstrated a ruptured aneurysm of the right posterior communicating artery with massive subarachnoid and intracerebral hemorrhage.

Case 14

A 29-year-old black man was found dead in his apartment by his wife. Cocaine was detected in his blood (0.1 mg/L) and urine. Autopsy showed a ruptured aneurysm of the left middle cerebral artery, with extensive subarachnoid hemorrhage at the base of the brain, cerebellum, and brain stem.

Case 15

A 31-year-old black woman felt disoriented and went to a nearby hospital. She was found to have intracerebral hemorrhage, and an intracranial shunt was placed, but the patient died. Cocaine was detected in blood on admission and in the bile postmortem. Autopsy revealed a ruptured 1.0-cm aneurysm of the anterior communicating artery, with intracerebral and intraventricular hemorrhage.

Case 16

A 44-year-old black woman was found dead at home by her husband. Cocaine was detected in blood (0.2 mg/L), bile, and urine. Autopsy revealed a ruptured 1.9-cm aneurysm, of the anterior cerebral artery, 2.5 cm distal to the anterior communicating artery.

Case 17

A 44-year-old black man with a history of intravenous drug abuse and hypertension was found unconscious on his bathroom floor. A CT scan at a nearby hospital showed subarachnoid hemorrhage. He died 36 h later. Cocaine was detected in blood (0.9 mg/L) at time of admission and in bile postmortem, in addition to methadone in blood (0.1 mg/dL) and liver, and ethanol (0.2 g/dL) in blood. Autopsy revealed bilateral subarachnoid hemorrhage. No aneurysm was found and there was no suggestion of trauma. Other findings included cirrhosis, ascites, hepatosplenomegaly, pleural adhesions, and needle tracks.

Case 18

A 33-year-old black man was found unconscious and brought to a nearby hospital, where he went into cardiopulmonary arrest and died. Cocaine was detected in blood (0.1 mg/L) and bile, amitriptyline in blood (3.0 mg/L). Autopsy showed massive intracerebral hemorrhage filling the right lateral ventricle without any obvious bleeding source. Needle tracks and skin ulcers were also found, along with pulmonary edema. There was no sign of trauma.

Case 19

A 28-year-old black woman was found unresponsive and brought to a nearby hospital, where she went into cardiopulmonary arrest and died. Cocaine or metabolites or both were found in blood, bile, and nostrils. Autopsy disclosed extensive intracerebral hemorrhage, fatty liver, and cardiac hypertrophy. There was no suggestion of trauma.

Discussion

Evidence of cocaine use was found in 15% of cases brought to the Office of the Chief Medical Examiner in New York City. Although deaths as a result of acute intoxication solely attributable to cocaine was a significant problem, deaths related to a combination of heroin and cocaine were three times more frequent. Classifying these deaths as acute narcotism should not result in attributing these deaths to heroin, but rather should raise the question of synergistic lethal effects of cocaine and heroin.

More than a third of the deceased with cocaine were homicide victims. This reflects the drug-homicide link previously reported, namely that one third of male homicide victims in Manhattan were directly related to drug dealing [*22*]. The large proportion of blacks and Hispanics found with cocaine in their bodies in this study probably reflects the confounding effects of increased homicide rates among blacks and Hispanics and not necessarily more cocaine use in blacks and Hispanics compared with whites in the general population. In other words, blacks and Hispanics may be more likely, because of more frequently being homicide victims, to come to the attention of the Medical Examiner's office.

Before discussion of the 19 cases of specific pathophysiologic interest (see Table 2), the number of fetal deaths should be noted. This supports the concern that cocaine use in pregnant women results in increased fetal and infant deaths [*23*]. It is believed that increased norepinephrine levels results in vasoconstriction, tachycardia, and an abrupt rise in blood pressure. Placental vasoconstriction also occurs, decreasing blood to the fetus. Increased norepinephrine levels also increase uterine contractility, thus increasing the rate of sponta-

neous abortion [*24*]. The deaths of newborns a few months old with cocaine in their bodies raises concerns about maternal cocaine use and presumably breastfeeding. Earlier studies of babies of cocaine using mothers following birth found only transient neurological abnormalities such as tremulousness, irritability, and muscular rigidity [*25,26*]. The finding of newborns dying in their cribs with cocaine should alert us to the possible implication of cocaine in deaths reported as Sudden Infant Death Syndrome (SIDS). Before a newborn death is classified as Sudden Infant Death, levels of cocaine should be assayed. These deaths may represent the tip of an iceberg in terms of the effect of cocaine on pregnancy in more subtle ways, as in neurobehavioral impairment.

There were six young men who died of acute cardiac events, three with myocardial infarction (Cases 1, 2, and 5) and one with myocardial ischemia, and findings suggestive of more serious myocardial damage, namely discoloration of focal myocardial softening (Case 6). Two cases of coronary thrombosis (Cases 3 and 4) did not have evidence of myocardial infarction but the thrombuses were clearly the cause of death. Cases with recent myocardial infarction had evidence of past myocardial infarction. Those without recent myocardial infarction, however (Cases 3 and 6), did have evidence of atheriosclerosis. The other without atheriosclerosis was the youngest of the six cases.

In the six cases, four had coronary thrombosis and all four of these had positive ethanol levels. Boag and Howard [*7*] noted that both ethanol and cocaine are arrhythmogenic, suggesting that there may be a synergistic effect. Both ethanol and cocaine increase available catecholamines in terms of cocaine inhibiting uptake and ethanol increasing release of catecholamines. Epinephrine is potentially arrhythmogenic since it decreases the myocardial refractory period and lowers serum potassium levels. Ethanol increases the circulation of free fatty acids which are arrhythmogenic and acid aldehyde, the major metabolite of ethanol increases myocardial norephrincprine. Whether the relationship between certain arrhythmias and thrombogenesis exists requires further study. Other mechanisms possibly related to myocardial infarction include the increase of cardiac output and oxygen demand coupled with coronary vasoconstriction, all the result of cocaine's decreasing norepinephrine uptake and desensitizing tissues to the action of catecholamines [*9*].

There were four cases of ruptured dissections of the ascending aorta (Cases 7 to 10) where there was no evidence of Marfan's syndrome, syphilis, or trauma. In fact, two other cases of aortic dissection in persons with cocaine in this study were excluded because of such factors. All of these persons complained of chest pains. Three died within hours of this and the fourth died within several days. None had other drugs of abuse in their bodies; one had evidence of having consumed ethanol.

There had been 2 previous reports of cocaine-associated ruptured aortic dissection. Barth and his colleagues described a previously healthy 45-year-old man who collapsed and died while smoking free-base cocaine [*16*]. There was no suggestion that the patient was in any major risk group for suffering an aortic dissection. In the second case, Mittleman and Wetli reported that a 38-year-old woman with ruptured dissecting aneurysm of the aorta had a history of hypertension and myocardial hypertrophy. The suspected link between cocaine is that of increased blood pressure and heart rate. In this study, there was no history of hypertension among the deceased with ruptured dissection of the aorta.

There were nine cases of cerebral hemorrhage, six with aneurysms of the cerebral arteries (Cases 11 to 16) and three with extensive hemorrhage without aneurysms or other vascular malformations (Cases 17 to 19). Eight were black, one was Hispanic, and four were women. In only one case (Case 17) was there a history of hypertension. The presence of cirrhosis and acites in this individual also raises the possibility that chronic alcoholism with a bleeding diathesis may have contributed to the subarachnoid hemorrhage. It is of particular interest that three of the cases reported here did not have aneurysms of the cerebral arteries or other vascular malformations. Again, the suggested link between cocaine and cerebrovascular accidents is the increase in blood pressure and heart rate. Even persons without preexisting pathology of the cerebral arteries or family history of such are therefore at risk.

TABLE 2—*Demographic and pathological findings associated with cocaine abuse.*

Case	Sex/Age	Race	Other Substances	Autopsy Findings[a]
1	M/40	B	ethanol	thrombosis LAD; atherosclerotic occlusion (>75%) L, R, LC; MI recent and old; cardiac hypertrophy
2	M/37	Hisp.	ethanol	coronary thrombosis; MI anterolateral wall LV acute; cardiac hypertrophy; MI old lower 1/3 IVS
3	M/35	B	ethanol	coronary thrombosis; atherosclerotic occlusion (50%) LAD; atherosclerotic occlusion 1.5 cm distal to ostium; atherosclerosis aorta with thrombolic occlusion proximal to the bifurcation
4	M/27	Hisp.	ethanol	thrombosis LAD
5	M/38	W	methadone lidocaine	MI recent and old; atherosclerotic occlusion (60 to 70%) LAD, LC; cardiomegaly; aneurysm LV anterior and posterior wall
6	M/33	W	none	diffuse occlusive coronary atherosclerosis; focal myocardial ischemia, cardiac hypertrophy
7	M/36	B	ethanol	rupture dissecting aneurysm ascending aorta
8	M/28	Hisp.	none	rupture dissecting aneurysm ascending aorta
9	F/45	Hisp.	none	rupture dissecting aneurysm ascending aorta
10	M/42	B	none	rupture dissecting aneurysm ascending aorta
11	M/44	B	ethanol quinine	rupture of aneurysm AC; cerebral hemorrhage; anoxic encephalopathy; pneumonia
12	M/27	Hisp.	morphine acetaminophen	rupture of aneurysm ACC (2-cm diameter); massive subarachnoid and intraventricular hemorrhages
13	F/29	B	lidocaine	rupture of aneurysm right PC, massive subarachnoid and intracerebral hemorrhages
14	M/29	B	none	rupture of aneurysm left MC; subarachnoid hemorrhage
15	F/31	B	morphine	rupture of aneurysm AC; intracerebral and intraventricular hemorrhages
16	F/44	B	none	rupture of aneurysm AC
17	M/44	B	ethanol methadone	subarachnoid hemorrhage; no aneurysm; cirrhosis; acites

18	M/33	B	amitriptyline	intracerebral hemorrhage; no aneurysm
19	F/28	B	none	intracerebral hemorrhage; no aneurysm; fatty liver, cardiac hypertrophy

"MI = myocardial infarction;
LV = left ventricle;
IVS = interventricular septum;
LAD = left anterior descending coronary artery;
L = left main, R = right main, and LC = left circumflex coronary arteries;
AC = anterior cerebral artery;
ACC = anterior communicating cerebral artery;
PC = posterior communicating cerebral artery; and
MC = middle cerebral artery.

In conclusion, cocaine use can have dangerous medical consequences. The cases reported in this study are probably an underestimate of these medical consequences. Future research should focus on persons presenting to emergency rooms and use routine toxicological assays for cocaine. In addition, prospective studies of cardiac tissue from persons dying of cardiac events attributable to cocaine should be done to determine the mechanism of action of cocaine in producing these deaths.

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Address requests for reprints or additional information to
Dr. Kenneth Tardiff
Cornell University Medical College
525 East 68th St.
New York, NY 10021